Swine Flu--What Should I Do?

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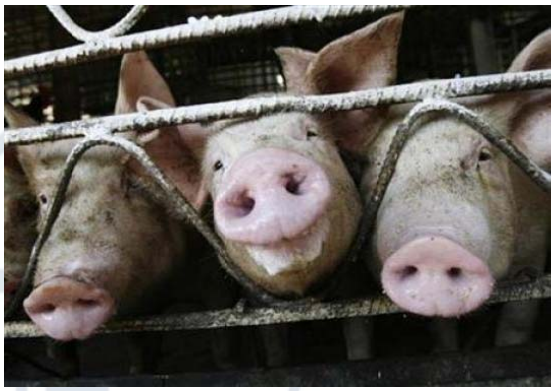
The news media run the swine flu epidemic as its lead story. Everyone from the Director of the World Health Organization to President Obama is warning of the dire consequences of swine flu. Our office is getting calls and emails about what to do. So what are the facts?



Indeed, there have been pandemics in the past. The best known are the bubonic plague between 1339 and 1351 AD and the Spanish Flu epidemic of 1918. However, there have been dire warnings of pandemics that never happened. Many remember the swine flu disaster of 1976 when the government killed dozens and injured hundreds with harmful vaccines while few died from the "eminent pandemic". President Ford was gearing up to force vaccination on the entire population when the vaccine began to create havoc. Claims of over $1.3 billion came from victims of the vaccine that caused severe paralysis, Guillain-Barre Syndrome, and death of 25. So is the fiasco of 1976 repeating itself, or is this for real? The call for mandatory vaccination is being discussed again--as soon as a vaccine can be prepared. One thing different now is that laws have been passed so those damaged by vaccines cannot sue the vaccine makers!

What is new about this virus is that it has a mixture of DNA from animals, birds, and humans! Normally viruses are species specific. Viruses that cause illnesses in hogs can rarely be transmitted to humans, but that virus usually cannot be transmitted human to human. Although some express confusion about how this virus could have mutated in a way that a hog virus and a bird virus could mix with a human virus and cause human-to-human transfer, it is known that mixing of viral DNA has been done in laboratories. Whether that is what happened in this current virus is not clear. Is this virus a natural mutation or was it made in a laboratory and somehow got into the population? Was it simply released in Mexico and spread from there? There is reason to wonder. Earlier this year, Baxter flu vaccines contaminated with H5N1 - otherwise known as the human form of avian flu, one of the biological weapons on earth with a 60% kill rate - were received by labs in the Czech Republic, Germany, and Slovenia. It was first given to animals and killed all that received it! The H5N1 virus on its own has killed hundreds of people, but it is less airborne and more restricted in the ease with which it can spread. However, when combined with seasonal flu viruses, which, as everyone knows are super-

airborne and easily spread, the effect is a potent, super-airborne, super-deadly biological weapon.

"In 1970 the discovery of a cell enzyme, called "reverse transcriptase" by Howard Temin and David Baltimore, allowed molecular biologists to detect so-called retroviruses in some animal cancers. It was soon recognized that retroviruses could be found normally in the genes of many animal cells, and that scientists could manipulate these viruses to produce detrimental effects on the immune system. In "species jumping" laboratory experiments, many viruses were transferred between different animal species and were adapted to human cells.

The earliest AIDS cases in America can be clearly traced back to the time period when the hepatitis B experiment began at the New York Blood Centre. The Centre began injecting gay men with multiple doses of the experimental vaccine in November 1978. The inoculations ended in October 1979, less than two years before the official start of the epidemic. The hepatitis B experiment, which inoculated over 1,000 healthy gay men in 1978 in New York, was a huge success with 96% of the men developing antibodies again the hepatitis virus. This high rate of success could not have been achieved if the men were immuno-suppressed, because immuno-suppressed people do not easily form antibodies to the vaccine. The experiment was followed by similar hepatitis B experiments using gay men in Los Angeles, San Francisco, Chicago, Denver and St. Louis, beginning in March 1980 and ending in October 1981, the same year the epidemic became official." http://www.newdawnmagazine.com/Article/The_Secret_Origins_of_AIDS.html

Except for the fact that the DNA of this virus is suspect, we should not expect to have an epidemic that kills many people. One of the reasons is that viruses usually do not kill people---they just make you feel bad. What killed the majority of people in 1918 was that the flu allowed people to get bacterial pneumonia from Streptococcus. That is what kills you. We are much better able to deal with bacterial pneumonia now than they were in 1918.

