Ebola Virus Disease;
The Scientific Data That is Not Being Discussed

The Concept of Emerging Infectious Disease

While each of us come and go about our daily business, there is a secret hidden war that is occurring all around us. It is a war between humanity, and the various populations of microorganisms all around us.

There are approximately 1,407 organisms (fungi, bacteria, parasites, protozoa and viruses) that can infect humans. Roughly 58% of these are considered to be animal diseases, and most of these have an Old World origin as a result of man's development of agriculture and animal domestication. In addition, there are now some 177 pathogens considered to cause newly emerging or reemerging diseases.

The term *emerging disease* defines an infectious disease that has newly appeared in a population, or is rapidly increasing in incidence or geographic range. During the past 30 years some 41 new infectious organisms or strains have jumped from their animal hosts into humans. This is typified by the period of 2012 to 2014, with the appearance of the Middle East Respiratory Syndrome Coronavirus (MERS-CoV), the lethal Bas-Congo rhabdovirus that causes hemorrhagic fever and kills within a few days, the large Sierra Leone-Liberia- Ebola outbreak, the continuing Kasai Oriental Province outbreaks of human Monkeypox, and cases of African Chikungunya virus in Florida with an epidemic currently underway in Puerto Rico.

One significant emerging infection is the Ebola Virus. The Ebola virus is nothing more than a collection of seven different proteins surrounding a strand of nucleic acid which contains the instructions for making copies of those proteins once the virus enters a human cell. As with all viruses known to science, the Ebola virus borders on the definition of what is considered a living organism.

The disease caused by this virus ; Ebola Virus Disease (EVD), features a narrow possible treatment window, is highly contagious, requires intensive clinical care, and mandates special considerations for patient transport and management to avoid secondary infection. As such, the World Health Organization has mandated the Ebola virus as a *Risk Group 4* Pathogen that requires Biosafety Level -4 (BSL-4) or equivalent laboratory containment.

Scientists who work with high concentrations of the Ebola virus do so only inside special BSL-4 laboratories in which they have complete skin surface and respiratory protection by means of wearing an encapsulating positive-pressure "space suit" with a separate filtered air supply.

Entry into a negative pressure BSL-4 facility is only through a hermetically sealed airlock. Before leaving the BSL-4 facility, the researcher must re-enter the airlock where the outside of the "space suit is sprayed with a mist of strong decontamination solution for several minutes. All equipment entering the BSL-4 area is sterilized with paraformaldehyde gas in another separate air lock before it is removed. All liquid waste is autoclaved to sterility and other materials are double bagged, passed through the airlock and immediately incinerated on-site.

Early Signs and Symptoms of Ebola Virus Infection

Following an average incubation period of 12.7 days (with 4.1% of patients demonstrating incubation periods longer than 21 days), the symptoms of EVD begin with the onset of a non-specific sudden flu-like illness characterized by sudden fever, headaches, and joint, muscle, and abdominal pain with sore throat, chest pain, hiccups, shortness of breath and difficulty in swallowing. Early infected cases may complain of nausea, vomiting, diarrhea and loss of appetite as well. The start of these symptoms is considered to be the start of a patient's infectious potential to other non-infected individuals.
On day 2-3 of symptoms, a Macular skin rash on the face and chest may appear in about 50% of cases and it is likely that this coincides with a burst of viral particles entering the blood from lymph nodes, the onset of systemic inflammation in the small blood vessels, and the start of a progression into multiple organ failure and possible death.

Current Outbreak

Sometime during December 2013, in the tiny village of Meliandou in southern Guinea in Africa, a small child who's name is now lost to history; came in close contact with a Fruit Bat or its blood or feces, and became infected with the Ebola virus. This event started a chain of infection that would soon progress into the largest known epidemic of EVD in virus's history.

