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Exposing the lies of Germ Theory and virology using their own sources.

Maurice Hilleman and the Avian Flu Pandemics

" Hilleman put down the paper: "My God," he said, "This is the pandemic. It's here!"

As I was investigating the origins of the flu "virus," I had come upon the infamous avian flu and decided to crack the egg open so to speak. I originally set out to uncover whether this would be the one "virus" that would finally provide evidence of both purification and isolation. < https://viroliegy.com/category/purification-isolation/ > Needless to say, I was not expecting to find such evidence as I already knew full well that the original strains of influenza < https://viroliegy.com/category/influenza/ > had never been properly purified nor isolated directly from the samples taken from sick patients. If the original influenza strain was never proven to exist, there was little chance a variant would be either.

My search for the origins of the avian flu lead me to originally look into the 1968 "Hong Kong" flu pandemic where the world encountered a flu strain that was said to have occurred due to a mixing of bird and human strains. This variant of the flu was ultimately labelled H3N2. I decided it was as good a place to start as any yet upon tracing the origins and uncovering the actual methods used for the supposed isolation of H3N2, I realized this was a more difficult task than I had initially thought it would be. This is partly due to the fact that the history of the 1968 H3N2 "virus" is heavily tied to the 1957 H2N2 "virus."

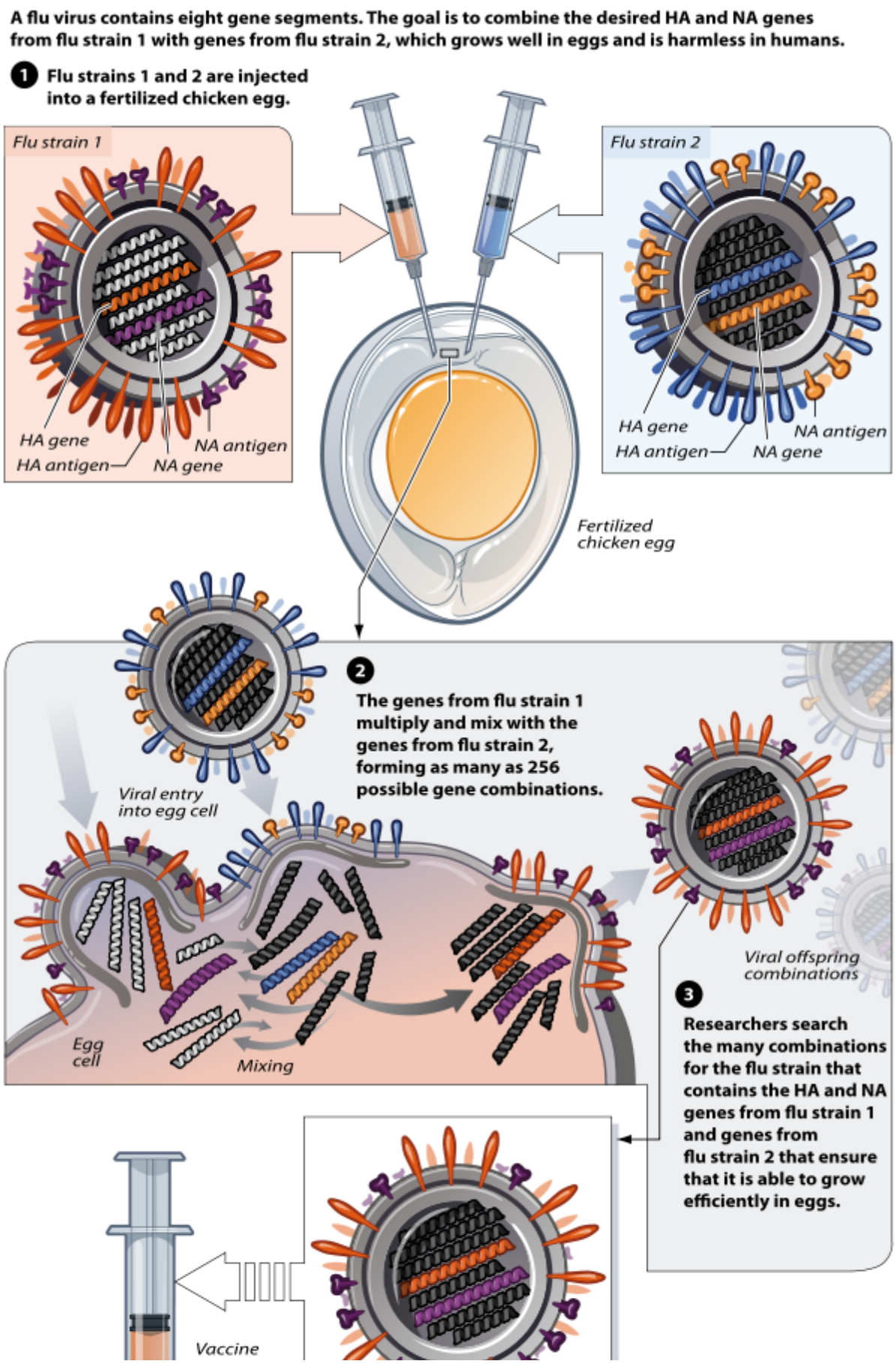
The Pandemics of 1957 (Asian Influenza) and 1968 (Hong Kong Influenza)

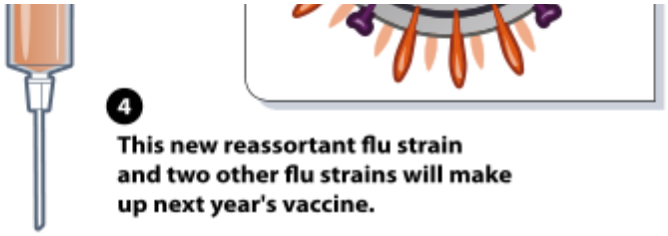
> "In 1957 a newly identified H2N2 influenza virus that is a reassortment between avian and human genes (see reassortment) was identified as the causative agent of influenza. Although the ensuing pandemic was not extraordinarily pathogenic, the increased mortality (70,000 deaths in the U.S. and over 1 million worldwide) is attributed to the lack of pre-existing immunity for HA and NA among humans. In 1968 the H2N2 Asian influenza virus was completely replaced by an H3N2 virus and this virus was also a reassortment between avian and human viruses. This virus was moderate in its pathogenicity (in U.S. 33,800 excess mortality) but the attack rate (40%) was highest in 10–14 year olds. Probably, preexisting antibodies to the N2 NA

> moderated the disease in older humans."
>
> R.A. Lamb, K.L. Roberts, in Reference Module in Biomedical Sciences, 2014

https://www.sciencedirect.com/topics/biochemistry-genetics-and-molecular-biology/influenza-a-virus-h2n2 < https://www.sciencedirect.com/topics/biochemistry-genetics-and-molecular-biology/influenza-a-virus-h2n2 >

In order to uncover the origin of the 1968 flu, it was clear that I needed to figure out the origin of the 1957 flu as well. This is due to the magical concept of reassortment. This theory involves the mixing of genetic material of one "virus" with that of another. The below illustration may help to visualize this process as done in a lab (the only place this actually occurs with "viruses").


A flu virus contains eight gene segments. The goal is to combine the desired HA and NA genes from flu strain 1 with genes from flu strain 2, which grows well in eggs and is harmless in humans.
1 Flu strains 1 and 2 are injected into a fertilized chicken egg.
Flu strain 1
HA gene
HA antigen
NA antigen
NA gene
Flu strain 2
HA gene
HA antigen
NA antigen
NA gene
Fertilized chicken egg
2 The genes from flu strain 1 multiply and mix with the genes from flu strain 2, forming as many as 256 possible gene combinations.
Viral entry into egg cell
Egg cell
Mixing
Viral offspring combinations
3 Researchers search the many combinations for the flu strain that contains the HA and NA genes from flu strain 1 and genes from flu strain 2 that ensure that it is able to grow efficiently in eggs.
Vaccine



The magic of nature at work.

In this case, we can see that the genetic material from birds and the genetic material from humans was said to have comingled and mixed together to ultimately create the H2N2/H3N2 strains. This can also be seen from a description of the 1968 H3N2 strain by the CDC:

> "The 1968 pandemic was caused by an influenza A (H3N2) virus comprised of two genes from an avian influenza A virus, including a new H3 hemagglutinin, but also contained the N2 neuraminidase from the 1957 H2N2 virus. It was first noted in the United States in September 1968. The estimated number of deaths was 1 million worldwide and about 100,000 in the United States. Most excess deaths were in people 65 years and older. The H3N2 virus continues to circulate worldwide as a seasonal influenza A virus. Seasonal H3N2 viruses, which are associated with severe illness in older people, undergo regular antigenic drift < https://www.cdc.gov/flu/about/viruses/change.htm >."

https://www.cdc.gov/flu/pandemic-resources/1968-pandemic.html < https://www.cdc.gov/flu/pandemic-resources/1968-pandemic.html >

The driving theory behind the 1968 H3N2 flu outbreak (referred to as the Hong Kong flu) was that it stemmed from the 1957 H2N2 flu outbreak (referred to as the Asian flu) which started in Singapore in February of that year. Sadly, I was unable to find out much about the exact methods used for the supposed isolation of the H2N2 "virus" as China was apparently not very forthcoming with that information.

However, I did find some interesting information about the man behind the supposed isolation of the "virus" in the US, Maurice Hilleman. Investigating this man helped to shed some light on the origins of these particular flu strains.

Confronting a Pandemic, 1957

> "Maurice Hilleman, a microbiologist at the Walter Reed Army Institute of Research in Maryland, read about the outbreaks in *The New York Times* <https://www.nytimes.com/1957/04/17/archives/hong-kong-battling-influenza-epidemic.html> on April 17 of that year. An article entitled "Hong Kong Battling Influenza Epidemic" stated that 250,000 people there were receiving treatment for the infection. Lines of people, including "many women" carrying "glassy-eyed children," were forming outside health clinics, the article noted. "I said, 'My God, this is the pandemic. It's here,' " Hilleman recalled in an interview <https://www.historyofvaccines.org/content/1957-asian-flu-pandemic> decades later.
>
> On April 18, Hilleman cabled a US military lab in Japan and managed to procure saliva from a patient infected in Hong Kong. His team quickly isolated the virus and tested it against hundreds of samples in Walter Reed's blood bank. The results confirmed Hilleman's fears: none of the samples neutralized the virus, a sign that none contained antibodies against it. This appeared to be a new influenza strain. Left unchecked, Hilleman predicted, it would reach the US within months, with disastrous consequences.
>
> Hilleman, who had previously worked on vaccines for other influenza strains, convinced pharmaceutical companies to start on a vaccine right

away, bypassing the US Division of Biologics Standards, the agency regulating vaccine development at the time. A Montanan who'd grown up on a farm, Hilleman also persuaded chicken farmers not to kill their roosters, as they usually did each spring—a move that ensured researchers had enough fertilized eggs to incubate the pathogen, then a standard step in vaccine development.

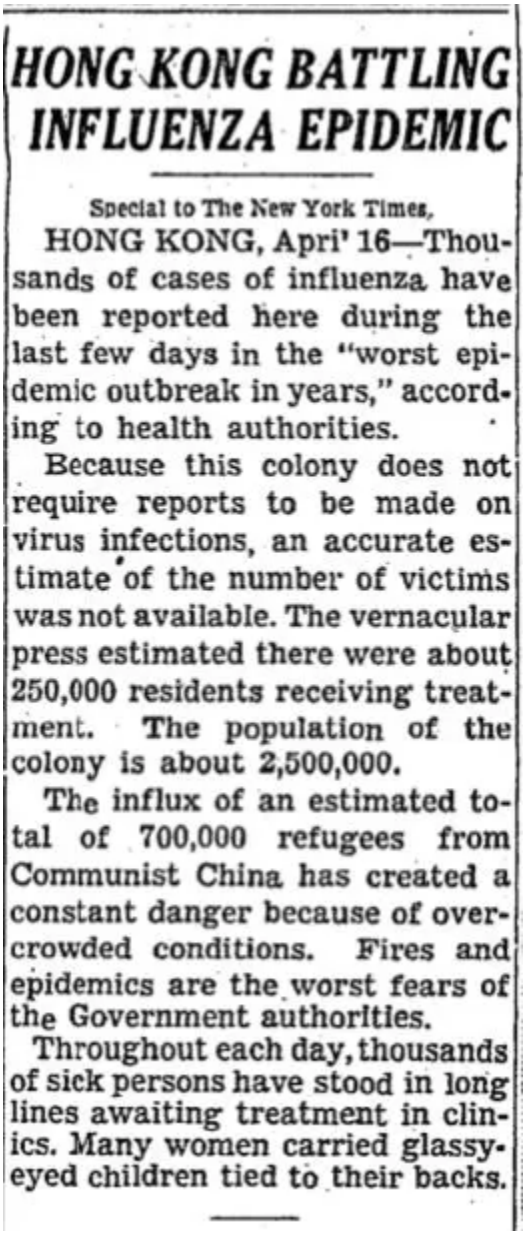
When the virus, later named H2N2, reached the US that summer, the country was ready. By the fall, several million people had received the vaccine and tens of millions more doses were distributed. Hilleman's vaccine likely saved hundreds of thousands of lives before the pandemic burned out in 1958, says Paul Offit < http://paul-offit.com/ >, a pediatrician and vaccine specialist at the University of Pennsylvania's Perelman School of Medicine who wrote about Hilleman in his 2007 book, *Vaccinated*. All told, around 100,000 people in the US and more than 1 million worldwide died from the disease, popularly known as the "Asian flu."

Hilleman went on to develop many more vaccines, including 9 of the 14 now routinely administered to US children. Writing in 2007, two years after Hilleman's death, National Institute of Allergy and Infectious Diseases Director Anthony Fauci described < https://www.jstor.org/stable/25478459?seq=1 > him as "perhaps the single most influential public health figure of the twentieth century."

https://www.google.com/amp/s/www.the-scientist.com/foundations/confronting-a-pandemic-1957-67564/amp < https://www.google.com/amp/s/www.the-scientist.com/foundations/confronting-a-pandemic-1957-67564/amp >

0:00 / 1:56

According to the above source and the accompanying video clip, Maurice Hilleman feared a flu pandemic on US shores based on nothing but a newspaper report. The very next day, he cabled Hong Kong and procured saliva from a sick patient, isolated the "virus," (methods unknown) and then determined it was ANTIGENICALLY different due to antibody testing on blood samples. Based on this information, Hilleman convinced farmers not to kill their roosters so that more CHICKEN (cough..Avian…cough) eggs would be available for the production of a rushed flu vaccine. Somehow, Hilleman had amazing foresight and predicted after reading one New York Times article (pictured below) that a pandemic was coming to the US. He then rushed the vaccine, bypassing safety standards, in order to combat it.



