Jeffrey Taubenberger's 1998 PBS Interview Concerning the 1918 Influenza Seems Strangely Familiar

Taubenberger discusses research that is directed toward reconstructing the hemagglutinin gene … a gene which he believes codes for a surface protein through which the 1918 virus supposedly gained entry to human cells. During the interview Taubenberger provides a theoretical overview that gives expression to the state of his understanding with respect to alleged pandemics such as the 1918 "Spanish Flu".

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Before being employed by National Institute of Allergy and Infectious Diseases, Taubenberger used to work for the Armed Forces Institute of Pathology (AFIP). The Institute has been in existence for about 130 years and began its operations during the Civil War as the result of an executive order by Lincoln which instructed the Army Surgeon General to study diseases that were connected to the battlefield.

The foregoing executive order was issued because more people were dying from various forms of pathologies that arose in conjunction with military conflicts than actually died as a result of the weapons that were being deployed during those engagements. Consequently, the Institute became a venue for collecting and studying samples taken from surgery as well as autopsies involving both human beings and animals that had roles of one kind or another within the military.

Taubenberger is a specialist in molecular pathology. This discipline develops methods for making diagnoses based on changes in genetic composition rather than -- as is the case in conjunction with traditional methods of pathology -- using microscopic examination of biological samples to do so.

Pathology samples are generally fixed in chemicals such as formaldehyde, and, then, embedded in wax. This makes the process of isolating DNA and RNA difficult to accomplish because the genetic material found within the samples that are fixed in the foregoing ways tends to become quite degraded over time.

RNA is much more fragile than DNA is. However, Taubenberger indicates that researchers have developed techniques which permit pathologists to help optimize – as much as possible – recovery efforts concerning those two molecules, and, consequently, the alleged 1918 flu virus served as an opportunity for using, exploring, and developing the kind of recovery techniques to which Taubenberger was alluding earlier that involve various kinds of molecules which are of interest to researchers.

Nevertheless, whatever the nature of the foregoing sorts of recovery techniques might be, unless one can show how those protocols are capable of zeroing in on RNA that is uniquely from alleged viral bodies rather than from other biological sources, then one is faced with a problem. More specifically, why should one suppose that whatever RNA is recovered through the foregoing sort of techniques is necessarily from viral bodies rather than from other biological components – such as tissue cells that have died and released their genetic contents into the samples that have been preserved?

Taubenberger said his recovery project was intended to "get a first direct look at the virus." However, for a number of reasons (some of which are noted in the following discussion), one might wish to question whether, or not, his research group actually ever came in contact with the alleged virus, and, therefore, in order to investigate such a possibility, let's take a look at various facets of Taubenberger's research that are touched upon in the 1998 Taubenberger interview.

According to Taubenberger, there were some 70 samples that were present in the Institute's archives that had been drawn from people who supposedly died from the influenza in 1918. These samples had been fixed in formalin and paraffin, and half of them were selected arbitrarily or randomly for purposes of study.

People died in different ways during the so-called Spanish Flu event of 1918. Some individuals died very quickly following the onset of symptoms, and this was quite different from the way people were believed to normally succumb to past cases of influenza.

Given that there were differences in the length of time that passed between, on the one hand, instances in which symptoms first began to appear, and, on the other hand, the point when life processes ceased in various patients, one query that could be explored is whether all the people who were dying in 1918 were necessarily dying from the same underlying pathology. For example, over the years, there have been a number of theories based on various kinds of evidence which suggest that whatever deaths occurred during 1918 might have been due to something other than -- or, perhaps, in addition to -- a suspected influenza virus.

Among the theories which have arisen over the years, are the following possibilities. (1) The forms of vaccines and medical treatments that were in use in 1918 often were injurious to patients in one way or another and, as a result, people might have died from the medical treatments they received rather than from a virus; or, (2) what had been diagnosed as cases of influenza were, instead, actually due to the work of the bacteria that is responsible for tuberculosis – something that was endemic in many places during the era of the "Spanish Flu and which can give rise to symptoms that are very similar to ones that are present in cases of influenza and, consequently, medical practitioners might have improperly diagnosed the nature of the problem with which they were dealing; or, (3) many people might have been developing bacterial infections of one kind or another due to the masks that were being worn to (supposedly) protect them against the alleged virus; or, (4) the pathology that was being referred to as the Spanish Flu might, actually, have been a form of poisoning that occurs when susceptible people are exposed to excessive amounts of certain kinds of electromagnetic radiation; or, (5) conceivably some combination of the foregoing possibilities came together in a sort of perfect storm of lethality, but, subsequently, were all subsumed in an undifferentiated fashion under the category of "death due to influenza".

To be sure, the aforementioned observed differences concerning the time intervals between symptom onset and death might have been a function of the extent to which individuals within the affected population could have possessed varying capacities of resistance to the pathology or pathologies to which they had been exposed. Nonetheless, as intimated previously, another way of accounting for the foregoing kinds of differences in temporal intervals between symptom onset and death is that an array of lethal causes might have been involved in the events of 1918, and some of those maladies might have been more lethal than others, and, if this were the case, then this might explain why some individuals died far more quickly than other individuals did.

Besides the issue of rapid rates of morbidity, another oddity concerning some of the people who became sick during 1918 had to do with the onset of pulmonary edema in which the lungs of patients would fill up with fluids generated by, among other things, the blood from hemorrhaging tissue. Such people died by drowning in their own fluids.

What was odd about the foregoing feature is there was very little, if any, inflammation that was observed prior to, or during, the rising, deadly onslaught of such bodily fluids. The

presence of pulmonary edema together with the absence of inflammation was not ordinary when compared with cases of influenza that had occurred in past years.

