

The Swine Flu (aka 'H1N1) That's Not A Flu At All

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The current Swine "flu" pandemic began in La Gloria, a pig-farming village in the Veracruz mountains of Mexico. Surely, if any place could unlock the true nature of the cause of the 2009 Pandemic, it would be found in La Gloria, whose villagers were certain that they were sickened by the surrounding pig farms, which they accused of polluting their air and water with pig waste. This is much like what happened in Haskell County, Kansas in 1918, original site of the Great Influenza Pandemic of 1918.

But Enrique Sanchez, top official from Mexico's Agriculture Department, could not find H1N1 in mucous samples taken from the pigs several weeks later, on April 30th. Common bacteria were also tested for. However no studies were done to rule out Swine tuberculosis which is predominantly avian and to a lesser extent bovine, and could have also accounted for the wholesale "respiratory" problems the villagers were experiencing.

Mexican health officials, overlooking the La Gloria situation initially, downplayed it, much as health officials everywhere are prone to do. Yet of 43 villagers whose mucous samples were taken, only one, a 5-year-old boy, was confirmed as having Swine Flu. So head investigator from the Biotechnology Institute of the National Autonomous University of Mexico there, Dr. Carlos Arias, told the AP:

"I cannot understand it. I could almost bet that there were more infections related to the virus."

Soon, more than half of La Gloria's 3,000 residents fell ill with FLU-LIKE illness, etiology unknown. 450 of the sickest of these were treated not with antivirals such as Tamiflu, but with antibiotics and masks. What was the diagnosis handed out? Why it was "acute respiratory infections", and by the time the mucous results came through in early April, most of the villagers had recovered, the more serious cases on antibiotics alone. The "virus" seemed to have left their systems. But the question lingers: since antibiotics don't cure "viruses" what infectious disease in these purported "Influenza" victims had been cured by the antibiotics given?

On June 3rd, 2009 in Global Research, a curious article by F. William Engdahl appeared, entitled "Sarkozy's Secret Plan for Mandatory Swine Flu Vaccination". In it Engdahl plainly stated that "The only problem with the Swine Flu (H1N1) Vaccine, is that to date, neither WHO nor the US Government's Center for

Diseases Control (CDC) have succeeded to isolate, photograph with an electron microscope, or chemically classify the H1N1 Influenza A virus". Furthermore there was no scientifically published evidence that French virologists have done so either. Therefore, mentioned Engdahl, "To mandate a vaccination for a putative (supposed or assumed to exist) disease that has never been characterized, is dubious to say the least."

Engdahl had done his homework. When questioned regarding the electron pictograph of H1N1 that the CDC recently came up with on their website, he revealed his source, German virologist Dr Stefan Lanka, an expert on the documentation of viruses, attesting to the fact that the H1N1 picture was bogus. The virologist wrote that he had "written the CDC many times as to who made the H1N1 photo's and whether they where scientifically documented as to chemical characteristics and other properties." There was never any reply. He concluded "If CDC refuses to cite the source of the photos, they are fake." Worse yet he said "The photos are merely liposomes, microscopic artificial sacs whose walls are a double layer of phospholipids, used to carry substances such as drugs, vaccines, and enzymes to specific cells or organs of the body. These have been artificially presented by a process where chick embryos or cell cultures are killed, reduced and then centrifuged with some solvent, to then, in a vacuum, be nanofiltered." As if this wasn't enough, the virologist testified that "Such a structure has never been characterized in either an organism or its fluids. Furthermore, if there wasn't for the centrifuge/solvent/nanofiltration manipulation, not to mention the precipitation procedure, such particles could never be presented under the electron microscope. In conclusion, without the isolation of the H1N1, there is no H1N1 infecting virus"

Engdahl wasn't finished. "Even more bizarre is the admission by the US Government's Food and Drug Administration, an agency responsible for the health and safety of its citizens, that the 'test' approved for premature release to test for H1N1 is not even a proven test. More to the point", continues F. William Engdahl, "there is no forensic evidence in any of the deaths reported to date that has been presented that proves scientifically that any single death being attributed to H1N1 Swine Flu virus was indeed caused by such a virus."

