

# INFLUENZA AND THE TUBERCULOSIS CONNECTION

*Part 2 of 2*

by Lawrence Broxmeyer, MD  
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The NY Institute of Medical  
Research, New York, USA  
Email: nyinstituteofmedical  
research@yahoo.com  
Website:

<http://drbroxmeyer.netfirms.com/>

As the greatest influenza pandemic of all time continued to rage, other doctors were speaking out. In 1920, with the killing fields of 1918–19 laid out before him, British physician Marcus Paterson took yet another jab at the "flu" epidemic.<sup>28</sup> He wrote: "During the time I was Resident Medical Officer at Brompton, it was usual to classify a rise of temperature in an ordinary case of 'chronic' pulmonary tuberculosis as influenza, even when there was no epidemic."

As for the well-known sick feeling (malaise) and preliminary symptoms of reactivated tuberculosis, Paterson asked: "Are these symptoms any different from those of the ordinary onset of influenza? They are not, simply because they denote not a particular disease, but a toxemia due to bacterial action."

Paterson's reference to the confusion of sorting out influenza from TB was pointed: "Surely there is no more emphatic testimony to the clinical difficulties of a differentiation between the two diseases when one group of clinicians describes a rise of temperature in a proven case of tuberculosis to be influenza, and another section terms an arrested case of tuberculosis with pyrexia [fever] an active case of tuberculosis."

Writing in 1920, after the greatest pound-for-pound health catastrophe ever, Paterson said what every physician still learns as a resident: "Before the war, it was usual to classify a sudden onset of acute symptoms as influenza, and it is very easy to appreciate the reason. When a person is taken ill, the patient's friends demand to know at once what is the ailment: hence it must be given a name, and 'influenza' is a good enough term for the moment."

What Paterson left out, as a given, was the hysteria in the family and immediate community that would intrude were the diagnosis of tuberculosis rendered. Instead, he stated that he knew of no physician who could differentiate clinically between tuberculosis and influenza. And he said that he "shudders to think" of the number of times that "tubercle bacillus have been classified as influenza without further investigation". To Paterson, this represented countless opportunities to cure the real cause, masked under the designation "influenza". His conclusion: "There is no research required here. It is a known fact that what is at present called influenza is often tuberculosis. But the knowledge is not practically applied. It is information of inappreciable value lying idle. It is no new discovery, or its import would ring throughout the world." In fact, for Paterson, much like it was for von Unruh, "influenza" was simply the first indication of tuberculosis.

Many years later, well after influenza was proclaimed "a virus", influential Johns Hopkins head of pathology Arnold Rich summed things up best:<sup>29</sup> "In relation to the question of the effect of influenza upon tuberculosis, it should be pointed out that in many cases in which pulmonary tuberculosis has been thought to have followed an attack of influenza, it is altogether probable that the supposed attack of influenza was, in reality, a manifestation of an existing tuberculous infection; for tuberculo-protein, whether absorbed from a

spreading lesion or injected into the body, can cause constitutional symptoms (fever, malaise, headache, joint pains, anorexia, prostration) quite like those of influenza." Rich concluded: "The writer has seen attacks closely simulating influenza occur in healthy, tuberculin-positive laboratory workers as a result of the accidental inhalation of the vapor of boiling tuberculin."

### Rockefeller Institute for Medical Research, NYC, 1931

American virologist Richard E. Shope, based in the Department of Animal Pathology at the Rockefeller Institute, was in direct communication with British investigators Smith, Andrewes and Laidlaw at Mill Hill, England, and sent samples of his flu virus and Pfeiffer's bacillus. But the British group, in return, wasn't being 100 per cent supportive.

Virologists like Shope and Laidlaw saw a great opportunity for virology provided by the 1918–19 pandemic. Shope began the first salvo on swine "influenza", again falling back on the stale conception that the mild disease and flu-like symptoms were created in pigs by what he believed to be a "filterable virus".<sup>30</sup> Shope had the singular advantage of realising that since 1918, pigs had been coming down with the same "influenza" each year. Having lived in Iowa,

he had grown up with the knowledge. But beginning his investigations in earnest, Shope became perplexed. Not a virus but a bacterium kept cropping up in swine mucous secretions, and it resembled Pfeiffer's bacillus or *Haemophilus influenzae* (H. flu) more than anything else. The problem was that he couldn't infect most of his subjects with the bacterium. So he took the mucous secretions of sick pigs and put them through a filter which he thought would only yield a virus. However, incredibly, even the filtrate from the discharge just gave low-grade symptoms. So if it wasn't a "virus" that had caused the deadly strains of flu, and if it wasn't a bacterium present in most malignant "flu", what could it be? To Shope, possibly both, working in conjunction with one another. In 1931, he introduced both the "virus" and Pfeiffer's bacillus into animals, which subsequently came down with just the deadly "flu"<sup>31</sup> complicated with pneumonia that killed between 50 and 100 million people in 1918–19.