A third, somewhat unique aspect of the patient histories that were being studied by Taubenberger in conjunction with the 1918 "Flu" had to do with the age of the individuals who were succumbing to whatever the pathology might have been that was stalking people during that time. Most of the cases he studied involved people who had been healthy and were young, rather than consisting of the sorts of elderly individuals who normally fell victim to influenza.

Therefore, in summary, there were at least three properties associated with some of the 70 cases that had been archived from 1918 that distinguished those cases from what might be considered to have been "normal" instances of influenza based on past clinical experience. First, the time interval between the onset of symptoms and the occurrence of death was extremely rapid in various cases; secondly, many of those cases involved pulmonary edema without being accompanied by any kind of inflammation, and, finally, many of the people who were dying were much younger in age than the individuals who normally were vulnerable to the ravages of influenza.

So, presumably, any explanation that proposes to account for what is transpiring in cases such as some of the ones that were occurring in 1918 will entail putting together a causal framework that might be capable of providing a degree of insight with respect to those cases that were exhibiting properties or characteristics that departed from what previous clinical experience had indicated was the normal course of events involving influenza. Such an explanation would need to answer at least the following questions – namely: Why was pulmonary edema showing up in 1918 patients without simultaneously being accompanied by inflammation, or why were some people succumbing quickly in 1918 relative to what seemed to have happened in the past with cases of influenza, and, finally, why did whatever was happening in 1918 seem to affect – in atypical fashion relative to cases of influenza in previous years -- young people rather than the elderly?

The foregoing questions will be re-visited toward the end of this article. However, let's leave aside -- at least for the time being -- the foregoing considerations and continue on with exploring the information that is being transmitted through Taubenberger's 1998 PBS interview.

For instance, according to Taubenberger, influenza viruses are believed to replicate very quickly. Yet, why – or how -- the foregoing characteristic is present is not addressed by Taubenberger.

What is said is the following: The process of rapid replication allegedly takes place within the cells of lung tissue, and, then, in about five day's time, viral bodies supposedly withdraw from the foregoing cells and move on to infect other cells and/or individuals. Consequently, according to virologists, after about a week one will not find any viral bodies present in lung tissue cells that had been infected previously by those alleged viral bodies.

As a result, Taubenberger wanted to examine samples of "influenza" patients who died in 1918 that -- according to the archived medical records -- had died within one week, or less, from whatever pathology had befallen them. In theory, such samples might provide him with an opportunity to access some of the replicated RNA material before it disappeared from a cell's interior.

One of the cases that met the foregoing conditions was accompanied by a sample that displayed strong histological features. In other words, when one looked at the tissue sample with a microscope, one could detect evidence that was interpreted to have been the result of primary influenza pneumonia.

Virology theory contends that the influenza virus consists of eight RNA fragments. These fragments supposedly vary in length, and are believed to run from approximately 1000 to 2500 base pairs per fragment.

In his PBS interview, Taubenberger indicates that the size of the fragments that he was able to recover from the 1918 patient lung tissue sample was only about 150 to 160 base pairs long. He admits in the interview that his research project consisted largely of trying to find ways to piece together different RNA fragments that were recovered from the sample being studied and, then, eventually, he hoped to arrive at a stage of research through which he would be able to come up with a model for the entire genome of the influenza virus.

Taubenberger's research is, to some extent, based on assumptions concerning the number and type of genes that are contained in different kinds of alleged influenza viruses. In other words, the number of genes (supposedly eight) is based on a theory about gene structure and function rather than being based on discoveries concerning the actual number, structure and function of genes "in the wild" that have been isolated, characterized, and sequenced in a rigorous methodological manner.

In the PBS interview, Taubenberger indicates that his research group first looked at segments of five different genes in order to attempt to develop a sense of what the overall genomic properties of the influenza virus might look like. However, given what has been said earlier in this article, Taubenberger and his associates weren't necessarily looking at subsections of the actual genes of an alleged influenza virus, but, instead, might only have been looking at theoretical constructions of those genes … theoretical constructions that might, or might not, accurately reflect the structure of certain facets of the contents that could have – possibly -- originally existed within the cell tissue samples being studied.

Taubenberger states that after completing the foregoing sorts of preliminary studies, his group began to narrow its focus on what was considered to be – at least theoretically -- one of the primary surface proteins of the influenza virus. The aforementioned protein supposedly is coded for by the hemagglutinin gene, and virologists believe that the hemagglutinin protein is the means by which influenza viruses gain access to the interior of a host that is allegedly being infected by such an agent.

Nonetheless, once again, all Taubenberger -- as well as his research associates -- might have accomplished is to have engaged reality through the lenses and filters of the theoretical framework to which virology gives expression. After all, among other things, no one, yet, has been able to capture the dynamics of a virus entering a cell through the activity of a hemagglutinin surface protein.

Consequently, one cannot be certain that the aforementioned sorts of cellular access events actually take place. Alternatively, if the foregoing dynamics actually do occur, one still does not know the details of those dynamics and whether, or not, the character of that activity accurately reflects the theory which virologists have put forth concerning how they believe influenza viruses are structured and function.

Notwithstanding the foregoing considerations, Taubenberger maintains that his research group has succeeded in putting together the genetic sequence that is alleged to code for the hemagglutinin protein. The sequence is said to be about 1800 bases in length.

However, as noted earlier, all one can really say is that the research group has come up with a "possible" sequence which is highly theoretical in nature. This is because Taubenberger and his associates have never actually isolated an influenza virus but, instead, have put forth various hypotheses concerning the nature of those sequences that is based on various theoretical principles for which there is a consensus, of sorts, by a certain number of practitioners within the field of virology.