HONG KONG BATTLING INFLUENZA EPIDEMIC

Special to The New York Times.

HONG KONG, Apri' 16—Thousands of cases of influenza have been reported here during the last few days in the "worst epidemic outbreak in years," according to health authorities.

Because this colony does not require reports to be made on virus infections, an accurate estimate of the number of victims was not available. The vernacular press estimated there were about 250,000 residents receiving treatment. The population of the colony is about 2,500,000.

The influx of an estimated total of 700,000 refugees from Communist China has created a constant danger because of overcrowded conditions. Fires and epidemics are the worst fears of the Government authorities.

Throughout each day, thousands of sick persons have stood in long lines awaiting treatment in clinics. Many women carried glassy-eyed children tied to their backs.

This next source provides further insight into Hilleman's predictive and persuasive powers:

The Man Who Beat the 1957 Flu Pandemic

"Twenty biomedical companies. Seventy nations. An aggressive search for COVID-19 treatments and vaccines is underway worldwide. Yet even 21st-century technology can't match one man who curbed a major influenza pandemic spreading across the United States in 1957.

Pioneering virologist Maurice Hilleman, now oft-forgotten, detected that pandemic from across the globe, convinced reluctant U.S. health officials to take notice, and single-handedly fostered a vaccine that became publicly available. All in just four months."

"Hilleman worked under the public radar yet touched most people's lives. He was chief of respiratory diseases at Walter Reed Army Institute of Research when a new H2N2 type of influenza, termed the Asian flu < https://www.cdc.gov/flu/pandemic-resources/1957-1958-pandemic.html >, hit in 1957—eventually causing more than 1 million deaths worldwide and killing an estimated 70,000 to 116,000 in the U.S. The number of American deaths could have reached 1 million, public health experts estimated, without the quick arrival of 40 million doses of vaccine that fall. With a reputation for emphasizing safety and reducing vaccine side effects, Hilleman nonetheless led that vaccine's rollout by ignoring anyone who might slow him down, including federal regulators."

"At first, Hilleman had a tough time convincing experts in the military Influenza Commission and U.S. Public Health Service the flu was a threat to the United States, according to Offit. On May 22, 1957, Hilleman sent out a press release from Walter Reed. He then predicted the flu would arrive in the U.S. in

> September, just as schools opened. "What pandemic?" some experts asked.

> Hilleman sent virus samples to six American-based companies that produced influenza vaccines. Flu vaccines had been available since the mid-1940s, so researchers weren't starting from scratch in 1957. Yet if there was "any hope of saving American lives, he would have to convince companies to make and distribute vaccine in four months. Influenza vaccine had never been made that quickly," Offit wrote. To do so, Hilleman ignored federal drug regulators: "I knew how the system worked," Hilleman said. "So I bypassed the Division of Biologics Standards, called the manufacturers myself, and moved the process quickly."

> The first flu vaccine lots were produced in June, within weeks of Hilleman's request. Vaccinations started in July. The influenza pandemic hit the U.S. in early September (just as Hilleman predicted). Forty million doses were given over the next three months. Today, the U.S. Food and Drug Administration and public health regulations require safety and efficacy tests for new vaccines, which take more time, though the FDA can authorize drugs under an Emergency Investigative New Drug category."

https://blogs.scientificamerican.com/observations/the-man-who-beat-the-1957-flu-pandemic/ < https://blogs.scientificamerican.com/observations/the-man-who-beat-the-1957-flu-pandemic/ >

Predicting a pandemic from media reports. < https://viroliegy.com/2021/09/18/drosten-sars-cov-2-pcr-paper/ > Quickly "isolating" a "virus" and basing the existence on antibody results. Bypassing federal regulations and rushing a vaccine. Sounds like a rather familiar scenario… 🤔

All of this leads to the question: did a new "virus" genetically mix between bird and man in 1957 and then antigenically shift into another strain in 1968 or could there be some other explanation for the chick…er, avian connection? The only references I can find providing any evidence as to how H2N2 was isolated comes from two sources. The first is a 1957 paper by K.A. Lim which gives a tiny bit of detail about the Asian flu isolation:

INFLUENZA OUTBREAK IN SINGAPORE

> "*Virus.*–The strain used was isolated during the present outbreak elsewhere

> in Singapore. This strain, similar to that recovered from a case in the Naval Base, has been designated A/Singapore/1/1957 by the World Influenza Centre. In this paper we shall call it Singapore virus.
>
> FM-1, PR8, ws, and LEE strains were kindly provided by Dr. Alick Isaacs of the World Influenza Centre.
> Allantoic fluids of eggs infected with these viruses were stored at -20°C until used. Haemagglutinin titres have been maintained for over a month.
>
> *Cells.*-Fowl red cells were stored in Alsever's solution. Before use cells were washed three times in saline and then made up in 0-1% suspension. A densitometer was used for standardisation."

https://doi.org/10.1016/S0140-6736(57)90893-0 < https://doi.org/10.1016/S0140-6736(57)90893-0 >

As can be seen, eggs were used for infection purposes along with Fowl red cells washed in saline. Whether these methods were used in the original isolation or not is unfortunately never stated nor are the step-by-step methods detailed and outlined. However, it can be seen that chicken eggs and Fowl cells were used in some way with this supposed "virus." It really should then come as no surprise where the "avian" connection could have come from.

Digging a little further, in Hilleman's own 1957 paper we can discover similar avian connections:

New Antigenic Variant in Far East Influenza Epidemic, 1957.

> "*Materials and methods.* Far East virus strains designated A- Japan-305-57

and A-Japan-307-57 were recovered in embryonated eggs from American military personnel by Drs. S. E. Grossberg and I. Gresser at U.S. Army 406th Medical General Laboratory, Zama, Japan, and were forwarded to this laboratory. The A-Malaya-309-57, 3 10-57, and 311-57 strains were recovered in Malaya by Dr. J. H. Hale of the University of Singapore. A-Hong Kong-304-57 virus was isolated in this laboratory from throat washings collected by Dr. C. Rraul of the 406th Medical General Laboratory from a patient in the Hong Kong outbreak."

"*Biological characteristics of thc Far East viruses.* The Far East viruses were readily recovered from patients' throat washings by passage in embryonated eggs inoculated into the amniotic cavity. Following initial recovery, the viruses grew readily in the allantoic cavity giving hemagglutination titers of 1:80 to 1:320 when tested with human "0" or with chicken erythrocytes and incubated at room temperature or at 4°C. Early small volume commercial pools of allantoic fluid harvested from embryonated eggs infected with the A-Japan-305-57 virus gave low titers in the range of 16 to 92 chicken cell agglutination (CC.4) units ml as compared with 300 or more units expected when strains well adapted to growth in eggs are employed."

https://doi.org/10.3181%2F00379727-95-23306 < https://doi.org/10.3181%2F00379727-95-23306 >

While I was unable to uncover the exact methods used for its isolation, it is quite obvious that in no instance were any particles assumed to be the H2N2 "virus" ever properly purified and isolated directly from the throat washings of the sick patients. It appears that the cultivation of the "virus" included the use of chicken eggs and

Fowl red cells. It doesn't take a rocket scientist to see the avian influence on these "viruses" nor how they were created from a mixture of genetic material from avian and human sources.

So how did this lab-created mixture of animal/human sources supposedly mutate to become the H3N2 "virus" that was ultimately the blame for the 1968 Hong Kong flu? Let's get a little background on the 1968 H3N2 pandemic to see if we can answer this:

The 1968 Pandemic Strain (H3N2) Persists. Will COVID-19?

> "1968 was a bad year for flu but, as pandemics go, it was pretty mild. Scientists called the flu strain that hit the world H3N2. It's still around.
>
> Globally, about one million people died until the outbreak faded during the winter of 1969-70. In the U.S., the death toll was approximately 100,000 – three or four times the average annual death toll for flu since 2010, according to CDC figures < https://www.cdc.gov/flu/about/burden/index.html >. Most of those deaths were among people age 65 or older."
>
> "The H3N2 flu originated in Hong Kong in July 1968, appeared in the U.S. in September and is still circulating as a type of Influenza A. H3N2 was present among the 2019 flu strains and in the swine flu < https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5915287/ > outbreak earlier in the decade."

"H3N2 is considered one of the most troubling flu strains because, like COVID-19, it is highly contagious.

Scientists suspect that H3N2 emerged through an antigenic shift, in which the hemagglutinin (H) H2 antigen on the surface of the virus mutated to become the H3 antigen. According to the CDC, "The H3N2 virus is comprised of two genes from an avian influenza A virus, including a new H3 hemagglutinin, but also contains the N2 neuraminidase from the 1957 H2N2 virus."

"The small changes that characterize antigenic drift generally result in viruses that are only minimally different, so a vaccine designed for one usually is effective for a slightly mutated version.

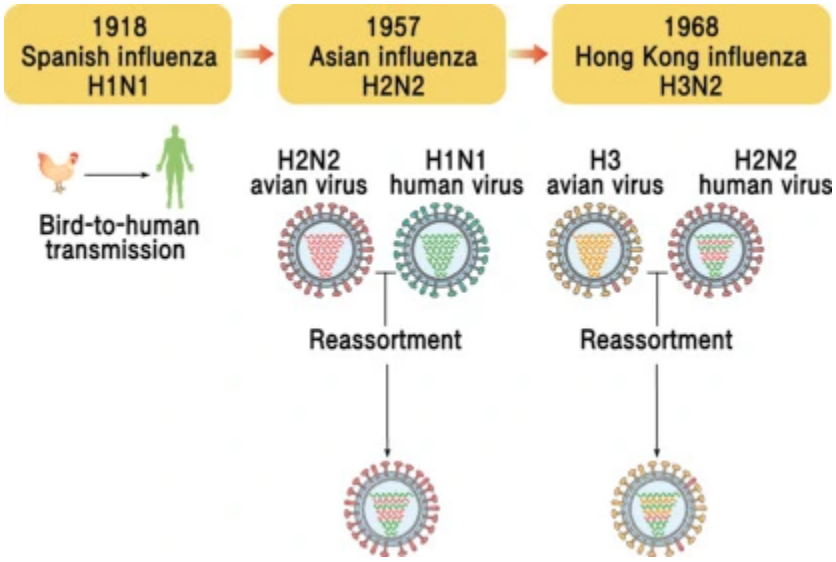
With the seasonal flus, like H3N2, antigenic drift is continual. The accumulated effects of antigenic drift, however, can result in viruses that are so different from the original virus that the immune system doesn't recognize them. Whether it will play a role in COVID-19 is still unknown.

Because H3N2 was closely related to the 1957 pandemic, many people were immune. This kept the 1968 H3N2 flu epidemic relatively mild, especially when compared to the 1918 Spanish flu. For some reason, however – possibly antigenic drift – the second wave of the H3N2 flu that struck in 1969 was more deadly."

> "Researchers speculated that (H3N2's) more sporadic and variable impact in different regions of the world were mediated by differences in prior N2 immunity. Therefore, the 1968 pandemic has been aptly characterized as 'smoldering,'" Kilbourne wrote."

> "In the 1960s, vaccines were evolving. The 1968 N3N2 pandemic triggered the development of trivalent vaccines and of subunit vaccines, which decreased adverse reactions. About the same time, the U.S. began recommending annual flu vaccination for high risk individuals."

https://www.biospace.com/article/the-1968-pandemic-strain-h3n2-persists-will-covid-19-/ < https://www.biospace.com/article/the-1968-pandemic-strain-h3n2-persists-will-covid-19-/ >



Magic!

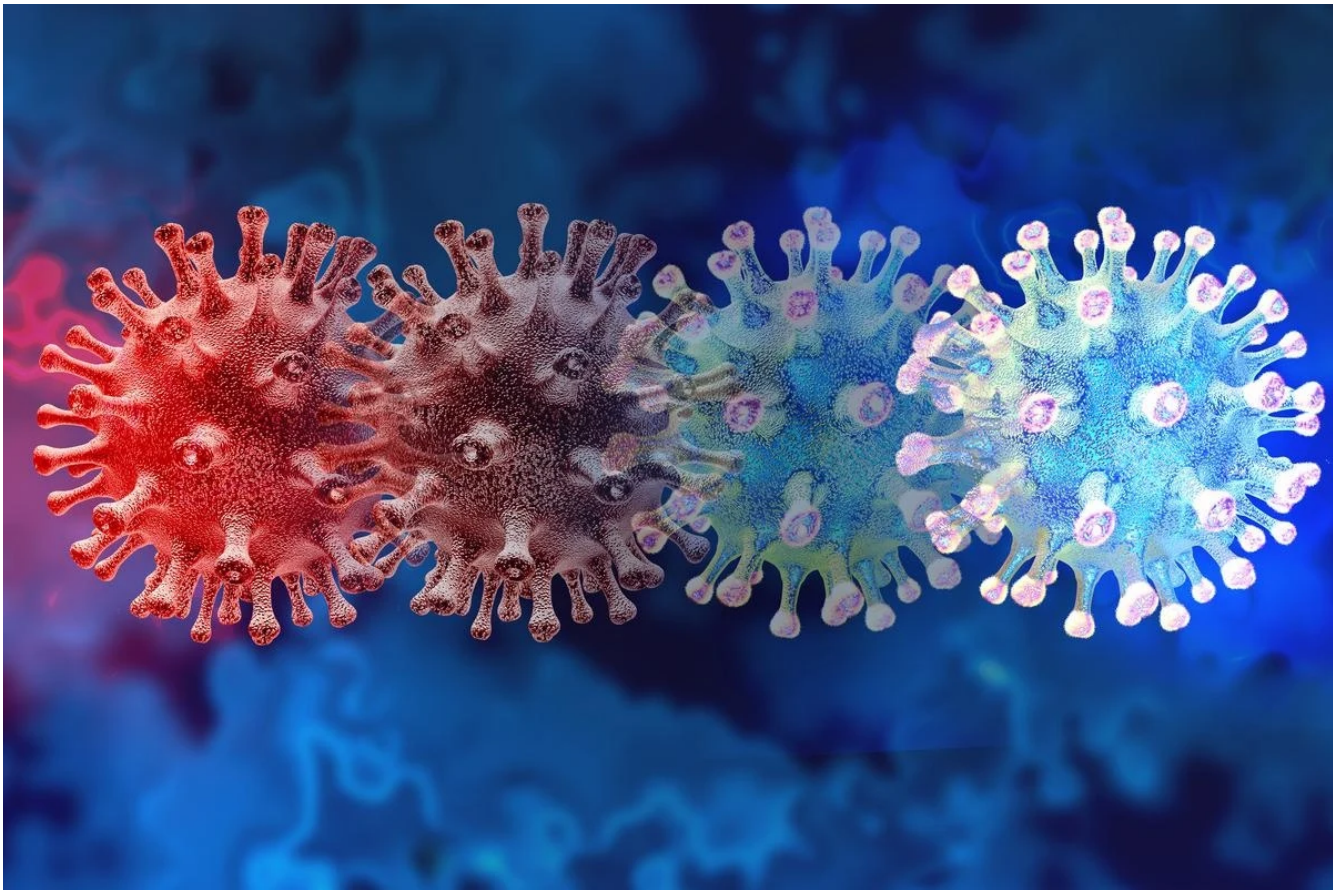
As can be seen from the above sources, the unproven concepts of ANTIGENIC SHIFT and DRIFT are how they sold the idea of differing versions of the same flu "virus" mutating over the proceeding decade to cover up for the vaccine failures

from 1957.