These thoughts were only strengthened when Engdahl looked into "Novavax, a US pharmaceutical company based in Rockville, Maryland, (who) conveniently enough just announced it is developing a vaccine for H1N1 based on 'virus-like particles'".

'Virus-like particles' do not mean virus. Viral-like, cell-wall-deficient forms of tuberculosis, for example, also appear virus-like and apparently.. so also did non-infectious liposomes.

Both the World Health Organization (WHO) and the Centers for Disease Control

(CDC) are fully aware of a far more serious and ongoing tuberculosis Pandemic in the world today. Yet they choose to downplay the link, disregarding the similar flu-like symptoms tuberculosis often begins with.

WHO freely admits that there were approximately 1.8 million deaths from tuberculosis in 2007, the most recent year for which data are available as well as that presently about one-third of the world's population, or two billion people, carry the TB bacteria.

The "H" and "N" of influenza sub-typing, revolves around two glycoproteins called Hemagglutinin (H) and Neuraminidase (N), both of which can be, and are, associated with infectious diseases such as the minuscule, viral forms of tuberculosis, a disease which ought to be high on the differential diagnosis for 'flu-like illness'. An August, 2008 Medline study in the Journal of Clinical Biochemistry showed that sputum neuraminidase levels over 1.0 mU per mL were proven associated with having tuberculosis in 92% of cases. Previous to this, bacteria closely related to TB were shown, through crystallization, to produce the same protein neuraminidase used to subtype 'Influenza'. Furthermore, as of 2006, it has become obvious in Menozzi's study that similar to Influenza, Tuberculosis not only uses Hemagglutinin to attach to the lung's epithelial cells it invades, but requires Hemagglutinin for dissemination of the disease to the rest of the body.

Khomenko's 1993 study, showed that the explosive contagiousness of just such influenza-like forms of tuberculosis are exactly the stuff that previous epidemics and pandemics could have been made of. Khomenko was mentioned by Nobel nominee Lida Mattman in her textbook. That is exactly why, that in response to the present world "flu" pandemic, Japan's Health Ministry's Tuberculosis Infection Diseases Control Division deputy director Takeshi Enami went hand in hand with Yoshio Nanba, director of The Office of Pandemic Influenza Preparedness and Response, to attend a news conference in Tokyo on May 1, 2009.

But back in the US, the CDC and NIH seem to feel differently, ignoring everything but "the virus". There was much the same "Influenza" talk when in 1990, a new multi-drug-resistant (MDR) tuberculosis outbreak took place in a large Miami municipal hospital. Soon thereafter, similar outbreaks in three New York City hospitals left many sufferers dying within weeks. By 1992, approximately two years later, drug-resistant tuberculosis had spread to deadly mini-epidemics in seventeen US states, and was reported, not by the American, but the international media, as out of control. Viral forms of swine, avian and human TB can be transmitted from one species to another. By 1993 the World Health Organization (WHO), proclaimed tuberculosis a global health emergency. (1)

1918 AND TODAY

No one can deny the similarities between the onset of the 1918 epidemic and that of today, yet a Press Release, issued on August 19, 2008, by the National Institute of Allergy and Infectious Diseases (NIAID), contains a striking finding and conclusion: The 20 to 40 million deaths worldwide from the great 1918 Influenza ("Flu") Pandemic were NOT due to "flu" or a virus, but to pneumonia caused by massive bacterial infection."(2)

Subsequently, a study published in JAMA by Talbot and Moore (3) in 2000 showed that Mexican immigrants to the US have the highest case rates for tuberculosis among foreign born persons. Mexico is the country where Swine Flu deaths were first documented.

The research of Lawrence Broxmeyer MD (4) first proclaimed that the 1918 pandemic was due to bacteria, particularly mutant forms of flu-like fowl, swine, bovine, and human tuberculosis (TB) bacteria:

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<http://drbroxmeyer.net/firms.com/001pdfBIRDFLUEDITORIALPUBLISHED.pdf>
).

These forms of tuberculosis are often viral-like, mutate frequently and can "skip" from one species to another. Moreover the antibodies from such viral TB forms react in the compliment fixation and later "viral" assays (5). They also grow on cultures which are supposed to grow only viruses.

