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Barbara Pfeffer Billauer†

“‘It is the beginning of scientific wisdom to recognize that not all questions have answers.’”††

Abstract

Biothreat projections for pandemic and bioterrorist planning are generated by a straight-forward computational system, rather than scientific evidence. The mathematical-modeling method typically uses three variables involved in disease transmission with values culled from assorted past epidemics, often assuming the absolute worst-case scenario. However, other important metrics, such as biological plausibility, relevant historical data, and modern medical practice, are often ignored. Past pandemic projections for avian flu, smallpox, anthrax, swine flu, and Ebola grotesquely overestimated the disease’s incidence and mortality illustrating deficiencies in current models, and in some cases, generating adverse health consequences more severe than any realistic epidemic. These catastrophic predictions generate fear and hysteria, thereby establish the predicate for maximum federal funding, impeding rational safeguards to our national security by inappropriately diverting resources from necessary, but less glamorous, quotidian public-health concerns.

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†† DAVID A. FREEDMAN, STATISTICAL MODELS AND CAUSAL INFERENCE 60 (2010).
Rather than modeling actual weapons of mass destruction, in essence, the exercise artificially creates weapons of mass hysteria (“WMH”).

Allocating billions of dollars to futile biodefense endeavors—or WMH—is clearly wasteful. It also breeds suspicion about the government’s ability to accurately predict and prepare for biothreats, damaging citizens’ confidence in governmental anti-terrorism programs and in homeland-security preparedness. Consistently overestimating epidemic risks suggests a systemic failure in methodology, highlighting the need for a scientific-statecraft paradigm to properly assess these risks.

For fear of being underprepared, the government continues to rely on worst-case scenario models. Trying to prove these models are flawed is fraught with political danger. What if these worst-case scenarios are in fact correct? Ignoring the possibility of calamity is a consideration no politician wishes to contemplate, even if overzealous projections ultimately cause more harm than more realistic and more prudent risk estimates.

This article demonstrates that worst-case scenario mathematical models for projection purposes are invalid, dangerous, and possibly motivated by their proponents’ personal interests by using past smallpox epidemics as a case-study. Further, this work discusses historical facts that seriously undermine the assumptions used in homeland-security planning for smallpox and raises considerations that may be useful in planning for other biothreats.

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I. Introduction

After the 2001 and 2002 anthrax scares, official American bioterror priority lists were updated. The Centers for Disease Control and Prevention ("CDC") in Atlanta, Georgia had refined its original list of high-profile bioweapons based on the conclusions of Donald Henderson and his Working Group on Civilian Biodefense, put forth in 1999. The group identified six agents as particularly fearsome, and prominently featured smallpox as the worst of the Class A bioterrorist agents. By 2001 the CDC dramatically

1. Cyril Wecht, Forensic Aspects of Chemical and Biological Terrorism 426 (2004) (explaining that the CDC divides bioweapons into three categories; six agents, including smallpox, are listed in the highest-priority category).

2. An original list of possible bioterrorist agents was developed by the World Health Organization in 1970.


4. Id.; see also N.Y. State Dep’t Health, Smallpox Questions and Answers: The Disease and the Vaccine, N.Y. State (Apr. 2003), https://www.health.ny.gov/publications/7004/ ("Smallpox is classified as a
augmented the list. Among the new diseases listed was Ebola, now also considered a Class A bioterrorist threat due to its perceived high case-fatality rate, ease of spread, and contagion.

To deal with the expected biothreats, states enacted myriad quarantine regulations, academic institutions composed manuals for disaster planning, public-health lawyers drafted model laws, government agencies developed contingency plans, Presidents Bush and Obama signed key Executive Orders, and millions of dollars were allocated to

Category A agent by the Centers for Disease Control and Prevention. Category A agents are believed to pose the greatest potential threat for adverse public health impact and have a moderate to high potential for large-scale dissemination. Other Category A agents are anthrax, plague, botulism, tularemia, and viral hemorrhagic fevers. BAYLOR COLL. MED., Potential Bioterrorism Agents, https://www.bcm.edu/departments/molecular-virology-and-microbiology/emerging-infections-and-biodefense/potential-bioterrorism-agents (explaining that Class A bioterrorist agents are the agents that the CDC believes pose the highest risk to national security and have the most potential to cause public hysteria).


6. BAYLOR COLL. MED., supra note 4 (noting that Ebola is a class A bioterrorist threat); Paolo Francesconi et al., Ebola Hemorrhagic Fever Transmission and Risk Factors of Contacts, Uganda, 9 EMERGING INFECTIOUS DISEASES 1430, 1436 (2003) (explaining that the original case-fatality rates were one hundred percent: “the higher death rate observed among primary and secondary case-patients (100%), in contrast with . . . the most recent patients (70.6%), could be explained by the treatment provided in the hospital, though this treatment was mainly supportive.”).

7. P. B. Jahrling et al., Lethal infection of primates with variola virus as a model for human smallpox, WORLD HEALTH ORG. (2017), http://www.who.int/csr/disease/smallpox/lethalinfection/en/ (“Smallpox virus is acknowledged to pose a significant threat as a bioterrorist weapon because of its contagiousness and the absence of effective antiviral drugs.”).


12. Jared P. Cole, CONG. RESEARCH SERV., RL33201, FEDERAL AND STATE QUARANTINE AND ISOLATION AUTHORITY 2 (2014); Executive Order 13295: Revised List of Quarantinable
academia to develop homeland-security programs. In 2005, the CDC proposed a regulation authorizing it to impound planes or ships with passengers who appeared ill with summary provisions allowing for up to a week’s detention. The regulation never passed, but the sentiment of the country was clear: better to restrict travel for anyone who might transmit a contagious disease than for a single American to succumb to one. By 2007, the United States operated more than 1356 level-three biosafety labs and fifteen level-four biosafety labs, presumably to devise treatment and preventive measures for bioterrorist and pandemic threats. To date, the results of their research are murky at best.