Only in March of 2014, did the World Health Organization recognized that a rapidly expanding EVD outbreak was occurring, but it waited until August 8 to declare it a public health emergency of international concern. By mid-September, it was clear that control efforts to control had failed and the epidemic had spread to Liberia and Sierra Leone. These three nations now face enormous challenges in halting the epidemic and dealing with their infected cases and rapidly collapsing infrastructure. In September 2014, the first case of Ebola arrived in the United States in Texas and by mid October, two secondary infections were documented in healthcare workers associated with the care of this U.S. index case.

Despite the expenditure of over 120 billion dollars by the US Federal Government over the last two decades in preparing for outbreaks of emerging infectious disease and bioterrorism, the initial response to the outbreak of Ebola in the United States has been badly designed, and poorly and incompetently implemented.

In their effort to minimize public concern or even panic, the leading health authorities of the United States have made far over reaching statements and assumptions that are not fully supported by the existing scientific literature on the subject. This article serves to outline and clarify some of these concerns by members of the scientific and medical community, and to outline still uncertain facts about the Ebola virus.

Public Statement One:
Fever is an early presenting sign of Ebola virus infection

U.S. health officials have repeatedly emphasized that fever is a reliable sign of individuals with EVD becoming infectious to others around them. Consequently, as a defense against the spread of the virus, the U.S. Government has ordered that passengers arriving from West Africa at five U.S. airports be checked for fever. As such, public health workers are screening the more than 1,000 air travelers who arrive each week in the United States from West Africa. The official assumption about the frequency of fever in Ebola patients has not been challenged publicly, but the "absolute" assumption that Ebola can be spread only when an infected person displays a fever is unsupported.

The largest study of the current outbreak in Liberia, Sierra Leone, and Guinea involving 3,343 confirmed cases of Ebola has found that fever is not a presenting

complaint in all cases of Ebola infection. Sponsored by the World Health Organization and published online in September by the New England Journal of Medicine, the study found that 87.1% of cases of early Ebola infections exhibited fever-but 12.9% did not. In the study, fever was defined as a body temperature of 38 degrees Celsius (100.4 degrees Fahrenheit).

Researchers studying an outbreak in Uganda in late 2000 and early 2001 reported that the commonest symptom of EVD infection was fever, but this only occurred in 85% of the cases. Another study on 24 confirmed cases of Ebola, found fever in only 88% of cases.

It seems that in some clinical cases, Ebola can present without fever, especially during the first initial phase of infection. The absence of fever is not a reliable indication that an individual is not infected**,** and the lack of fever should not be used to assess the level of infectiousness of an infected case to others. The concept is not fully supported by published, peer reviewed scientific data.

New England Journal of Medicine; 371:1481-1495 October 16, 2014.

Public Statement Two;
Airborne transmission of the Ebola virus cannot occur.

Field observations during epidemic human outbreaks of EVD indicate that human-to-human secondary transmission is linked to improper hypodermic needle use, direct contact with Ebola infected tissue or fluid samples, and close unprotected contact with infected patients. However, while it is *presumed* that the Ebola virus infects through either the skin or with contact with mucous membranes, the only two routes of exposure that have been extensively experimentally verified in animal models, are direct injection, droplet exposure, and aerosol inhalation.

While classical epidemiologic evidence indicates that aerosol exposure is not an important means of virus transmission in human-to-human epidemics of EVD, infective Ebola virus particles are present in the oral fluid of infected patients, and experimental studies have verified that Ebola infection can be effectively transmitted by oral or conjunctival droplet exposure in monkey and other animal models.

Formenty P, Leroy EM, Epelboin A, et al. Detection of Ebola virus in oral fluid specimens during outbreaks of Ebola virus hemorrhagic fever in the Republic of Congo. *Clin Infect Dis* 2006;42:1521-1526.

Jaax NK, Davis KJ, Geisbert TJ, et al. Lethal experimental infection of rhesus monkeys with Ebola-Zaire (Mayinga) virus by the oral and conjunctival route of exposure. *Arch Pathol Lab Med* 1996;120:140-155.

Aerosol models of Ebola transmission have been developed in knock-out guinea pigs and monkeys. In addition, mouse-adapted Ebola models of airborne transmission have been developed which show liver damage in all aerosol challenged mice, as well as lung lesions in two of the three strains tested.