As late as 1944, Shope insisted that the pandemic influenza was this meld of "virus" plus Pfeiffer's bacillus.<sup>32</sup> But as opposed to what's written in today's revisionist history, the idea wasn't really his. When Shope was a teenager, the announcement that hog cholera (swine flu) was due to the combined action of a bacterium and a virus stimulated French-Canadian microbiologist Félix

d'Hérelle in 1917 to publish his discovery of bacteriophages—viruses which live in and can destroy or alter the shape of bacteria—followed by his 1921 classic book on such bacterial viruses.<sup>33</sup>

Actually, only some time after joining Rockefeller in 1928, Richard Shope had switched his interest from tuberculosis to virology. Originally he was looking for simply a bacterial cause such as Pfeiffer's bacillus for "swine influenza", as the medical orthodoxy of his day dictated. There is no evidence in the literature that Shope even knew about the filterable forms of *H. influenza* (Pfeiffer's bacillus) which also appeared "viral". To accept Shope's conclusion, an exception had to be made. There wasn't a single cause behind the 1918–19 pandemic, but two: a virus and a bacterium.

### UK Medical Research Council, Mill Hill Farm, 1932

British virologists Smith, Andrewes and Laidlaw had started out by falsely trying to link the virus which caused dog distemper to human influenza. Nor was the fact lost on many that Patrick Playfair Laidlaw's group seemed to fly directly in the face of Shope's multifactorial conclusion, which said that both a virus and Pfeiffer's bacillus from swine were necessary to acquire the flu. Just the "virus" itself was

necessary, claimed the British trio.<sup>34</sup>

Christopher Howard Andrewes, who would subsequently receive the lion's share of credit for discovering the human influenza virus, thought that he had discovered the viral cause of rheumatic fever at the Rockefeller Institute as far back as 1923.<sup>35</sup> When that didn't pan out, and back in England, he, in a similar way to Shope in America, put his efforts into finding the virus behind cancer but again they proved wrong. But when Walter Fletcher, first secretary of Britain's Medical Research Council (MRC), started a new program, Andrewes soon found himself at the Mill Hill farm under virologist Patrick P. Laidlaw and his colleague Wilson Smith. The MRC was originally founded as a consequence of the recommendations of the Royal Commission on Tuberculosis, but under Fletcher it had apparently gone viral.

When influenza appeared in epidemic proportions during the autumn of 1932, Andrewes and Smith worked out a plan of action designed to reveal their hypothetical virus. In the midst of this, Andrewes began to feel unwell with flu-like symptoms. Immediately, Smith, having passed Andrewes's respiratory secretions through a filter, began to inject these intracerebrally and intratesticularly into mice, rabbits and guinea pigs. Nothing happened straight away, but just afterwards Laidlaw was informed

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by the director of Wellcome laboratory, one of their reference labs, that some of their ferrets appeared to be suffering from influenza at the same time as the epidemic of influenza was raging among Wellcome's staff. It turned out that these Wellcome ferrets didn't have influenza but were suffering from distemper. Not to be dismayed, Smith then inoculated several other ferrets through the nose with filtrates from Andrewes, and shortly afterwards influenza appeared. Subsequently, Wilson Smith himself came down with a flu that the rest of the group suspected was from a ferret. From the filtrate of this strain, WS (Wilson Smith) was isolated.

As head of a government lab, Dr William Crofton actually examined the famed WS strain that had afflicted Wilson Smith. Today, this strain is still being used in research; it bears the alphanumeric notation A/WS/1933, for Influenza A/Wilson Smith/1933. Crofton, of all people, knew that there was nothing "viral" about the WS strain, nor anything to do with "influenza". During the summer and winter of 1918, he was isolating Pfeiffer's bacillus from 100 per cent of influenza cases. But to do so, he was using improved special-growth agents and a sufficiently high-powered microscope. Crofton's "moist-chamber method" kept his culture medium warm and moist. If the microbe wasn't kept warm, Crofton found, then it couldn't be isolated in every case. Pfeiffer's bacillus was clearly pleomorphic (many forms) with viral-like forms that could easily pass through a filter.