It appears, however, that the bioterror-prevention tactics were for naught. In the ensuing decade, no bioterrorist-mediated event materialized and none appear on the event-horizon. Mother Nature, however, filled the gap. In 2014, she unleashed an epidemic of unseen proportions in recent times: the Ebola virus swept through Africa. In March, the World Health Organization (“WHO”) reported its outbreak in four districts of Guinea with eighty-six suspected cases and fifty-nine deaths. More cases were...
reported in neighboring Liberia and Sierra Leone. Not long afterwards, Liberia declared an international state of emergency, generating fear that cascaded out of control; apocalyptic projections predicted 1.4 million cases by January, 2015. The WHO re-sounded the pandemic warning on August 8, 2014, allocating billions of dollars and deploying thousands of personnel to infection zones as top-tier health officials around the world made potentially risky decisions.

The United States mounted an unprecedented response effort in terms of funding, allocation of person-power, and deployment of healthcare workers and auxiliary personnel. A White House press release announced that the U.S. would be “leveraging[ing] the unique capabilities of the U.S. military and broader uniformed services to help bring the epidemic under control,” which “would entail command and control, logistics expertise, training, engineering support and 350 million dollars.” The WHO...


24. Id. (“U.S. Africa Command will . . . facilitate coordination with U.S. government and international relief efforts . . . which will involve an estimated 3,000 U.S. forces . . . [and] . . . transportation of equipment, supplies and personnel . . . . Command engineers will build additional Ebola Treatment Units in affected areas, and the U.S. Government will help recruit and organize medical personnel to staff them. Additionally, the Command will . . . train up to 500 health care providers per week, enabling healthcare workers to safely provide direct medical care to patients. The United States Public Health Service Commissioned Corps is preparing to deploy 65 Commissioned Corps officers to Liberia to manage and staff a
allocated more aid in October, after the CDC published its September Mortality and Morbidity Report ("MMWR") that estimated approximately eight-thousand cases would occur in Liberia and Sierra Leone by September 30, noting the figure could be 21,000 when correcting for underreporting. To account for underreporting, the MMWR used a correction factor of 2.5, asserting that "for every case reported and recorded in publicly available case counts, an additional 1.5 cases are not recorded." The CDC concluded that "without additional interventions or changes in community behavior . . . by January 20, 2015, there will be a total of approximately . . . 1.4 million [cases]." The CDC used a ninety percent case-fatality rate estimate for the Zaire subtype of Ebola and predicted there would be 1.26 million deaths; the WHO used an estimated case-fatality rate of seventy percent, and predicted approximately one million deaths.

More than a year later, after WHO declared the epidemic over, a grand total of 28,638 cases and 11,315 deaths were reported worldwide. Of these fatalities, eighty-seven percent were in Liberia and Sierra Leone.

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25. Press Release, White House Office of the Press Secretary, FACT SHEET: The U.S. Response to the Ebola Epidemic in West Africa (Oct. 6, 2014), available at https://obamawhitehouse.archives.gov/the-press-office/2014/10/06/fact-sheet-us-response-ebola-epidemic-west-africa ("The United States already has committed more than $350 million toward fighting the outbreak in West Africa, including more than $111 million in humanitarian aid, and the Department of Defense (DoD) is prepared to devote more than $1 billion to the whole-of-government Ebola response effort . . . . U.S. actions have galvanized millions of dollars in international funding and in-kind support.").

26. Id.

27. Id. (noting 550,000 of the fatalities would be in Liberia and Sierra Leone).


31. Id.
Sierra Leone, it barely topped thirty-five percent, similar to the case-fatality rates for hantavirus and Legionnaires’ disease.\(^{33}\)

The press and public-health experts criticized WHO for its inadequate response efforts,\(^{34}\) yet the projection-versus-reality mismatch and resultant hysteria was never addressed. It appears that the CDC’s and WHO’s estimation methodologies were flawed due to a lack of critical information and rapidly changing parameters on the ground.\(^{35}\) Had this been the only incident of gross over-projection, one might attribute the mishap to an anomaly. However, the consistent pattern of over-projection calls into question the efficacy of mathematical modeling as a pandemic-projection tool. We saw similar overestimates for swine flu,\(^{36}\) avian flu,\(^{37}\) anthrax, and Middle East Respiratory Syndrome (“MERS”).\(^{38}\) An all-but-certain and omnipresent bioterrorist-attack clarion call emerged:

[a]s the 20th century drew to a close, most biological[...]defense professionals, both military and civilian, were in agreement that the

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33. Id.


35. Josh Michaud & Jennifer Kates, Measuring the Impact of Ebola: Will it Reach 1.4 Million?, KAISER FOUN.D.N (Sept. 29, 2014), http://kff.org/global-health-policy/perspective/measuring-the-impact-of-ebola-will-it-reach-1-4-million/ (“The model...which assumes no change in the trajectory of the epidemic...translates...to an equivalent [and absurd] rate in the U.S. population...[of] almost 45 million people nationally, and more than 90,000 in Washington, DC alone.”); see Donald G. McNeil, Jr., Fewer Ebola Case Go Unreported Than Thought, Study Finds, N.Y. TIMES (Dec. 16, 2014), https://www.nytimes.com/2014/12/16/science/fewer-ebola-cases-go-unreported-than-thought-study-finds.html (“[R]esearchers said they had too little data to predict how many West Africans could eventually be infected, but enough to show that the dire predictions were inaccurate...Epidemics surge and wane...so extrapolating an estimate from one month’s data to an entire outbreak was ‘not legitimate.’”).


38. CTRS. DISEASE CONTROL & PREVENTION, Revised Technical Instructions for Panel Physicians related to Severe Acute Respiratory Syndromes based on Executive Order 13674 (last updated Aug. 21, 2014), https://www.cdc.gov/immigrantrefugeehealth/exams/ti-panel/revised-technical-instructions-panel-physicians-respiratory-syndromes-executive-order-13674.html (stating that Executive Order 13674, which was signed on July 31, 2014, shortly after the Ebola epidemic began “gives clear legal authority...to make MERS a communicable disease of public health significance.” (emphasis added)).
probability of a bioterrorist event occurring the United States was not a matter of *if*, but *when* . . . . *If* is now behind us, and we are left with the burning issues of ‘*who, where, when next, and why*.’

These prophecies were based on mathematics and statistics, not science. For example, one commentator claimed that influenza pandemics “typically occurred every 10-50 years throughout recorded history,” and as the last pandemic was in 1957, “we are ‘due’ for one.” In response, millions of dollars were allocated for flu vaccines—vaccines that later went unused.