However, the genetically altered viruses like the AIDS virus have killed many. That is the reason for current concerns.

In 1897, the German company Bayer patented aspirin. Their patent expired in 1917, just at the end of World War I. Many of the returning American soldiers brought it back to their families. It was the first time that there had been widespread use of aspirin with the flu. It is known that when a virus attaches to a cell, it cannot duplicate if there is a fever, but it will make a million copies of itself if the temperature is low. Thus lowering temperature with drugs allows viruses to multiply! It is also known that aspirin and drugs like it suppress the immune system making it easier for bacteria to grow. This makes it easier for pneumonia to occur. It is not clear how much aspirin contributed to the spread of the 1918 flu. A current problem is that the antiviral drugs, Tamiflu® and Relenza® lower body temperature. It is not uncommon to see people get the flu and start one of these drugs. They feel better. Then a week later, they have pneumonia.

Since 2003, there have been multiple warnings that the H5N1 bird flu virus would kill millions of people. Only 257 people are known to have died from the bird flu! Over 1,000,000 people get malaria every year, but there are no dire warnings from the World Health Organization or President Obama about malaria!

Can there be other reasons that we are being frightened about a flu pandemic? The Bush administration bought $1.4 billion of Tamiflu®--"to combat the bird flu." The Obama administration wants to buy enough to treat 25% of the American population. Other governments are stockpiling it as well. This is despite the fact that Tamiflu® does not work for the bird flu and is not likely to work for the swine flu either. "After following WHO protocols in treating 41 victims of the H5N1 bird flu virus (19% of the worldwide cases of bird flu reported to date), Nguyen Tuong Van, MD, who runs the intensive care unit of the Center for Tropical Diseases in Hanoi, Vietnam concluded that Tamiflu, the drug most widely stockpiled around the world to combat a potential bird flu pandemic, is "useless." (Wikipedia) Thus, the American taxpayers paid billions of dollars for a drug to treat about 100 cases per year of the bird flu. Someone made a lot of money from a drug that does not work for an epidemic that never happened. They are making even more money this year. If only we were using that money for something useful like treating malaria!

So what if this new virus does cause widespread flu? What if I am wrong about this being history repeating itself? What should you do?

There are several simple steps you can take to help prevent yourself from getting colds and the flu.

1. Vitamin D is made when you expose significant amounts of your skin to the sun. It takes about 20 minutes a day to make what you need. The vitamin D blocks the sites on cells where viruses normally attach. Thus taking vitamin D3 in 2000 to 5000 international units per day significantly reduces your chances of getting a viral infection. It is interesting to note that "flu season" is during the cold months when you do not expose your skin to the sun. It is also interesting that the bubonic plague of the 1340's was also known as the "Little Ice Age" because of unusually cold temperatures in Europe. The 1918 flu epidemic occurred in the winter. You can do a blood test to see if your vitamin D levels are low, but in my practice, I have never seen a normal test in someone who was not taking supplements.
2. Iodine kills all single-celled organisms and viruses. They can never become resistant to it. If you have enough, you body will put 30 times as much iodine in the parts of the body exposed to the outside world as it has in the blood. This envelopes you in a shield of protective iodine to keep you from getting infections. You should take 12.5 mg of iodine per day. The brands are iThroid® (RLC Labs) and Iodoral®.
3. Vitamin C is important to keep the body working correctly. The only species that cannot make their own vitamin C are humans, guinea pigs and primates.

You need about 5000 mg of powdered vitamin C or 1000 mg if taken with phosphatidyl choline. The book Curing the Incurable, Vitamin C and Infectious Disease and Toxins by Thomas Levy, MD looks at 1200 medical articles about killing infections with vitamin C. We have been successful in curing even penicillin-resistant staph (MRSA) with IV vitamin C.