Yet, science requires more than consensus. One must be able to empirically demonstrate that the working hypothesis which is being used to explain certain kinds of phenomena can be verified independently by means of real world data that is capable of being replicated in a variety of experimental circumstances.

Unfortunately, in many respects, virology gives expression to a set of theories concerning the way its proponents believe certain dimensions of reality operate. As a result, virology doesn't necessarily accurately capture the facet of reality to which its theories are alluding.

As an addendum to the foregoing claim, one might note in passing that despite a lot of early hype on the matter, nonetheless, virology failed miserably to come up with a defensible viral theory of cancer during the 1970s and 1980s. Moreover, as the Perth Group in Australia -- along with Peter Duesberg, Kary Mullis, and others -- has shown, through a variety of empirical venues, virology also struck out with respect to being able to provide a verifiable explanation for precisely how HIV causes AIDS, and, yet, despite such a monumental failure, many virologists continue to engage life through their best, blustery, Wizard of OZ, knob turning, lever pulling, smoke generating, pay no attention to the man behind the curtain modes of behavior.

Furthermore, since the HIV causes AIDS debacle (which led to the deaths of millions of people in Africa and elsewhere through the ill-advised use of poisonous anti-viral medicines such as AZT), many virologists have been making a very good living promoting various modalities of fear-porn as they sought to transmit their alleged concerns to fellow human beings with respect to all manner of alleged imminent viral pandemics [such as: West Nile Virus (1999), SARS (2003), Swine Flu (2009), MERS (2012), Avian Flu (2013), Zika Virus (2015-2016), and so on] that, supposedly, were, or are, invading humanity. Moreover, virologists and other researchers were not shy to recommend that everyone urgently needed to be treated by means of one brand, or another, of virology-based vaccinations and pharmaceuticals despite the fact that none of their pronouncements – either with respect to the alleged pandemics or the proposed treatments for those putative pandemics – accurately reflected what actually transpired in the real world during the aforementioned time periods.

During his PBS interview, Taubenberger stated he felt that the complete reconstruction of the entire set of genetic instructions for the influenza virus (and not just the hemagglutinin gene on which he was focused prior to 1998) is likely to take years to complete since the fragments being studied are so small that the process of reassembling them is very time intensive. One should point out once again, however, that the foregoing

sorts of efforts will not necessarily involve reassembling the actual genetic sequence of some viral entity (For example, see my article: *The Deadliest Flu: The Complete Story of an Influenza Pandemic (?)*, which is a critical reflection on a CDC paper that purports to provide an account of the subsequent work of Taubenberger and others concerning their contention that they have "discovered" the viral agent that, supposedly, was responsible for the 1918 flu).

Instead, as intimated previously, he appears to be interested in developing a theory about what he and his associates believe such a sequence might look like, and this assumes, of course, that such an entity actually exists. In short, Taubenberger's research group is engaged in a process of interpreting certain kinds of data and, therefore, the group is not necessarily pursuing a course of research that is capable of uncovering the actual nature of the dynamics that give expression to the 1918 phenomena which they are seeking to explain.

In many respects, Taubenberger and his associates appear to have become entangled in a game of conceptual will-o'-the-wisp. If so, then the foregoing sorts of understanding which are guiding his research team could be nothing more than a series of variable glimpses into a mist of elusive data that is heavily shaped by theoretical considerations that could be distorting the nature of what actually might have happened in 1918.

According to Taubenberger, his research group believes that it can assert, with some degree of definitiveness, that the entity which they believe they have been studying is an influenza virus. More specifically, they claim that the agent they have been studying is a type A influenza and belongs to the subtype H1N1 where H and N stand for proteins that supposedly permit such an alleged virus to, respectively, be able to gain access to (i.e., infect), as well as to be able to exit, a given cell on its way to infect other cells or organisms.

Virologists maintain that there are three types of influenza viruses – namely, A, B, and C. These types of influenza are further sub-categorized according to the kind of hemagglutinin (H) and neuraminidase (N) proteins that are believed to be present on the surface of any given influenza virus.

While such influenza types and subtypes give expression to virology theory, nonetheless, no one has seen viruses entering or exiting cells via, respectively, H and N proteins. Therefore, there appears to be an absence of the requisite kinds of data which might be able to definitively verify any of the aforementioned theoretical pronouncements of virology.

Currently, virologists claim there are 14 different kinds of hemagglutinin protein subtypes and 9 different subtypes of neuraminidase proteins which differentiate one type of influenza from another type of influenza. The virus that is believed to have been present in the lung tissue samples from patients who died during 1918 is thought to be the H1N1 subtype, and this belief rests on the sorts of antibodies which were found in people who had been alive during 1918 but were able to survive whatever took place at that time.

Although there are theories within virology and immunology about how, and why, antibodies emerge, there is no reliable empirical data which actually captures the process of antibodies coming into existence. The evidence all has to do with finding antibodies at one point in time but not another, and, then, coming up with a theory for why such

antibodies are found at one time but not another, or why those antibodies exist in some people but not others.

Virologists not only believe that influenza viruses infect human beings, but, as well, such individuals also are of the opinion that those presumed viral agents are able to infect chickens, ducks, and a variety of birds as well as pigs and horses. Furthermore, based on the study of serum drawn from human beings who lived during 1918 and were able to survive whatever transpired during that year, virologists maintain that the antibodies in circulation in those individuals are a closer match to alleged swine influenza bodies that virologists believe were discovered in the 1930s than the aforementioned 1918 antibodies were a match to the human influenzas that were supposedly discovered in the 1930s.