Similar hyperbole emanated from the now largely-discredited Dark Winter (“DW”) war game, a pandemic-projection tool ostensibly designed to sensitize the government to the threat of a smallpox terrorist attack and the country’s lack of preparation. Some in the media and scientific community heavily criticized DW as a fable, questioning its basic assumptions, but DW nonetheless successfully launched the government-spending juggernaut. In fact, shortly after presentation and publication of...
the fable, which projected three million cases of smallpox and one million deaths in six weeks, Congress enacted Operation BioShield, a government program allocating billions of dollars to address projected risks from this hypothetical cataclysm.\textsuperscript{46} Allocations to biodefense over the past fifteen years have burgeoned substantially, now reaching about eighty billion dollars.\textsuperscript{47}

As a further consequence, smallpox vaccines are still routinely administered to soldiers.\textsuperscript{48} Even though the threat from Iraq’s weapons-of-mass-destruction program has been eliminated—along with Saddam. Many in the military oppose vaccination,\textsuperscript{49} citing to the fact that soldiers and their spouses have experienced serious adverse reactions.\textsuperscript{50} Some allege that vaccine, a shortage resulting from limited vaccine-manufacturing capacity, which was directed to other pandemic scares).

46. Sherwood Ross, America the Beautiful’s Germ Warfare Rash, MWC NEWS (Nov. 9, 2013), http://mwcnwes.net/focus/politics/33240-germ-warfare.html (explaining that Project BioShield was enacted in 2004 and allocated five billion dollars to development and production of vaccines—which ordinarily take months to produce, and are mired in FDA regulations—to be used in the event of a terrorist attack, and that intelligence reports on terrorist activities are ineffective and have been wrong or were misinterpreted); Peptide Therapeutics Group, OraVax, Inc. Awarded Contract to Develop, Manufacture and Manage A National Stockpile of Smallpox Vaccine for Civil Defense, PRNEWS.COM (Sept. 20, 2000), http://www.prnewswire.com/news-releases/oravax-inc-awarded-contract-to-develop-manufacture-and-manage-a-national-stockpile-of-smallpox-vaccine-for-civil-defense-73342157.html (noting that one of the first allocations for bioterrorism-defense funding, which was made on the recommendations of the Working Group on Civilian Biodefense, was a $343 million contract with OraVax, who would be responsible for “production in large-scale cell cultures, using modern methods for the manufacture of live-viral vaccines.”).


48. DAVID KOPLOW, SMALLPOX: THE FIGHT TO ERADICATE A GLOBAL SCOURGE 27 (2004) (explaining that the Department of Defense contracted “with a Maryland firm called BioReliance to deliver 300,000 doses of an improved vaccine for $22.4 million (approximately $70 per dose). This inventory, to be administered . . . to service members deployed to locations of greatest threat.”).


50. \textit{Id.}; Samuel A. Bozette et al., A Model for a Smallpox-Vaccination Policy, 348 NEW ENGL. J. MED. 416, 417 (2003); Barbara P. Billauer, The Specter of Bioterrorism: Real
one or more members of the Working Group on Biodefense helped institutionalize the government’s initial bioterror list, deriving financial or other benefits from it, such as lucrative consulting contracts. Lamentable as this may be, some perversely give it a positive spin:

Polemists in the public- and health field... argue that the furor generated by fears of bioterrorism has created a valuable opportunity to secure funding for notoriously underfunded public-health systems... In this way, the threat of bioterrorism may be harnessed to create historic opportunities for the diversion of federal spending away from military defenses towards strengthening broadband public-health capabilities.

One wonders, however, whether fraudulent resource allocation can ever be proper.

Despite alarmist overfunding, when Ebola struck, the government ignored its pandemic-planning initiatives, including its successful past experience with severe acute respiratory syndrome (“SARS”). During the SARS epidemic, the Department of Health issued and enforced travel advisories. No one who was infected was allowed to travel. But when Ebola struck, the State Department rejected calls for restricting travel from West Africa, and sick citizens returned to the United States, infecting others. Perhaps not surprisingly, two new Ebola cases developed on American soil. This situation reignited debates about quarantine and isolation that had been resolved in prior pandemic-response initiatives—or


52. Toyin Ajayi, Smallpox and Bioterrorism, 3 STAN. J. INT’L RELATIONS 1, 5 (2002).

53. See CTRS. DISEASE CONTROL & PREVENTION, Remembering SARS: A Deadly Puzzle and the Efforts to Solve It, https://www.cdc.gov/about/history/sars/feature.htm (last updated Apr. 26, 2013) (showing that during the 2003 outbreak of severe acute respiratory syndrome (SARS), U.S. patients were isolated until they were no longer infectious).


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so we thought.\textsuperscript{57} And, fifteen years into government allocations and Biosafety Labs directed to produce antidotes, no vaccine or drug or antiserum was available to address Ebola. Only by the close of the epidemic did we see commercial ventures beginning to produce antidotes.

The significance of these issues from a national-security perspective is manifold. Faulty threat assessments can result in misallocating resources and diverting attention from real threats, and illusory bio-threats may desensitize the population to future warnings. These factors may help explain why returning Ebola-exposed Americans refused to abide by isolation or quarantine orders.\textsuperscript{58} In the case of Ebola, further transmission in the United States was limited, but we may not be so lucky the next time.

As national security becomes an ever-increasing concern,\textsuperscript{59} prioritization of spending and efficient risk communication become paramount concerns. Wasted money and “crying wolf” are as detrimental to the country’s security as are insufficient resource allocation, inefficient use of resources and poor risk communication.\textsuperscript{60} It is therefore important to institute a “scientific-statecraft” paradigm in which science-based policy helps effectuate national-security objectives. This will allow the country to responsibly reevaluate response methodologies before the next outbreak using valid and reliable scientific evidence, rather than relying on sterile and hysteria-mongering mathematical projections.

The purpose of this article is to demonstrate the differences and deficiencies between abstract mathematical modeling—especially the mix-and-match, worst-case-scenario approach used in DW—and realities of real-life epidemics using our experiences with smallpox for illustration. This review demonstrates the futility of relying on pure mathematical modeling tools for pandemic projection and planning without real-time upgrades and integration of biological and physical constraints—including data regarding diseases’ particle-transport properties and weather conditions,\textsuperscript{61} which of course, can never be accurately predicted.\textsuperscript{62}

\textsuperscript{57} Id.