4. Nano-particles of silver kills microorganisms efficiently, even resistant ones like MRSA and pseudomonas. Take one teaspoonful, three times a day. I use it when I need an antibiotic, not every day.
5. Essential oils are extracted from plants or manufactured in a lab. The natural ones are therapeutic. The manufactured ones smell nice but have no health benefits. About 95% of those sold in the US have no effect. However, the pure essential oils are very powerful. The spices are particularly great at killing microorganisms. It is interesting that the spice traders did not get the bubonic plague. Make a tea with a few drops of therapeutic cinnamon and clove oil mixed with a tablespoon of coconut oil and a tablespoon of raw, local honey.
6. Methylene blue has been used for years to kill viruses in our blood supply. See the references below. Professor Charles McWilliams says, "Here's the down and simple: tell you viral patients to get unadulterated 3% hydrogen peroxide, like that found in valu-mart or dollar stores. The cheaper the better. Also, get a one-dollar bottle of methylene blue. If not available at the drug store, go to the pet shop for the same. As a tonic, four times daily: a full glass of water, 1 tablespoon of H2O2, and a few drops of methylene blue. Drink!" Your tongue and urine will be blue, but that is better than a few days in bed with the flu.
7. I also use the Tennant Biomodulator® to be sure the voltage in every organ is normal. It is hard to have infections if voltage (and thus oxygen levels) is normal.
8. Wash your hands and clothing frequently.

These things are easy and inexpensive. Most are in stock in our office. In the unlikely event the flu spreads, you will have taken precautions to keep from getting it or at least shortening its course. I have put more reading material and references below if your want more information.

Be Well!

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References:

Epidemics/Pandemics

Between 1339 and 1351 AD, a pandemic of plague traveled from China to Europe, known in Western history as The Black Death. Carried by rats and fleas along the Silk Road Caravan routes and Spice trading sea routes, the Black Death reached the Mediterranean Basin in 1347, and was rapidly carried throughout Europe from what was then the center of European trade. Eventually, even areas of European settlement as isolated as Viking settlements in Greenland would be ravaged by the plague. By the time these plagues had run their course in 1351, between 25 and 50% of the population of Europe was dead. An equally high toll was exacted from the populations of Arabia, North Africa, South Asia, and East Asia. This paper will examine the role of trade in the spread of the plague.

Note: Europe is a crucial focus to plague as a trade related issue for this reason. Since plague is not native to the European region, the relationship between Medieval trade and Medieval Europe's greatest ecological disaster become obvious. 2. Description The Black Death was actually a combination of three different types of plague: bubonic, pneumonic, and septicaemic, with bubonic being the most common. The bacteria that cause the diseases live in the digestive tract of fleas, most frequently rat fleas. Plague bacteria can continue to survive in places like rat burrows (dark, moist environments) even after the plague has killed off all the rats in an infected group. Thus, the plague can lay dormant for a lengthy period until a new group of rodents moves into an infected burrow. Rats were extremely important to spreading the plague, but they were not the only means of dissemination. Through the spread of fleas among rodent populations, plague outbreaks can strike both urban and rural areas. Furthermore, the plague-bearing fleas could be transmitted to virtually any type of household or farm animal (1).

Bubonic Plague has an incubation period, from infection to the first symptoms, of approximately six days. The initial symptom is a blackish pustule forming over the point of the bite, followed by swollen lymph nodes near that bite. This is followed by subcutaneous hemorrhaging, which produces bruise-like purple blotches, called buboes, on the victim's skin. It is from this word, buboe, that the bubonic plague takes its name. The hemorrhaging causes an intoxication of the nervous system, which produces neurological and psychological disorders, including insomnia, delirium, and stupor (2). These disorders, particularly delirium, might be behind the bizarre *danse macabre* performed by plague victims that are described in medieval chronicles. Bubonic Plague is the least toxic of the three types, but still kills 50 to 60% of its victims (3).

Septicaemic plague is, like the bubonic plague, carried by insects. Its distinguishing feature is its rapidity - death occurs within a day of infection, even before buboes have had time to form. This form of the plague is the rarest rare, but is almost always fatal (4).

Pneumonic plague differs from the other two forms in that it can be spread from person to person. After a two to three day incubation period, victims suffer a sharp drop in body temperature, which is followed by severe coughing and discharge of bloody sputum. This sputum contains the plague bacteria, making for an airborne transmission. As in bubonic plague, neurological and psychological disorders follow. Pneumonic plague is rarer than bubonic, but is fatal in over 95% of its victims (5).