In a supportive 16-page-paper which appeared in Population and Development Review, University of California demographers Andrew Noymer and Michael Garenne came up with convincing statistics showing that undetected tuberculosis may have been the real killer in the 1918 flu epidemic (6).

THE ROOTS OF HISTORY

Noymer's TB hypothesis stands sound against history. Few flu "experts" are aware that in medical texts printed circa 1918 "Influenza" was attributed not to a virus but a bacteria called Mycobacterium influenzae, discovered by Pfeiffer and Canon (7) in 1892.....not exactly a coincidence since Richard Pfeiffer worked, at one time, for Robert Koch, the discoverer of tuberculosis, a disease also caused by another bacillus called Mycobacterium tuberculosis. Both mycobacteria stained best with carbol-fuchsin, a bacterial stain commonly used in the staining of mycobacteria as it has an affinity for the mycolic acids found in their cell walls. Mycobacteria such as tuberculosis are particularly deadly because they share properties of the fungi ("myco-") as well as bacteria. TB was, not all that long ago, referred to as "captain of the men of death". And it never lost its potential to kill.

Mycobacterium influenzae was considered by most to be the cause of influenza until 1933. But there were serious diagnostic problems with *Mycobacterium influenzae*. Stengel and Fox warned (8) about them in their widely quoted W.B. Saunder's 1915 version of "A Textbook of Pathology". Problems with identification revolved mainly in that although the bacterial influenza occurred abundantly in the sputum of flu patients at first, it decreased in quantity as the cases advanced. And when purulent expectoration stopped, whether the disease was still active or not, *Mycobacterium influenzae* "disappears entirely"(8).

In 1933 English physicians Wilson Smith, Christopher H. Andrewes, and Patrick P. Laidlaw(9) removed secretions from the throat of a human with flu-like symptoms thought to have "influenza", and then filtered out a suspected infectious agent, which by virtue of the fact that it went through a filter was falsely proclaimed, from the onset, to be "a virus". Injecting it into ferrets, the ferrets then developed the same flu-like symptoms which Smith, Andrews and Laidlaw summarily declared as "influenza". In addition Sir Christopher Andrewes suggested, with the help of Burnet and Bang, that the term "myxovirus", meaning "mucous virus", be incorporated into a family name for the Influenzas. This, one imagines, was because the organism came from mucous secretions.

But to government pathologist and pioneer physician/researcher William M. Crofton, who by virtue of his office as County pathologist had examined some of Laidlaw's human "Flu" samples, Laidlaw's entire study was flawed. Crofton found Laidlaw's Flu samples to be laced with the bacillus *Mycobacteria influenzae*, which by then had been renamed *Haemophilus Influenzae*. To Crofton this bacilli was the common denominator for the Pandemic of 1918 he had witnessed, although the bacteria could be accompanied by an array of opportunistic organisms such as Staph and Strep. Crofton publically and personally confronted and challenged Laidlaw to come to his laboratory for the proof that his Influenza samples weren't viral. Crofton was convinced by the confirmation of scientists like Calmette at Pasteur regarding how certain forms of tuberculosis, appearing both minuscule and viral, could pass through the smallest of filters. Crofton himself then established that tuberculosis could disappear into tissues as viruses did, and then go through filters which stopped cold even most of those "now invariably called viral disease". "Surely, then", Crofton concluded, "Tuberculosis has more right to be considered a true virus than these(10)." So, at a time when viral forms of TB were scantily being documented, Crofton struggled to link H. Flu with the TB it so often infected in coordination with, as historical and political momentum carried Laidlaw's study through for posterity. A great opportunity was missed to correct the record.

Frank Macfarlane Burnet was the first to grow "Influenza" in a laboratory setting; in 1940 he grew influenza in embryonated chicken eggs, using the allantoic sac. He obviously considered such a technique "Influenza" specific, never bothering to consider that such an allantoic site might also be an excellent site for culturing the viral or Cell-Wall-Deficient (CWD) forms of tuberculosis and the mycobacteria.

Viral TB, in fact grows there as early as 6 hours after introduction into such a place in chicken embryos(11).