\textsuperscript{60} See Mutsvara, \textit{supra} note 34.


\textsuperscript{62} Henderson et al., \textit{supra} note 3, at 2127 (noting the preference of the virus for low humidity and cold).
II. Background

Gloom and doom scenarios about terrorists using smallpox as a bioweapon abound. These scenarios come from politicians and reputable scientists alike, and prestigious journals publish them. For example, one eminent author writes, alarmingly, that:

[t]he former Soviet Union developed variola virus, which causes smallpox, for use as a biologic weapon, and supplies may have fallen into other hands. As Lev Sandakhchiyev, the director of Russia’s Vektor Institute, has warned, “All you need is a sick fanatic to get to a populated place.” U.S. experts agree.

The author does not tell us that this pronouncement is based on information from the now largely-discredited Soviet defector Ken Alibek, also known as Colonel Kanat Zhon Alibekov, whose financial conflicts of interest should have barred him from publishing in any reputable scientific journal.

Here’s another example:

[t]he potential of smallpox as a biological weapon is most dramatically illustrated by two European smallpox outbreaks in the 1970s. The first occurred in Meschede, Germany in 1970(-1). This outbreak illustrates that smallpox virus in an aerosol suspension can spread widely and infect at very low doses. Another outbreak occurred in Yugoslavia in February 1972(-1). Despite routine vaccination in Yugoslavia, the first case in the 1972 outbreak resulted


67. Id. (“His most sensational research findings . . . have not withstood peer review by scientific specialists. His promotion of nonprescription pills—sold in his name over the Internet and claiming to bolster the immune system—was ridiculed by some scientists. He resigned as executive director of a Virginia university’s biodefense center 10 months ago while facing internal strife over his stewardship . . . . By his count, Alibek has won about $28 million in federal grants or contracts.”).
in 11 others; those 11, on average, each infected 13 more. Other outbreaks in Europe from 1958 on showed that such explosive spread was not unusual during the seasonal period of high transmission . . . . One can only speculate on the probable rapidity of spread of the smallpox virus in a population where no one younger than 25 years of age has ever been vaccinated and older persons have little remaining residual immunity.68

We are not told, however, that the two epidemics referred to in this quote do not support claims that smallpox outbreaks typically lead to mass casualty scenarios. Could an unknowing reader possibly guess that these two epidemics together produced a total of only 195 cases?69 And here’s another:

[s]milkpox is the ultimate weapon of mass destruction. It has killed more people throughout history than any other infectious disease, including the bubonic plague. The U.S. population grows more vulnerable to the potential ravages of its effects as time goes by.70

The CDC officially addressed the issue in 1999, when a meeting of experts found that “of all the potential biological weapons identified, smallpox was unanimously determined to pose the greatest threat to the United States.”71 This fear ostensibly stemmed from difficulties inherent in diagnosis and rapidly containing a disease most physicians had never seen.72 The CDC disregarded contrary views—even when voiced by eminent scientists.73 For example, they failed to acknowledge that one model required that before the risks associated with the smallpox vaccine could be justified, a smallpox bio-agent would need to be released at ten airports simultaneously.74 The hysteria spiraled: the Bush administration even

69. See infra Part V.E, V.F.
70. Ajayi, supra note 52.
71. Id.
72. Id.
73. See, e.g., Jonathan Tucker, Historical Trends Related to Bioterrorism: An Empirical Analysis, 5 J. EMERGING INFECTIOUS DISEASES 498, 503 (1999) (“Few terrorist groups possess the scientific-technical resources required for the successful large-scale release of a biological agent.”); Martin Eichner & Klaus Dietz, Transmission Potential of Smallpox: Estimates Based on Detailed Data From an Outbreak, 158 AM. J. EPIDEMIOLOGY 110, 116 (“Fenner et al. who already discussed a possible bioterrorist attack with variola virus in 1988 concluded, ‘the risk of any such act leading to the reestablishment of endemic smallpox should not be exaggerated.’”).
74. Bozette et al., supra note 50, at 419.
ignored the CDC’s recommendation against mass smallpox vaccination and began buying up existing stores and contracting for more.\(^75\)

So starts the saga of the modern terrorization of smallpox, resulting in the American government’s initial procurement of over 300 million vaccine doses.\(^76\) The original order was more than enough for one dose for every citizen—including the fifty million for whom administration of the vaccine was medically contraindicated due to pre-existing or immunosuppressive conditions,\(^77\) or because they were taking drugs that would make them more susceptible to known adverse events associated with the vaccine,\(^78\) or other ethical considerations.\(^79\) The cost: roughly two billion dollars.\(^80\) Vaccine hoarding continues—even in the face of negative data, and the cost keeps escalating. The goal is, and was, to protect all citizens by mass

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79. Would the eleven million illegal aliens be included in any vaccination campaign? Would the 2.2 million prisoners be administered the vaccine, which by definition would be experimental? IRB regulations prohibit experimental drug use on prisoners, leaving this a vexing bioethical problem. The data are silent on these questions.

vaccination. However, the scientific support is wanting: adequate scientific support from public-health experts establishing that mass vaccination would be the best mechanism to protect the public did not (and does not) exist.

III. Are Models Beautiful?

A. Re-Analysis of Mathematical Modeling of Disease Projections

So, what would a smallpox outbreak look like? That question has spawned a new growth industry since September 11, 2001—the vaccine model industry. At least six United States research groups are building epidemic computer models, enabling them not only to project its possible course under different scenarios, but also to test the effect of vaccination and quarantine strategies. Many of these models are still in development and their results are contradictory, but none are as gloomy as DW, the hypothetical fantasy war game discussed earlier, which projected a runaway smallpox epidemic. DW’s assumptions and its projected results have since been debunked, although U.S. policy, as well as those of other countries, still rely on it.