Twenhafel NA, Shaia CI, Bunton TE, et al. Experimental aerosolized guinea pig-adapted Zaire ebolavirus (variant: Mayinga) causes lethal pneumonia in guinea pigs. *Vet Pathol* 2014 May 14.

Zumbrun EE, Abdeltawab NF, Bloomfield HA, et al. Development of a murine model for aerosolized ebolavirus infection using a panel of recombinant inbred mice. *Viruses* 2012;4:258-275.

Concern over the possibility of Ebola aerosol transmission first emerged following the 1989 outbreak of the Reston ebolavirus (RESTV) at a primate quarantine facility in Reston, Virginia. At the time, some scientists thought that the infection was aerosol transmitted between monkeys and several animal care workers showed antibody levels indicating they had been exposed. Fortunately this strain of Ebola proved to be non-pathogenic to humans. Nevertheless, the strain was assigned a Level-4 Biosafety mandate by the CDC. This concern was reinforced in 2008 when RESTV was detected in pigs in the Philippines and specific RESTV antibodies were found in pig farmers, confirming their exposure to the virus.

Later experimental challenge studies in 5-week-old pigs, with exposure of animals by the nasal route showed virus replication in internal organs and viral shedding from the nose and mouth in the absence of clinical signs of disease in the infected pigs. Although non-pathogenic to humans, these observations confirm not only that asymptomatic infection of pigs with RESTV occurs, but that affected animals pose a transmission risk to farm, veterinary, and abattoir workers.

Extending these studies further, researchers documented the possibility that pigs are susceptible to infection with the pathogenic Ebola Zaïre virus. This human pathogen was shown to be able to replicate and induce disease in domesticated Landrace Strain pigs, and the infection could be aerosol transmitted to uninfected animals after mucosal membrane exposure to the virus. Replication of Ebola virus to high levels was observed in the respiratory tract, and the infected animals developed severe lung pathology. Shedding from the mouth and nasal mucosa was detected for up to 14 days after infection, and airborne transmission was confirmed in all uninfected pigs housed with the inoculated animals.

Kobinger GP Leung A, Neufeld J, et al. Replication, pathogenicity, shedding, and transmission of Zaire ebolavirus in pigs. *J Infect Dis* 2011;204:200-208.

While it must be emphasized that airborne droplet and particle transmission between humans has not been evident in epidemiological studies of Ebola outbreaks in Africa, aerosol droplet transmission has been demonstrated in animal models. It is therefore irresponsible for government health officials to emphatically state that aerosol transmission does not occur.

The main criteria for the physiology of airborne transmission of the Ebola virus from animals to humans and from humans to humans exist. These include susceptible cells for infection in the upper human airway, the ability of large aerosol particles to penetrate into the upper airway, and the ability of only a few particles of Ebola virus to initiate an infection. Uncertainty remains in the amount of Ebola virus actually shed by infected humans in their respiratory secretions and the amount of respiratory shedding that occurs during different phases of the disease. This is a factor that may actually vary between the different Ebola strains found in nature. From a biomedical viewpoint, aerosol transmission of the Ebola virus from animals to humans and from humans to humans may be possible under certain conditions.

Public Question;

Can animals infected with Ebola show signs of infection and transmit the disease to man?

