

Failed Attempts to Prove Contagion: 1958 Paper on Speculations Regarding Causes of Influenza

Influenza cannot be “transmitted” and appears simultaneously in places faster than available means of travel and contagionists wrongly turn effects and consequences into causes through misinterpretation of observations coupled with speculative unproven theories of disease.

Shaykh Ḥāfiẓ al-Ḥakamī (رَحِمَهُ اللهُ) stated:

The intent is that the negation of contagion¹ is absolute, it is upon its totality and within it is singling out Allāh (سُبْحَانَهُ وَتَعَالَى) with full disposal [of all affairs] in His creation...

And within that lies strengthening of the heart of the believers, aiding of them with the strength of reliance and soundness of certainty, and proof for them against the polytheists and all of the stubborn opposers.²

The superstition of contagion rests upon dismissing or ignoring the multi-factorial, multi-causal and often the very hidden nature of disease causation as a result of which people fall into **confusing association with causation**, wherein exaggeration is made in coincidental encounters, turning them into causes without any evidence at all. The Prophet (صَلَّى اللهُ عَلَيْهِ وَسَلَّمَ) alluded to this affair in

¹ This speech might be perplexing to those who have been indoctrinated in the speculative sciences of the materialists and naturalists and they are advised to explore this matter with an open mind so that the superstitious beliefs they currently think are established scientific realities might become clear to them, particularly in the pseudoscience of virology.

² Ma‘ārij al-Qubul of al-Ḥakamī.

what he brought of the most perfect guidance, and this was the very mistake of the bedouin whose observational error [of confusing coincidence with causation] he pointed out with a very simple statement, “*Who/what passed it to the first one?*”

In support of these truths, I present below excerpts from another paper from 1958 written by a Dr. Richard Shopes, a Professor and member of the Rockefeller Institute of Medical Research, titled “**Influenza: History, Epidemiology and Speculation**” which can be found on the Pubmed archives here:

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1951634/>

Public Health Rep. 1958 Feb; 73(2): 165–179.

The two main points extracted are that historically speaking influenza appeared simultaneously in places too far from each other to be explained by the available means of transport and that experiments mimicking real life situations repeatedly failed to prove contagion. Further, attempts to pinpoint an exact cause have been elusive and remain speculative, and are not free of mistakes of logic, causation and of misinterpretation of observations being made therein.

Influenza

History, Epidemiology, and Speculation

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I have selected three outbreaks of pandemic influenza to discuss, for comparative purposes, and to use historically in connection with my consideration of the present influenza outbreak. I have chosen one from olden days, before the speed of modern travel entered to confuse the epidemiological picture (1789), one from the beginning of the bacteriological era (1889), and one modern one (1918).

Pandemic of 1789

The 1789 outbreak of influenza as it occurred in the United States was well described by Robert Johnson in his inaugural dissertation for the degree of doctor of medicine at the University of Pennsylvania in 1793. To orient you as to the time of this influenza outbreak, it came in the year that Washington was inaugurated President, that the first Congress met in New York, and that the French Revolution began. The first steamboat did not cross the Atlantic until 1819, and the first steam train did not run until 1830. Air travel, of course, was not even dreamed of. This outbreak occurred before modern means of rapid travel were available and when a man could go no faster than his horse could gallop. Despite this, according to Johnson, the influenza of 1789 spread like wildfire.

Now Johnson, in his definition of influenza, characterized it, among other things, as "a disease capable of being propagated by contagion." In spite of this conception, he could not completely rationalize the speed of its dissemination on the basis of transmission by contagion alone and visualized the importance of a "vicious quality of the air."

It became obvious to people in the days of travel by horse and ship that “contagion” could not explain the simultaneous appearance of disease across very large geographical areas. So they were forced into incorporating some reality into the explanation, which at that time was to invoke the quality of the air, though that in itself is only one factor of many in the mechanisms of disease causation.

Johnson supported his contention about the spread of influenza by citing examples from the pandemic of 1782, in which he felt transmission by contact did not play the essential role. He stated, “Influenza appeared at London between the 12th and 18th, at Oxford in the third week, and at Edinburgh on the 20th day of May.” He doubted that the disease could have been transferred to these three cities in such rapid succession “by things imbued with the contagion or by persons labouring under the complaint.”