Conventional mathematical models identify key parameters involved in epidemic transmission, and selected variables are plugged into the program. The computer then spits out risk estimates without a person ever setting foot in a quarantined town or village. In the words of one expert:

81. CDC Update, supra note 75.
83. See Chris Barrett et. al, If Smallpox Strikes Portland, 292 SCIENTIFIC AMERICAN 56 (2005) (explaining that epidemiologists use mathematical models, which are straight-forward computational systems, to study the spread of infectious diseases.).
86. See JOHNS HOPKINS CTR. FOR CIVILIAN BIODEFENSE ET AL., DARK WINTER: BIOTERRORISM EXERCISE (2001) (hereinafter Dark Winter or DW).
87. Enserink, supra note 85, at 1593.
89. Michaud & Kates, supra note 35.
controversy exists regarding the best method of protecting the public against the potential release of smallpox as a biological weapon. Infectious disease modeling plays an important role in this dialog, and . . . is critical to producing appropriate predictive models and understanding which controls will work best under varying conditions.90

According to one public-health expert, “[m]odeling smallpox poses even more unusual challenges to assure that proper biological, social and spatial factors affecting the spread are accounted for.”91 Many smallpox projections the “gloomers and doomers” espouse used cherry-picked variables from different epidemics strung together in one mishmashed model,92 thereby creating artificial worst-case scenarios.93 In other words, the data culled for each parameter were selected from different epidemics and mixed and matched to artificially create one biologically implausible model. This method is programmed to produce the most extreme and catastrophic calculations possible,94 generating fear and hysteria that fed the cry for maximum federal funding.95

I argue that misused modeling was the root cause of the Ebola overestimates. In estimating the Ebola impact, models relied on one parameter—the reproductive rate, which at the time was “doubling every 24 days in Liberia and every 30 days in Sierra Leone.”96 The model assumed that rate was invariable, i.e., it would be constant throughout the epidemic.

90. Donald K. Milton, What was the primary mode of smallpox transmission? Implications for biodefense, 2 FRONTIERs in CELLULAR & INFECTION MICROBIOLOGY (2012) (internal citations omitted).
92. See Dark Winter, supra note 86.
93. See generally Kaplan et al., supra note 84.
94. Milton, supra note 90 (“The rapidity with which smallpox would spread in a developed nation is not known and is a major source of uncertainty in models used for public health planning.”) (citations omitted); see Barret et al., supra note 94, at 56 (A more sophisticated modeling method allows for variable cost estimate projections, i.e., the modelers can alter the model to reflect a mild, moderate, or severe epidemic—arbitrarily determined based on the lowest, highest and midrange estimates mathematically possible, but again without regard to biologic plausibility. A third and even more sophisticated model considers the likelihood and length of contact between individuals. This method, produced at Los Alamos National Laboratory by Stephen Eubank, more accurately captures replication and transmission rates by simulating the behavior of individuals in a community. It claims to have data on each of the 1.6 million people in Portland, Oregon and their interaction patterns.).
95. KING, supra note 13, at 435.
96. Michaud & Kates, supra note 35.
In actuality, the reproduction rate was not invariable; it later decreased, as it did in every epidemic I reviewed.97 To properly evaluate mathematical modeling, it is necessary to properly identify parameters involved in spread. The critical parameters are the number of cases each person infects (“reproductive number”), the number of generations over which the infection is transmissible (“transmission rate”), and the case-fatality rate—of the people infected, the number who died.98 Other factors also influence spread, although they are generally excluded for want of accurate measurements.99 After the variables are identified, appropriate data are “plugged into” the model. If the model, even if using appropriate parameters, uses inappropriate data, the projections will be flawed.100

B. Re-Modeling Dark Winter (DW)

As noted, the inferno that fueled the smallpox hysteria and continues to keep it smoldering is the Dark Winter (“DW”) war-game.101 In DW, an illustrative group of governmental and media guinea pigs—some of whom appear to have been scientifically illiterate—were exposed to an imaginary fable loosely based on an amalgam of worst case data from several past epidemics.102

The exercise took place on June 22 and 23, 2001, but the game forecasted that the hypothetical outbreak was to occur about a year and a


98. Milton, supra note 90; See also Pourbohloul et al., supra note 82, at 1250.

99. See, e.g., Milton, supra note 90, at 4 (stating that particle size, weather conditions, proximity and duration of exposure between patient and others were not reported).

100. The gloom and doom scenarios have even infected high school students, who cite these statistics with wild abandon. See., e.g., JOE FETSC, SMALLPOX MARTYR BIO-TERRORISM SCENARIO MODELING COMPUTER SYSTEMS LAB (Jan. 27, 2010), available at https://www.tjhsst.edu/~rlatimer/techlab10/Per5/SecondQuarter/FetschPaper Q2-10.pdf (“[S]mallpox has a fatality rate between 30 and 40 percent and spreads like wildfire both locally and globally, traveling around the world in a month because of the 2 week incubation period in which no symptoms are shown from the infected person, as they travel around, moving to uninfected cities or healthy sections of a population before the sudden outbreak catches them by surprise.”).

101. See generally Dark Winter, supra note 86.

102. See Reuven Ben-Shalom, The Tragedy of Scientific Ignorance: Shirking scientific research and failing to implement methodologies derived from it leads to mediocrity, JERUSALEM POST, at 19 (Jan. 26, 2017), http://www.jpost.com/Opinion/The-tragedy-of-scientific-ignorance-47972 (arguing that the media is scientifically illiterate).
half hence, in December, 2002. The locales were selected, ostensibly, because normally weather conditions at that time would have been expected to be conducive to spread. At the outset, the participants were primed with the claim that “of all potential biological weapons, smallpox is historically the most ominous and feared.”

DW’s smallpox outbreak begins with the emergence of twenty index cases in Oklahoma City on December 1, 2002. By December 9, the fable forecasts that fifty-three cases were diagnosed across three non-contiguous states. At the end of six weeks, three million people were affected and one million were dead. The game does not explain how or where the cases were infected, other than to state vaguely that some imaginary aerosolized biological weapon had caused the infections. These projections were modeled on at least ten faulty assumptions, eight of which are mentioned here. All ten are then discussed in greater depth below.

First, the projections assumed that the initial attack was from weaponized smallpox, similar to what the former Soviet Union would have developed in a secret bioweapons program. However, to the best of our knowledge no such weapon exists, nor is it even realistically or scientifically feasible. Even now, fifteen years later, it is clear that DW was not based on what any hostile country actually had or currently has, but on what DW’s creators believed such countries might have had—even though such beliefs lacked evidence.

