

Promising Cure to URTI Pandemics, Including the Avian Flu (H5N1): Has the Final Solution to the Coming Plagues Been Discovered? (Part I)

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Abstract

BACKGROUND: Recently Rentz (2003) published a convincing retrospective, peer-reviewed treatise on a highly advanced, effective and safe virotoxic oligodynamic silver (Ag^+) hydrosol, making the case that it is the agent of choice to combat SARS.¹ The works of Goetz (1940),² Berger *et al* (1976),³ Simonetti *et al* (1992),⁴ Russel *et al* (1994),⁵ and Crocker and Grier (1998)⁶ collectively established that electrolytically produced oligodynamic Ag^+ hydrosol provides the ideal speciation of bioactive Ag^+ completely harmless to mammals, in contrast to other colloidal silver or silver salt speciations that are predominantly inactive and potentially toxic to mammals. They also established that oligodynamic Ag^+ hydrosol possesses fabulous virotoxic properties. Comprehensive studies conducted by NASA (circa 1970) on a crude oligodynamic Ag^+ hydrosol preparation offer a compelling argument that today's highly advanced oligodynamic Ag^+ hydrosols may be the solution to lessening the impact of viral plagues. With today's advancement in Ag^+ processing technology, at least one commercially available, cost-effective oligodynamic Ag^+ hydrosol is (a) proven to be over 95% bioactive per volume, with (b) an unprecedented surface area of activity (i.e., $\geq 6 \text{ km}^2$ per gram Ag), that provides (c) an ideal concentration factor of < 25 ppm, in (d) an ideal liquid medium.

CONCLUSION: The pharmacology of advanced oligodynamic Ag^+ hydrosol shows great promise to easily overwhelm key defensive mechanisms of URTI resistance in general, including H5N1. Additional clinical studies are warranted to further demonstrate the efficacy and compatibility of *per os* virotoxic oligodynamic Ag^+ hydrosol, as well as investigational intravenous and nebulized (aerosol) protocols.

Key words: URTI pandemics, H5N1, oligodynamic Ag^+ hydrosol pharmacology, microbial defenses, microbial resistance, conventional treatments, effective protocol development, hypomolarity, sorbitol.

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Introduction

During the past 300 years, humanity has suffered ten pandemics and several epidemic scares. Could the solution to pandemics have a simple answer? Has nature always held the key to defeating pathogens in a simple Ag⁺ molecule?

Commonly known as the flu or the grippe, influenza is a contagious disease of the upper respiratory tract caused by viruses from the orthomyxoviridae family, all RNA viruses.⁷ Generally, human Influenzavirus starts with direct human contact with sick poultry and other animals.^{8,9}

The orthomyxoviridae family is divided into three types – Influenzavirus A, B, and C. Each type is differentiated by its respective “antigenic” nucleoprotein and matrix protein dissimilarities.¹⁰

Because two main “antigenic” surface glycoproteins of Influenzavirus A are prone to significant mutation, this virus requires further classification into subtypes. The Influenza A virus is a species of the genus influenzavirus A. There are multiple subtypes of Influenza A. The H5N1 virus is a direct descendent of the Spanish Flu virus, a subtype of Influenza A. Influenzavirus B and C do not have subtypes. However, Influenzavirus B and the subtypes of Influenzavirus A are further classified into strains.¹¹ The H5N1 subtype alone has more than 400 different strains.¹² Only Influenzavirus A may cause pandemics.

Influenzavirus A infects people, wild birds, domestic poultry, horses, pigs, and other animals. Wild birds are the viruses' natural hosts.¹³ Influenzaviruses B and C only infect humans.¹⁴ Influenza-virus B may cause epidemics, and Influenzavirus C is not capable of causing either, and typically causes only mild infections.¹⁵ Global pandemics of Influenzavirus A typically occur when three conditions are met:

1. A new type of Influenza A virus is introduced into the human population.
2. The new subtype causes serious human illness.
3. The virus sustains itself when it develops the means to easily spread from person to person (i.e., goes airborne or “sheds” easily from person to person).