Unfortunately, during the interview, Taubenberger does not spell out what is meant by the idea that the so-called "matches" between certain types of influenza and antibodies circulating in the blood stream are a better fit when considered in conjunction with alleged swine influenza bodies of the 1930s rather than in relation to presumed human influenza bodies of the 1930s. Antibodies can be quite promiscuous with respect to the kinds of entities with which they manifest some degree of affinity, and, therefore, one cannot be certain – as some virologists seem to be -- that the reason why there is a some amount of affinity between antibodies from 1918 and swine influenza bodies from the 1930 is necessarily because the 1918 antibodies were formed due to, or response to, an encounter with some sort of swine flu entity either just prior to, or during, the events of 1918.

In fact, if -- contrary to current theories and models of virology -- one were to entertain an hypothesis that the 1918 influenza virus did not necessarily exist, then, one would have to come up with a different theory to account for why antibodies of a certain kind might exist at one time rather than another. After all, if the 1918 influenza virus did not exist, and if influenza was caused by something other than a virus, then, making the sort of claims that some virologists seem inclined to make concerning the alleged significance that is supposedly demonstrated through the presence of alleged matches between particular kinds of antibodies and certain kinds of swine viruses becomes something of a problem.

Among other things, the foregoing conceptual crisis would force one to search for some alternative reason or set of reasons to account for why antibodies of a particular kind can be found in the serum of some people but not others. In other words, one would have to ask: Why do certain antibodies arise if this is not in response to the presence of some sort of viral agent?

Notwithstanding the foregoing considerations, Taubenberger and his research associates believe that the aforementioned purported antibody-swine flu match indicates that the 1918 flu did not come directly from avian sources but, instead, arose through some sort of mammalian connection. In other words, they believe that the path of viral transmission might have started with avian organisms, and, then, emerged, at some point, within mammalian organisms -- such as swine -- and, then, somehow, got passed on to human beings.

However, at the present time, there is no detailed account that is capable of providing a viable explanation for the supposed process through which various genetic fragments might be able to make the jump from avian hosts to swine hosts, and then, subsequently, to human hosts. Although, in general terms, the foregoing sort of transition phenomenon is

presumed to have transpired through some modality of recombinant DNA or RNA processes, nonetheless, this presumption is unaccompanied by any sort of account concerning a demonstrable, step-by-step dynamic that gives expression to the proposed series of transitions in genetic material that runs from avian, through swine, and, eventually to human beings.

The foregoing issue is crucial. In other words, based on antibody data (which, as previously suggested, does not necessarily mean what some researchers believe that data signifies), Taubenberger stipulates that prior to 1918, viruses had been circulating within human populations in a relatively non-lethal form except in conjunction with a small fraction of individuals who, for various reasons, might have been susceptible to those kinds of influenza agents, and, therefore, one needs to ask the following questions: How did the 1918 influenza virus acquire its alleged lethality, and what was the nature of the biological or molecular mechanism that underlies such supposed lethality?

According to Taubenberger, viruses tend to be genetically unstable, and, as a result, undergo regular transitions with respect to certain aspects of their structure and function. Taubenberger describes such transitions as "… presumably an adaptation of the virus, to evade the host immune response, so that the influenza virus that was circulating last year is not the same as the influenza virus that is circulating this year" and concludes by saying: "So they're very clever in that sense."

To be sure, changes in genetic sequences might give expression to some form of genetic instability, but determining the cause of those changes tends to be quite another matter. One cannot assume – as Taubenberger seems to -- that changes in the genetic sequence of a virus are due to some sort of, apparently, intentional or logistical viral strategy which seeks to adapt to a host's immune response by bringing about changes that enable successive generations to evade that same kind of immune response.

Viruses are not necessarily "very clever" in the foregoing sense." More specifically, if one were to assume that changes in genetic sequence occur among viruses, then, although some of those changes might confer a "novel" advantage of some sort, nonetheless, other changes might not necessarily confer any kind of advantage, or those changes could introduce something that is decidedly a disadvantage to the virus.

Therefore, whether or not a presumed virus acquires some sort of new "trick" that permits the immune responses of a host to be evaded will depend on the nature of the changes in genetic sequence that either do, or do not, occur. Yet, such changes do not necessarily have anything to do with some kind of adaptive strategy of 'cleverness' that is supposedly actively transpiring within a given viral entity.

In other words, changes in genetic sequence within a proposed virus could be a reflection of nothing more than – to use Taubenberger's way of stating things -- the inherent genetic instability of those entities. If so, then, as previously indicated, whatever changes occur in genetic sequence do not necessarily have anything to do with cleverness or adaptive, evolutionary strategies but merely give expression to the alleged virus's on-going susceptibility to genetic instability which arbitrarily moves the genome of the alleged virus in one direction rather than another … sometimes with felicitous results, and sometimes with problematic results, and, sometimes with the sort of variance that has no appreciable impact concerning issues of adaptability.

Taubenberger maintains that while mutations do tend to occur on a regular basis, most of these changes will not lead to substantially different structural or functional forms. However, he believes that every so often, substantial changes do occur, and this takes place he supposes as the result of some sort of recombinant exchange dynamic that takes place between two different species.

As a result, he maintains that the foregoing sorts of recombinant changes could give rise to a form of virus that has not previously been encountered. Furthermore, he believes that this sort of virus might pose a threat for any species that did not have the capacity to defend against the presence of that kind of an agent.

Of course, not all changes in genetic sequence will necessarily give rise to a variant that carries potential lethal implications in conjunction with human beings. Moreover, for a virus, the essence of adaptation is a function of being able to replicate and continue on, and such a capacity is quite independent of any potential that might bring about biological mayhem in the organisms that are being engaged by the virus.

In short, the capacity of a virus to inflict pathology on its host – or, in conjunction with some degree of vulnerability or susceptibility in a host to the properties of a virus that will generate a dynamic that results in death or disease -- is not necessarily adaptive. On the other hand, the capacity of a virus to be able to replicate is quintessentially adaptive in nature.