> ANTIGENIC SHIFT: the process by which two or more different strains of a "virus," or strains of two or more different "viruses," combine to form a new subtype having a mixture of the surface antigens of the two or more original strains

> ANTIGENIC DRIFT: random genetic mutation of an infectious agent resulting in minor changes in proteins called antigens, which stimulate the production of antibodies by the immune systems of humans and animals. These mutations typically produce antigens to which only part of a population may be immune.

With these two concepts introduced for the 1957 and 1968 Asian flu "virus," virologists can cover up the fact that no two genomes for any "virus" are ever 100% identical. It provides them the ability to state that multiple versions of the same strain exist yet can potentially be "more contagious, infectious, or deadly." Sound familiar?



B.1.1.7 (Alpha), B.1.351 (Beta), P.1 (Gamma), B.1.427 (Epsilon), B.1.429 (Epsilon), and B.1.617.2 (Delta). BA.1 (Omicron) and BA.2 (Stealth Omicron). XD and XF (both Deltacron). XE (Omicron + Stealth Omicron).

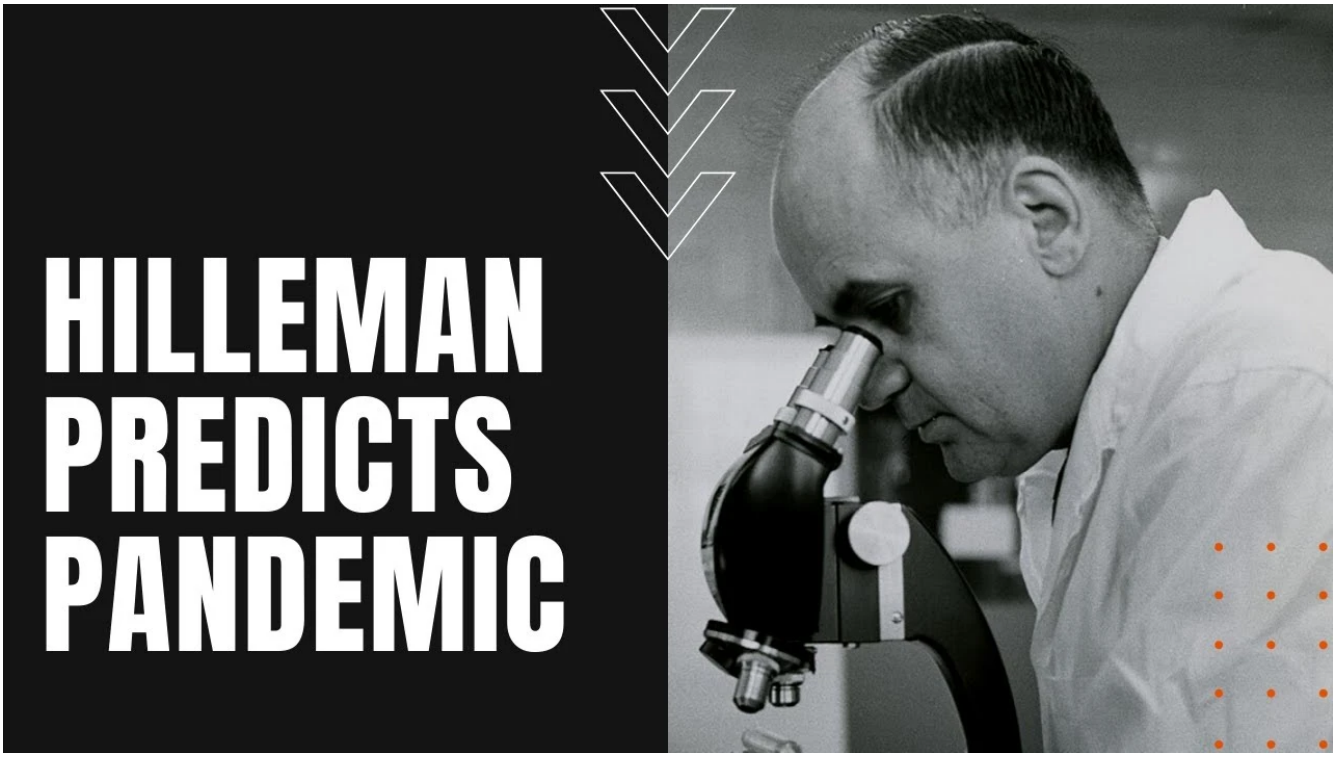
Antigenic shift and drift is how they get to claim new versions of the same "virus" exist every year and cover up for the inevitable vaccine failures. And who was responsible for the creation of these two concepts? If you guessed Maurice Hilleman, you've been paying attention!

> "In 1948, Hilleman left industry to concentrate on basic research at the Walter Reed Army Institute of Research in Washington. Among many other achievements, he discovered antigenic drift in influenza viruses, the phenomenon by which point mutations allow the virus to evade the full force of the continuously evolving herd immunity. He also discovered antigenic shift, that rare genetic reassortment that can occur during

> influenza double-infections and that leads to influenza strains against which herd immunity does not yet exist."

https://www.nature.com/articles/4341083a < https://www.nature.com/articles/4341083a >

From this 1957/1968 flu "pandemics," we see Maurice Hilleman's antigenic shift concept in action where a flu "virus" was created through a reassortment of avian and human influenza "viruses." We also get his antigenic drift concept of the same "virus" mutating and becoming more infectious/deadly. This of course led to trivalent vaccines (the combination of 3 strains in one shot) and subunit vaccines (containing only the antigenic parts of the pathogen). Hilleman was very instrumental in vaccine production for Merck and the US Army. In fact, he is credited with developing more than 40 vaccines, including measles, mumps, hepatitis A, hepatitis B, meningitis, pneumonia, *Haemophilus influenzae* bacteria, and rubella. It seems Hilleman was a very busy man.



Since I was unable to determine the exact isolation methods for H2N2, I hoped H3N2 might provide some insight into how a new flu "virus" jumped from bird to man. Was the H3N2 "virus" ever properly purified/isolated and proven pathogenic? Highlights from a few studies help to answer these questions.

From this first source from 1969, it is shown that the H3N2 "virus" is yet another tissue culture creation from monkey kidney tissues. No EM images of purified/isolated particles were presented to confirm the size/morphological charateristics of the assumed "virus" particles as no purification/isolation took place. Indirect non-specific antigenic tests were done with material supplied by the CDC to relate this monkey tissue culture goo to other A2 strains:

National Influenza Experience in Hong Kong, 1968

> "Although its origin is uncertain, the 1968 influenza epidemic in Hong Kong may have spread from the mainland of China. It began in Hong Kong on 13 July and reached its maximum intensity in 2 weeks, lasting some 6 weeks in all. About 15 % of the population was affected, but the mortality rate was low and the clinical symptoms were mild. The causative strain was isolated on 17 July and, because of its antigenic deviation from 1967 A2 strains, was sent to the World Influenza Centre in London and the International Influenza Center for the Americas in Atlanta, Ga., It was then proved to be a distinct antigenic variant of A2 virus, and the World Health Organization warned of its possible world-wide spread on 16 August."

> "Ever since our virus laboratory started functioning as a National Influenza Centre of the World Health Organization in 1963, we have been aware of the emergence of influenza virus mutants in this part of the world. Eleven years after the Asian influenza epidemic, a new virus variant was isolated in the summer of 1968 in Hong Kong. The origin of this variant is not known. There

was no official information on an influenza epidemic from the health authorities of mainland China, but prior to the outbreak in Hong Kong, travellers reported an increased incidence of influenza-like infections in the neighbouring Chinese province. For various reasons, virus isolations were not carried out on arriving travellers to confirm these reports."

"In contrast to the cold-season occurrence of influenza epidemics in Hong Kong, an epidemic broke out in mid-summer of 1968 (Fig. 1). It was first observed on 13 July, when there was a sudden increase of patients with influenza-like symptoms at the Government clinics. The epidemic soon reached its maximum intensity in the week of 27 July and gradually subsided in the following 3 weeks (Fig. 2). Altogether, the outbreak lasted for about 6 weeks. It was reported that the disease affected all age-groups and the clinical symptoms were considered mild, lasting for 3-5 days. There were no observable excess deaths during the epidemic.

The data for the 1968 epidemic in Hong Kong are far from complete, because the figures supplied by 9
of the Government clinics represented only a small proportion of the affected people. Many attended private clinics or sought relief from Chinese herbalists. No useful information could be derived from absenteeism data for factories and schools; the majority of the labour force are workers who are paid daily and who do not report sick unless they have severe symptoms, and the schools were closed for the summer vacation. However, it was suggested that about 15 % of the population was affected in this epidemic.

On 17 July, the laboratory isolated the virus strain in primary monkey kidney

> tissue culture and made a preliminary identification of subtype A2. Its haemagglutinating activity was inhibited to only a low titre of 1:80 by the polyvalent A2 antiserum. The antiserum, which was supplied by the National Communicable Disease Center in the USA, had a haemagglutination-inhibiting titre of 1:640 against the 1967 A2 strains. No dissimilarity of such magnitude had been observed in the previous A2 strains which were isolated between 1962 and 1967 in Hong Kong. Consequently, the virus strain was immediately dispatched to the World Influenza Centre in London in the form of infected tissue culture. In the following week, 5 more lyophilized strains were sent to the World Influenza Centre and also to the International Influenza Center for the Americas in Atlanta, Ga. The strain was later confirmed to be a distinct antigenic variant of A2 virus. Following a warning of its possible spread issued by the World Health Organization on 16 August, the virus was found to be causing epidemic outbreaks in other parts of the world in the later part of 1968."

https://www.google.com/url?sa=t&source=web&rct=j&url=https://apps.who.int/iris/bitstream/handle/10665/262470/PMC2427693.pdf&ved=2ahUKEwiImJjtk_P2AhV7LTQIHT46Bp0QFnoECC0QAQ&usg=AOvVaw2pqvWZnmSbnpXHMBMV-Osy < https://www.google.com/url?sa=t&source=web&rct=j&url=https://apps.who.int/iris/bitstream/handle/10665/262470/PMC2427693.pdf&ved=2ahUKEwiImJjtk_P2AhV7LTQIHT46Bp0QFnoECC0QAQ&usg=AOvVaw2pqvWZnmSbnpXHMBMV-Osy >

In this next source also from 1969, we see again that the "virus" is a tissue culture creation. The exact procedure for its creation is unknown as it is never revealed what exact steps were used in the isolation of this "virus." China was not forthcoming with information it seems and most of what the WHO knew came from a single newspaper article (why is it always a single newspaper article…). They prepared sera in ferrets for antigenic tests and determined the antigenic pattern was different from A2. They also mention unknown factors that might influence disease such as Farr's 1885 observation that the drop in temperature during the months of December – February determine to a great extent mortality even without influenza epidemics. No "virus" necessary:

Origin and Progress of the 1968-69 Hong Kong Influenza Epidemic

> "The Hong Kong strain of influenza virus A2 may have originated in the mainland of China but this is not certain. It caused a very large epidemic in Hong Kong and spread rapidly to countries as far as India and the Northern

Territory of Australia-as happened in the 1957 epidemic. Later its progress slowed down but epidemics occurred in many countries in the northern hemisphere in the winter of 1968-69. In all these countries except the United States of America the disease was mild and not associated with a large increase of deaths. In the United States of America, however, the number of "excess deaths" was similar to the number in 1957-58."

"In 1968, our first intimation of a possible new epidemic strain of influenza virus was a report in The Times of London for 12 July that a widespread outbreak of acute respiratory disease was occurring in south-eastern China. Five days later the health authorities in Hong Kong and Dr Chang, Director of the Influenza Centre there, reported a sudden increase in influenza-like illness and, most important, the isolation of viruses which by preliminary tests appeared to be similar to influenza virus A2. The strains were despatched as infected tissue-culture fluids on wet ice to the World Influenza Centre, where strain-specific sera were prepared in ferrets, and by this means it was determined that the antigenic pattern of the Hong Kong strain differed markedly from previous strains of virus A2. Similar findings were obtained in the International Influenza Center for the Americas to which Dr Chang had also sent specimens.

ORIGIN OF THE EPIDEMIC

We are dependent on a single newspaper report that the outbreak in Hong Kong was immediately preceded by an epidemic of acute respiratory disease in south-eastern China. There is no information on the etiology of this outbreak in China but its close temporal relationship to subsequent events makes it possible that it was due to the Hong Kong strain. It will have escaped none of the members of the Conference that the 1957 pandemic first

came to light in southern China, and the experience in 1968, though very tenuous, adds a little more information to the often-expressed hypothesis that strains of influenza virus which have the capacity to spread widely and rapidly often arise in that part of the world. Unfortunately contact between health authorities in China and other countries is even more difficult than in 1957 and it is impossible to obtain information on the possible origin or behaviour of the epidemic prior to its appearance in Hong Kong."