So, in 1938, Crofton cornered Andrewes, who was presenting a paper before the Epidemiology Section of the Royal Society of Medicine: "I asked him how then he knew that his colleague Wilson Smith, from whom was first isolated the virus, had, in fact

influenza, and how he and his fellows dared to advertise to the four corners of the earth that at long last the [viral] cause of influenza had been discovered. I told him that I had ascertained that Wilson Smith had, in fact, influenza, because he was swarming with [bacterial] influenza bacilli. I asked him why no cultures on proper medium were made from the infected ferret to ascertain if the Pfeiffer bacillus could be grown, as it would have been inevitably if, in fact, influenza had been transmitted. Andrewes replied not one word, and the authorities of the section would not publish my criticism."<sup>36</sup>

Apparently, the fix was already in through powerful governmental forces at Mill Hill's MRC. Christopher Andrewes, flushed with victory, suggested with the help

of Burnet and Bang that the term "myxovirus", meaning "mucous virus", be incorporated into a family name for the influenzas.<sup>37</sup> This, one imagines, was because the organism came from mucous secretions.

By May 1935, Laidlaw in "Epidemic Influenza: A Virus Disease"—an article seemingly titled to assure himself—would give the only credence he saw fit to Crofton and the many scientists like him by saying that although he thought that it was "gradually" being proven that a "filterable virus" was primarily responsible for epidemic influenza, "Pfeiffer's bacillus or *Haemophilus influenzae* is still regarded by many observers as the prime cause of the disease and many of its complications".<sup>38</sup>

Crofton was convinced by scientists like Albert Calmette at Pasteur that, like Pfeiffer's bacillus, certain forms of another mycobacterium, tuberculosis, appearing both minuscule and viral, could pass through the smallest of filters. So, at a time when viral forms of TB were being documented scantily, Crofton struggled to link Pfeiffer's bacillus with the TB with which it so often infected in coordination. Pfeiffer's strongly resembled TB; it was just smaller.

#### Rockefeller Institute, NYC, 1933

After the pandemic of 1918–19, the name of Pfeiffer's bacillus, *Mycobacterium influenzae*, was officially changed to *Haemophilus influenzae*. American bacteriologist Margaret Pittman, who later defined the two major strains of *H. influenzae*, both encapsulated and unencapsulated, pinpointed the influenza bacillus's name change to the 1920 report by Charles-Edward A. Winslow and colleagues for the Society of American Bacteriologists' nomenclature committee.<sup>39</sup> In the same 1931 Rockefeller Institute

paper, Pittman admitted that pleomorphist Philip Hadley, poster scientist for the many forms in bacterial life cycles, contributed "new and important knowledge concerning variations in bacteria", knowing full well that her own findings on the different virulence of "smooth" and "rough" forms of *H. influenzae* were probably held up for decades by Koch and Winslow's monomorphist dogma, which held enormous sway.

By any stretch, C.-E. A. Winslow was far from the ideal choice to head a panel to rename *Mycobacterium influenzae* to *Haemophilus influenzae*. Not only was he an avid one-form-only bacteriologist,<sup>40</sup> leaving very little room for a microbe which could appear in both fungal and bacterial forms, but his unique thought processes became

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obvious when, as Professor of Public Health at Yale, he said he believed that posture was a neglected cause of tuberculosis, a disease known since antiquity for its spine-bending changes.

Yet Winslow and his colleagues decided to ignore Pfeiffer's bacillus's previously documented fungal forms, concluding that, unlike the mycobacteria, it stained Gram-negative and liked haemoglobin. But neither was conclusive enough to warrant the name change that Winslow had in mind.