Later in his thesis he wrote, “On the 2d day of May 1782, the late Admiral Kempenfelt sailed from Spithead with a squadron under his command, of which the Goliath was one, whose crew was attacked with the influenza, on the 29th of that month: the rest were affected at different times: and so many of the men were rendered incapable of duty by this prevailing sickness, that the whole squadron was obliged to return into port about the second week in June, not having had communication with any shore, and having cruised solely between Brest and the Lizard.”

Still another example was cited as follows, “About the 6th of May [in the same year 1782], Lord Howe sailed for the Dutch coast, with a large fleet under his command: all were in perfect health: towards the end of May the disorder first appeared in the Rippon, and in 2 days after in the Princess Amelia. Other ships of the same fleet were affected with it at different periods: Some indeed not until their return to Portsmouth about the second week in June. This fleet also had no communication with the shore until their return to the Downs, on their way back to Portsmouth, towards the 3d or 4th of June.”

Robert Johnson, in his 1793 dissertation, gave examples of how contagion cannot explain the outbreak of disease. He then gave some rationalisation:

Johnson rationalized his views concerning the multiplicity of foci of origin of influenza during a pandemic by contending, “The morbid matter exciting the disease must have originated at some time and somewhere: and a cause like to that which gave rise to it in any one country, at any one point of time, might produce it in another country at the same time, under similar circumstances.”

This explanation here is what the Messenger (ﷺ) said to the bedouin when he said: “*And who/what gave it to the first one?*” Which means that just as the first camel got scabies without a prior one to allegedly “infect” it, then likewise, the second, third, fourth and the rest of them. They were all subjected to the same factors, causes and conditions from their surroundings, drink, diet and environment which led to them developing the disease and their interactions were coincidental.

The same applies to other disease conditions, such as influenza, colds, leprosy³ and so on.

Johnson, being perplexed, still cannot let go of the notion of contagion and states:

He continues, “I do not assert, nor do I wish to be understood to mean, that the influenza is not at all contagious: on the contrary, I am possessed of facts which prove in the most incontestable manner, that it may be, and often is, propagated from one person to another by means of contagion. But I mean, and the arguments which I have adduced, I trust, will warrant the conclusion, that the disease often does arise from some vicious quality of the air, or exhalation in it, as well as from a matter arising from the body of a man labouring under disease.”

The facts possessed by Johnson to suggest that influenza “may be and often is propagated from one person to another” do not constitute evidence for this claim because the “transmission” of influenza through contagion was falsified a century and half later by experiments and in any case, his facts would simply be resting on the confusion between association and causation.

After this Shopes discusses the 1918 Influenza and follows the same path of Johnson regarding the 1789 pandemic:

³ See <https://abuiyaad.com/w/leprosy-contagion> and also <https://abuiyaad.com/a/qurtubi-contagion> towards the end of the document, the section on leprosy and contagion.

However, certain discrepancies enter to spoil the perfection of the case-to-case transfer explanation for the spread of influenza during the second wave of the 1918 pandemic. These have to do with certain flukes in distribution, certain skips of large bodies of population. For example, Boston and Bombay had their epidemic peaks in the same week, while New York, only a few hours by train from Boston, did not have its peak until 3 weeks later (10). In like manner, Seattle, Los Angeles, and San Francisco had their epidemic peaks some 2 weeks earlier than Pittsburgh, which is just an overnight run from the infected eastern seaboard cities. In some respects, the epidemiologist had an easier time getting the pandemic disease transferred over long distances than in taking it to communities nearby. Thus, though it got

In the light of these various epidemiological ambiguities one cannot help wondering whether perhaps more than one mechanism of dissemination may have been operating during the 1918 pandemic to account, on the one hand, for the lightning-like spread of disease over large distances and, on the other hand, for its slower diffusion over relatively small distances. The suggestion is apparent that extensive and widespread preseeding of virus in a masked or occult form, with its almost simultaneous provocation to infectivity by a stress common to wide geographic areas, might better account for the appearance of extremely rapid dissemination over great distances than does the view that case-to-case transfer was the responsible mechanism.

So here Shope is now saying it could be the case that the alleged virus⁴ is preseeded over large geographical areas—meaning it is

⁴ There is such thing as a “pathogenic virus” and virologists confuse an effect with its cause, similar to how the presence of ash after a fire is blamed for

already present, in circulation—and then an environmental, geographical stress factor which covers large areas provokes the alleged virus into “infectivity”. Thus, people become ill, not because of contagion, through passage from one person to another, but by a combination of causes that affect them all in a similar manner at the same time, but in many different places.