103. See, e.g., Oklahoma Ice Storms, 2000-2007, OKLAHOMAN (Jan. 25, 2009, 12:00 AM), http://newsook.com/article/3340695 (documenting weather that would have theoretically scuttled the effectiveness of any attack); see infra note 213, at 41.

104. Tara O’Toole et al., Shining Light on “Dark Winter” 34 CLINICAL INFECTIOUS DISEASES 972, 972; see also Dark Winter, supra note 92.

105. See Dark Winter, supra note 92.


107. Id.

108. See infra, Section VI.A.7. The belief that smallpox can be weaponized is predicated on the idea that smallpox can be transmitted in an airborne fashion, which, though theoretically possible, practically never occurs (if it can occur at all). CTRS. DISEASE CONTROL & PREVENTION, Transmission: How Does Smallpox Spread? (June 7, 2016), https://www.cdc.gov/smallpox/transmission/index.html (stating that airborne transmission of smallpox has “rarely” occurred). When it is said to be responsible for an outbreak, its effects have historically been limited to one generation of spread. Duffy, supra note 106.

109. Even Colin Powell’s speech at the United Nations attempting to cajole international support for the US attack on Iraq claimed at most “Iraq had the wherewithal” to stage a smallpox attack, a meaningless if not reckless statement. Transcript of Powell’s U.N. Presentation, CNN (Feb. 5, 2003), http://www.cnn.com/2003/US/02/05/sprj.irq.powell.transcript.05/ (emphasis added).
Second, the program assumes that 228 million United States citizens were highly susceptible to infection.  

Third, the program assumes that each infected person infected ten others, i.e., had a replication rate of ten.  

Fourth, that the epidemic lasted three to four generations. The explicit prediction of three thousand people infected in the first generation and three million four generations later—giving an implicit assumption that the replication rate remained constant over the four generations of transmission.  

Fifth, the scenario states the CDC had twelve million vaccine doses available at the time of the outbreak, and that new vaccine would not be available for at least four weeks; the incorrect assumption that the United States would have a dearth of available vaccine was probably the most explosive factor and induced the most hysteria as reported in the fable, and as apparently was experienced by the participants. In reality, the CDC actually had fifteen million doses on hand in December 2002 and the Department of Defense ("DOD") coincidently found another seventy-five to ninety million a few months later. Finally, shortly after the exercise Anthony Fauci of the National Institute of Health determined that dilution of the vaccine is effective at a 1:5 or 1:10, generating up to 150 million doses using the CDC’s then available stockpile and another 375 million using DOD’s supply.  

The sixth assumption was that there would be a delay in diagnosis. The game’s authors state that the United States is

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110. Duffy, supra note 106. 
111. Id. With a replication rate of ten, there would have to be three hundred index cases, not twenty as the scenario states, or else the epidemic would have had to run five or six generations—virtually unheard of in the last century—rather than four, as the program postulates. In essence, any real event associated with the DW data would indicate that it is presented beginning at the second generation, not the first as represented. 
112. Id. 
113. O’Toole et al., supra note 104. 
114. Larry Kerr, Panel III—Partnerships Against Bioterrorism, NA’T’L A.C.A.D. PRESS (2005), https://www.nap.edu/read/11300/chapter/6; see also infra, Section V.B. Available research disclosed that as of 1962, dilution was effective at a titer of 1:30. It appears no one even researched the possibility before the war game was developed or presented, nor even by American scientists afterwards. See Douglas & Edgar, infra note 248, at 614; see also, Smallpox in Bradford, Medical Memoranda, Smallpox Vaccination in a University and Economy in Use of Vaccine, 1 B. MED. J. 614, 614 (Mar. 3, 1962) [hereinafter Vaccination in a University]. 
a highly mobile society. By the time the first victims are diagnosed with smallpox . . . the disease will already have begun spreading to a second generation of victims. Some of the initial victims . . . will have travelled [sic] to other cities by that time. Since few American doctors have ever seen a case of smallpox, and since the initial symptoms resemble flu, diagnosis is liable to be slow.\textsuperscript{116}

The seventh faulty assumption was that the case-fatality rate would be thirty percent; in the last century’s reality, the worst-case case-fatality rates for smallpox range from fifteen to twenty percent.\textsuperscript{117}

An eighth faulty assumption was that the weather in Oklahoma City on the projected date of the attack would have fostered spread—if anything, the actual weather on the projected day and locale of the attack would have significantly curtailed transmission,\textsuperscript{118} something no planner or terrorist could possibly have forecasted.

\textbf{C. Parameters for Evaluating Disease Transmission}

\textbf{1. Exposure and Attack Rate}

The critical feature in disease causation is adequate exposure to a noxious agent. Whether an individual contracts a disease depends on whether the target organ receives sufficient exposure to the pathogen, or adequate dose,\textsuperscript{119} which explains why brief contact with many diseases usually does not cause illness.\textsuperscript{120} Indeed, exposure to small amounts of smallpox was the preferred means of preventing the disease during the eighteenth and nineteenth centuries.\textsuperscript{121}

Whether exposure is adequate depends on the route, duration, and amount of exposure, for, as Paracelsus said, “the dose makes the poison.”\textsuperscript{122}

\begin{footnotes}

\textsuperscript{116} Duffy, \textit{supra} note 106.

\textsuperscript{117} \textsc{Dan Longo et al.}, \textsc{Harrison’s Textbook of Internal Medicine} 1774 (18th ed. 2011) (indicating that the world-wide, and presumably historical, case-fatality rate was 20 percent).

\textsuperscript{118} \textsc{Oklahoma Ice Storms, supra note 103}.

\textsuperscript{119} Gordon G. Heiner et al., \textit{A Study of Intrafamilial Transmission of Smallpox}, 94 \textsc{Am. J. Epidemiology} 316, 321 (1971) (“[T]he effect of certain differences in exposure patterns was clearly detectable. Contacts who had ‘constant’ exposure, e.g., mothers, other female relatives and young children, showed significantly higher attack rates than persons whose exposure was only ‘daily.’”).

\textsuperscript{120} \textit{Id.} (“Contacts whose duration of exposure was seven days or more showed significantly higher attack rates than persons exposed for less than seven days.”).