None of these plagues is native to Europe. Plague bacteria normally resides in Central Asia, Yunan China, Arabia, East Africa, and limited areas of Iran and Libya. One reason for this is climactic. The weather in Northern Europe is hostile to the plague bearing fleas to such an extent that regular outbreaks would not be possible even in the summers, let alone the winters of 14th Century Europe (6). Their spread to Europe from these areas has always been through global commerce - trade which carried with it plague- bearing rats and fleas.

The first instance of a plague outbreak in Europe was Justinian's Plague, which raged from 541 to 544 AD, with sporadic lesser outbreaks of the plague lasting until the end of the Eighth Century. Outbreaks of Justinian's Plague almost invariably followed the same pattern. It is believed to have been carried down the

Nile, from East Africa into the Mediterranean Basin. It rapidly spread along the trading routes from Egypt's main port, Alexandria, to Central and South Asia, Arabia, North Africa, and much of Southern Europe. Justinian's Plague was quite terrible - between years 541 and 542 about 40% of Constantinople, the central trading port of the Mediterranean world then, died of the plague. By the end of 544, it is estimated that 20 to 25% of the population of Europe south of the Alps had been killed by the plague. The spread of the plague into Northern and Central Europe was limited, probably by the lack of a significant trade infrastructure to the North in what was the ensuing Dark Ages (7).

By the 14th Century, things had changed. Due to technological innovations in agriculture, such as the three-field planting system, the population of Europe had risen to a level that it had not seen in millennia, during the Roman Empire (8). This is despite the "Little Ice Age," a period of climactic deterioration and generally colder weather, which would not end until the mid-16th Century (9). A well-developed trade network in expensive luxury goods existed, carrying products by three main routes. The first was entirely overland, running from Northern China, through Central Asia, and then to the Black Sea. This was the famous "Silk Road." Spice trading ran along a route from South Asia to the Persian Gulf, and thence overland by caravan to the Levant. A second spice route ran by sea from South Asia to the Red Sea and Egypt (10).

From the Black Sea, the Levant, and Alexandria (still Egypt's main port), goods were picked up most often by Italian middlemen, who plying routes along the Mediterranean Basin, which was the nexus of European trade, delivered goods to Italy, Catalonia, and Southern France. By the 14th Century, there were also developed routes through the Straits of Gibraltar to England and Holland. From England and Holland, trade by sea extended into the Baltic, reaching lands as far away as Muscovy.

The great pandemic of plague, known in Europe as the Black Death, is thought to have begun in China in the early 1330s. Reliable chronicles tell of an outbreak of the plague in China, beginning in 1331. Sources in Latin, Arabic, and Chinese tell of numerous natural disasters such as floods and earthquakes. These events might have destroyed the habitats of the plague-bearing rodents, forcing them into contact with other rodent populations and thus spreading their fleas (11). By the 1350s, two-thirds of China's population lay dead (12) The influenza pandemic of 1918-1919 killed more people than the Great War, known today as World War I (WWI), at somewhere between 20 and 40 million people. It has been cited as the most devastating epidemic in recorded world history. More people died of influenza in a single year than in four-years of the Black Death Bubonic Plague from 1347 to 1351. Known as "Spanish Flu" or "La Grippe" the influenza of 1918-1919 was a global disaster.

http://www1.american.edu/TED/bubonic.htm

The Grim Reaper by Louis Raemaekers:

In the fall of 1918, the Great War in Europe was winding down and peace was on the horizon. The Americans had joined in the fight, bringing the Allies closer to victory against the Germans. Deep within the trenches these men lived through some of the most brutal conditions of life, which it seemed could not be any worse. Then, in pockets across the globe, something erupted that seemed as benign as the common cold. The influenza of that season, however, was far more than a cold. In the two years that this scourge ravaged the earth, a fifth of the world's population was infected. The flu was most deadly for people ages 20 to 40. This pattern of morbidity was unusual for influenza, which is usually a killer of the elderly and young children. It infected 28% of all Americans (Tice). An estimated 675,000 Americans died of influenza during the pandemic, ten times as many as in the world war. Of the U.S. soldiers who died in Europe, half of them fell to the influenza virus and not to the enemy (Deseret News). An estimated 43,000 servicemen mobilized for WWI died of influenza (Crosby). 1918 would go down as unforgettable year of suffering and death and yet of peace. As noted in the Journal of the American Medical Association final edition of 1918: *"The 1918 has gone: a year momentous as the termination of the most cruel war in the annals of the human race; a year which marked, the end at least for a time, of man's destruction of man; unfortunately a year in which developed a most fatal infectious disease causing the death of hundreds of thousands of human beings. Medical science for four and one-half years devoted itself to putting men on the firing line and keeping them there. Now it must turn with its whole might to combating the greatest*