Furthermore, some of the filamentous forms of tuberculosis mentioned by Corper (12) appear similar to those attributed to "Influenza" by biochemist and Influenza guru Burnet (13).

It wasn't because Burnet didn't know that bacteria could assume viral forms. The first, and for a time the only virologist in Australia, it was Burnet who discovered bacteria in viral forms of Q-fever (14). And in that same paper, proclaiming Virology as an Independent Science, and after admitting that viruses, including influenza, are composed of the same sorts of material as bacteria, Burnet struggles to differentiate Influenza by the fact that it "probably has no DNA" and was therefore exclusively RNA.(Ibid.). But, according to Xalabardar, some cell-wall-deficient mycobacterial forms also are exclusively RNA. Furthermore, points out Xalabardar, such cell-wall-deficient tubercular forms are true antigens, all of which, similar to Influenza, induce the production of specific antibodies detectable by complement fixation tests, such as those originally used to detect Influenza (5).

CONCLUSION

In a landmark study(15), Dr. Robert Donaldson, working out of the Pathological Society of Great Britain had ruled out that the mycobacteria now referred to as H. Influenza by itself was behind 1918, perhaps because of its disappearing nature. Yet at the same time he quickly added that there wasn't "the slightest shred of evidence" that the disease was due to a "virus" or influenza. Nor was Donaldson ever able to refute Broxmeyer and Noymer's feelings that TB was behind the many deaths in the pandemic, specifically because it is well known that secondary bacterial infections, be they from opportunistic Haemophilus influenza or any other common bacteria, are a common secondary manifestation in TB-infected lungs. During the pandemic, one-third of patients who had Haemophilus influenza were also found to have tuberculosis - keeping in mind, as always, that many other cases with TB went undiagnosed.

In order to understand why we have this emphasis by those virologists invested in a 'killer flu' in the US today, one must look back historically at the science itself. Until the late 1940s influenza 'viruses' were studied as infections, which, although filterable, were conceived of as analogous to bacteria, a kind of ultra or viral-like bacteria. Not to be deterred, and still seeing Influenza as a great opportunity for virology, in 1941 virologist Hirst claimed that influenza "virus" could agglutinate (or clot) red blood cells of fowl and other animal species(16). Such a hemagglutinin discovery, in turn, led to fast assays of what is thought to be Influenza. The "H" in H1N1 comes to us through Hirst, who showed that "virus" particles first adsorbed to the red cells and, after a certain time, eluted again as a

result of what could be interpreted as an enzymatic reaction. But 6 years later, Middlebrook and Dubos(17) made this seem nothing more than a cheap hat trick by showing that similarly red blood cell agglutination could be produced by sera from patients with tuberculosis. Takahashi and Ono(18,19) reviewed similar red cell agglutination occurring in the presence of tuberculous serums.

As Influenza historian van Helvoort aptly pointed out (20), indeed, in the 1930s and 1940s the concept of 'filterable viruses', including Influenza, were subject to such criticism that Virology's very foundations were threatened. Dogmatized statements like those coming from pioneer virologist Andre Lwoff in 1957 : "Viruses should be considered as viruses because viruses are viruses"(21) were totally unacceptable, and did little to help the situation. So it was in 1952 that Cornelius P. Rhoads, Director of Sloan-Kettering Institute for Cancer Research in New York City, remarked in a conference introduction that the term "viruses", such as that of Influenza, had achieved "a high professional status with doubtful credentials"(22).

Perhaps in his authoritative text, *The Pathogenesis of Tuberculosis*, Johns Hopkin's head of pathology, Arnold Rich summed things up best:

"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." (23)

As Professor Hans Rosling (<http://www.youtube.com/watch?v=V8bUtbODV-Q> <http://www.youtube.com/watch?v=V8bUtbODV-Q>) has so aptly pointed out: during the initial 13 days that WHO started gaining data on Swine Flu Deaths, April 24-May 06, 2009, 31 people died of Swine Flu. 29 of these were in Mexico and 2 in the US. During this same 13 day window, 63,000 people, around the world died of tuberculosis. (<http://www.who.int/research/en>).

What we have today, is a pandemic with "flu-like" symptoms. And flu-like symptoms doesn't necessarily mean "Influenza" is its underlying cause.