History

Influenza was coined as the name of a predictable/seasonal disease in 15th century Italy. It derives its name from people who associated it with unfavorable astrological *influences*. The term was adopted by the medical community in the 18th century into “influenza di freddo” (meaning “influence of the cold”). Influenza reaches a peak in the winter months consistently six months after the maximum of solar radiation. It is unclear why the phenomenon of influenza is so consistently seasonal in nature.¹⁶

Three pandemics have taken place over the last 100 years. The Spanish Flu (a subtype of Influenza A known as H1N1) of 1918 to 1919 was the worst recorded human disaster in history. Over 500 million people suffered morbidity, and up to 100 million may have died, including over 500,000 Americans. In subsequent decades, two more pandemics arose. In 1957, the Asian flu broke out, a subtype of Influenza A known as H2N2. Up to 1.5 million may have died, including approximately 70,000 Americans. In 1968, the Hong Kong Flu broke out. A subtype of Influenza A known as H3N2, the virus killed up to a million people, including around 34,000 Americans.^{17,18}

Despite the Swine Flu (1976), Russian Flu (1977) and the Hong Kong Flu (1997) causing world-wide scares of a looming pandemic, no pandemic has occurred since 1968.¹⁹ This suggests that the world may be overdue for an influenza pandemic.

In any given year, up to 20% of Americans suffer from the flu, with over 100,000 requiring hospitalization. On average, 36,000 people per year die from the viral infection, predominately from a resulting pneumonia. Many would argue that it is not about if the next pandemic is coming, but when. And, when it comes, what are we going to do about it? Deal with it conventionally, or intervene with a highly effective and safe, virotoxic oligodynamic Ag⁺ hydrosol?²⁰

We will see shortly that the pharmacology of picoscalar oligodynamic Ag⁺ hydrosol make it an excellent candidate to thwart pandemics.

Impact

We are currently in phase 3 of an H5N1 pandemic alert. This is the half-way mark to a full blown pandemic.²¹ To date, there have been a total of 135 deaths from H5N1.²² So far, two cases involved documented human-to-human transmission. Due to the specific histology of the Influenza A virus, plus

the compounding issue of air-travel and rapid transit that facilitates contagion, it is expected that future pandemics will more easily infect numbers equivalent to or greater than the Spanish Flu of 1918.²³ The Center for Disease Control (CDC) predicts that should H5N1 go pandemic, a *medium-pandemic* would infect up to 35% of the US population, and up to 207,000 would die.²⁴ Should H5N1 evolve into a *severe-pandemic*, up to 90 million Americans are expected to contract the disease, and 2 million could die.²⁵

The symptoms of influenza may include acute respiratory distress, arthralgia, diarrhea, eye irritation, extreme chills, fatigue, fever, gastrointestinal pain, myalgia, nasal congestion, sneezing, sore throat, and unproductive cough.²⁶ The course of influenza is much more substantial than the common cold, and typically occurs over one to two weeks or more. Although healthy people will contract the flu, the young, the old and the chronically ill are the most prone to complications and death. Complications include pneumonia, bronchitis, conjunctivitis, sinusitis, otitis media, and also exacerbation of other chronic illness present, such as asthma.²⁷

Essentially the dynamics of influenza mutation evolve the viruses' proteinaceous antigenic profile. Two key mutagenic mechanisms involve “antigenic drift” (the most common) and “antigenic shift” (the least common). Antigenic drift describes “point mutations” that induce small, gradual changes in antigens on the surface of the virus that create new viral strains. In contrast, antigenic shift describes a substantial “genetic reassortment” between human and animal influenza genes that abruptly create novel viral subtypes. Only antigenic shift creates an environment for pandemics relative to influenza viruses. While Influenza A may undergo either form of mutation, Influenza B only undergoes antigenic drift. This is the reason why only Influenza A has the potential to generate pandemics. Even though both forms of mutations *may* produce novel antigens unrecognizable to antibodies conditioned by contact with previous influenza strains, antigenic shift virtually assures non-recognition by conditioned or naive antibodies.²⁸