enemy of all--infectious disease," (12/28/1918).. http://virus.stanford.edu/uda/

Aspirin

Medicines containing derivatives of salicylic acid, structurally similar to aspirin, have been in medical use since ancient times. Salicylate-rich willow bark extract became recognized for its specific effects on fever, pain and inflammation in the mid-eighteenth century. By the nineteenth century, pharmacists were experimenting with and prescribing a variety of chemicals related to salicylic acid, the active component of willow extract.

A French chemist, Charles Frederic Gerhardt, was the first to prepare acetylsalicylic acid in 1853 (patented under the name aspirin on March 6, 1899 [8])

In 1897, scientists at the drug and dye firm Bayer began investigating acetylsalicylic acid as a less-irritating replacement for standard common salicylate medicines. By 1899, Bayer had dubbed this drug *Aspirin* and was selling it around the world.[12]The name Aspirin is derived from A = Acetyl and "Spirsäure" = an old (German) name for salicylic acid.[13] Aspirin's popularity grew over the first half of the twentieth century, spurred by its effectiveness in the wake of the Spanish flu pandemic of 1918, and aspirin's profitability led to fierce competition and the proliferation of aspirin brands and products, especially after the American patent held by Bayer expired in 1917.[14][15] Wikipedia. Note from Dr. Tennant: This Wikipedia article suggests that aspirin was an effective treatment of the flu. See my discussion above that it may have assisted in spreading the flu!

Deaths from bacterial pneumonia during 1918-19 influenza pandemic.
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Deaths during the 1918-19 influenza pandemic have been attributed to a hyper virulent influenza strain. Hence, preparations for the next pandemic focus almost exclusively on vaccine prevention and antiviral treatment for infections with a novel influenza strain. However, we hypothesize that infections with the pandemic strain generally caused self-limited (rarely fatal) illnesses that enabled colonizing strains of bacteria to produce highly lethal pneumonias. This sequential-infection hypothesis is consistent with characteristics of the 1918-19 pandemic, contemporaneous expert opinion, and current knowledge regarding the pathophysiologic effects of influenza viruses and their interactions with respiratory bacteria. This hypothesis suggests opportunities for prevention and treatment during the next pandemic (e.g., with bacterial vaccines and antimicrobial drugs), particularly if a pandemic strain-specific vaccine is unavailable or inaccessible to isolated, crowded, or medically underserved populations.

Epidemic influenza and vitamin D. Cannell JJ, Vieth R, Umhau JC, Holick MF, Grant WB, Madronich S, Garland CF, Giovannucci E **Epidemiol Infect** (2006 Dec) 134(6):1129-40 ISSN: 0950-2688

Abstract: In 1981, R. Edgar Hope-Simpson proposed that a 'seasonal stimulus' intimately associated with solar radiation explained the remarkable seasonality of epidemic influenza. Solar radiation triggers robust seasonal vitamin D production in the skin; vitamin D deficiency is common in the winter, and activated vitamin D, 1,25(OH) 2D, a steroid hormone, has profound effects on human immunity. 1,25(OH) 2D acts as an immune system modulator, preventing excessive expression of inflammatory cytokines and increasing the 'oxidative burst' potential of macrophages. Perhaps most importantly, it dramatically stimulates the expression of potent anti-microbial peptides, which exist in neutrophils, monocytes, natural killer cells, and in epithelial cells lining the respiratory tract where they play a major role in protecting the lung from infection. Volunteers inoculated with live

attenuated influenza virus are more likely to develop fever and serological evidence of an immune response in the winter. Vitamin D deficiency predisposes children to respiratory infections. Ultraviolet radiation (either from artificial sources or from sunlight) reduces the incidence of viral respiratory infections, as does cod liver oil (which contains vitamin D). An interventional study showed that vitamin D reduces the incidence of respiratory infections in children. We conclude that vitamin D, or lack of it, may be Hope-Simpson's 'seasonal stimulus'.