Although there is considerable evidence indicating that recombinant processes do occur, nonetheless, the notion that those recombinant processes will necessarily give rise, at some point, to something that is, on the one hand, capable of evading the capacity of organisms to defend against the presence of such entities, and, on the other hand, will be capable of being highly lethal in relation to its impact on a given organism is really nothing more than a conjecture. Consequently, even though Taubenberger – along with other researchers -- has put forth a hypothesis which contends that the foregoing sort of 'substantial' recombinant event occurred in connection with 1918, nonetheless, he has not provided evidence which demonstrates that such an event actually did occur.

In fact, during the PBS interview, he indicates that he actually is searching for the foregoing sort of evidence. Consequently, although – as noted earlier -- he does refer to a certain amount of data involving antibody titers in blood serum that had been drawn from people who lived during -- but survived – the 1918 event, nevertheless, at best, that sort of data is only suggestive – and can even be ambiguous with respect to its significance concerning the possible relationship between swine influenza viruses and human beings -- and, therefore, the presence of the sorts of antibody data to which Taubenberger is alluding does not necessarily support his contention that the existence of those antibodies means that they came into existence as a result of earlier encounters with swine flu antigens.

During the PBS interview, Taubenberger refers to three alleged pandemics – namely, events in 1918, 1957, and 1968 – which he believes give expression to the possibility that some sort of recombinant set of events occurred which gave rise to novel viruses of one kind or another that had lethal properties in all three of those instances. However, in each case, Taubenberger fails to put forth any evidence to persuasively demonstrate that what he believes was responsible for those three events – namely, changes in genetic sequence due to recombinant dynamics – is what actually happened.

Furthermore, one might note in passing that there is a certain amount of evidence to indicate that the events of 1918, 1957, and 1968 might not have been due to a viral agent at all. For example, in the book: *The Invisible Rainbow: A History of Electricity and Life*, Arthur Firstenberg puts forth considerable evidence in support of the possibility that the three "pandemics" cited by Taubenberger (as well as a number of other outbreaks of "influenza" that occurred prior to 1918 and after 1968) might have been due to various kinds of changes in electromagnetic radiation that were being introduced into the Earth's environment at those times (e.g., numerous new sources of powerful radio frequencies had come on line in many geographical locals just prior to and during 1918 and were being beamed throughout the world; or, in the case of 1957 there were many powerful radar facilities that were being deployed in various parts of the world, or, in the case of 1968, numerous communication and intelligence satellites had been, and were being, launched by various military groups as well as by an array of corporations and, as a result, such technology was bathing the Earth – and its life forms – in an array of electromagnetic radiation).

Radiation poisoning has been demonstrated to be capable of producing many of the same sorts or symptoms that are present in cases of influenza … symptoms that, for nearly a hundred years, have been attributed to a viral agent of some kind. In fact, although abundant evidence currently exists which is capable of demonstrating that electromagnetic radiation can bring about flu-like symptoms as well as many other kinds of pathological conditions (see the work of, among others, Samuel Milham, Olle Johansson, Martin Pall, and Devra Davis), nonetheless, to date, no one has been able to properly isolate an influenza virus which can be shown to be infectious or lethal (and the notion of "isolates" that appears in the virology literature is a bastardized version of the sort of rigorous methodologies that are needed to properly isolate, sequence, and demonstrate that such isolated agents actually exist as well as that they are actually infectious and lethal).

The foregoing considerations give expression to a very critical issue. If viruses, of one kind or another, cannot be shown (following proper isolation and sequencing) to be the cause of, say, influenza, then, one must look to some other sort of environmental trigger (e.g., chemical, electromagnetic, or biological) to account for the existence of those maladies.

Yet, if something other than a virus plays a role in the onset of influenza, then, the nature of the dynamic with which human beings are presently faced changes in substantial ways. For instance, instead of trying to come up with some kind of virology-based vaccine or virology-based pharmaceutical elixir, and, then, insisting that people – as a matter of public health – must become vaccinated with, or must ingest, such an anti-viral concoction, then, perhaps, the proper way of treating such maladies lies in another direction.

More specifically, if viruses do not have a causal role to play with respect to the occurrence of diseases such as influenza (and, to date, the viral theory of influenza rests on evidentially problematic grounds), and if, furthermore, viruses do not have a role to play in pathologies like SARS, MERS, Zika, and so on (and, once again, there has been no proper process of virus isolation that identifies different kinds of viruses as causing the foregoing maladies), then public health in those circumstances need not depend on discovering and mandating certain kinds of virology-based vaccines or pharmaceuticals.

Instead what is required is a shift in the nature of the paradigm through which those diseases are explored. In other words, if the nature of the problem with respect to the foregoing sorts of maladies is not a function of the role that different kinds of infectious agents of a viral nature play, then, perhaps the problems associated with, for example, influenza, might be better resolved if one were to suppose that the diseases mentioned previously might be due not to viruses but, instead, could be due to, for example, the impact that different kinds of electromagnetic and/or chemical poisoning are having on the environment along with the ecologies that reside in the environment.

If the latter possibility were the case, then the onus of responsibility for combating those pathologies would no longer be a matter of trying to foist off some sort of mandated vaccine or pharmaceutical program onto the people and, then, proceeding to try to argue that resolving those health crises requires individuals to do their civic duty and take their medicine in order to protect others. Instead, the responsibility for combating the aforementioned diseases shifts to those who are poisoning the environment through chemical, electromagnetic, or biological means, and, therefore, what must be mandated are not various kinds of vaccines or pharmaceuticals but, rather, mandates should be issued which require various environmental polluters to cease and desist with respect to the activities which are poisoning human beings.