DISCUSSION

"Though, therefore, the virus has been spread widely through the world, many of the countries in which it was detected did not experience typical large epidemics, and in many of those in which epidemics did occur, their influence on absence from work and on death rates was slight or absent. The United States of America was the exception to the general rule, and the difference there is one of the most striking features of the epidemiological behaviour of the Hong Kong strain. Such differences have rarely been reported in the past."

"It would seem that some additional factor must have been operative in Merseyside. Semple (1951) pointed out that immediately before and during the epidemic period Merseyside experienced the coldest spell for many years, the weekly mean temperature from mid-December to mid-January being 4.4°F to 7.5°F (2.5°C to 3.6°C) below the mean temperatures for the corresponding weeks in the previous 20 years. Over two-thirds of the deaths were in persons, mainly women, 65 years of age or more and most of these deaths were in persons over 75 years of age.

> As long ago as 1885 Farr showed from statistics of deaths in England and Wales that "the degree down to which mean monthly temperatures fall in December, January, or February determines, to a great extent, the mortality of winter," even when epidemics of influenza are absent. He went on to suggest minimum night temperatures for the bedrooms of the very old and very young.
>
> In 1950-51, therefore, 2 explanations of the abnormally high death rate in Merseyside were possible: a more virulent strain, or an exceptional climatic condition occurring as the epidemic developed. Perhaps there were also other factors which were not identified.
>
> What was the important factor in the epidemic of Hong Kong influenza in the United States of America last winter? Its identification might go far to improving our knowledge of the behaviour of influenza and might lead to the development of more effective means of preventing influenza deaths if not of preventing the disease."

https://apps.who.int/iris/bitstream/handle/10665/262531/PMC2427756.pdf?sequence=1&isAllowed=y < https://apps.who.int/iris/bitstream/handle/10665/262531/PMC2427756.pdf?sequence=1&isAllowed=y >

In this final study, we actually get a bit more detail on the creation of this avian "virus." Unfortunately, there is no way that I could find out about how the "virus" was originally "isolated" in China but here we can see what the US did to "isolate" it themselves. This time it is stated that the throat washings were cultured in both monkey kidney tissues and chicken embryo amniotic-allantoic fluid. "Viruses" resembling the Hong Kong strain were said to be recovered. Monospecific antisera were prepared in chickens by a single intravenous injection of 5 ml. infected allantoic fluid. 24 of the first 39 clinical specimens submitted to the International Influenza Center for the Americas yielded influenza "viruses" in chicken allantoic fluid harvests from primary inoculation. Individual strains showed some variation in avidity by H.I. tests, but all were immunogenic and stimulated broadly reacting antibodies in chickens.

Again, no purification methods were described, no electron microscope pictures showing the particles assumed to be A2 were provided to confirm isolation, and no tests were done to prove pathogenicity. Everything relied on INDIRECT antigenic evidence attempting to relate the invisible "viruses" created from chicken embryos

and monkey kidney tissue cultures. It should go without saying that in order for any antibody result to have any meaning in order to be used as evidence, the particles assumed to be the "virus" must be purified and isolated first. It is well known that antibodies are not specific < https://viroliegy.com/2021/11/12/antibody-specificity/ > and that they can react to any similar proteins within the sample. Without purifying and isolating the "virus" particles from everything else first, the theoretical antibodies < https://viroliegy.com/category/antibodies/ > could be attaching to anything within the sample that is not the theoretical "virus." Thus, the antibody results are meaningless.

I have provided the full 1968 study along with the antibody results in order to show the incredible lengths virologists go to be able to claim an invisible "virus" exists within their culture soup:

THE HONG KONG/68 INFLUENZA A2 VARIANT

> A new influenza variant has been associated with an extensive outbreak of influenza in Hong Kong during July, 1968. Representative isolates have been classified in the A2 subtype because of antigenic overlapping with previous human A2 strains. However, their behaviour under laboratory conditions, reciprocal hæmagglutination-inhibition reactions with other influenza-A-virus strains, and measurements of immunological responses all support the conclusion that the hæmagglutinin of Hong Kong/68 variants has undergone considerable antigenic change from that of earlier A2 influenza strains. The neuraminidase of the new variants was similar to that of 1964 and 1967 strains of A2 virus.

INTRODUCTION

IN July, 1968, an outbreak of influenza occurred in Hong Kong and spread rapidly to all age-groups, resulting in the largest outbreak in that area since 1957. Viruses recovered by the National Influenza Center, University of Hong Kong, were identified with reference polyvalent antiserum as influenza A2. Additional studies revealed these strains to represent a major antigenic variation within the subtype.

Viruses antigenically closely related to the A2/Hong Kong/68 strains have been isolated subsequently from
influenza outbreaks in other areas, including Singapore, Taiwan, the Philippines, Northern Territory of Australia, San Diego, Hawaii, Thailand, Teheran, Saigon, India, the United Kingdom, and Sweden A number of isolates have also been recovered from individuals in the United
States who travelled in these areas.

We present here laboratory data on the biological and antigenic characteristics of the influenza A2/Hong Kong/68 variants.

METHODS

Virus Isolation
Throat-swab or throat-washing specimens were inoculated into primary rhesus-monkey kidney-tissue cultures and/or
the amniotic-allantoic cavities of embryonated eggs.

Haemagglutination-inhibition (H. I.) Tests

Infected allantoic fluids were used as haemagglutinating antigens. Monospecific antisera were prepared in chickens by a single intravenous injection of 5 ml. infected allantoic fluid. Serum was collected 9 days later. Both animal and human sera were treated with receptor-destroying enzyme (R.D.E.) of *Vibrio cholera* to remove non-specific serum inhibitors. H.I. tests were performed by the microtitre method using 4 haemagglutination units of antigen.

Neutralisation Tests

Neutralisation tests were done in embryonated eggs or rhesus kidney-tissue cultures using virus doses of approximately 30 I.D'50 (1 I.D'50 is the dose estimated to infect 50% of eggs or cultures). Sera were heat inactivated at 56°C for 30 minutes.

Neuraminidase-inhibition Tests

Virus concentrates, prepared as described by Laver and Webster were digested with 0.05% pronase (British Drug Houses Ltd.) in 0.01 M phosphate buffer at pH 7.2 for 1 hour at 37°C to destroy the viral haemagglutinin, thus avoiding non-specific inhibition of the enzyme by antibody to the haemagglutinin. Neuraminidase-inhibiting antisera were obtained from infected ferrets or from rabbits immunised as described by Laver and Webster with virus concentrates or with pure A2/57 neuraminidase derived from the recombinant virus X7(Fl). Enzyme activities were assayed by the method described by Warren which was modified by Laver and Webster using fetuin as substrate. Neuraminidase-inhibiting antibody was tested as described by Webster and Pereira, except that serum dilutions and virus preparations containing 1.2 enzyme units were incubated for 16-18 hours at 4°C before addition of substrate.

Chicken antiserum†	Swine	PR/8	FM/1	Denver/1	Japan/305	Japan/170	Taiwan/1	Tokyo/3	AA/7	Aichi/2	Hong Kong/1	Hong Kong/8	Equi 1/Prague	Equi 2/Miami	Equi 2/Mil	Equi 2/Ohio	Equi 2/Riche	Equi 2/France
Hæmagglutination inhibition:																		
A/Swine/1967/31	*160**	10														..	..	..
A/PR/8/34		*5120*		10												..	..	..
A1/FM/1/47		10	*320*													..	..	..
A1/Denver/1/57				*160*												..	..	..
A2/Japan/305/57					*320*	160	80	10	20	10	10	10				..	..	..
A2/Japan/170/62					640	*640*	320	80	80	20	20	40				..	..	..
A2/Taiwan/1/64					80	160	*640*	40	40							..	..	..
A2/Tokyo/3/67					40	160	160	*640*	160							..	..	..
A2/Ann Arbor/7/67					40	80	80	40	*160*							..	..	..
A2/Aichi/2/68..							10	20		*320*	320	320		10	10	10		10
A2/Hong Kong/1/68					40	80	80	10	20	1280	*1280*	1280		20	20	10	10	20
A2/Hong Kong/8/68					10	80	40	40	40	320	320	*640*		10	10	10		10
A/Equi 1/Prague/1/56													*160*					
A/Equi 2/Miami/1/63										10	10	10		*640*	320	320	320	160
A/Equi 2/Milford/2/63										20	20	20		160	*320*	160	160	160
A/Equi 2/Ohio/1/63	..	..	..	..			..	..	..					20	20	*20*	10	20
A/Equi 2/Richelieu/1/63	..	..	..	..			..	..	..	20	20	20		640	320	320	*320*	320
A/Equi 2/France/1/65	..	..	..	..			..	..	..					160	80	160	80	*80*
Neutralisation:																		
A2/Hong Kong/8/68	..	..	..	..	..	..	..	..	..	..	..	*640*	..	..	10	..	..	..
A/Equi 2/Milford/2/63	..	..	..	..	..	..	..	..	..	..	..	20	..	..	*1280*	..	..	..

* Values are reciprocals of serum dilutions. Blank spaces denote <10. .. denotes reaction not tested. † Treated with R.D.E. for H.I. tests; heat inactivated neutralisation. Figures in italic type indicate homologous antiserum titres.

TABLE II—SIMILARITY COEFFICIENTS OF TYPE-A INFLUENZA STRAINS WITH 1968 HONG KONG VARIANTS

Virus	A/Swine/1976/31	A/PR/8/34	A1/FM/1/47	A1/Denver/1/57	A2/Japan/305/57	A2/Japan/170/62	A2/Taiwan/1/64	A2/Tokyo/3/67	A2/Ann Arbor/7/67	A2/Hong Kong/1/68	A2/Hong Kong/8/68	A/Equi 1/Prague/1/56	A/Equi 2/Miami/1/63	A/Equi 2/Milford/2/63
A/Swine/1976/31	..													
A/PR/8/34	i	..												
A1/FM/1/47	i	i	..											
A1/Denver/1/57	i	i	i	..										
A2/Japan/305/57	i	i	i	i	..									
A2/Japan/170/62	i	i	i	i	1·4	..								
A2/Taiwan/1/64	i	i	i	i	4·0	2·8	..							
A2/Tokyo/3/67	i	i	i	i	16·0	5·7	8·0	..						
A2/Ann Arbor/7/67	i	i	i	i	5·7	2·8	5·7	4·0	..					
A2/Hong Kong/1/68	i	i	i	i	i	22·6	i	i	i	..				
A2/Hong Kong/8/68	i	i	i	i	i	11·3	22·6	i	11·3	1·0	..			
A/Equi 1/Prague/1/56	i	i	i	i	i	i	i	i	i	i	i	..		
A/Equi 2/Miami/1/63	i	i	i	i	i	i	i	i	i	45·6	45·6	i	..	
A/Equi 2/Milford/2/63	i	i	i	i	i	i	i	i	i	16·0	22·6	i	1·4	

By definition, a coefficient of 1·0 indicates strains indistinguishable under test conditions. i=indeterminate.

RESULTS

Viruses resembling the Hong Kong strains were readily isolated from throat swabs and throat washings by inoculating tissue-cultures or eggs. The recovery-rate was high; 24 of the first 39 clinical specimens submitted to the International Influenza Center for the Americas yielded influenza viruses in allantoic fluid harvests from primary inoculation. Haemagglutinin titres of 1/128 or greater were common after 1-2 passages. Individual strains showed some variation in avidity by H.I. tests, but all were immunogenic and stimulated broadly reacting antibodies in chickens.

Reciprocal H.I.-antibody titres with the Hong Kong isolates and selected human and equine influenza strains are shown in table 1. Although the Hong Kong isolates stimulated antibodies which reacted with most earlier A2 strains, the Hong Kong antigens were not consistently inhibited by antisera prepared with these same strains. Low, but reciprocal, cross-reactions also occurred between Hong Kong and equine-2 strains. Neutralisation tests in eggs confirmed the Hong Kong/equine-2 cross-reactions observed in H.I. tests (table 1).

Similarity coefficients based on H.I. titres (table 1) of Hong Kong variants with other influenza A strains are recorded in table 2. A value of 1 indicates strains which are indistinguishable by reciprocal H.I. reactions. Increasingly larger values indicate greater antigenic dissimilarity between strains. Coefficients for virus pairs cannot be determined (i=indeterminate) unless both strain-specific antisera inhibit the heterologous viruses. Similarity coefficients indicate that the Hong Kong strains are related to about the same degree to equine-2 viruses as they are to the earlier human A2 viruses.

This apparent relationship between human and equine strains was further investigated by testing paired sera from horses with laboratory-confirmed equine-2 influenza in 1963 and from recent human cases of influenza associated with the Hong Kong variant (table ill). Horses responded to equine-2 infection with antibody rises both to the equine subtypes and the Hong Kong variant. Increases in H.I. and neutralising antibodies to the Hong Kong antigen were consistently equal to or greater than the antibody increases measured by the infecting equine-2 antigen. All human serum pairs showed fourfold or greater antibody response to the infecting Hong Kong

variant, but antibody titres to equine antigens were not detected.

When a number of A2 strains were compared by the neuraminidase-inhibition test with avian and equine strains (table iv), a gradual antigenic shift in the neuraminidase was shown within the A2 subtype. As previously reported, some of the earlier A2 strains cross-reacted with an avian strain A/turkey/Massachusetts/65 but the A2/Hong Kong/68 strain no longer shows this cross-reaction. There was no cross-reaction between any of the equine and A2 strains, but the antibody content of both equine antisera may have been too low to reveal minor cross-reactions.

Sera*	Acute/convalescent H.I. titres with antigen:			Acute/convalescent tissue-culture neutralisation titres with antigen:	
	Equi 1	Equi 2	A2/HK/68	Equi 2	A2/HK/68
Horse A (3 yr.) ..	80/2560	0/20	10,320	0/80	10,320
Horse B (8 yr.) ..	40/80	0/20	10/160	40/80	10/160
Horse C (3 yr.) ..	10/320	0/20	0/40	0/20	0/20
Horse D (4 yr.) ..	40/40	0/20	0/40	0/40	0/40
Patients† (22–49 yr.)	0/0	0/0	0/40		

0 = < 10.