On the epidemiology of influenza. Cannell JJ, Zasloff M, Garland CF, Scragg R, Giovannucci E Virol J (2008) 5:29 ISSN: 1743-422X

Abstract: The epidemiology of influenza swarms with incongruities, incongruities exhaustively detailed by the late British epidemiologist, Edgar Hope-Simpson. He was the first to propose a parsimonious theory explaining why influenza is, as Gregg said, "seemingly unmindful of traditional infectious disease behavioral patterns." Recent discoveries indicate vitamin D upregulates the endogenous antibiotics of innate immunity and suggest that the incongruities explored by Hope-Simpson may be secondary to the epidemiology of vitamin D deficiency. We identify - and attempt to explain - nine influenza conundrums: (1) Why is influenza both seasonal and ubiquitous and where is the virus between epidemics? (2) Why are the epidemics so explosive? (3) Why do they end so abruptly? (4) What explains the frequent coincidental timing of epidemics in countries of similar latitude? (5) Why is the serial interval obscure? (6) Why is the secondary attack rate so low? (7) Why did epidemics in previous ages spread so rapidly, despite the lack of modern transport? (8) Why does experimental inoculation of seronegative humans fail to cause illness in all the volunteers? (9) **Why has influenza mortality of the aged not declined as their vaccination rates increased?** We review recent discoveries about vitamin D's effects on innate immunity, human studies attempting sick- to-well transmission, naturalistic reports of human transmission, studies of serial interval, secondary attack rates, and relevant animal studies. **We hypothesize that two factors explain the nine conundrums: vitamin D's seasonal and population effects on innate immunity, and the presence of a subpopulation of "good infectors**." If true, our revision of Edgar Hope-Simpson's theory has profound implications for the prevention of influenza.

West Nile virus in plasma is highly sensitive to methylene blue-light treatment.

Mohr H, Knuver-Hopf J, Gravemann U, Redecker-Klein A, Muller TH **Transfusion (2004 Jun) 44(6):886-90** ISSN: 0041-1132

Abstract: BACKGROUND: The epidemic of West Nile virus (WNV) in the US resulted in cases of transfusion-transmitted WNV. Effective pathogen reduction methods could have removed this infectious agent from the blood supply. We have evaluated the efficacy of photodynamic treatment of fresh frozen plasma (FFP) with methylene blue (MB), a decontamination method applied in several European countries.

STUDY DESIGN AND METHODS: FFP units (300 ml each) were spiked with WNV. MB was added, and the units were illuminated with white or monochromatic yellow light. WNV infectivity was determined by bioassay. WNV-RNA was quantitated by real-time PCR. The inactivation of WNV was investigated under standard and under suboptimal conditions, respectively. In addition, rechallenge experiments with multiple additions of WNV at maximal load (approx. 105 CFU/ml) and repeated illumination without replenishing MB were performed.

RESULTS: Complete inactivation of WNV was achieved by MB (0.8-1 mmol/l) and illumination with white light (30,000-45,000 Lux) within 2 min. White yellow light 20-40 J/cm(2) (2.5-5 min) were sufficient for inactivation by 5.75 log10-steps. The rechallenge experiments revealed the substantial reserve capacity of the procedure to inactivate WNV. Quantitative PCR indicated that the viral RNA

was rapidly destroyed.

CONCLUSION: All experimental data demonstrate the enormous potency of photo treatment with MB to inactivate WNV in plasma.

Mammalian genotoxicity assessment of methylene blue in plasma: implications for virus inactivation [see comments]

Wagner SJ, Cifone MA, Murli H, Dodd RY, Myhr B **Transfusion (1995 May) 35(5):407-13** ISSN: 0041-1132

Abstract: BACKGROUND: The risk of adverse consequences of virus-inactivation procedures for plasma and cellular blood components must be less than the risk of transfusion-associated viral disease. Previous studies demonstrated that methylene blue, which is currently used in Europe for virus inactivation in fresh-frozen plasma, can elicit mutations in bacterial test systems. This study investigates the potential for methylene blue genotoxicity in two mammalian test systems.