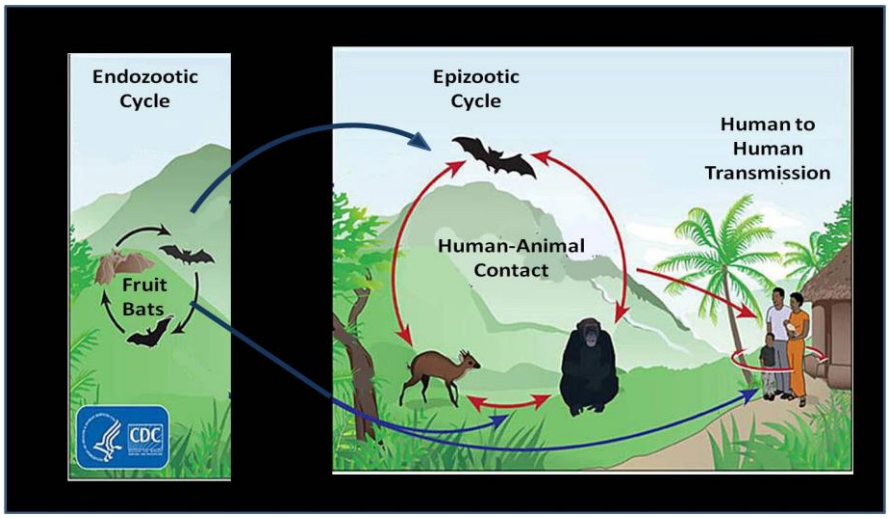
Bats are recognized as reservoir hosts for many viruses that can cross species barriers to infect humans and other mammals. Bats in the suborder Megachiroptera (flying foxes and fruit bats) and the *Pteropodidae* family, have been implicated in Ebola virus outbreaks. Replicating Ebola virus has been recovered from experimentally infected insectivorous bats in the laboratory, but no pathologic lesions were observed, indicating the animals may act as vectors (or carriers) of the virus. In one experiment, the virus was seen in the lining of the lungs of a bat sacrificed on day 8 post-inoculation. In addition, virus was recovered from the feces of a fruit bat on day 21 post-inoculation.

Swanepoel R, Leman PA, Burt FJ, et al. Experimental inoculation of plants and animals with Ebola virus. Emerg *Infect Dis* 1996;2(4).

This is significant, as the presence of Ebola virus implies that respiratory, oral, or guano spread of infection could occur in the confined spaces where bats roost. Isolation of the virus from bat feces suggests the existence of mechanism for Ebola transmission to other animals by skin or mucous membrane contact. It should be noted that human Rabies is not considered to be an aerosol transmitted agent, but human Rabies infections have occurred by the inhalation of dried infected bat guano in caves.

This study spurred further efforts to demonstrate bats as a reservoir host for the *Filoviridae*. Sampling studies conducted between 2001 and 2003 in Gabon and the Republic of the Congo found evidence of asymptomatic infection by Ebola virus in three species of fruit bat, indicating that these animals are likely the reservoir for this deadly virus**.**

Leroy EM, Kumulungui B, Pourrut X, et al.. Fruit bats as reservoirs of Ebola virus. *Nature.* 2005;438:575-576.



Indirect evidence also exists. Between May and November 2007, Ebola reemerged in the Occidental Kasaï province of the Democratic Republic of Congo (DRC), causing 186 deaths. The local African population described a massive annual fruit bat migration by the Lulua River, and these were massively hunted by villagers as a source of protein. Contact tracing demonstrated that the first human victim in this outbreak bought freshly killed bats from hunters to eat.

Leroy EM, Epelboin A, Mondonge V, et al. Human Ebola outbreak resulting from direct exposure to fruit bats in Luebo, Democratic Republic of Congo, 2007. *Vector Borne Zoonotic Dis.* 2009;9:723-738.

There is a disturbing indication that Ebola may be harbored across a much larger geographic range then previously assumed. Antibodies to the Ebola Zaire and Reston viruses were detected in 3.5% of the bat samples from four Districts in Bangladesh. Antibody evidence of the deadly Zaire Ebola virus strain has also been reported in orangutans in East Kalimantan, Borneo, although this requires a confirmatory expedition.

Olival KJ, Islam A, Yu M, et al. Ebola virus antibodies in fruit bats, Bangladesh. *Emerg Infect Dis* 2013;19(2).

Humans and monkeys are end hosts for Ebola virus infection, and while fruit bats appear to be a major natural reservoir, the involvement of other species in Ebola virus transmission remains unclear. Dogs and pigs are so far the only *domestic* animals identified that can be infected with the Ebola virus.

Weingartl HM, Nfon C, Kobinger G. Review of Ebola virus infections in domestic animals. *Dev Biol* (Basel). 2013;135:211-218.