Then Shopes goes on to discuss attempts to prove contagion.

Efforts to Prove Contagiousness

With all of the observed clinical and epidemiological evidence pointing to the likelihood that the 1918 pandemic influenza was highly contagious and spread from sick to well easily and apparently at the very first available opportunity, one would have anticipated that proof of its contagiousness by transmission tests in human volunteers would have been extremely easy. However, such did not prove to be the case: in not a single controlled experiment was it possible to demonstrate the transmissibility of the disease.

Note that clinical and epidemiological evidence cannot prove contagion for reasons we have discussed elsewhere, because it is by and large simply a matter of confusing association with causation, upon which a large part of “epidemiology” rests.

causing the fire due to its frequent presence at scenes of fires. The components of what are alleged to be “viruses” are simply fragments and debris, breakdown products of dying or dead cells which the body expels, and likewise microvesiccles of various sorts which have a number of roles. These are misunderstood and wrongly abstracted into a “pathogenic virus” by fanatical, zealous germ theorists and virus hunters whose dogmas and beliefs were falsified in the early 20th century, no different to how the religion of the Jahmites was falsified in the 2nd century hijrah, yet despite that, there continued people after them propagating the same falsehoods in disguised form, such as the Mu‘tazilah and Ash‘ariyyah, centuries later.

The most carefully planned and conducted experiments were those carried out by the Navy and the Public Health Service. In the series of experiments conducted in Boston during November and December 1918, 62 volunteers between 15 and 34 years of age were used (18). Thirty-nine of these had no history of having had influenza at any time, although apparently some degree of exposure had occurred. Filtered and unfiltered secretions from the upper respiratory tracts of patients with typical influenza were sprayed into the nose and throat and instilled into the eyes of some of the volunteers; direct swabbing from nasopharynx to nasopharynx was the method of exposure for others; and in one experiment freshly drawn citrated blood was injected subcutaneously. The results were summarized as follows: "In only one instance was any reaction observed in which a diagnosis of influenza could not be excluded, and here a mildly inflamed throat seemed the more probable cause of the fever and other symptoms. Nothing like influenza developed in the other volunteers."

In an attempt to imitate nature more closely, 10 volunteers were exposed to patients with acute influenza in hospital wards. Each volunteer was placed very near the patient, shook hands with him, chatted with him for 5 minutes, and then received the patient's breath full in his face five times while he inhaled. Finally the patient coughed five times directly in the subject's face. Each volunteer did this with each of 10 different patients, all of them acutely ill for not more than 3 days. All patients used had typical acute cases selected from a distinct focus or outbreak of disease. None of the volunteers developed the disease.

A second series of similar experiments was carried out in San Francisco during the same period also with completely negative results (19).

Shopes then summarises the results of all the research and experimentation that was done to find the cause of the 1918 Influenza:

Much work was expended during the 1918 pandemic in an effort to determine the causative agent of the outbreak. Prior to the 1918 studies, *Hemophilus influenzae* had been generally regarded as the agent responsible for influenza. It seems quite natural, therefore, that much of the 1918 investigative work should have been concerned with a further study of the relationship of this bacterium to the disease. The results obtained were frequently confusing and contradictory, which is not surprising in view of the fastidious character of the organism and the technical difficulties associated with its isolation from the respiratory tract. It is difficult to give an accurate appraisal of the significance of the large amount of work done during the 1918 pandemic in trying to prove or disprove the etiological relationship of the Pfeiffer bacillus to influenza. About all that can be said is that the role of the organism was more controversial after the smoke of the 1918 pandemic studies had cleared than it had been before.

With the failure to gain clear-cut evidence that *H. influenzae* was the cause of the 1918 pandemic, the view was rather widely held and was frequently expressed that a virus was probably the etiological basis for the disease. This actually constituted no more than an ungrounded opinion, for consideration of the data on the subject published from 1918 investigations reveals that no one adduced good evidence to incriminate a virus as the causative agent. The upshot of a terrific amount of effort during the 1918 influenza pandemic to learn the cause of the disease was to weaken the view that Pfeiffer's bacillus was the etiological agent and to substitute no other in its place.

The conclusion is that no clear-cut evidence had been provided for a bacterial or viral causation of the Influenza, and if anything had been achieved, it was that the view of bacterial causation by *Hemophilus influenza* had been weakened and no substitute cause was given or proven and the view regarding a virus was an ungrounded opinion.