STUDY DESIGN AND METHODS: Different concentrations of methylene blue were prepared in plasma (heat-treated at 56 degrees C for 1 hour to reduce cytotoxicity) and used, without illumination, in an in vitro mouse lymphoma cell assay designed to detect forward mutations in the gene encoding thymidine kinase. The assay was performed in the presence or absence of rat liver S9 microsomal fraction. Similarly prepared samples of methylene blue in heat-treated plasma were used in an in vivo mouse micronucleus assay. Each system included a negative vehicle control (heat-treated plasma without methylene blue) and a positive control consisting of a known genotoxic agent.

RESULTS: Intravenous administration to mice of 62 mg per kg of methylene blue did not increase the frequency of micronuclei in polychromatic red cells harvested from bone marrow. However, methylene blue concentrations of 10 micrograms per mL (with S9 activation) and 30 micrograms per mL (without S9 activation) significantly increased the thymidine kinase mutation frequency of mouse lymphoma cells to approximately 110 x 10(-6), from a spontaneous frequency of 28 x 10(- 6).

CONCLUSION: Methylene blue is mutagenic in cultured mammalian cells. In contrast, results from the mouse micronucleus assay suggest that the genotoxicity is not expressed in vivo. Considerably more investigation will be required to assess the genotoxic potential of intravenously administered methylene blue used in virus-inactivation procedures, because of the likelihood of the formation of methylene blue photoproducts or the impact of metabolic conversion of methylene blue to leukomethylene blue in vivo.

No evidence for neoantigens in human plasma after photochemical virus inactivation.

Mohr H, Knuver-Hopf J, Lambrecht B, Scheidecker H, Schmitt H **Ann Hematol (1992 Nov) 65(5):224-8** ISSN: 0939-5555

Abstract: Photodynamic virus inactivation of human fresh plasma mediated by visible light in the presence of the phenothiazine dyes methylene blue or toluidine blue was investigated to determine whether it influences functional, structural, and immunological properties of plasma proteins. The activities of the coagulation factors I, VIII, IX, X, and XI were affected to a certain degree, while those of most other plasma proteins were not. The elution profiles obtained by ion exchange chromatography of untreated and photodynamically treated plasma were almost identical. Using a number of antisera against human plasma and single plasma proteins, different immunochemical techniques revealed identical patterns for untreated and treated plasma. Thus, there was no indication that the photodynamic virus inactivation procedure applied considerably influences the properties of plasma proteins.

Protective effect of vitamin C on protein activity in plasma during virus inactivation

Li Y, Li MY, Jiang RJ, Jia WX **Zhongguo Shi Yan Xue Ye Xue Za Zhi (2006 Apr) 14(2):392-6**
ISSN: 1009-2137 Published in Department of Microbiology West China College of Preclinical and Forensic Medicine Sichuan University Chengdu 610041 China.

Abstract: To determine whether addition of vitamin C (Vit C) to single-unit plasma could influence the efficacy of inactivating viruses and could maintain the activity of plasma proteins by methylene blue (MB)-light treatment. Vesicular stomatitis virus (VSV) Indiana strain was used as the indicating virus. Human plasma containing VSV was added with different concentrations of Vit C and final concentration 1 micromol/L MB and irradiated by fluorescence at an intensity of 40,000 lx, samples were collected at different times for detection. Cytopathic effect was used to test the effect of virus inactivation. A segment of the nucleic acid encoding capsid protein of VSV was amplified with RT-PCR. Some methods, such as the Clauss method, the one-stage method, microimmunoelectrophoresis, were used to investigate the changes of plasma components. The results showed that when the VSV plasma was added with 240 micromol/L Vit C and treated by MB-light irradiation for 60 min, the titer of VSV decreased by more than 8 lg TICD50/ml. Meanwhile, target segment amplification of VSV was also negative. The recovery rates of fibrinogen and coagulation factor VIII (FVIII: C) were 83.55% and 81.67% respectively, which had significant difference comparing with the routine MB-fluorescent light treatment. Most of plasma proteins were not affected significantly. No change in immunogenicity of these proteins was observed by using microimmunoelectrophoresis. It is concluded that virus inactivation is not influenced and plasma proteins are effectively protected by Vit C. Vit C can be used as a protector and is beneficial to improving the quality of plasma subjected to MB- photodynamic treatment.