In 2009, a survey in Gabon found an over 30% exposure of the Ebola virus in dogs during the Ebola outbreak in 2001–2002, and as previously noted, pigs in the Philippines have been reported to be infected with the non-human pathogen Ebola Reston virus, suggesting that other interim or amplifying hosts may exist. Ebola infections in dogs appear to be asymptomatic, but it is uncertain if they can shed and transmit the Ebola virus to humans.

Pigs experimentally infected with the lethal Zaire EBOV can develop clinical disease and can transmit lethal Zaire-Ebola virus to naive pigs and monkeys; although the same study failed to demonstrate transmission in that manner between the monkeys.

Kobinger GP Leung A, Neufeld J, et al. Replication, pathogenicity, shedding, and transmission of Zaire ebolavirus in pigs. *J Infect Dis* 2011;204:200-208.

Any pig-human transmission role during the Ebola outbreaks in Africa requires further study. Pig farms in outbreak areas should be considered potential sites of Ebola virus infection, and the attendant risk should be managed.

Public Statement Three;
Quarantine would not be effective

Quarantine, also known as enforced isolation, has been dramatically effective in decreasing the spread of the Ebola virus during its periodic outbreaks. In the field, the establishment of strict quarantine measures to prevent further virus transmission is still a major way to fight the infection. U.S. law permits the enforced individual quarantine of individuals infected with the Ebola virus.

The subject of a National quarantine directed towards travelers from West African Ebola endemic areas to the United States has for some reason, become a topic for much debate. In contrast to recent statements by leading health authorities stating that a National quarantine would not be effective and even counterproductive, other governments often quarantine areas where the disease

is occurring to slow its spread outside of an initial area. The argument against a National quarantine is inexcusable in light of the size of the current West African epidemic. No quarantine will ever be completely effective, but several centuries of experience in infectious disease has proven the value of National quarantines by slowing the entry of disease carrying individuals into a country.

The infected nurse from Texas who boarded a commercial airline flight required the contact tracing of over 800 other individuals. It would not take too many more cases of this nature before the ability of public health authorities to perform contact tracing would be overwhelmed. Therefore, it is essential to try and limit the number of possible Ebola infections entering any country from outside its borders.

Sompayrac, Lauren (2002). *How pathogenic viruses work* (3. print. ed.). Boston: Jones and Bartlett Publishers. p. 87 *and 89*

Alazard-Dany N, Ottmann Terrangle M, Volchkov V (2006). "[Ebola and Marburg viruses: the humans strike back]". *Med Sci (Paris)* (in French) **22** (4): 405–10.

Public Statement Four;
Patients not showing early symptoms of Ebola infection cannot transmit the disease to others.

Perhaps the most uncertainty about Ebola infection concerns the time, duration, and amount of virus that is shed from infected patients, most particularly with respect to aerosol and droplet transmission and the amount of virus shedding that may occur during early infection.

Although the Ebola virus is transmitted by unprotected physical contact with infected persons, little data exists on which specific bodily fluids are infected, the amount of virus in various body fluids, or on the actual risk of shed particle and droplet transmission. To address this problem in 2007, researchers analyzed clinical specimens taken from 26 laboratory-confirmed human cases of Ebola, as well as environmental specimens collected from an Ebola isolation ward. Virus was detected in 16 of 54 clinical specimens, including saliva, stool, semen, breast milk, tears, nasal blood, and skin and mucous membrane swabs; during the acute phase of illness. EBOV was also detected in two of 33 environmental specimens taken from surfaces in the Ebola isolation ward.

Bausch DG, Towner JS, Dowell SF, et al. Assessment of the risk of Ebola virus transmission from bodily fluids and fomites. *J Infect Dis* 2007;196(Suppl 2):S142-S147.

The researchers concluded that human EBOV infection is accompanied by viral shedding in a wide variety of bodily fluids during the acute period of illness.
Little definitive data exists concerning the amount of virus shed during the early phases of Ebola virus infection.