Antigenic shift also virtually assures vaccine failure until the new subtype can be collected and successfully manufactured into an updated vaccine, typically realizable only after four to six months post-pandemic onset.²⁹

Discussion

Influenza viruses have envelopes to their outer surface that contain from 18% to 37% lipids by weight. This is a critical aspect of viral defenses. As a NASA-commissioned study showed, crude oligodynamic Ag⁺ preparations have a difficult time penetrating these waxy envelopes.³⁰ Picoscalar oligodynamic Ag⁺ hydrosol, which is over 95% bioactive, would not suffer the same handicap as did NASA's 1970 technologically crude Ag⁺ hydrosol preparation. There is an exchange between host cells and the virus of both lipids and proteins. The virus incorporates host cell lipids from plasma membranes into its envelope. The envelope, which may be either pleomorphic or filamentous, ranges from 20 to 300 nm in length. Influenza projects roughly 500 distinct surface projections of hemagglutinin and neuraminidase in a ratio of approximately 4.5 to 1.

Contrarily, host membranes will acquire viral proteins in their own membranes post-infection.^{31,32} This illustrates an important and likely cause of autoimmune

sequelae and may be a key etiology underlying post-viral syndromes in general. *The Journal of Nanotechnology* recently reported that picoscalar silver ions may easily defeat viral adsorption mechanisms by denaturing surface/envelope proteins.³³ Conceptually, if oligodynamic Ag⁺ hydrosol were given early in the course of infection, the powerful denaturing actions upon viral proteins might block host membranes from incorporating the Ag⁺ cleaved/highly fragmented protein residues rendered inert. Some day in the future, oligodynamic silver hydrosol may be recognized as a preventative for certain autoimmune sequelae that would otherwise manifest in ever-escalating anti-self antibody production over many years and decades post infection.

Influenza viruses are composed of nucleocapsids containing nucleoproteins. The nucleocapsids have a helical symmetry.³⁴ The nucleic acid is composed

of seven to eight linear negative-sense stranded RNA segments.³⁵ These related genes and their transcriptional end-products are prime targets for the denaturing activity of oligodynamic Ag⁺ hydrosol for all microbial life. Specifically for H5N1, these targets are as follows:³⁶

- Eight Genes;
- Hemagglutinin;
- Neuraminidase;
- Nucleoprotein;
- Matrix proteins M1 and M2; and
- RNA polymerase.

Amantadine, oseltamivir (Tamiflu®),^a ribavirin, rimantadine, and zanamivir are the central conventional drug treatments proven effective in influenza.



The majority of these drugs are expensive, and insurance coverage is often determined by whether or not the physician can successfully make the case that their use would make a significant difference, such as in an immune-compromised individual.³⁷

Drug side reactions for typical dosages of amantadine and rimantadine in young, healthy adults include CNS and gastrointestinal disturbances, which are typically mild and cease upon drug discontinuation. Serious delirium, hallucinations, agitation, seizures, and renal failure may occur as well, especially in the elderly and the sick.³⁸ Twenty percent of people taking amantadine and two percent of those taking rimantadine suffer from adverse CNS events.³⁹

Side reactions for oseltamivir include nausea and vomiting.⁴⁰

Side effects of ribavirin may be among the most serious of the five drugs. Significant numbers of patients,

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especially infants, experienced serious lung sequelae such as apnea, atelectasis, bacterial pneumonia, bronchospasm, cyanosis, dyspnea, pulmonary edema, hypoventilation, pneumothorax, and dependency upon a ventilator. Equally alarming were cardiovascular events that included arrhythmia, bradycardia, cardiac arrest, digitalis toxicity, and exacerbation of congenital heart disease.⁴¹

In patients with COPD, zanamivir has a significant record of inducing bronchospasm and deteriorating

respiratory function. For these reasons, the drug is contraindicated in patients with underlying airway disease.⁴²