Toward the latter part of his 1998 PBS interview, Taubenberger returns to the idea of evolutionary adaptation. For example, after mentioning how there are many bacteria which can be found on our skins and within various parts of the gastrointestinal tract that are well-adapted to the surrounding biological environment and which actually perform many useful functions for their hosts – such as generating vitamin K – he goes on to allude to different kinds of bacteria and viruses that are not well-adapted to their hosts and, as a result, those entities take on what Taubenberger believes to be is an adversarial relationship with their hosts.

Taubenberger does not explain how bacteria and their hosts came to work out adaptive solutions which serve their mutual interests – or how they discover ways that, at least, do not adversely affect one another. Furthermore, he does not mention the fact that there are many different kinds of agents that have been found on, say, human skin – such as staphylococcus aureus – that, under the right circumstances, are potentially harmful but which, for unknown reasons, are not always active, and, therefore, contrary to what Taubenberger claims, do not automatically take on an adversarial relationship with their hosts.

In any event, Taubenberger indicates that if an agent -- virus ‘x’ -- were to behave in an overly aggressively manner with respect to their hosts, then, the infected individuals will die too quickly. As a result, this sort of aggressive activity would tend to prevent that virus from being able to move on to other hosts.

Taubenberger alludes to the idea that the alleged 1918 virus seems to have avoided the foregoing sort of problem and, instead, was able to work out a good evolutionary strategy. In other words, although he believes that the virus killed a lot of people, nevertheless, it somehow managed to constrain its activities in ways that only lethally affected somewhere between 2 and 5 percent of the population.

According to Taubenberger, by behaving in the foregoing manner, such a strategy provided the virus with an opportunity to move from host to host and, thereby, spread all over the world since only a relatively small percentage of the host population succumbed to the alleged onslaught of that virus. One wonders, however, whether the aforementioned 2-5% solution is the product of an evolutionary strategy that emerged in some inexplicable manner or whether that percentage merely reflects the possibility that 2-5% of the population is, for whatever reasons, vulnerable to the presence of certain kinds of agents and, therefore, the 2-5% figure might have nothing to do with some sort of viral evolutionary strategy but, instead, just gives expression to the manner in which viral agents with certain kinds of properties interact with susceptible biological systems in a given set of contingent circumstances and, in certain instances, leads to a series of complex interactions that result in the demise of some of those organisms.

Taubenberger maintains that as a virus is transmitted from locale to locale in different regions of the world, people eventually would have developed an effective immune response to the virus. He further contends that such a state of affairs of general immunity would have placed the virus under "enormous pressure" to undergo mutation so that it could change some facet of its genetic composition – such as the part of the genome that gave expression to one or another protein on its surface – in order to be able to find new ways of infecting human hosts.

Notwithstanding Taubenberger's foregoing account, one might suppose that mutations either occur, or they don't. One does not need to assume that there is some sort of "pressure" that is present which induces a given virus to mutate.

Taubenberger's use of the term "pressure" might merely be his way of framing the discussion by means of a theory which seeks to advance the possibility that there is some kind of "force" in existence which is capable of inducing organisms to move in – or mutate in -- new directions that will prove to be adaptive. However, over a period of several billion years, the primary lesson of life on Earth would seem to be that, sooner or later, almost all species tend toward extinction irrespective of whatever changes might, or might not, take place with respect to their genomes.

As far as we know, to whatever extent viruses exist, they consist only of a glycoprotein coating which houses either an RNA or DNA-based genomic reservoir which codes for a small number of genes that, under the right circumstances, supposedly enable those viruses to go about the business of replicating themselves by hijacking the machinery of a host cell or organism. Whether the foregoing entities can be considered to be alive in some sense is a debatable issue, but irrespective of their existential status, there is nothing in their molecular or genetic composition which would seem to suggest that there is some underlying force or pressure within them, or working through them, that requires mutations of a certain kind to emerge … namely, mutations that would allow those entities to find new ways to infect and/or inflict damage on a host.

However, Taubenberger resorts to the idea of viruses operating under an 'extreme pressure' to bring about adaptive mutations of certain kinds in order to account for why, after 1918, the alleged pandemic did not continue on but, eventually, petered out. Presumably, the virus had undergone some sort of mutation that would permit it to continue to circulate within the human population but, in the process, had – due, perhaps,

to the immune responses of host organisms – lost the ability to have anything more than a limited capacity for lethality with respect to all but a small percentage of human beings who were somehow vulnerable to such a viral presence.

Yet, to suppose, as Taubenberger does, that a virus must mutate if it is to continue on is not necessarily true. Indeed, until one knows why some people are either more vulnerable than others -- or vulnerable at all -- to the presence of a viral agent, one cannot necessarily suppose that the virus will have to mutate in order to continue to be able to infect a host.

Thus, irrespective of whether, or not, antibodies arise in conjunction with the presence of a given viral agent -- and leaving aside the issue of whether, or not, the presence of those antibodies helps confer sufficient immunity to prevent all of a virus's genetic potential from being able to express themselves -- it might be that some small percentage of a previous viral population will continue to exist even if such entities were to have lost their capacity to act in a lethal manner with respect to most individuals within a host population. A virus – to whatever extent it exists – has certain capabilities that (given the right opportunity) will be expressed, but in other circumstances might just remain inactive.

If the right kind of conducive circumstances do not arise, then, even if the virus was not able to fully express itself, nonetheless, it might continue to exist for an indeterminate or indefinite period of time quite independently of whether, or not, a host actively engages – or is engaged by -- such an agent. The entity just wouldn't replicate, and since viruses – to whatever extent they exist – are not necessarily "alive," then whether or not replication continues to occur is not necessarily a matter of "life and death" for such an entity.