\textsuperscript{121} Stefan Riedel, \textsc{Edward Jenner and the history of smallpox and vaccination}, 18 \textsc{Baylor Univ. Med. Ctr. Proceedings} 21, 22 (2005).

\textsuperscript{122} Juliana Anderson, \textit{Paracelsus}, \textsc{Toxicology} (Nov. 12, 2013), http://www.toxipedia.org/display/toxipedia/Paracelsus (explaining that “the dose makes the poison,” means “the amount of a substance the person is exposed to is as important as the substance”).

\end{footnotes}
Exposure is also influenced by the noxious agent’s hardiness, its method of spread, and whether environmental conditions foster the agent’s proliferation. Because exposure is difficult to assess, mathematical models often omit this variable. Other variables also impact transmission, such as the virus’s pathogenicity. A virus’s pathogenicity may be gleaned by its attack rate, based on how many exposed people actually become sick. Because it is difficult to assess how many people are exposed to a virus but do not fall ill, models also typically omit this variable.

2. Reproduction Rate and the Transmission Rate

The key parameter in modeling bioterrorism transmission is the number of individuals the first case—called the index case—infects. The reproduction rate, \( R_0 \), is sometimes referred to as the transmission rate and describes a disease’s tendency to spread. But because the reproduction rate “is a function of the contact rate between individuals, it can be affected by changes in the environment,” and hence varies from epidemic to epidemic. Consequently, mathematical models use the average number of new infections each case creates, a purely artificial number. For projecting smallpox, researchers used historical data and outbreaks in developing countries, which is likely irrelevant as well. Further, although the rate generally decreases after the first generation, models use a single reproduction rate throughout the epidemic, i.e., the same value is used.

123. See Kaplan et al., supra note 84, at 1038 (discussing generally the factors that affect the spread of disease).

124. See Pourbohloul et al., supra note 82, at 1249.


127. Pourbohloul et al., supra note 82, at 1253 (“The epidemic potential of disease is commonly estimated by using the basic reproductive number \( R_0 \), the number of secondary infections arising from a single infection in a relatively naïve [susceptible or unvaccinated] population.”).

128. Milton, supra note 90.

129. Enserink, supra note 85, at 1592.

130. Id. at 1593.

131. See, e.g., Raymond Gani & Steve Leach, Transmission Potential of Smallpox in Contemporary Populations, 414 NATURE 748, 748 (2001); Richard Harling et al., Interim smallpox guidelines for the United Kingdom: Developing new policies from old evidence, 325 BRITISH MED. J. 1371, 1371 (2002) (“[T]he underlying evidence relies on historical data, which were collected in a different, now outdated context and are often incomplete.”).
artificially used in each generations of spread. This is not how Mother Nature works and, I suggest, is the single most significant cause of model failure.

The reproduction rate is also a function of the virus’s transmission characteristics, and the nature and number of human interactions. In the context of smallpox, “researchers can’t agree on the right value. Past outbreaks yielded varying results, and the number can differ from population to population.” As one group of researchers noted, “models must balance biological realism against limitations of knowledge, and uncertainties need to be accurately communicated to policy-makers.”

Another group of researches notes that “[s]mallpox poses the particular challenge that key biological, social, and spatial factors affecting disease spread in contemporary populations must be elucidated largely from historical studies undertaken before disease eradication in 1979.”

Notwithstanding variability in reproduction rates from a theoretical and historical perspective, bioapocalyptic reproduction rates are commonly used in epidemic planning. DW assumed a replication rate of ten; an assumption which was seriously contested as a replication rate of two is considered more representative of actual past epidemics. Interestingly, concocting an epidemic model with a replication rate of ten for every generation creates a geometric increase in spread, further artificially—and exponentially—inflating the predictions.

132. See Kaplan et al., supra note 84, at 1039.
133. Pourbohloul et al., supra note 82, at 1255.
134. Enserink, supra note 85, at 1593.
135. Milton, supra note 90 (“Infectious disease modeling plays an important role in this dialogue, and the biology of the transmission pathway, the focus of this review, is critical to producing appropriate predictive models and understanding which controls will work best under varying conditions.”).
136. Ferguson et al., supra note 91.
137. Eichner & Dietz, supra note 73, at 116 (agreeing with Fenner that the threat of a bioterrorist attack using smallpox should not be exaggerated).
138. O'Toole et al., supra note 104, at 975.
139. Enserink, supra note 85, at 1593 (“The most contested assumption in Dark Winter, however, is the [replication rate] for smallpox . . . .A team led by CDC’s Martin Meltzer . . . concluded after a similar analysis of many more past outbreaks that the average rate of transmission was lower than 2.”); see also Bozette et al., supra note 50, at 417, 419 (describing other models that estimate that the second-generation attack rate was even lower, at 1.8, “with fewer than two second-generation cases” and estimating these data from “the average number of next-generation cases of smallpox arising from the current generation of cases” in twenty-five reported post-WWII outbreaks.).
140. Id. (“When [the reproductive number] is high—say 10 to 13, as for measles—a disease will spread exponentially; when it’s between 1 and 2, it will just keep going, and below 1, it will peter out.”).
3. Transmission Potential and Susceptibility

While the reproduction rate reflects the number of people each case infects, the transmission potential tells us the number of people each case is capable of infecting. This figure includes the length of time ("duration") an infected person is in proximity to a potential victim enabling sufficient exposure and the proximity of contact. Most smallpox infections result from close contact, usually two meters or less, with infected individuals for several hours. And historical data attests to the fact that household members and hospital contacts are by far at the highest risk for infection.

An infected person’s mobility also impacts the number of people with whom he or she comes in contact and is thus able to infect. A very sick person confined to bed is not likely to have as many contacts as some of the forecasters would have us expect. Extrinsic factors such as weather, cultural factors such as social gatherings, and even regulated conduct such as traffic light timings all affect duration of exposure—and hence transmission capacity.

Of course, if transmission can be airborne, the attack rate is multiplied because person-to-person contact is no longer necessary. For this reason, multiple attempts to weaponize the smallpox virus into an aerosolized version have been undertaken. To date, it appears that all attempts have failed.