When it is considered that as few as 10 Ebola virus particles can cause human infection, this important question needs to be better clarified, especially using sensitive laboratory techniques and referencing these with the very early signs of disease in infected patients.

Replication of the Ebola virus in the skin

One of the target organs for infection by the Ebola virus is the skin, where the virus invades and replicates inside specialized cells called Langahan's cells.
The presence of infectious Ebola virus in the skin of infected cases and the clinical possibility of transmission via skin shedding was suggested in 1995 during an Ebola outbreak in the Democratic Republic of Congo. During the outbreak, researchers examined the possibility of using skin swabs as a positive alternative diagnostic method for EVD. Testing showed abundant viral proteins and Ebola viral particles in the skin of Ebola patients reaffirming the role of contact transmission in EVD.
The timing of when live viral particles appear in the skin has not been well defined during early infection. Epidemiological studies suggest this is not a factor for early case human-to-human transmission, but further studies need to be done.

Zaki SR, Shieh WJ, Greer PW, et al. A novel immunohistochemical assay for the detection of Ebola virus in skin: implications for diagnosis, spread, and surveillance of Ebola hemorrhagic fever. Commission de Lutte contre les Epidémies à Kikwit. *J Infect Dis* 1999;179 (Suppl 1):S36-S47.

Sstability of the Ebola virus outside the body

The environmental decay of the Ebola virus and its ability to remain infectious when outside the body of a living host is dependent upon a number of factors including exposure to the Ultraviolet Radiation of sunlight, oxidation by the atmosphere and osmotic stress caused by the ambient humidity in the air.

As part of its offensive biological warfare program, the former U.S.S.R. successfully weaponized large quantities of the Marburg filovirus, a close relative of the Ebola virus.. However, Soviet scientists found it difficult to stabilize the aerosol decay rate of concentrated Ebola virus as a small dry particle preparation suitable for offensive aerosol dissemination (K. Alibek, personal communication, 1998).

The inherent high biological decay rate for the Ebola virus in natural aerosols could be delayed however, if surrounded by suitable organic material which may act as a stabilizing agent, such as bat guano, or droplet mucous. Further studies are needed in this area.

Important Unanswered Questions

Current epidemiological data indicates that once a primary human infection develops, classical secondary transmission occurs through contact with an infected person or their bodily fluids. It is agreed that transmission in a medical setting can be especially prevalent without proper precautions by workers, who otherwise are at high risk.

It is assumed that humans infected with EBOV are not shedding infective virus during the incubation period before the onset of high fever, muscle pain, and general malaise. This needs to be better documented in both animal models and human cases, especially in connection with respiratory droplet, urine, feces, and skin viral shedding. A detailed study of the timing and the quantitative amount of viral shedding in human Ebola patients should be undertaken as patient's progress through their disease course. This should include viral quantization in skin, saliva, and respiratory droplets, and the stability of Ebola virus when disseminated in these media. Further studies should include an assessment of the amount of live virus shedding in recovered survivors.

Safe management of Ebola patients and outbreaks.

The Ebola virus is classified as a BSL-4 infection and is required to be contained and handled in a BSL-4 laboratory environment. Realizing the dangers and uncertainties associated with this viral disease, a previous generation of physicians and scientists acted to establish a special U.S. Army military team designed to aeromedically transport Ebola infected patients to a specialized BSL-4 medical treatment facility in the United States.

In 1978, the U.S. Department of Defense created the *Aeromedical Isolation and Special Medical Augmentation Response Team* (AIT-SMART). This was a rapid response unit with worldwide airlift capability designed to safely evacuate and manage contagious patients under high-level biological containment.

The team used a Transit Isolator for patient transport with the interior of the isolator maintained at a pressure negative to the external environment by a high-efficiency particulate air (HEPA) filtered blower. While moving or attending to the isolator, team members wore protective Tyvek suits sealed to provide positive-pressure, and HEPA-filtered *Racal* respirators and hoods

Throughout its existence the AIT-SMART was associated with a BSL-4 Medical Containment Suite (MCS) at the US Army Medical Research Institute for Infectious Diseases (USAMRIID) for ICU-level patient care under full BSL-4 conditions [(51)]. The MCS was built in 1969 and became operational in 1972 and it was the final destination for Ebola or other highly contagious patients transported by the AIT-SMART. The unit's Aircraft Transit Isolator could be attached directly to an access port situated on the external wall of the main USAMRIID building. This allowed movement of the patient into the MCS BSL-4 medical care suite without exposing the patient to the environment.