Readers interested in the subject of Influenza/TB can also go to Dr. Ron Paul, MD's take at: [HYPERLINK](#)

"<http://informationclearinghouse.info/article22507.htm> \\
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Other relevant links include:also applicable to the current Swine Flu epidemic

include: (<http://www.frequencyfoundation.com/2009/07/bird-flu-influenza-and-1918-case-for.html>)

And:

(<http://www.frequencyfoundation.com/2009/07/swine-flu-do-we-really-have-right.html#links>)

REFERENCES

1. Talay F Kumbetli S Altin S Factors associated with Treatment Success for Tuberculosis Patients: a Single Center's Experience in Turkey Jpn. J. Infect. Dis., 61,25-30, 2008.
2. DM Morens et al. Predominant role of bacterial pneumonia as a cause of death in pandemic influenza: Implications for pandemic influenza preparedness. The Journal of Infectious Diseases DOI: 10.1086/591708 (2008).
3. Talbot EA Moore M McCray E Binkin NJ Tuberculosis Among Foreign-Born Persons in the United States, 1993-1998 JAMA. 2000;284:2894-2900.
4. Broxmeyer L. Bird flu, influenza and 1918: The case for mutant Avian tuberculosis. Med Hypotheses. 2006;67(5):1006-15. Epub 2006 Jun 27
5. Xalabardar C, Formas L. Publicaciones Del Instituto Antituberculoso Francisco Moragas. 7, Barcelona; 1970. p. 183.
6. Noymer A, Garenne M. The 1918 influenza epidemic's effects on sex differentials in mortality in the United States. Popul Dev Rev 2000;26(3):56581.
7. Chester FD A Manuel of Determinative Bacteriology Macmillan & Company Ltd, London 1901 p.351. 401ppgs.
8. Stengel A Fox H A text-book of Pathology 6th Edition Philadelphia and London WB Saunders & Co 1915 1039 ppgs P298
9. Smith W, Andrewes C, Laidlaw P. A virus obtained from influenza patients. Lancet 1933;2:6668.
10. Crofton WM The True Nature of Viruses 2nd Edition, Staple Press, London 166pgs 1939
11. (Balan V.F. (1991): The use of chicken embryos for the culture of L-forms of mycobacteria tuberculosis . Problemy Tuberkuleza i Bolezni Legkikh, 2, 5960.)

12. Corper HC. In discussion following mutation forms of the tubercle bacillus. JAMA 1926; 9(October):121011.
13. Burnet F. Filamentous forms of influenza virus. Nature 1956; 177(4499):130.
14. Burnet F. Virology as an independent science. Med J Australia 1953; 40(223):8415.
15. Donaldson R. The bacteriology of influenza: with special reference to Pfeiffer's Bacillus. In: Crookshank, editor. Influenza. London: Heinemann; 1922. p. 139-144.
16. Hirst GK. The agglutination of red cells by allantoic fluid of chick embryo infected with influenza virus. Science ;xciv:223. 1941.
17. Pound A. Observation on the agglutination and haemolysis of red cells treated with extracts of Mycobacterium tuberculosis: an evaluation of methods. J Pathol Bacteriol 1952;64(1):131-143.
18. Takahashi Y, Ono K. Hemagglutination reaction by the phosphatides of the tubercle bacillus. Kekkaku no Kenkyu (Tuberculosis Res) 1957;7:1.
19. Takahashi Y, Ono K. Study on the passive hem agglutination reaction by the phosphate of M. tuberculosis. 1. The reaction and its specificity. Am Rev Resp Dis 1961;83(2):
20. van Helvoort T. History of virus research in the 20th century: the problem of conceptual continuity. Hist Sci 1994;32(2):185-235.
21. Lwoff A. The concept of virus. The third Marjory Stephenson Memorial Lecture. J Gener Microbiol 1957;xvii:239-53.
22. Rhoads CP. Introduction {to a conference on viruses as causative agents in cancer}. Ann NY Acad Sci 1952;liv:87-23.
23. Rich, AR The Pathogenesis of Tuberculosis 2nd Printing. Charles C. Thomas Publisher. Springfield, Illinois 1946. 1008 pps.P.627

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