Several reports substantiating influenza multiple drug resistance (MDR) are documented. The CDC reports that high rates of resistance to amantadine and rimantadine occur in the H3N2 strain of influenza A: China (74%), Hong Kong (70%), Taiwan (23%), and South Korea (15%). The overall resistance rate for the US is four percent.⁴³ This has caused the international community to stockpile oseltamivir. However, oseltamivir resistant H5N1 was isolated from a Vietnamese girl in February 2005.⁴⁴ Alarmingly, a recent publication by de Jong from Oxford University has shown these stockpiles could be powerless to stop an H5N1 pandemic. When oseltamivir was given in its current proper dose within the first 48 hours of flu onset, it still failed to prevent death!⁴⁵ Over time, the pharmacodynamics of these drugs makes them less able to affect the newly mutated proteins of the latest version of the influenza virus.

Prevention with vaccinations is mired by similar difficulties. Even when an effective vaccine is developed, it is typically effective for a single year at best, due to the high rate of influenza mutation.⁴⁶ In fact, the CDC lists the flu vaccine as being only 16% to 63% effective

a. Tamiflu® is a registered trade mark of Roche Holding.

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in general. In 2003, the flu vaccine's effectiveness was virtually nil.⁴⁷ Other complications stemming from vaccination programs can sometimes be quite embarrassing. For example, during the US Swine Flu scare of 1976, 24% of the US population became inoculated, resulting in 25 deaths nationwide. This iatrogenic mortality rate exceeded the death rate from the Swine Flu itself. Additionally, a significant number of those inoculated were maimed with Guillain-Barre Syndrome (GBS).⁴⁸

The results of influenza virus mutation and the limitations of conventional treatment clearly illustrate why oligodynamic Ag⁺ hydrosol may be the medicine of choice to thwart pandemics. All current scientific evidence supports that oligodynamic Ag⁺ hydrosol will denature virtually any protein or nucleic acid^{49,50,51} that lacks sufficient antioxidant protection or lacks sufficient surveillance by metallothioneins.⁵² Viruses possess neither system of defense, and their endlessly evolving cycle of viral protein mutations cannot shield their recurrent vulnerability to the denaturing action of oligodynamic Ag⁺. The evidence is quite compelling that, over the past century, the Orthomyxoviridae family [e.g., Influenza (species unidentified),^{53,54,55} Influenza A (strains not identified),^{56,57} Influenza A (Okuda strain),⁵⁸ and Influenza B (*Haemophilus influenzae*)]⁵⁹ has been unable to outwit the virotoxic effects of oligodynamic Ag⁺.

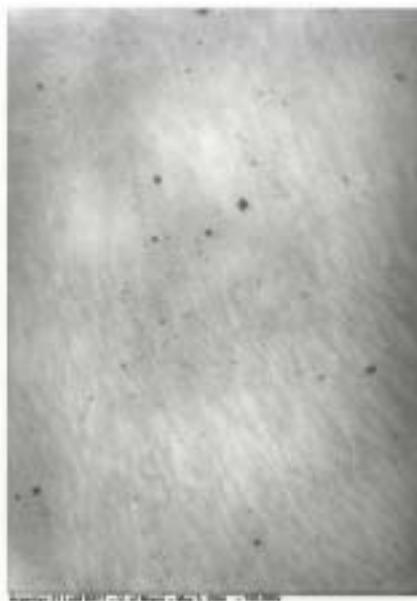
Picoscalar oligodynamic Ag⁺ hydrosol may be the safest and durable denaturing agent of lower life forms' essential proteins.⁶⁰ Comprehensive evidence suggests that the fate of spent picoscalar silver in humans is easily and harmlessly eliminated.⁶¹

In view of all of the above, it would be prudent for physicians to consider using oligodynamic Ag⁺ hydrosol to treat influenza, especially MDR influenza cases.