The ATI-SMART was a well designed self-contained military unit capable of transporting a highly contagious patient using a variety of global USAF rotary-wing and fixed wing assets, while providing maximum microbiological security and critical care nursing.

This unique concept combined the BSL-4 MCS critical care unit with several suites of BSL-4 laboratories staffed by highly experienced researchers in exotic

diseases. USAMRIID provided full clinical and pathology laboratories, a large experimental animal colony with strain mice, Guinea Pig, and non-human primate models, along with scientists and physicians highly experienced in disease assessment, pathogenesis, and experimental vaccine development, and Intensive care physicians and nurses from the Walter Reed National Military Medical Center who were well practiced in providing clinical care under BSL-4 conditions.

Clayton AJ. Containment aircraft transit isolator. *Aviat Space Environ Med*, 1979;50:1067-1072.

Christopher GW, Eitzen EM Jr. Air evacuation under high-level biosafety containment: the aeromedical isolation team. *Emerg Infect Dis* 1999;5:241–246.

Marklund LA. Patient care in a biological safety level-4 (BSL-4) environment. *Crit Care Nurs Clin N Am* 2003;15:245-255.

In 2010, the AIT-SMART was decommissioned and this unified capability was lost. With the dissolution of both the AIT and the BSL-4 patient treatment facility at USAMRIID, the United States under CDC recommendations, became relegated to managing EVD patients under BSL-3 conditions as has previously been done in African outbreaks.

As recently witnessed, a collaborative effort with the CDC has created a "Serious Communicable Disease Unit" at the Emory University Hospital. However this facility operates at BSL-3 and is one of only four such small-bed facilities in the United States**.**

Kroll, D. Should We Be Concerned About American Ebola Patients Coming To Emory Hospital? www.forbes.com., Aug 01, 2014.

For aeromedical transportation, US Air Force's Critical Care Air Transport Teams (CCATTs), are planning to use a Gentex® Patient Isolation Unit (PIU). The PIU is a temporary, single-use, portable structure designed to temporarily isolate a highly infectious patient, but without an AIT-SMART capability. However, the PIU represents only an enhanced patient isolation capability.

In addition, there are no provisions to replace the USAMRIID BSL-4 MCS intensive care unit, although this could be reinstituted with airflow reversion changes.

The response of the CDC to the management of the first Ebola case in Texas was to promote inadequate and inexcusable guidelines that did not provide adequate health worker protection. Proper science, previous experience, and caution, were ignored.

Hundreds of workers in Africa, and now two in the U.S., have caught the disease by using inadequate levels of protection. Yet the CDC (Centers for Disease Control and Prevention) asserts that every hospital in the U.S. can be adequately prepared. This is the same organization that approved a nurse with a potential Ebola exposure to take a commercial flight, who became symptomatic.

During recent Congressional Testimony some congressmen complained that the CDC did not have enough money. But none mentioned that the existing Aeromedical Isolation and Special Medical Augmentation Response Team (AIT-SMART), was dismantled in 2010 and this unified capability with USAMRIID's BSL-4 laboratories staffed by highly experienced researchers in exotic diseases was lost.

Te Ebola virus is classified as a BSL-4 level infective agent. At present, outbreaks in the United States are being managed at only the BSL-3 level. This can be done, but it is dependent on a high level of experience in health care practitioners, as well as adequate personal protective equipment, and a procedural protocol that is based on the side of caution. Sending in a rapid CDC response team from Atlanta is not a substitute for proper protective equipment, negative-pressure isolation rooms, and proper and well practiced staff and decontamination procedures. Neither are "tear sheets" instructing air travelers from epidemic areas to check their temperature.