Tuberculosis specialist Stephen Maher wrote in 1913: "All non-acid coccal and bacillary derivatives of the tubercle bacillus are, strange to say, Gram-negative."<sup>41</sup> Krylow confirmed Maher's observation that TB could stain Gram-negative.<sup>42</sup> The Gram stain, developed by Hans Christian Gram, separates bacteria based on their cell walls. The thick layers in "Gram-positive" cell walls stain purple, while the thin "Gram-negative" cell walls appear pink. Like other bacilli, cultures of TB, on the other hand, could be Gram-positive when young but might become Gram-negative as they aged. Hans Much saw this in 1907<sup>43</sup>, and Chandrasekhar reported it in 1983.<sup>44</sup> Therefore, the fact that Pfeiffer's influenza bacillus was Gram-negative still didn't rule it out as a bacillary derivative of the tubercle bacillus. Furthermore, the organism was arbitrarily named *Haemophilus influenzae* (from the Greek *haemophilus*, meaning "blood-loving"), but it grew on the same blood-based cultures on which *Mycobacterium tuberculosis* had long thrived.<sup>45</sup>

To many in the lay and scientific communities, bacterial names and classifications are hallowed ground. But Sneath and Brenner's 1992 paper<sup>46</sup> for the American Society for Microbiology clarified that there was no such thing as an official classification of bacteria or "approved lists" and that even *Bergey's Manual* was not "official" but merely the best consensus at the time. Therefore, Sneath and Brenner said, bacterial lists and classifications (called "taxonomy") are partly a matter of judgement and opinion, as is all science, and, until new information is available, different bacteriologists may legitimately hold different views. In the same vein, George Fox and colleagues remind us that even regarding today's sacred 16S rRNA sequence identity as a criterion for species identification, 16S rRNA may not be sufficient alone to guarantee species identity.<sup>47</sup>

Regrettably, no attempt at bacterial nomenclature, since its inception, has left room for a microbe such as

Pfeiffer's, which exists in more than one form, both bacterial and fungal, true to its original mycobacterial designation. The price of this to the world's future health and welfare would be substantial. Today, we are carefully taught that Pfeiffer's bacillus, historically *Mycobacterium influenzae*, was erroneously thought to have caused and been behind the Great Pandemic of 1918–19. We are not taught that Pfeiffer's bacillus itself has a viral cell-wall-deficient phase which goes right through a filter. Nor are we taught that its original difficulty in cultivation on normal media alone, without blood's haemoglobin, almost classified it, *a priori*, as a "virus" in the minds of those who relentlessly tracked a virus for influenza.

It was in 1952 that Cornelius P. Rhoads, Director of the Sloan-Kettering Institute for cancer research in New York City, remarked in the introduction to a conference on viruses and cancer that the term "virus" had achieved "a high professional status with doubtful credentials".<sup>48</sup>

Papers such as that of Peter Palese,<sup>49</sup> of the Mount Sinai School of Medicine in Manhattan, remind us that, even in 1992, millions in China already had antibodies to H5N1, meaning that they had contracted it and that their immune system had little trouble fending it off.

And as Dutch science historian Ton van Helvoort aptly pointed out, by the 1950s the word "virus" had become so mouldable a concept that one could speak of virus workers without the existence of any consensus whatsoever of what viruses were.<sup>50</sup> Extremely supportive of this mouldability among virologists was biophysicist Max Delbrück's subtitle for "Viruses 1950", a conference held at the

California Institute of Technology: "Proceedings of a conference on the similarities and dissimilarities between viruses attacking animals, plants, and bacteria, respectively".<sup>51</sup> Indeed, in the 1930s and 1940s, the concept of "filterable virus" was subjected to such criticism that its very foundations were threatened. Statements like those coming from pioneer virologist André Lwoff in 1957, such as "viruses should be considered as viruses because viruses are viruses", were unacceptable.<sup>52</sup>

Also under the gun was Thomas M. Rivers, "the father of modern virology", who said that viruses could be differentiated by three things; namely, their invisibility under the ordinary microscope, their ability to pass through the finest filters because of this small size, and their inability to propagate themselves in the absence of susceptible cells.<sup>53</sup> But English bacteriologist Frederick

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Twort, working in 1915 on Johne's bacillus, presently itself suspected of being a tuberculosis-like mycobacterium, had to add special factors before this bacterium would grow on a lifeless medium.<sup>54</sup> Henry Sweany's 1933 study showed that certain virus-like forms of tuberculosis met Rivers's criteria as well.<sup>55</sup> Rivers, who would become head of the Rockefeller Institute, was wrong. In fact, in 1951, Emmy Klieneberger-Nobel showed just the opposite: that some bacteria could pass through filters that some of the larger viruses could not.<sup>56</sup> Wade and Manalang, as noted in part one, proved this with mycobacterial forms of Pfeiffer's.<sup>57</sup>