Preview of Part II - Today's Most Promising Treatment

NASA's research concluded that, *in vitro*, a minimum of 50 ppb of oligodynamic Ag⁺ hydrosol was necessary to exert meaningful virotoxic effects over four hours.⁶² More recent *in vivo* human studies suggest 1 ppm to 10 ppm as a suitable plasma concentration for oligodynamic Ag⁺ for viral infections such as HIV.⁶³

Picoscalar, or near picoscalar oligodynamic Ag⁺,⁶⁴ acquires a surface area capable of exerting virotoxic events many orders of magnitude^{65,66} superior to any previous Ag⁺ compound. The quintessential mechanism of picoscalar Ag's oligodynamics is *particle charge*.⁶⁷⁻⁷⁰ By volume, at least one highly advanced picoscalar silver product contains over 95% bioactive Ag⁺⁷¹ as opposed to under two percent for most other forms of historical silver-based drugs.⁷²



**Picoscalar Silver Hydrosol Lot #BR11,
University of Miami, May 5th, 2004;
100,000X**

In the case of H5N1, the "chief battlefield" for oligodynamic Ag⁺ hydrosol's virotoxicity would be (a) just prior to viral adsorption into host cells, or (b) when it intercepts already replicated virus cores budding back into the extra cellular fluid of the host. Timing with respect to the course of the viral disease is important in investigational intravenous administration of Ag⁺ hydrosol.

For example, during initial/preliminary stages of influenza virus adsorption into host cells, a simple 60 cc IV push administered over 20 minutes and repeated two or three times weekly with a p.o. follow-up is all that is typically necessary. Recovery is essentially rapid post-IV push over the next 24 hours. But when the viral load has already become significant (late-stage), and an "en masse" viral exodus to infect new host cells is underway, 750 cc to 1500 cc IV drips rendered isotonic with sorbitol may be called for once or twice weekly (with at least a 72 hour hiatus between IV drip

administrations). IV drips with sorbitol aim to build plasma Ag⁺ concentrations rapidly up to 1 ppm to 10 ppm over three hours. Typically, only one IV drip with sorbitol given over three hours is required in the most severe cases. However, in cases requiring more therapy, one follow-up IV push of 60 cc 72 hours post-IV drip should resolve the case within the next 72 hours, providing supportive p.o. and nebulized administration care continue with respiratory therapy until full recovery. If the IV push fails to resolve the case, one more IV drip with sorbitol over three hours may be advisable 72 hours post-IV push.

Best pre- and/or post- follow-up dosage schedules to IV administration include the following:

(a) *Per os* dosages ranging from one teaspoon to one tablespoon taken on an empty stomach every 20 to 60 minutes during initial stages (first week) of the flu – reducing dosages accordingly with symptom alleviation; discontinue p.o. dosages until three to four hours post-prandial. If symptoms do not show clear improvement within 24 hours, then in addition to continuing the upper p.o. dosage, begin

(b) Investigational nebulized dosages of 5 cc given once or twice daily or even hourly alongside respiratory therapy if case continues to deteriorate. Immediately reduce dosages accordingly as symptoms improve.

(c) In severe cases that are *slowly deteriorating*, or in cases where rapid improvement is deemed medically necessary, an investigational slow IV push as described above may be required.

(d) In critical cases with *rapid deterioration*, investigational use of IV drips administered as described above may be prudent.

All dosages are for an average +75 Kilo adult patient, with investigational nebulized or investigational IV dosages being cut by one-half for patients approximately 37 Kilos in size. For toddlers less than 20 Kilos, the dosages are further reduced to just one-quarter of the adult amounts.

Uneventful Jarisch-Herxheimer Effects (die-off) are to be expected in select subpopulations and managed if required, as will be covered in Part II.

Part II: Pharmacology, Therapeutic Index, Case Histories; Protocol Proposal and Procedure for Follow-on Studies; and Adjunctive CAM Prevention and Treatment will appear in the April issue of *The Townsend Letter*.

Correspondence

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