Even though the United States has had strong infection control procedures and guidelines in place since the emergence of other infectious diseases such as human immunodeficiency virus (HIV) and viral hepatitis, additional research and data collection needs to be undertaken to gain a true consensus on the necessary levels of precautions and best treatment practices for patients infected with hemorrhagic fevers.

As new antiviral treatment approaches are developed it is likely they will have to be administered as soon after patient infection as possible. This highlights the need for a rapid medical response unit able to insert into remote areas for on-site diagnosis and the initiation of projected anti-viral therapy. Follow on actions

would include the isolation transportation of cases to a dedicated BSL-4 patient treatment facility for disease assessment.

In this light, it is reasonable to suggest that the present lack of a dedicated AIT-SMART should be reassessed. This is with respect to its possible upgrading into a unit capable of performing foreign conflict inserts for rapid on-site diagnosis, to quickly initiate on-site antiviral therapy and intensive care, and to conduct a controlled and biosecure patient evacuation to a dedicated BSL-4 level MCS and research facility. In addition we suggest that this team be given the training for the added capability to conduct Field Epidemiological Surveys.

In addition, the current practice of caring for previously BSL-4 classified Ebola patients under BSL-3 conditions should be continuously reassessed with respect to health worker safety in the United States.

Paramount to the management of Ebola cases is a coherent explanation to the citizens of any country by that nation's national health authorities. The citizens of any nation are not stupid. When they see healthcare givers wearing respiratory protection while managing Ebola patients and they are told there is no risk of aerosol transmission; this breeds mistrust in the population.

The best way to confront national fear is by discussing all the facts with transparency, repeated communication, and the truth.

The United States has a sophisticated Heath Care system. With proper Public Health leadership based on firm scientific data, there is no reason to fear a large uncontrolled community outbreak of Ebola Virus Disease in the United States. That is if well formulated rules and guidelines are stringently followed. However, the current Ebola outbreak serves to provide an important warning.

Mankind as a species has doubled its numbers over the last 30 years, and we are spread out over the world at a density unimaginable only a few centuries ago. As a result, a large number of new viruses are entering the human population virtually every day. With modern air travel, a new deadly infectious pathogen is only 24-hours away from its remote lair deep in some jungle to the center of a major modern metropolitan city. Ebola is now in the public mind, but there are other known and unknown viral infections just as bad if not worse, lurking in nature.

The only real defense that we have is to maintain our public health capabilities, ensure that the next generation of public health workers are well trained, and to perform constant worldwide surveillance for new and unusual infectious disease outbreaks. This requires the highest level of Public Health leadership. At the time of this writing, the position of Surgeon General of the United States remains vacant.

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Dr. Hatfill is a medical doctor and a scientist, with a background in military special operations and training in emergency and tropical medicine and pathology. After serving as an overwinter physician in Antarctica, he obtained separate Master's degrees in microbial genetics, medical biochemistry, and hematological pathology with 16 published scientific research papers and over 15 years of clinical experience in Africa.

His past postdoctoral Fellowships include a Clinical Research Scientist position at Oxford University in England, the National Institutes of Health in the Department of Molecular and Cellular Biophysics, and the National Research Council where he studied the Ebola Virus under BSL-4 conditions at the United States Army Institute for Infectious Diseases at Fort Detrick, Maryland.

In 2000, he was certified by the United Nations as a biological weapons inspector for the UNMOVIC commission in Iraq.

He is currently an Adjunct Assistant Professor in two Departments at the George Washington University: School of Medicine and Health Sciences, in Washington D.C., and is the Chairman of the *Asymmetrical Biodiversity Studies and Observation Group* in Kuala Lumpur, Malaysia. He is the Medical Director of a London –based company with extensive contracts in the Middle East and Africa, and operates a remote jungle training facility to test new equipment and to train other scientists to conduct research in high biodiversity areas. Dr. Hatfill is a National Fellow of the *Explorers Club* in New York City and a Board Member of *Doctors for Disaster Preparedness*.