The life cycle of a virus – to whatever extent it exists -- is digital in nature. It is either on or off … that is, it either replicates or it doesn't.

Whatever else happens with respect to such an entity – in the way of lethality or infectivity or pathology – will be a matter of the particular manner in which a given virus and a given host interact with one another during the time in which the two are in contact. Conceivably, a virus could remain inactive or dormant even though the circumstances that are necessary for replication are not present, and, yet, such a body might still continue to inhabit a host just as bacteria like staphylococcus aureus can be found in human beings in a non-active or non-problematic state.

Consequently, Taubenberger's notion that viruses must mutate in order to continue their existence is little more than a conjecture. While the possibility that he mentions is consistent with the theory of viruses as well as an evolutionary framework, there is not any evidence which is capable of definitively demonstrating the truth of the conceptual thrust of his conjecture concerning the existence of some sort of pressure that induces a virus to continue to mutate in ways that are increasingly adaptive in some sense of the word.

Indeed, one might suppose that developing some sort of capacity for lethality is actually counterproductive for a virus's continued viability. Viruses appear to complete their life-cycle via replication and not through inflicting pathology.

There is no evident evolutionary purpose that appears to be served by enhancing the capacity of a virus to inflict pathology. Being able to gain access to the interior of a cell or to be able to find a way out of that cell or to be able to borrow some of a cell's potential to replicate does not necessarily require the virus to be able to "infect" that cell in

pathological manner and, thereby, cause some sort of disease anymore than DNA or RNA needs to inflict damage on a cell in order to be able to replicate.

Taubenberger's 1998 PBS account of the 1918 pandemic leaves unanswered a number of questions. For example, what was the specific nature of the recombinant event(s) involving -- at least, possibly, initially -- birds and mammals (such as swine) and, then, how did the process of species jumping continue on by, allegedly, making the transition from the foregoing sorts of mammals to human beings? One also would like to know the precise character of the dynamics of lethality that supposedly arose in an unknown manner, and, therefore, one might ask whether the lethality came from birds, or mammals, or, in some unanticipated way, emerged during the time when the jump was made to human beings? Finally, one might also ask why and how such a lethal agent suddenly appeared to vanish.

Apparently, Taubenberger is putting forth nothing more than a narrative which has been woven from various assumptions and conjectures based on a hermeneutical engagement of different kinds of empirical data. Indeed, in many respects, virology – and any discipline (for instance, molecular pathology) that has a potential for contributing to the development of virology -- appears to be nothing more than a theoretical narrative which seems to be masquerading as a set of scientific discoveries.

Taubenberger states that: "Historically, it seems that most new influenza viruses emerge in Asia, in the Far East, which is another thing that's unusual about the 1918 virus because everything we know historically suggested that it actually originated in the United States." One might wonder, however, about why different kinds of influenza supposedly have such an inclination to begin in Asia.

Could the foregoing sort of asymmetry in racial or ethnic susceptibility be a function of certain kinds of environmental conditions (e.g., electromagnetic, chemical, as well as biological)? Or, could such a racial or ethnic asymmetry be due to some sort of genetic vulnerability that is more pronounced in Asians relative to other racial and ethnic groups? Or, perhaps such an asymmetry might be due to some sort of systemic iatrogenic issue in which various kinds of pneumonia and respiratory diseases are being misdiagnosed as, or confused with, influenza, and, as a result, one is being given a distorted impression of what is actually taking place or whether there is any actual kind of asymmetry in susceptibility to influenza that is present.

Nonetheless, notwithstanding the foregoing sorts of considerations, Taubenberger's claim that the 1918 event started in the United States is not necessarily capable of being verified. More specifically, there is a considerable body of evidence (e.g., see *Virus Mania* by Torsten Engelbrecht and Claus Köhnlein, as well as *The Invisible Rainbow* by Arthur Firstenberg) indicating that large numbers of people were dying all over the Earth from influenza-like maladies at roughly the same time in 1918, and, indeed, even Taubenberger states during the PBS interview that the spread of influenza took place with an incredible rapidity that occurred "within a period of a month or so in the fall of" that year.

Consequently influenza-like deaths were taking place in many locations around the world in a fashion that seemed to be faster than could be accounted for by any possible route of surface transmission that was available at that time (e.g., horses, automobiles, trains, or ships). On the other hand, the seemingly inexplicable rapidity of disease transmission in 1918 would be quite consistent with the possibility that the deaths being

attributed to the "Spanish Flu" were actually due to the generation of electromagnetic frequencies that were poisoning people all over the world in a, more or less, simultaneous fashion at roughly the speed of light.

The explanation which Taubenberger offers as a way of trying to account for why influenza tends to emerge in Asian societies rather than in Western nations is zoonotic in nature. In other words, he contends that the cultural eating habits of many Asians involves going to so-called wet markets where various exotic life forms are available for purchase and consumption.

Presumably, somewhere along the line -- during or following the aforementioned visits to the so-called wet markets -- influenzas supposedly made a species jump from birds to mammals of one kind or another, or, a species jump allegedly transpired between mammals of one kind to other mammals such as human beings. Yet, as intimated previously, Taubenberger really doesn't appear to have any concrete evidence that is capable of demonstrating the validity of his zoonotic hypothesis.

Taubenberger goes on to indicate that during the 1950s "influenza viruses could be cultured and characterized in the laboratory." Technically speaking, however, viruses are not living and, therefore, do not need to be cultured. Indeed, short of a fully functioning host, there is no medium in which one could place a virus in order to help it grow and replicate.