* All sera treated with R.D.E. for H.I. or heat inactivated for neutralisation.

† Geometric mean titres of 26 pairs of human sera. Convalescent titres of individual sera range from 10 to 320.

Equi 1 = A equine 1 Prague 1 56.
Equi 2 = A equine 2 Milford 2 63.
A2 HK 68 = A2 Hong Kong 8 68.

TABLE IV—THE NEURAMINIDASES OF HUMAN AND ANIMAL INFLUENZA-A VIRUSES

Source of neuraminidase †	Neuraminidase-inhibition titres with the following antisera *:						
	Anti-pure A2/57 neuraminidase	A2/Sing/1/57	A2/Eng/12/64	A2/HK/1/68	AO/Bel	A/Eq 1/Prague	A/Eq 2/Miami
Human:							
A2/Singapore/1/57	*2500*	*250*	30				
A2/England/12/64	500	10	*1500*	50			
A2/Tokyo/3/67 ..	300	10	1000	200			
A2/Hong Kong/1/68	50	10	1500	*200*			
A2/Hong Kong/8/68	80	10	1000	250			
AO/Bel					3000		
A1/FM/1/47 ..					500		
Animal:							
A/Turkey/Mass/65	5000	150	800				
A/Equi 1/Prague/56						120	
A/Equi 2/Miami/63							150

* Antisera used were from hyperimmunised rabbits except for those for A2/Hong Kong/1/68, equine-1 and equine-2 viruses, which were post-infection ferret sera.

† The virus preparations used in neuraminidase-inhibition tests were treated with pronase to destroy hæmagglutinin.

Serum dilution producing 50% inhibition of neuraminidase activity.

Blank spaces denote titres less than 1/10.

DISCUSSION

Several antigens are associated with the influenza virion: an internal soluble (S) antigen and the external viral (V) antigens including the viral haemagglutinin and neuraminidase which are distinct structural components of the lipoprotein envelope. All influenza-A viruses share a common ribonucleoprotein S antigen. They are divided into subtypes according to host range and similarity of viral antigens.

The extent of antigenic change is reflected in results of H.I. tests on human sera reported by several laboratories. Sera tested were collected from the

general populations in different areas, from persons recently immunised with several different influenza-vaccine formulations, and from confirmed cases of influenza during the 1967-68 outbreak in the United States. Antibody responses to A2/Hong Kong/68 were absent or minimal in all groups.

The Hong Kong variants show a definite relationship to A2 strains by H.I., neutralisation, and neuraminidase-inhibition tests and should be classified as members of this subtype. However, the magnitude of antigenic drift is greater than has been previously demonstrated within the A2 subtype.

The antigenic relationship between the Hong Kong-like isolates and the A/equine-2 strains also indicates the unique nature of these A2 variants. Although the heterotypic titres of monospecific antisera are low, reciprocal cross-reactions both by H.I. and neutralisation tests are unequivocal. The high levels of antibody titres to Hong Kong strains after A/equine-2 infections of horses offer further confirmation of this antigenic relationship. However, neuraminidase studies suggest that the similarity of the two viruses is limited to the viral haemagglutinin. The neuraminidase antigens of the Hong Kong isolates are related only to those of other human A2 strains.

The reciprocal crossing reported here for the Hong Kong isolates and the A/equine-2 strains confirms and extends the earlier reports of minor antigenic similarities between influenza viruses of both species. At this point, there is no evidence to suggest that these interspecies antigenic linkages have any aetiological significance in human epidemics or equine epizootics of influenza. However, these findings underscore previous

> comments made by Tumova and Pereira regarding the classification of influenza-A viruses. As new strains from different species are studied, a continuous spectrum of antigenic variation may become apparent within the whole influenza-A family."

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It is obvious to anyone looking at this both logically and critically why there is such a heavy connection between birds and influenza. Not only are many influenza "viruses" created by being grown and cultivated in chicken embryos, this same process is used to create the toxic injections. Flu vaccines are still regularly made in chicken eggs as they are cheap to make and produce in large quantities. There has been a mixing of bird and human genetic materials associated with the flu for the majority of the existence of the "virus," as can be seen by the highlights from this last source:

A brief history of bird flu

> "The HA and NA surface proteins are antigenic, very diverse, encoded on separate segments and split into 18 and 11 subtypes, respectively. Apart from the recently discovered bat-specific H17, H18, N10 and N11 proteins [5,6],

all of the subtypes have been found in avian species, whereas only a subset of the others have been detected in mammals. The other six segments are often considered as encoding the 'internal' genes. Although there is continuous global circulation of IAV in humans, due to the connectivity of the population [7], the majority of the diversity is in avian species and the reservoir population is avian [2]. Therefore, understanding the general global patterns of IAV epidemiology in birds will help elucidate the origins of past pandemics and could help inform predictions about future events."

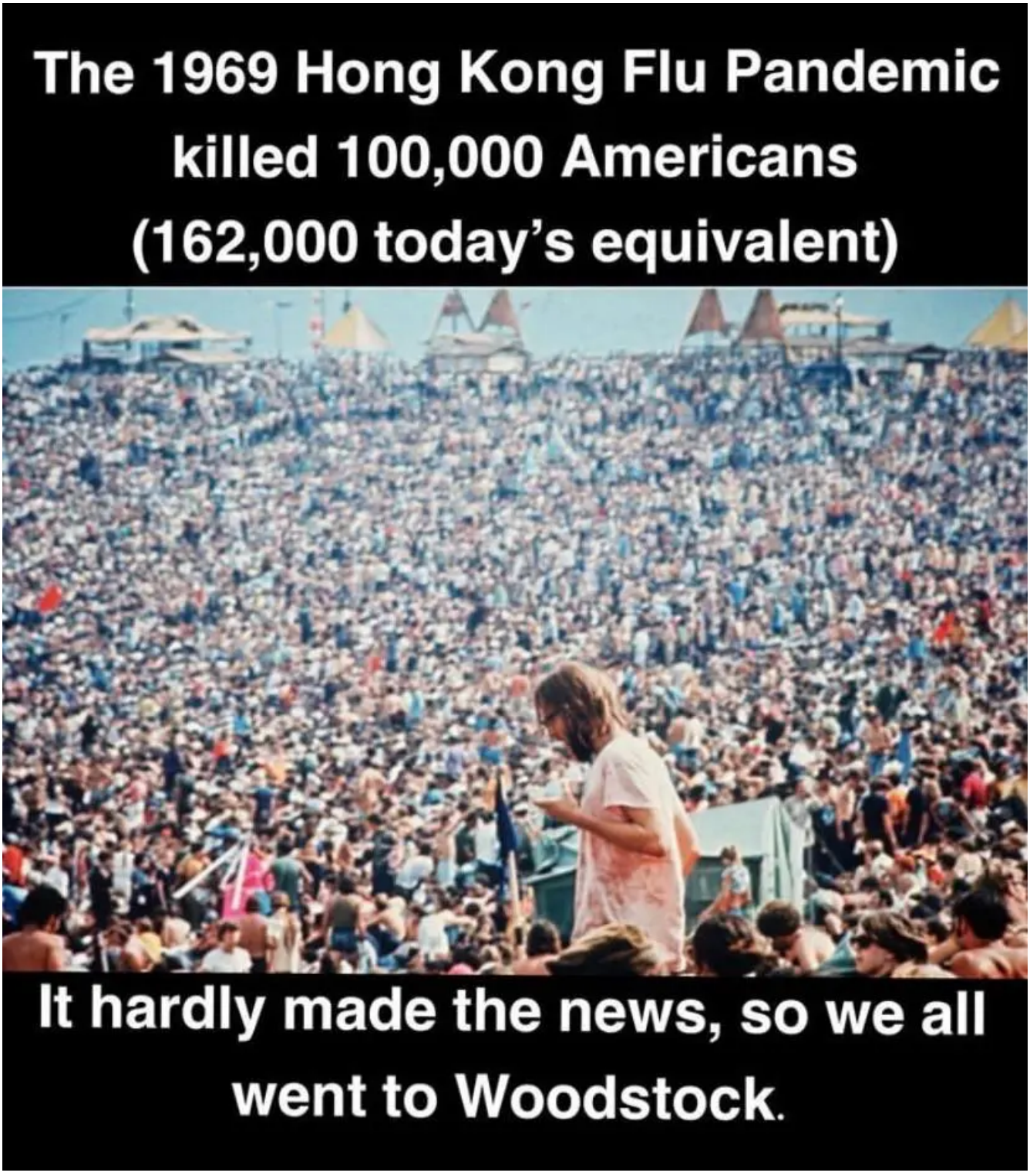
"In the nineteenth and early twentieth centuries, these outbreaks were termed 'fowl plague', and it was not until 1955 that Schafer determined that 'fowl plague virus' (FPV) was indeed a type of IAV, with similar internal antigens to human and swine influenza viruses [15]. Sequencing studies performed many years later resulted in the identification of the highly pathogenic avian influenza (HPAI) virus strains responsible for these outbreaks as H7 subtype IAVs, including A/chicken/Brescia/1902 (H7N7) [16], A/FPV/Weybridge/1927 or A/FPV/Dutch/1927 (H7N7) [13,17] and A/chicken/FPV/Rostock/1934 (H7N1) [18]. In 1959, an antigenically different HPAI H5 subtype was found in a chicken farm in Scotland (represented by A/chicken/Scotland/1959 (H5N1) [17]), while in 1961 an H5N3 strain was isolated from a wild common tern (Sterna hirundo) in South Africa (A/tern /South Africa/61 (H5N3) [19]). Because of the highly pathogenic phenotype of these first H5 and H7 isolates, it was parsimonious to consider all H5 and H7 viruses to be similarly virulent. However, this was reconsidered after the isolation of low pathogenic avian influenza (LPAI) H5 and H7 strains from ducks in the 1950/1960s and from turkeys in the 1960s/early 1970s (e.g. A/turkey/Ontario/77332/66 (H5N9) [20] and A/turkey/Oregon/71 (H7N3) [21]). Since then, an enormous variety of LPAI and HPAI H5 and H7 subtypes

have been isolated from domestic and wild birds, as well as the viruses bearing the majority of all other possible combinations of H1–H16 and N1–N9 surface glycoproteins [2,22–24]."

"Since then, three other human IAV pandemics have occurred: H2N2 in 1957 (Asian flu), H3N2 in 1968 (Hong Kong flu), and H1N1 again in 2009 (swine flu). In each case, IAV strains bearing segments coding for antigenically novel NA and/or HA surface protein(s) rapidly spread through a human population with no or little prior immunity. The relationship between fowl plague, avian influenza and human influenza was not apparent before the 1950s, but by 1967 Pereira, Tumova & Webster suggested that the human H2N2 and H3N2 pandemic viruses might have had an avian origin on the basis of antigenic cross-reactivity [31]."

"As soon as IAVs were sequenced (e.g. [18]), phylogenetic analyses started to show how avian and human viruses were related, and how this relationship could vary according to the segments involved. Such studies unambiguously confirmed the avian virus origin of the human 1957 and 1969 pandemic glycoprotein genes [32,33]."

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6553608/ <
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6553608/ >



In Summary:

- In 1957 a newly identified H2N2 influenza "virus" that is a reassortment between avian and human genes was identified as the causative agent of influenza

- In 1968 the H2N2 Asian influenza "virus" was completely replaced by an H3N2 "virus" and this "virus" was also a reassortment between avian and human "viruses"
- According to the CDC, the 1968 flu pandemic was caused by an influenza A (H3N2) "virus" comprised of two genes from an avian influenza A "virus," including a new H3 hemagglutinin, but also contained the N2 neuraminidase from the 1957 H2N2 "virus"
- Seasonal H3N2 viruses, which are associated with severe illness in older people, undergo regular antigenic drift < https://www.cdc.gov/flu/about/viruses/change.htm > (more on this later)
- Maurice Hilleman, a microbiologist at the Walter Reed Army Institute of Research in Maryland, read an article entitled "Hong Kong Battling Influenza Epidemic" in The New York Times on April 17 of that year
- The article stated that 250,000 people there were receiving treatment for the infection
- On April 18, Hilleman cabled a US military lab in Japan and managed to procure saliva from a patient infected in Hong Kong and his team quickly "isolated" the "virus" and tested it against hundreds of samples in Walter Reed's blood bank
- The results confirmed Hilleman's fears: none of the samples neutralized the "virus," a sign *(i.e.assumption)* that none contained antibodies against it and that it was a new strain
- *Reading media reports in order to determine a "virus" sounds rather familiar…*
- Hilleman, who had previously worked on vaccines for other influenza strains, convinced pharmaceutical companies to start on a vaccine right away, bypassing the US Division of Biologics Standards, the agency regulating vaccine development at the time
- He also persuaded chicken farmers not to kill their roosters, as they usually did each spring—a move that ensured researchers had enough fertilized eggs to incubate the pathogen, then a standard step in vaccine development
- By the fall, several million people had received the vaccine and tens of millions more doses were distributed
- According to Paul Offit, a member of the CDC and a childhood immunization advocate who co-developed the "rotavirus" vaccine, Hilleman's vaccine likely saved hundreds of thousands of lives before the pandemic burned out in 1958
- *Or it could be said that Hilleman's pre-emptive vaccine caused the disease which had not been in the US until after the vaccine distribution…chicken/egg*

scenario?