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Methylene blue photoinactivation abolishes West Nile virus infectivity in vivo.

Papin JF, Floyd RA, Dittmer DP Antiviral Res (2005 Nov) 68(2):84-7 ISSN: 0166-3542

Abstract: The prevalence of West Nile virus (WNV) infections and associated morbidity has accelerated in recent years. Of particular concern is the recent demonstration that this virus can be transmitted by blood products and can cause severe illness and mortality in transfusion recipients. We have evaluated methylene blue (MB)+light as a safe and cost-effective means to inactivate WNV in vitro. This regimen inactivated WNV with an IC50 of 0.10 microM. Up to 10(7)pfu/ml of WNV could be inactivated by MB+light with no residual infectivity. MB+light inactivated three primary WNV isolates from the years 1999, 2002 and 2003 and prevented mortality in a murine model for WNV infection. Since MB is already approved for human use at a dose of 100mg/kg/day, we conjecture that MB+light treatment of blood products for high-risk patients will be efficacious and suitable for use in resource-limited settings.

Inactivation of dengue virus by methylene blue/narrow bandwidth light system.

Huang Q, Fu WL, Chen B, Huang JF, Zhang X, Xue Q **J Photochem Photobiol B (2004 Dec 2) 77(1-3):39-43** ISSN: 1011-1344

Abstract: Peracetic acid was one of the most commonly used disinfectants on solid surfaces in hospitals or public places. However, peracetic acid is an environmental toxin. Therefore, safer, alternative disinfectants or disinfectant systems should be developed. Because photodynamic virus

inactivation with methylene blue (MB)/light system has proven effective in blood banking, MB was selected as a photosensitizing agent, dengue virus as a model virus for enveloped RNA viruses, and an in-house fabricated narrow bandwidth light system overlapping the absorption spectrum of MB as the light source. Dengue virus was mixed with different concentrations of MB, and illuminated by the narrow bandwidth light system under different illumination distances and times. The amount of dengue virus remaining was evaluated by plaque forming assays. Results showed that the concentration of MB working solution, illumination intensity of light source, illumination distance and time were four key factors affecting efficiency of virus inactivation using the MB/narrow bandwidth light system. Dengue virus could be completely inactivated at 2.5 m in 5 min when MB >/= 1.0 microg/ml. However, when the distance reached 3.0 m, only greater concentrations of MB (2.0 microg/ml) could completely inactivate virus in a reasonably short time (20 min), and smaller concentrations of MB (1.0 microg/ml) could only completely inactivate virus using longer times (25 min). The results of this virus inactivation model indicate that our MB/narrow bandwidth light system provides a powerful, easy way to inactivate dengue viruses.

Anti-herpes simplex virus activities of Eugenia caryophyllus (Spreng.) Bullock & S. G. Harrison and essential oil, eugenol.

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Abstract: In this study, an extract from the flower buds of Eugenia caryophyllus (Spreng.) Bullock & S. G. Harrison and the essential oil, eugenol, were evaluated for their anti-herpes simplex virus properties on standard HSV-1(F), standard HSV-2(G) and ten HSV isolates. The plaque reduction assay showed that HSV-1(F), HSV-2(G), two HSV-1 isolates (2, 30) and four HSV-2 isolates (1, 2, 3, 21) were inhibited by E. caryophyllus. Only HSV-1 isolates 1 and 30 were inhibited by eugenol. Thus, strains or isolates of viruses may affect the range of inhibition. Moreover, particles of HSV standard strains were directly inactivated by E. caryophyllus and eugenol. The total virus yield of HSV standard strains and isolates at 30 h also declined after treatment with E. caryophyllus and eugenol. The E. caryophyllus extract exerted higher antiviral replication on HSV-2(G) than on HSV-1(F). The inhibition of the viral yield of HSV-1 isolates was higher than standard HSV-1(F) and standard HSV-2(G) was also inhibited more than most of the HSV-2 isolates. The anti-HSV activity of eugenol against HSV-1(F) and HSV isolates was stronger than with the E. caryophyllus crude extract. However, the percentage inhibition was more pronounced on HSV-1(F) than on HSV-2(G). Moreover, HSV-1(1) and HSV-2(1, 32) could not replicate when eugenol was included in the assay.

Dr. Tennant's note: eugenol is an active ingredient in clove.