In fact, if a given virus is functional, then, one does not need to place such a virus in some sort of medium culture. All one has to do is take a virus that has been properly isolated – and, therefore, separated from everything else including a culture medium of some kind – then, expose a potential host to that isolated virus and, finally, just wait to see what takes place.

This is what transpires in the wild, so to speak. Introducing cultured mediums into the research process merely obfuscates the character of whatever dynamics might follow.

According to Taubenberger, various attempts were made to exhume bodies of individuals in Alaska and elsewhere who supposedly died of influenza during 1918. However, while those exploratory expeditions were able to bring forth live bacteria through the use of various kinds of culture mediums, no one had been able to induce influenza viruses to surface.

In passing, Taubenberger mentions the work of a Canadian researcher, Dr. Kirsty Duncan, who has been attempting to locate the bodies of individuals who had died from influenza in 1918 but who had been buried in very cold – i.e., frozen – conditions. He notes that she is hoping to be able to uncover functional viruses from the foregoing sorts of cold storage exhumations.

Taubenberger contends that he feels the aforementioned research venture is not likely to succeed. He goes on to indicate that influenza viruses are quite fragile and that although bodies frozen in permafrost might retain some fragments of viral RNA, nonetheless, those samples would be unlikely to contain "live" or viable viral entities because of – as previously noted -- the fragile character of the influenza virus.

While Taubenberger mentions the extremely fragile nature of influenza viruses in the foregoing overview, nonetheless, he doesn't actually go into any sort of detail about the

kind of environmental conditions that are necessary in order for a virus to be able to "survive" – i.e., be in a position to replicate when conditions are right. Presumably, the understanding which the aforementioned sort of missing information might help engender would be of value if one wanted to try to figure out the nature of the dynamic through which influenza viruses and human beings tend to engage one another, and, furthermore, such information also would be of value if wished to determine what kinds of conditions might be more conducive or less conducive to such alleged viruses becoming active within a host – human or otherwise.

Taubenberger believes that, generally speaking, societies in 1998 are in a much better position than they were in 1918 to be able to deal with potential pandemics. He feels this is the case because, among other things, "…we know that influenza viruses exist, and we can analyze them and watch their emergence and evolution." In addition, Taubenberger maintains that societies also are better prepared to deal with potential forthcoming pandemics due to (1) advancements in medical treatment such as drugs that, supposedly, are able to thwart the capacity of influenza viruses to, for example, replicate, as well as due to (2) the emergence of influenza vaccines which Taubenberger claims "are obviously the most important factor of our current armamentarium against influenza viruses."

However, as noted previously, neither Taubenberger, nor anyone else, has actually gone through the necessary set of rigorous procedures which are capable of properly isolating, characterizing, or sequencing the alleged1918 influenza virus, nor, in addition, has he or other researchers also been able to go on to reliably demonstrate that such isolated virus are both infectious as well as lethal [See my article: "The Deadliest Flu: The Complete Story of a Virus Influenza Pandemic (?)"]. Moreover, the antiviral treatments that are used to treat various viruses have proven, quite frequently, to be quite hazardous in their own right (for example, consider the deadly impact that the use of AZT had on the treatment of alleged cases of HIV).

Finally, notwithstanding Taubenberger's foregoing claim to the contrary concerning the alleged essential role of vaccines, there is considerable evidence that flu vaccines (e.g., see *Jabbed* by Brett Wilcox; *The Vaccine Court* by Wayne Rohde; Dissolving *Illusions: Disease, Vaccines, and the Forgotten History* by Dr. Suzanne Humphries and Roman Bystrianyk; *Vaccines: A Reappraisal* by Dr. Richard Moskowitz, Vaccine Epidemic, edited by Louise Kuo Habakus and Mary Holland, as well as *What Really Makes You Ill? – Why Everything You Thought You Knew About Disease Is Wrong* by Dawn Lester and David Parker) are neither safe nor effective. In this respect, one might consider, among other possibilities, the fiasco that arose in 1976 with respect to so-called swine flu in which hundreds of cases were documented in which human beings suffered from Guillain-Barré Syndrome, instances of transverse myelitus, or death as a result of the flu vaccines that were given in 1976.

One might also note in closing – and as was intimated to be a topic that would resurface toward the beginning of this article -- that early in the PBS interview Taubenberger listed a number of features that were atypical with respect to cases of influenza that had been encountered prior to the 1918 event. More specifically, he indicated that: (1) the death of many individuals took place very rapidly following the onset of symptoms; (2) a substantial number of the cases that occurred in 1918 exhibited signs of pneumonia edema without any accompanying inflammation; (3) a large proportion of the cases he studied involved

individuals who had been healthy and were young, rather than the sort of elderly people who, in the past, normally fell victim to influenza; (4) the "influenza" that occurred in 1918 seemed to emerge, more or less, simultaneously in different parts of the world rather than following some sort of epidemiological path that moved from one location to the next via individuals who were traveling by foot, or via horses, trains, or ships.

Nothing which Taubenberger stated in the 1998 PBS interview is capable of providing an answer to any of the foregoing anomalies that he, himself, introduced into the discussion and which seemed to differentiate the 1918 event from previous bouts of influenza. While he offers a lot of conjectures during his interview, nevertheless, he does not provide much in the way of substantive, definitive information that is capable of addressing the four aforementioned anomalies that apparently were uniquely characteristic of the 1918 "influenza" event and do so in a satisfactory manner.

Finally, as indicated earlier in this article, during the 1998 PBS interview, Taubenberger attempted to describe some of his research concerning the hemagglutinin gene and, in the process, sought to link that work to the events of the 1918 "Flu". However, at best, his research only appears to advance a theoretical narrative, of sorts, concerning what he believes transpired in 1918 rather than giving expression to a fully delineated account of the 1918 phenomenon that is capable of being empirically substantiated.