- The 1957 Asian flu was said to have caused more than 1 million deaths worldwide and killing an estimated 70,000 to 116,000 in the U.S
- The number of American deaths could have reached 1 million, public health experts estimated, without the quick arrival of 40 million doses of vaccine that fall
- Hilleman led that vaccine's rollout by ignoring anyone who might slow him down, including federal regulators
- "I knew how the system worked," Hilleman said. "So I bypassed the Division of Biologics Standards, called the manufacturers myself, and moved the process quickly."
- The first flu vaccine lots were produced in June, within weeks of Hilleman's request
- Vaccinations started in July and the influenza pandemic hit the U.S. in early September (just as Hilleman predicted)
- Forty million doses were given over the next three months.
- In just four months, Maurice Hilleman single-handily:
    1. Detected a pandemic from across the globe from a single newspaper article
    2. Convinced reluctant U.S. health officials to take notice
    3. Convinced chicken farmers not to kill their roosters in order to have more fertilized eggs
    4. Isolated the "virus" from a saliva sample (something said to be difficult for respiratory "viruses") from Japan
    5. Bypassed federal regulations and fostered a vaccine that became publicly available less than two months later in June before the "pandemic" hit in September
- Scientists called the flu strain that hit the world in 1968 H3N2
- Globally, about one million people died until the outbreak faded during the winter of 1969-70
- The H3N2 flu originated in Hong Kong in July 1968, appeared in the U.S. in September and is still circulating as a type of Influenza A
- Scientists suspect that H3N2 emerged through an antigenic shift
- The small changes that characterize antigenic drift generally result in "viruses" that are only minimally different
- With the seasonal flus, like H3N2, antigenic drift is continual
- The accumulated effects of antigenic drift can result in "viruses" that are so different from the original "virus" that the immune system doesn't recognize them

- For some reason, however – possibly antigenic drift – the second wave of the H3N2 flu that struck in 1969 was more deadly
- Researchers speculated that (H3N2's) more sporadic and variable impact in different regions of the world were mediated by differences in prior N2 immunity
- The 1968 N3N2 pandemic triggered the development of trivalent vaccines and of subunit vaccines
- About the same time, the U.S. began recommending annual flu vaccination for high risk individuals
- Hilleman was responsible for the concepts of antigenic shift and drift
- It was claimed that he demonstrated that influenza A "viruses" underwent "gradual and progressive minor antigenic characteristics called 'drift and shift,' which are the basis of modern influenza vaccine strategies"
- According to K.A. Lim's 1957 paper, the strain used was isolated during the present outbreak elsewhere in Singapore
- Allantoic fluids of eggs infected with these "viruses" were stored at -20°C until used
- Fowl red cells were stored in Alsever's solution and were washed three times in saline before use and then made up in 0-1% suspension
- According to Hilleman's 1957 paper, Far East "virus" strains designated A-Japan-305-57 and A-Japan-307-57 were recovered in embryonated eggs
- The Far East "viruses" were readily recovered from patients' throat washings by passage in embryonated eggs inoculated into the amniotic cavity
- Following initial recovery, the "viruses" grew readily in the allantoic cavity giving hemagglutination titers of 1:80 to 1:320 when tested with human "0" or with chicken erythrocytes and incubated at room temperature or at 4°C

Bull. Org. mond. Santé
Bull. Wld Hlth Org. } 1969, **41**, 349-351

National Influenza Experience in Hong Kong, 1968

W. K. CHANG[1]

Although its origin is uncertain, the 1968 influenza epidemic in Hong Kong may have spread from the mainland of China. It began in Hong Kong on 13 July and reached its maximum intensity in 2 weeks, lasting some 6 weeks in all. About 15 % of the population was affected, but the mortality rate was low and the clinical symptoms were mild. The causative strain was isolated on 17 July and, because of its antigenic deviation from 1967 A2 strains, was sent to the World Influenza Centre in London and the International Influenza Center for the Americas in Atlanta, Ga., It was then proved to be a distinct antigenic variant of A2 virus, and the World Health Organization warned of its possible world-wide spread on 16 August.

- Although its origin is uncertain, the 1968 influenza epidemic in Hong Kong may have spread from the mainland of China
- The causative strain was "isolated" on July 17th and, because of its antigenic deviation from 1967 A2 strains, was sent to the World Influenza Centre in London and the International Influenza Center for the Americas in Atlanta, Ga.
- It was then "proved" to be a distinct antigenic variant of A2 "virus," and the World Health Organization warned of its possible world-wide spread on August 16th
- Eleven years after the Asian influenza epidemic, a new "virus" variant was "isolated" in the summer of 1968 in Hong Kong
- The origin of this variant was not known
- There was no official information on an influenza epidemic from the health authorities of mainland China, but prior to the outbreak in Hong Kong, travellers reported an increased incidence of influenza-like infections in the neighbouring Chinese province
- For various reasons, "virus isolations" were not carried out on arriving travellers to confirm these reports
- It was first observed on 13 July, when there was a sudden increase of patients with influenza-like symptoms at the Government clinics
- The epidemic soon reached its maximum intensity in the week of 27 July and gradually subsided in the following 3 weeks
- Altogether, the outbreak lasted for about 6 weeks
- It was reported that the disease affected all age-groups and the clinical symptoms were considered mild, lasting for 3-5 days

- There were no observable excess deaths during the epidemic
- The data for the 1968 epidemic in Hong Kong were far from complete, because the figures supplied by 9 of the Government clinics represented only a small proportion of the affected people
- However, it was suggested that about 15 % of the population was affected in this epidemic
- On 17 July, the laboratory isolated the "virus" strain in primary monkey kidney tissue culture and made a preliminary identification of subtype A2
- The antiserum, which was supplied by the National Communicable Disease Center in the USA, had a haemagglutination-inhibiting titre of 1:640 against the 1967 A2 strains
- The "virus" strain was immediately dispatched to the World Influenza Centre in London in the form of infected tissue culture
- The strain was later confirmed to be a distinct antigenic variant of A2 "virus"

Bull. Org. mond. Santé
Bull. Wld Hlth Org. } 1969, 41, 345-348

Origin and Progress of the 1968–69 Hong Kong Influenza Epidemic [1]

W. CHARLES COCKBURN, P. J. DELON & W. FERREIRA

The Hong Kong strain of influenza virus A2 may have originated in the mainland of China but this is not certain. It caused a very large epidemic in Hong Kong and spread rapidly to countries as far as India and the Northern Territory of Australia—as happened in the 1957 epidemic. Later its progress slowed down but epidemics occurred in many countries in the northern hemisphere in the winter of 1968–69. In all these countries except the United States of America the disease was mild and not associated with

- The Hong Kong strain of influenza "virus" A2 may have originated in the mainland of China but this is not certain
- In all these countries except the United States of America the disease was mild and not associated with a large increase of deaths
- In the United States of America, however, the number of "excess deaths" was similar to the number in 1957-58
- According to the WHO n 1968, the first intimation of a possible new epidemic strain of influenza "virus" was a report in The Times of London for July 12th that a widespread outbreak of acute respiratory disease was occurring in south-eastern Chin

- The "isolated" strains were despatched as infected tissue-culture fluids on wet ice to the World Influenza Centre, where "strain-specific" sera were prepared in ferrets, and by this means it was determined that the antigenic pattern of the Hong Kong strain differed markedly from previous strains of "virus" A2
- Theu were dependent on a single newspaper report that the outbreak in Hong Kong was immediately preceded by an epidemic of acute respiratory disease in south-eastern China
- There was no information on the etiology of this outbreak in China but its close temporal relationship to subsequent events made it possible that it was due to the Hong Kong strain
- The experience in 1968, though very tenuous, added a little more information to the often-expressed hypothesis that strains of influenza "virus" which have the capacity to spread widely and rapidly often arise in that part of the world
- However, the WHO admitted that it was impossible to obtain information on the possible origin or behaviour of the epidemic prior to its appearance in Hong Kong
- In many of those in which epidemics did occur, the influence on absence from work and on death rates was slight or absent
- The United States of America was the exception to the general rule, and the difference there was one of the most striking features of the epidemiological behaviour of the Hong Kong strain
- In a related flu outbreak in Merseyside from 1951, it was admitted that some additional factor must have been operative to cause a worse toll
- Semple (1951) pointed out that immediately before and during the epidemic period Merseyside experienced the coldest spell for many years
- Over two-thirds of the deaths were in persons, mainly women, 65 years of age or more and most of these deaths were in persons over 75 years of age
- In 1885, Farr showed from statistics of deaths in England and Wales that "the degree down to which mean monthly temperatures fall in December, January, or February determines, to a great extent, the mortality of winter," even when epidemics of influenza are absent
- He went on to suggest minimum night temperatures for the bedrooms of the very old and very young
- In 1950-51, therefore, 2 explanations of the abnormally high death rate in Merseyside were possible:
    1. A more "virulent" strain

2. An exceptional climatic condition occurring as the epidemic developed

- They even questioned whether there were also other factors which were not identified
- What was the important factor in the epidemic of Hong Kong influenza in the United States of America last winter?
- The WHO admitted to not knowing and that its identification might go far to improving their knowledge of the behaviour of influenza

Public Health

THE HONG KONG/68 INFLUENZA A2 VARIANT

MARION T. COLEMAN Ph.D. WALTER R. DOWDLE Ph.D.
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W. K. CHANG M.D.
OF THE GOVERNMENT VIRUS UNIT,

revealed these strains to represent a maj variation within the subtype.

Viruses antigenically closely related to t Kong/68 strains have been isolated subsec influenza outbreaks in other areas, includin Taiwan, the Philippines, Northern Territory San Diego, Hawaii, Thailand, Teheran, S the United Kingdom, and Sweden A numb have also been recovered from individuals i States who travelled in these areas.

We present here laboratory data on the b antigenic characteristics of the influenza A2/ 68 variants.

METHODS

Virus Isolation

Throat-swab or throat-washing specimens w

- Representative isolates have been classified in the A2 subtype because of antigenic overlapping with previous human A2 strains
- However, their behaviour under laboratory conditions, reciprocal hæmagglutination-inhibition reactions with other "influenza-A-virus" strains, and measurements of immunological responses all support the conclusion that the hæmagglutinin of Hong Kong/68 variants has undergone considerable antigenic change from that of earlier A2 influenza strains
- "Viruses" recovered by the National Influenza Center, University of Hong Kong, were identified with reference polyvalent antiserum as influenza A2
- Additional studies revealed these strains to represent a major antigenic variation within the subtype
- "Viruses" antigenically closely related to the A2/Hong Kong/68 strains have been isolated subsequently from influenza outbreaks in other areas
- Throat-swab or throat-washing specimens were inoculated into primary rhesus-monkey kidney-tissue cultures and/or the amniotic-allantoic cavities of embryonated eggs

- Infected allantoic fluids were used as haemagglutinating antigens
- Monospecific antisera were prepared in chickens by a single intravenous injection of 5 ml. infected allantoic fluid
- Both animal and human sera were treated with receptor-destroying enzyme (R.D.E.) of *Vibrio cholera* to remove non-specific serum inhibitors
- Neutralisation tests were done in embryonated eggs or rhesus kidney-tissue cultures
- For Neuraminidase-inhibition tests, "virus" concentrates were digested with 0.05% pronase in 0.01 M phosphate buffer at pH 7.2 for 1 hour at 37°C to destroy the "viral" haemagglutinin, thus said to avoid non-specific inhibition of the enzyme by antibody to the haemagglutinin
- Neuraminidase-inhibiting antisera were obtained from infected ferrets or from rabbits immunised with "virus" concentrates or with pure A2/57 neuraminidase derived from the recombinant "virus" X7(Fl)
- The recovery-rate was high; 24 of the first 39 clinical specimens submitted to the International Influenza Center for the Americas yielded influenza "viruses" in allantoic fluid harvests from primary inoculation
- Haemagglutinin titres of 1/128 or greater were common after 1-2 passages
- Individual strains showed some variation in avidity by H.I. tests, but all were immunogenic and stimulated broadly reacting antibodies in chickens
- Although the Hong Kong isolates stimulated antibodies which reacted with most earlier A2 strains, the Hong Kong antigens were not consistently inhibited by antisera prepared with these same strains
- Low, but reciprocal, cross-reactions also occurred between Hong Kong and equine-2 strains
- Neutralisation tests in eggs confirmed the Hong Kong/equine-2 cross-reactions observed in H.I. tests
- *Cross-reactions = not specific*
- Similarity coefficients indicated that the Hong Kong strains were related to about the same degree to equine-2 "viruses" as they are to the earlier human A2 "viruses"
- This apparent relationship between human and equine strains was further investigated by testing paired sera from horses with laboratory-confirmed equine-2 influenza in 1963 and from recent human cases of influenza associated with the Hong Kong variant
- When a number of A2 strains were compared by the neuraminidase-inhibition test with avian and equine strains, a gradual antigenic shift in the neuraminidase was shown within the A2 subtype
- There was no cross-reaction between any of the equine and A2 strains, but

the antibody content of both equine antisera may have been too low to reveal minor cross-reactions

- Several antigens are associated with the influenza "virion:" an internal soluble (S) antigen and the external "viral" (V) antigens including the "viral" haemagglutinin and neuraminidase
- They are divided into subtypes according to host range and similarity of "viral" antigens
- *In order for virologists to know for a fact that these antigens react to any "virus," the "viral" particles would need to be properly purified and isolated directly from a human sample first which is never done which is why these antigens are only "associated"*
- Sera tested were collected from the general populations in different areas, from persons recently immunised with several different influenza-vaccine formulations, and from confirmed cases of influenza during the 1967-68 outbreak in the United States
- Antibody responses to A2/Hong Kong/68 were absent or minimal in all groups
- The magnitude of antigenic drift is greater than has been previously demonstrated within the A2 subtype
- The antigenic relationship between the Hong Kong-like isolates and the A/equine-2 strains indicated the unique nature of these A2 variants
- Although the heterotypic titres of monospecific antisera were low, reciprocal cross-reactions both by H.I. and neutralisation tests are unequivocal
- The reciprocal crossing reported for the Hong Kong isolates and the A/equine-2 strains confirmed and extended the earlier reports of minor antigenic similarities between influenza "viruses" of both species
- There was no evidence to suggest that these interspecies antigenic linkages have any aetiological significance in human epidemics or equine epizootics of influenza
- As new strains from different species are studied, a continuous spectrum of antigenic variation may become apparent within the whole influenza-A family

THE ROYAL SOCIETY PUBLISHING | PHILOSOPHICAL TRANSACTIONS B

A brief history of bird flu

Samantha J. Lycett, Florian Duchatel, and Paul Digard

- Apart from the recently discovered bat-specific H17, H18, N10 and N11 proteins, all of the subtypes have been found in avian species, whereas only a subset of the others have been detected in mammals
- Although there is continuous global circulation of IAV in humans, due to the connectivity of the population, the majority of the diversity is in avian species and the reservoir population is avian
- It was not until 1955 that Schafer determined that 'fowl plague virus' (FPV) was indeed a type of IAV, with similar internal antigens to human and swine influenza "viruses"
- Sequencing studies performed many years later resulted in the identification of the highly pathogenic avian influenza (HPAI) "virus" strains responsible for these outbreaks as H7 subtype IAVs, including:
    1. A/chicken/Brescia/1902 (H7N7)
    2. A/FPV/Weybridge/1927
    3. A/FPV/Dutch/1927 (H7N7)
    4. A/chicken/FPV/Rostock/1934 (H7N1)
- Other IAV's include:
    1. A/chicken/Scotland/1959 (H5N1)
    2. A/tern/South Africa/61 (H5N3)
    3. A/turkey/Ontario/77332/66 (H5N9)
    4. A/turkey/Oregon/71 (H7N3)
- An enormous variety of LPAI and HPAI H5 and H7 subtypes have been isolated from domestic and wild birds, as well as the "viruses" bearing the majority of all other possible combinations of H1–H16 and N1–N9 surface glycoproteins
- The relationship between fowl plague, avian influenza and human influenza

was not apparent before the 1950s, but by 1967 Pereira, Tumova & Webster suggested that the human H2N2 and H3N2 pandemic "viruses" might have had an avian origin on the basis of antigenic cross-reactivity
- As soon as IAVs were sequenced, phylogenetic analyses started to show how avian and human "viruses" were related, and how this relationship could vary according to the segments involved
- Such studies unambiguously confirmed the avian "virus" origin of the human 1957 and 1969 pandemic glycoprotein genes

In April of 1957, Maurice Hilleman read an article in The New York Times and decided a flu pandemic from Asia was imminent. He convinced chicken farmers not to kill their roosters in order to have a sufficient supply of eggs for the creation of vaccines and then bypassed federal regulations in order to quickly produce said vaccines in a matter of months. The vaccines were given out in the beginning of July and by September, the predicted pandemic hit. The H2N2 "virus" that was isolated by Hilleman was said to have been a reassortment of avian and human flu "viruses," thus fulfilling Hilleman's own antigenic shift theory.