To say that the history of the theoretical underpinning of virology has been a tortuous one is probably an understatement, and, incredibly, many virologists even to this day persist in using the flawed reasoning that that which passes through a microfilter is a virus. Certainly, one exception to these is world-renowned virologist/molecular biologist Stefan Lanka, who, to this day, questions the very existence of H1N1 and its allied forms as a swine flu virus altogether. Lanka, after reviewing the data, came to the conclusion that studies, micropictographs and even the data have been manipulated. Lanka was the first to isolate a maritime virus. Through personal communication, he has assured this author that his repeated investigative letters to the National Institutes of Health (NIH) regarding supportive material for the "influenza virus" have gone unaddressed and unanswered.<sup>58</sup>

### Challenging the Medical Orthodoxy

Shannon Brownlee and Jeanne Lenzer are not new to writing incisive warnings regarding public health. Among their writings listed on Medline is a *British Medical Journal* treatment entitled "Doctor takes 'march of shame' to atone for drug company payments".<sup>59</sup> That influenza vaccination and oral antiviral studies have been consistently pushed and paid for by pharmaceutical companies themselves is no secret. But once in a while, unsponsored papers such as Brownlee and Lenzer's 2009 article in *The Atlantic* clear the misinformation.<sup>60</sup>

"Whether this season's swine flu turns out to be deadly or mild, most experts agree that it's only a matter of time before we're hit by a truly devastating flu pandemic—one that might kill more people worldwide than have died of the plague and AIDS combined. In the US, the main lines of defense are pharmaceutical—vaccines and antiviral drugs to limit the spread of flu and prevent people from dying from it. Yet now some flu experts are challenging the medical orthodoxy and arguing that for those most in need of protection, flu shots and antiviral

drugs may provide little to none. So where does that leave us if a bad pandemic strikes?..."

The authors added: "But what if everything we think we know about fighting influenza is wrong? What if flu vaccines do not protect people from dying—particularly the elderly, who account for 90 percent of deaths from seasonal flu? And what if the expensive antiviral drugs that the government has stockpiled over the past few years also have little, if any power to reduce the number of people who die or are hospitalized? The US government—with the support of leaders in the public-health and medical communities—has put its faith in the power of vaccines and antiviral drugs to limit the spread and lethality of swine flu. Other plans to contain the pandemic seem anemic by comparison. Yet some top flu researchers are deeply skeptical of both flu vaccines and antivirals. Like the engineers who warned

for years about the levees of New Orleans, these experts caution that our defenses may be flawed, and quite possibly useless against a truly lethal flu. And that unless we are willing to ask fundamental questions about the science behind flu vaccines and antiviral drugs, we could find ourselves, in a bad epidemic, as helpless as the citizens of New Orleans during Hurricane Katrina."

My paper has intentionally questioned perhaps the most fundamental question about the science behind influenza and its vaccine and antiviral cures: is it really a virus at all? For if it is indeed a form of viral, cell-wall-deficient mycobacteria such as *Mycobacterium influenzae* or *Mycobacterium tuberculosis*, as many physicians and scientists in the past have suggested, we could indeed find ourselves in the midst of another devastating, infectious Katrina. ∞

### About the Author:

Lawrence Broxmeyer, MD, is currently an internist and medical researcher. He was on staff at New York affiliate hospitals of SUNY Downstate, Cornell and New York universities for approximately 14 years. In conjunction with colleagues in San Francisco and at the University of Nebraska, he first pursued, as lead author and originator, a novel technique to kill AIDS mycobacteria and tuberculosis with outstanding results (see *The Journal of Infectious Diseases* 2002 Oct 15; 186[8]:1155-60). He contributed a chapter regarding these findings to Sleator and Hill's textbook *Patho-Biotechnology* (Landes Bio-science, 2008). He has also written many peer-reviewed articles, available on PubMed at <http://tinyurl.com/3u2ly8s>. Dr Broxmeyer's research covers the most challenging medical problems of our times, including AIDS and Alzheimer's disease. His two-part article "The Untold Truth About Cancer" was published

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Dr Broxmeyer can be contacted by email at [nyinstituteofmedicalresearch@yahoo.com](mailto:nyinstituteofmedicalresearch@yahoo.com) and via his website <http://drbroxmeyer.netfirms.com/>.

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