After this Shopes goes on to develop the claim that a causative agent was eventually provided through the workings of nature, and he goes on the trail of Swine flu, asserting that the causative agent, another bacterium, came from swine, given that outbreaks would occur in families following outbreaks in pigs. However, this epidemiological observation is not proof, since localised environmental conditions and geographical stresses affecting both animals and humans could be the true underlying causes, alongside individual susceptibility to illness that varies from person to person and which itself has its own causes returning to diet, habits and lifestyle. Further, the presence and isolation of bacteria is not proof of causation, since bacteria come to the scene of diseased and damaged tissue and are not the primary cause of disease. Here is what Shopes writes next:

The late Dr. Paul A. Lewis and I began our studies of swine influenza during the autumn of 1928, and we were elated and pleased when we isolated from our very first cases of the disease an organism that was, so far as we could tell, like the non-indol-producing strains of Pfeiffer's bacillus (23). We named this organism *Hemophilus influenzae suis*. We isolated the same organism from field outbreaks of swine influenza again in 1929 and in 1930. It was the only organism we found with any regularity, and sometimes it was the only one present in the respiratory tracts of sick swine. Unfortunately, so far as assigning it etiological importance was concerned, *H. influenzae suis* administered in pure culture to susceptible swine produced no illness. We were thus faced with the dilemma of having found an organism that seemed always to be present in cases of the disease, that was demonstrable at the sites of the influenza lesions in the respiratory tract, but that failed to induce disease when administered to normal swine.

As we have pointed out elsewhere, bacteria do not cause disease, but they are a consequence of disease states, their role is a janitorial one, one of cleaning up. They come to diseased or dying tissue and in order to maintain the integrity of the body, they break the dead tissue down, and either repurpose the broken down components or prepare them for elimination. As for what caused the tissue or cells to die, then it reduces to improper nutrition and/or toxicity of various sorts.

If you take bacteria that are feasting on and breaking down dead organic matter and completely separate them from that matter until you only have a pure culture of that bacteria, and then place them on healthy, living tissue, nothing will happen. This is why the germ theorists were being exposed, ridiculed and disproven in the

early 20th century by their opponents who would take glassfuls of cholera bacteria and drink them without becoming ill at all.

However, not satisfied, the germ hunters tried to find some other particle to blame the disease on, and this is where we come to the “virus”. Shopes continues:

It was subsequently found that a filtrable virus, differing from any hitherto known, was important in the causation of swine influenza (24). This virus, however, was not the sole cause of swine influenza: when the virus was administered alone to susceptible swine it produced a disease that was clinically much milder than the true swine influenza as seen under natural conditions.

In reality, all that happened here is that, having failed to induce disease with the bacteria, they were simply looking at debris, fragments, breakdown materials which the body expels as part of a cleaning, detoxification mechanism, in which built up waste, morbid materials, dead and diseased tissues and linings are expelled from the body through a collection of mechanisms and symptoms which we call a “cold” or a “flu”, and they called this a “filterable virus”. They wrongly treated the effect, or the outcome or the end-result of these mechanisms to be the cause, similar to how ash is mistaken as the cause of a fire, when it was the end result of the fire and the fire was caused by something else.

So when they took this material which they called a “virus” and injected it into animals, they were effectively poisoning the body of the animal, as injection of foreign material directly into the tissue or blood is not a normal, natural route. Naturally the body is going to react and work to expel the foreign materials, and therefore some mild symptoms may be observed. This is really a pre-programmed response, in-built into the body. The true ailment was the poisoning and what we perceive as the

“disease”—symptoms such as fever—that is actually the in-built healing, detoxifying mechanism.

In short, bacteria which are living organisms and the “virus” were blamed for illness when in reality, they were the consequences of disease states created by other factors and conditions which were ignored, keeping in mind there had just been a huge world war. The word “virus” is an incorrect label to what are individual elements such as proteins, vesicles, genetic fragments, it is a theoretical abstraction wherein dead, inert, lifeless matter is constructed into a particle, an agent, called the “virus” and in the modern era, theoretical genetic sequences are created through sleight of hand tricks in laboratories and use of computer software.

In conclusion, this is another paper through which we can glean some realities regarding how germ theorists and virus hunters are misguided in their beliefs while they confuse association with causation and wrongly reverse and flip effects as causes, which is what they do with bacteria and “viruses”.

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13 Rabī‘ al-Thānī 1443 / 18 November 2021—v.1.0