A little over a decade later in 1968, a new flu pandemic hit the US from Hong Kong. The "virus" was said to be related to the H2N2 strain from 1957. It was said to be isolated from the throat washings which were cultured in both monkey kidney tissues and chicken embryo amniotic-allantoic fluid. It was given the name H3N2 as it was said to be a mutation of the 1957 strain. In other words, Hilleman's theory of

antigenic drift came in to play in order to explain why people came down with the same disease but were not immune to the "virus" even though they had been vaccinated against it since 1957. Thus, Maurice Hilleman is intimately connected to the Asian/avian flu pandemics.

How was this man able to predict and defeat this avian-related pandemic? Maybe it has to do with his upbringing on the family farm:

> "Before Hilleman, who died in April 2005, became the world's most prolific vaccinologist, with a portfolio including vaccines for measles, hepatitis, meningitis, and more, He was a farm boy. His family sold fruit, poultry, and eggs to make ends meet through the Great Depression."

https://mag.uchicago.edu/science-medicine/man-who-developed-40-vaccines < https://mag.uchicago.edu/science-medicine/man-who-developed-40-vaccines >

> "Hilleman, the youngest of eight siblings, grew up with his aunt and uncle on a farm outside the small eastern Montana community of Miles City. There he learned to raise chickens — a skill he'd later credit for knowing egg production well enough to help avert an influenza epidemic in the United States."

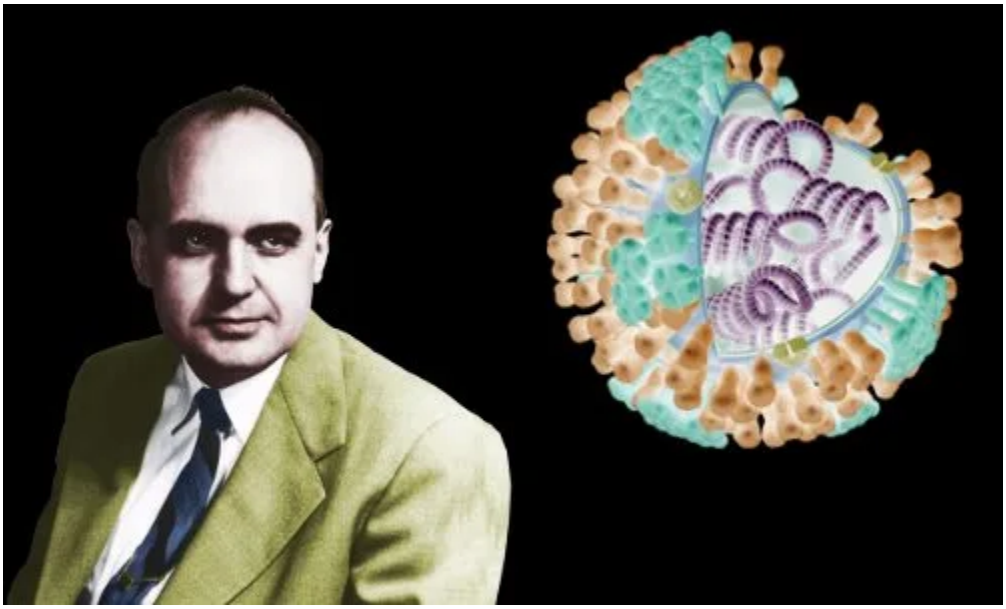
> "Hilleman's ability to marshal people and resources were key in 1957, when, acting on reports from Hong Kong signaling a looming flu pandemic, he convinced U.S. chicken farmers not to kill their roosters as part of a public health mobilization. The onetime farm boy knew the seasonal cycles of poultry production, and his foresight on the need for enough fertilized

> eggs ensured the country had adequate raw materials for mass vaccination — using a vaccine he developed."

https://www.google.com/amp/s/www.washingtonpost.com/history/2020/05/23/vaccines-maurice-hilleman-children/%3foutputType=amp < https://www.google.com/amp/s/www.washingtonpost.com/history/2020/05/23/vaccines-maurice-hilleman-children/%3foutputType=amp >

> "Maurice Hilleman, PhD, who was trying to use measles virus obtained from Enders to develop a vaccine for Merck, went in search of chickens known to be free of the leukemia virus for use in developing a measles vaccine. He eventually traveled to Kimber Farms in California, where the director of poultry research, W.F. Lamoreux, initially refused to sell the farm's flock of specially bred leukemia-free chickens.
>
> Hilleman prepared to leave empty-handed, only to find that Lamoreux was happy to sell him the chickens when he realized that Hilleman, like Lamoreux, was a native of Montana. Hilleman bought the farm's entire flock of leukemia-free chickens for $1 each. The descendents of that original flock are still being used to create vaccines at Merck."

https://www.historyofvaccines.org/content/flock-chickens-changes-everything < https://www.historyofvaccines.org/content/flock-chickens-changes-everything >



Hilleman was a farm boy who raised and sold poultry and eggs. He credited his chicken-raising skills for allowing him to know egg production well enough in order to create the vaccine for the 1957 Asian flu. He was instrumental in securing chickens for a cheap price at $1 each to be used in the creation of vaccines for Merck and the descendants of those chickens are still being used today. It makes one wonder if Hilleman had a bad encounter with a chicken on the farm at a young age and swore revenge on all future chicken offspring.

In any case, it is clear that neither H2N2 or H3N2 were ever properly purified and isolated directly from the fluids of sick patients and then proven pathogenic in a natural way by Hilleman or anyone else. From what I can gather, these "viruses" are the exact same tissue/cell culture and chicken embryo processes used to create a mish-mash of human and avian genetic material in the laboratory. The usual indirect non-specific antibody results were used to claim these "viruses" were related yet distinct entities. For good measure, Hilleman's unproven theories of antigenic shift and drift were used to explain away the vaccine failures and the inability to produce the same genome every time. However, the only antigenic shift, drift, and reassortment that occured was in the mixing of human genetic material in embryonated chicken eggs.

36 comments

Jeffrey Strahl

April 5, 2022 at 5:57 pm

Wow, so many things, so many "facts" i've taken for granted for over 60 years just went POOF. Thanks again, Mike!

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2015&_wpnonce=baaf45a9de >
Liked by 1 person

Mike Stone < https://viroliegyhome.wordpress.com >

April 5, 2022 at 6:55 pm

My pleasure! 🙂

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2018&_wpnonce=a9527b1dde >
Like

Jeffrey Strahl

April 6, 2022 at 5:06 am

FWIW, over the last few days i've noticed in my mainstream email and web groups ("mainstream" within the "resistance," that is) a growing number of criticisms of movement luminaries for hanging on to the virus/pandemic narrative.

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2036&_wpnonce=77827e2f6f >
Liked by 1 person

ClariFire

April 6, 2022 at 5:53 am

That's a great trend, and it's information like on this site that serves as the solid underpinning driving that trend, as well as the continuous refinement of the arguments and their presentation. We are the yin seed in the middle of the fullest part of the yang trend of modern medicine and scientism. ☯ The spark and kindling that ignites the countertrend.

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2037&_wpnonce=d65c5474b9 >
Liked by 2 people

Mike Stone < https://viroliegyhome.wordpress.com >

April 6, 2022 at 12:29 pm

Beautifully said. ❤

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2044&_wpnonce=5c261ff119 >
Like

Mike Stone < https://viroliegyhome.wordpress.com >

April 6, 2022 at 12:29 pm

That is excellent to hear! We need to weed out those who appear to be for truth but are in fact pied piping to lead people back to virology.

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2043&_wpnonce=a0ae64ff8d >
Like

NiciesMan

April 5, 2022 at 7:46 pm

I have heard some more evidence from a proponent of the virus theory:
Firstly, a relative of mine got sick and was diagnosed with "covid," and she was put on a ventilator. She then was said to have got an infection in her arm, and she was transfered to another hospital, I believe because the other hospital had some blood-cleaning substance or device. As the story goes, she survived, but (as the claim goes) if the doctors were completely wrong about what causes disease, the treatment would not have worked.
Second, I was told that many studies have been performed on animals where a solution supposedly containing a virus was injected, and then the animal became ill, whereas a control group injected with a placebo was not ill.
Here are some links related to this last claim: https://www.nature.com/articles/s41586-020-2787-6 < https://www.nature.com/articles/s41586-020-2787-6 > !
https://www.frontiersin.org/articles/10.3389/fmicb.2021.626553/full <
https://www.frontiersin.org/articles/10.3389/fmicb.2021.626553/full >

Thanks, and have a great day.

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2035&_wpnonce=8ad66ebaf9 >
Like

Mike Stone < https://viroliegyhome.wordpress.com >

April 6, 2022 at 12:27 pm

If a treatment is able to relieve symptoms, that does not mean the cause is known or identified. The effect does not prove cause. For instance, Ivermectin is a drug primarily used for parasites and worms. However, people are using it for "Covid-19." Does Ivermectin working to relieve symptoms mean a "virus" was present or was it

parasites causing their symptoms? Maybe neither a "virus" nor parasites were the cause and it was some other unknown factor? Maybe Ivermectin only suppressed the healing proces? There are many unknowns. The takeaway is that the effect of a drug/treatment working does not prove the cause. There will be many who will see no benefit to treatment whatsoever.

I will look at the animal studies but in every study I have read, they use toxic cell cultured goo and infect the animals in grotesque and unnatural ways. Purified/isolated particles assumed to be "viruses" are never used. They also are unable to recreate the human disease in most cases.

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2042&_wpnonce=f1c1182150 >
Liked by 1 person

Jeffrey Strahl

April 6, 2022 at 11:51 pm

Surprise, injecting toxic cell culture into an animal's brain results in the animal getting sick. 🙂

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2063&_wpnonce=4d15d0213b >
Liked by 1 person

Mike Stone < https://viroliegyhome.wordpress.com >

April 7, 2022 at 1:25 am

Shocking right? 😉

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2069&_wpnonce=aa52709cf9 >
Like

ClariFire

April 6, 2022 at 12:55 pm

First issue is that medicine only knows diseases as sets of symptoms, and they do know how to stop many symptoms pretty well. However, they miss that most of the symptoms are the cleaning, or the repair. There's no benefit to stopping a cleaning or repair process, other than instant convenience.

What causes disease is the body itself. The question is, what creates the need for the body to that? Simply stopping a cleaning or repair process doesn't imply you have an answer to that question. People need to undo that 180 degree flip before they can shake themselves out of the medical paradigm. Symptoms are good; but symptoms are also a sign that something has priorly gone wrong. Like fire trucks showing up at a house. Bad sign but given the fire people are glad they're there.

Wound infection is generally caused by the premature sealing of wounds. There are toxic byproducts that would naturally drain out but instead get stuck inside.

For the animal studies, try a search on this site. Usually it's something like they contained toxins or were put in brains or lungs in large amounts thus causing damage, or the symptoms produced were grossly dissimilar to those in humans.

Not to mention, animal-to-human transmission is supposed to be this big event, yet they expect to just take some animals and infect them with some human "virus"? Not making sense.

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2048&_wpnonce=e3d5bc6531 >

Liked by 2 people

Jeffrey Strahl

April 6, 2022 at 11:56 pm

Excellent points. Much of treatment is because of a desire for instant convenience, people not wanting to take their time detoxifying, totally intent on resuming "normal" routines with no breaks, of course a lot of that is because people cannot afford time off due to stressful schedules. An artifact of a unhealthy lifestyle.

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2064&_wpnonce=cd62afc94a >
Liked by 1 person

ClariFire

April 7, 2022 at 12:21 am

Oh wow that actually connects an important dot: the system of modern medicine was brought in alongside the rat-race work culture around the industrial revolution, which precludes taking 6-8 weeks off to do a proper fast or other detox until the circumstances have gotten truly dire and you're laid up in a hospital bed.

Medicine for the cattle class suppresses symptoms until all the productive juice has been squeezed out of the subjects for their role as a cog in the machine, then they're discarded. Though it seems even the elite have started to drink their own Kool-Aid on this broken paradigm.

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2065&_wpnonce=c28ee0e3a4 >
Liked by 1 person

ClariFire

April 5, 2022 at 11:01 pm

A fascinating window into how the medical myth factory works.

Lionizing some lonestar as a way to sweep the miracles under the rug is becoming

all too familiar.

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2034&_wpnonce=ee9d273750 >
Liked by 2 people

Mike Stone < https://viroliegyhome.wordpress.com >

April 6, 2022 at 12:15 pm

Yes, and there are many parallels to today. It's not surprising Hilleman and Fauci were friends. I imagine Fauci learned a great deal from Hilleman on how to successfully sell a "pandemic."

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2040&_wpnonce=5c46c7a557 >
Liked by 1 person

Alex

April 6, 2022 at 7:25 am

Excellent work again Mike, keep going.

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2041&_wpnonce=675a235186 >
Liked by 1 person

Mike Stone < https://viroliegyhome.wordpress.com >

April 6, 2022 at 12:45 pm

Thanks, I appreciate it! 🙂

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2047&_wpnonce=4313f7e593 >

Like

Federico

April 6, 2022 at 1:33 pm

Hi. What do you think about this Hilleman's paper about adenovirus?
Biophysical Characterization of the RI (RI-67) Viruses. *
https://www.semanticscholar.org/paper/Biophysical-Characterization-of-the-RI-(RI-67)-Hilleman-Tousimis/c1bfa5852ea3a2222815b663c0cc893549766465 <
https://www.semanticscholar.org/paper/Biophysical-Characterization-of-the-RI-(RI-67)-Hilleman-Tousimis/c1bfa5852ea3a2222815b663c0cc893549766465 >
Clearly they started from an Hela cell colture in order to "purify" the virus, not from a pure sample. They use a Gradacol membrane in order to filtrate the "virus", and they found out "numerous spherical bodies of uniform size and shape and also a few "doughnut" forms" on electron microscope. In your opinion are they artifacts, cellular debris or what else?
I found out that Hilleman used this Gradacol membrane in other sudies definition: "collodion membrane prepared from a solution of collodion in alcohol and ether in such a way as to have a predetermined average pore diameter and used especially in ultrafiltration (as of a virus suspension)"
https://www.merriam-webster.com/dictionary/gradocol%20membrane <
https://www.merriam-webster.com/dictionary/gradocol%20membrane >

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2049&_wpnonce=4d0f435957 >
Liked by 1 person

Mike Stone < https://viroliegyhome.wordpress.com >

April 6, 2022 at 3:12 pm

I'll have to give it a read. The original "Adenovirus" paper never properly purified and isolated a "virus" nor proved pathogenicity:

https://viroliegy.com/2022/03/26/the-adventitious-adenovirus/ <
https://viroliegy.com/2022/03/26/the-adventitious-adenovirus/ >

I doubt Hilleman did either, especially based on the fact that, as you stated, he started from a cancerous cell line.

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2054&_wpnonce=6d326ce4db >
Like

nestorseven

April 6, 2022 at 2:46 pm

Over the last 2 years, 95% of seasonal (every year) flu cases have virtually disappeared so I guess we can conclude that 60 years of vaccines have done the trick. But wait, we still have millions of flu cases…they have just been rebranded as covid…same symptoms, same outcomes. Why would these super smart medical experts change the game plan? To sell more injections like mRNA gene therapy that they never question.

I am tired of all the Asian and bird flu types. Why no new flu beginning in the US? They always start in the countries where there is the greatest possibility that environmental conditions are the main cause of sickness. When I start hearing about the Kokomo flu or the Knob Noster flu, then I might get concerned.

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2051&_wpnonce=a42b3f3f66 >
Liked by 2 people

Mike Stone < https://viroliegyhome.wordpress.com >

April 6, 2022 at 3:14 pm

I don't even think we can conclude that 60 years of vaccines worked. All we can conclude is that if you ignore the flu, it goes away…

…but then comes back disguised as a different "virus." 😉

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2055&_wpnonce=57d06370bb >
Like

Lynn Wright

April 6, 2022 at 10:32 pm

nestorseven, I hear you! I want a hamster flu from Bakersfield! Ugh, this is all so comical.

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2059&_wpnonce=ad2ad66fef >
Liked by 1 person

NiciesMan

April 6, 2022 at 2:59 pm

So, you mean the solutions used caused "covid" symptoms by being harmful (toxic) to the animals, but it not being "covid" that caused it?

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2052&_wpnonce=0b14d1681f >
Liked by 1 person

Mike Stone < https://viroliegyhome.wordpress.com >

April 6, 2022 at 3:09 pm

Yes, the cell culture supernatant is full of toxic materials and it is injected into animals in an unnatural way. Having cultured goo shoved into the nasal cavities is not natural. Injecting anything into the nose can cause symptoms in the upper respiratory system, even water.

On top of that, they are never able to recreate the human disease. It usually amounts to lethargy, weight loss, and ruffled fur, none of which are symptoms of "Covid."

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2053&_wpnonce=665f2a5945 >
Like

NiciesMan

April 8, 2022 at 11:38 pm

I was told that the two substances (the "covid" one and the placebo) used in the study were the same, but a small bit of a "covid" solution was dropped into one. How would adding a small amount of the "covid" concoction cause disease, while the base liquid doesn't?
Thanks again

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2116&_wpnonce=2aaf3f693a >
Like

Mike Stone < https://viroliegyhome.wordpress.com >

April 8, 2022 at 11:55 pm

Is there a particular study you are referring to and are you asking about the controls?

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2120&_wpnonce=d58d67776c >
Like

Damion

April 6, 2022 at 8:51 pm

Great work Mike I enjoy reading ✌🏾👍🏾👏🏾👏🏾

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2056&_wpnonce=ef49f34d0f >
Liked by 1 person

Mike Stone < https://viroliegyhome.wordpress.com >

April 7, 2022 at 1:17 am

Thank you! I appreciate it. 🙂

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2066&_wpnonce=40037befb0 >
Like

Lynn Wright

April 6, 2022 at 10:49 pm

Fantastic work as always Mike! I really loved this post, the similarities between then and now are amazing.Only now we have de novo assemblers that are able to generate in silico "variants" til the cows (or chickens?) come home. Over 10 million

scariants now in GISAID for SARS CoV-2. Just this morning I watched a short, recent clip of The Donald once again bragging about how he pressed the FDA and the vaccine manufactureres to warp speed-up the process of vaccine creation. My bet is in his mind he truly believes he has helped (or perhaps single handedly, because, after all, he IS The Donald) to save humanity from this latest scourge. Braggadocious, as was Hilleman. And it does appear as if Fauci is just carrying the viroLIEgy torch onward, repeating the same old 20th century nonsense into the 21st. It's just all so amazing how they make sh!t up over and over again. Rehashing. Oh, and the cartoons showing us how it all works in biology, in the human body. The merging of strains, different viruses from different species, into one scary mess. i tell you if we weren't all indoctrinated with germ theory from the get go, and this story was a sci-fi novel or movie, people would have a hard time believing it. I'd go for aliens from outer space first.

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2060&_wpnonce=9d51ff3d92 >
Liked by 1 person

Mike Stone < https://viroliegyhome.wordpress.com >

April 7, 2022 at 1:21 am

Aliens definitely feel more realistic at this point than the lies of "virology." I just can not fathom how people ever take this seriously. The only thing I can think of is the indoctrination from birth. It is hard to break. As you said, without it, people would see this for the fiction it is.

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2067&_wpnonce=faf27aa77c >
Like

Pingback: Gain of Fiction – ViroLIEgy < https://viroliegy.com/2022/04/07/gain-of-fiction/>

NiciesMan

April 9, 2022 at 12:42 am

I linked a study (I think it was a particular one), but it was claimed that this was done many, many times, and in various countries.
Yes, I am talking about the control group.
Thanks; God bless.

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2123&_wpnonce=8e0c937667 >
Like

Mike Stone < https://viroliegyhome.wordpress.com >

April 9, 2022 at 1:09 pm

I'm not sure which study as I don't see any linked on this comment page. The controls are rarely detailed. Are you referring to challenge studies or seeing CPE in cell cultures?

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2149&_wpnonce=20bc4f334c >
Like

PC

April 9, 2022 at 7:58 am

Thanks .Another great article.

I had a quick look recently into the avian flu in birds as they are killing chickens.

Avian Influenza

Again a perfect example of virology at its best , another fabricated virus and the testing.

The first description of avian influenza (AI) dates back to 1878 in northern Italy, when Perroncito [Perroncito E. Epizoozia tifoide nei gallinacei. Annali Accad Agri Torino 1878;21:87-126] described a contagious disease of poultry associated with high mortality. The disease, termed "fowl plague", was initially confused with the acute septicemic form of fowl cholera. However, in 1880, soon after its first description, Rivolta and Delprato [as reported by Stubs EL. Fowl pest, In: Biester HE, Devries L, editors. Diseases of poultry. 1st ed. Ames, IO: Iowa State College Press; 1943. p. 493-502] showed it to be different from fowl cholera, based on clinical and pathological properties, and called it Typhus exudatious gallinarum. In 1901, Centanni and Savunzzi [Centanni E, Savonuzzi E, La peste aviaria I & II, Communicazione fatta all'accademia delle scienze mediche e naturali de Ferrara, 1901] determined that fowl plague was caused by a filterable virus; however, it was not until 1955 that the classical fowl plague virus was shown to be a type A influenza virus based on the presence of type A influenza virus type-specific ribonucleoprotein [Schäfer W. Vergleichender sero-immunologische Untersuchungen über die Viren der Influenza und klassischen Geflügelpest. Z Naturf 1955;10b:81-91]. The term fowl plague was substituted by the more appropriate term highly pathogenic avian influenza (HPAI) at the First International Symposium on Avian Influenza [Proceedings of the First International Symposium on Avian Influenza. Beltsville, MD. 1981, Avian Dis 47 (Special Issue) 2003.] and will be used throughout this review when referring to any previously described fowl plague virus.

https://pubmed.ncbi.nlm.nih.gov/18533261/ <
https://pubmed.ncbi.nlm.nih.gov/18533261/ >

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All bird keepers (whether you have pet birds, commercial flocks or just a few birds

in a backyard flock) must keep a close watch on them for signs of disease and maintain good biosecurity at all times. If you have any concerns about the health of your birds, seek prompt advice from your vet.

You should register your poultry, even if only kept as pets, so we can contact you during an outbreak. This is a legal requirement if you have 50 or more birds. Poultry includes chickens, ducks, turkeys, geese, pigeon (bred for meat), partridge, quail, guinea fowl and pheasants.

You can sign up to our alerts service to keep up to date with the latest news. You can also get Defra email alerts or subscribe to Defra's RSS feed by copying and pasting this RSS feed URL into your feed reader.

https://www.gov.uk/guidance/avian-influenza-bird-flu < https://www.gov.uk/guidance/avian-influenza-bird-flu > (list the area 'affected')

Biosecurity measures

Click to access biosecurity-poultry-guide.pdf < https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/1064069/biosecurity-poultry-guide.pdf >

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And how to watch for 'symptoms', it has to be a virus, rarely the poor condition.

https://cs-tf.com/avian-influenza-in-chickens/ < https://cs-tf.com/avian-influenza-in-chickens/ >

Click to access usda-avian-influenza-diagnostics-testing-factsheet.pdf < https://www.usda.gov/sites/default/files/documents/usda-avian-influenza-diagnostics-testing-factsheet.pdf >

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And what they call isolation and testing.

“The avian influenza (AI) virus is usually isolated and propagated by inoculating either swab or tissue samples from infected birds into the chorioallantoic sac of embryonating chicken eggs. This is the accepted method, but occasionally an isolation may only be successful when inoculated either into the yolk sac or onto the chorioallantoic membrane of embryonating chicken eggs. Chorioallantoic fluid is harvested from eggs with dead or dying embryos and is tested for the presence of hemagglutinating antigen. If hemagglutination-positive, this indicates that the isolate may be the AI virus. The presence of the AI virus may be confirmed by either an agar gel immunodiffusion (AGID) assay, RT-PCR specific for AI virus, or a commercially available immunoassay kit specific for type A influenza. Instructions for AI virus primary isolation and propagation, preparing antigen for an AGID test, setting up an AGID test, and interpreting results are given.”

https://pubmed.ncbi.nlm.nih.gov/18370039/ <
https://pubmed.ncbi.nlm.nih.gov/18370039/ >

and wikipedia, a load of antiscientific nonsense.Just astonishing what the so called ‘scientist ‘ can make up , hoping and knowing, by throwing in a lot of jargon they make it sound so scientific and complicated that no-one will question it.

– https://en.m.wikipedia.org/wiki/Avian_influenza < https://en.m.wikipedia.org /wiki/Avian_influenza >

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wonder why they do not apply same to humans , best way to stop the pandemics is to kill the ‘ infected ‘ one. 😉
from one of the links. Now here is an idea as seems that tamiflu does not work.

"As of now, there are no drugs that cure bird flu in chickens. Therefore, the best way to treat chickens suffering from the flu and prevent the spread is to kill them and dispose of their remains properly.
If one chicken in your flock gets affected, the chances are that the rest are equally affected too. A laboratory test would most likely prove that. In this case, what remains is to cull the entire flock and start raising a new one."

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2141&_wpnonce=29363ef459 >
Like

NiciesMan

April 9, 2022 at 8:19 pm

https://www.frontiersin.org/articles/10.3389/fmicb.2021.626553/full <
https://www.frontiersin.org/articles/10.3389/fmicb.2021.626553/full >
I'm referring to that, I guess.

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2156&_wpnonce=c877758657 >
Like

Mike Stone < https://viroliegyhome.wordpress.com >

April 9, 2022 at 9:46 pm

That is a review of many animal models. The problem is that they still admit that no animal model faithfully recreates the "Covid" symptoms as seen in humans. This is from a review, also in Frontiers, a few months after the one you supplied:

"Although the existing experimental animals CAN NOT COMPLETELY REPLICATE HUMAN DISEASE FEATURES, they actually provide useful tools to address the

need for greater understanding of COVID-19."

https://www.frontiersin.org/articles/10.3389/fmicb.2021.770935/full < https://www.frontiersin.org/articles/10.3389/fmicb.2021.770935/full >

In other words, the experimental disease they create in animals in a lab is not the same as that seen in humans.

As for why the animals getting inoculated with cell culture goo may get sick versus a placebo such as saline, the cell culture is made up of toxic substances such as human fluids, animal cells, antibiotics/antifungals, fetal bovine blood, various chemicals and nutrients, etc. They are having this goo shoved into their nasal cavities. In the case of mice, they are genetically altered beforehand. Nothing about the animal models are natural.

★< https://viroliegy.com/2022/04/05/maurice-hilleman-and-the-avian-flu-pandemics/?like_comment=2157&_wpnonce=e7d0966479 >
Like

VIROLIEGY < HTTPS://VIROLIEGY.COM/ >