4. Generations of Transmission and Case-Fatality Rate

The number of generations of transmission—how many times the virus can be spread from one person to the next before the virus is no longer transmissible—is a crucial factor in modeling. Another model parameter

141. Kaplan et al., supra note 84; R. H. Henderson & M. Yekpe, 90 Smallpox transmission in Southern Dahomey. A study of a village outbreak, AM. J. EPID. 423 (1969) ("The observed behavior of smallpox in this outbreak suggests that transmission occurring from casual contact is a rare event."); Milton, supra note 90, at 5 ("Susceptible individuals . . . would have needed to spend between 1.7 and 16.7 hours in the patient’s room to have a 63% probability of becoming infected.").

142. Enserink, supra note 85, at 1593.

143. Andrea Ammon et al., Early Disease Management Strategies in Case of a Smallpox Outbreak, in POXVIRUSES 414 (Andrew Mercer et al. eds., 2007) ("The European outbreaks between 1950 and 1971 showed that 55% of the infected persons contracted smallpox at a hospital, 20% in the family, 14% at their working place or school and 3% of the infected persons were working in a laundry, while 8% were unidentified contacts.").

144. Jahrling, supra note 7, at 1.

145. See, e.g., id. at 2 (showing that Jahrling tried to infect monkeys with smallpox and was only successful when the monkeys were both injected with the virus at high concentrations and subjected to aerosolized virus).

146. See generally Nicholas C. Grassly & Christophe Fraser, Mathematical models of infectious disease transmission, 6 Nature Reviews Microbiology 477, 484 (2008)
is the case-fatality rate—the percentage of infected people who die—which is a function of the virus’s lethality, the victim’s susceptibility and medical care rendered. 147

Counter-intuitively, case-fatality rate appears to be indirectly proportional to generational duration; the faster the virus kills off its host population, the fewer susceptible members of the population there are left to infect. Once the virus runs out of susceptible members of the population, it loses the ability to transfer to other hosts, 148 as smallpox needs a human host reservoir to remain viable; smallpox has no known animal reservoir, and unlike bacteria, cannot form spores to preserve itself for “better times.” Epidemiologists have calculated that smallpox’s continued spread requires a population of around two-hundred thousand people living within fourteen days of travel from each other. 149 Without this feeding ground, the virus cannot continue its life cycle and dies out. 150

The 1918 Spanish influenza pandemic is illustrative of this counter-intuitive concept. The virus was successful due to its extremely low lethality; its case-fatality rate was only 2.5 percent. 151 However, its infection rate was the highest in modern history, with 500 million infections worldwide—about a third of the world’s population. 152 But because the Spanish flu does not kill most of those it infects, a large number of infected people are available to spread the disease to others over multiple generations. 153 (Think of making a one-dollar profit from one million people to making a thousand-dollar profit from ten.) This feature helps explain why

(explaining that viruses can essentially run out of susceptible individuals, making the number of generations an important factor in modeling epidemics).


148. Henderson and Yekpe, supra note 141.


150. Id. See also FRANK FENNER ET AL., SMALLPOX AND ITS ERADICATION 192 (1988) (“It seems likely that a disease as lethal as smallpox must have exerted some selection for more resistant genotypes within populations in which it had been endemic.”).


152. Id.

153. See id. (stating that the Spanish flu killed an estimated fifty million people).
Ebola, with its high case-fatality rate, petered out far sooner than expected; it simply exhausted all susceptible hosts in the feasible transmission area.

5. Extrinsic Factors and Secular Trends

Finally, a disease’s incidence is dependent on genetics, nutrition, and even weather. The weather not only influences the agent’s longevity, but also contact frequency between carriers and potential patients.

To illustrate, as noted earlier the DW attack was planned to occur in early December, 2002, originating in Oklahoma City. Theoretically, this would be an optimum time for a bio-terrorist smallpox attack; the virus loves cold and dry weather. The fable’s creators, nor any would-be terrorist, could not possibly have known at the time the program was run that, in early December, 2002, an ice and snow storm would virtually shut Oklahoma City down. On December 3, 2002, icy winds “left a damage footprint in a narrow band from west central to north central Oklahoma. Areas north of the icing region generally received 2-6 inches of snow, with some areas reporting more than eight inches.” The south sustained moderate to heavy rainfall, but the storm caused major damage to the electrical distribution systems throughout Oklahoma City. Had a real terrorist attack been orchestrated for early December in Oklahoma City, it would have flubbed dramatically; the virus would have died quickly, because people would not have ventured from their houses, thus impeding the imagined explosive spread.

The unpredictability of weather, a critical factor in transmission and epidemic success, makes bioterrorism a poor-choice of weapon for cash-strapped terrorists. With no guarantee that the attack can achieve even a modicum of success, why would a terrorist group expend huge sums of
capital to create a bioweapon, cultivate the bacteria, create a deployment system, and protect themselves only to see the anticipated event fizzle out because of rain or snow.

IV. Truth? or Consequences

A. A Statistical Summary of Post-World War II Epidemics

I suggest governments reevaluate policies that use simple non-real-time mathematical models for pandemic projections. To illustrate the flaws in current policies, I detail below stark statistical data. Then, as a case-study, I investigate what actually happened in smallpox epidemics post-World War II ("WWII") to contrast reality and history with DW's projections and government assumptions based thereon.

Frank Fenner's work provides a comprehensive review of smallpox epidemics sequestered by time period; other works provide additional detail. From 1959 on, Fenner tallied thirty-four European smallpox epidemics and concluded that all these outbreaks together resulted in only 573 cases over the next thirty years. Many outbreaks consisted of a single case. Two-thirds of outbreaks led to no more than five cases. Fenner also states that most post-1959 outbreaks—including those in Western, Central, and Eastern Africa and Indonesia—were associated with case-fatality rates in the range of five percent to fifteen percent.

Mack identifies an additional fifteen smallpox epidemics in Europe between 1950 and 1971, and tallied 936 confirmed cases, amounting to fewer than twenty cases per year, with a case-fatality rate of sixteen percent. He also calculated that these outbreaks gave rise to, on average, “1.6 cases from the general public and 2.4 cases from hospital contacts,” and that in only two of these outbreaks were there more than two deaths—and these occurred in hospital settings. He also noted that eighty-four

164. See D.J. Muscatello et al., Translation of Real-Time Infectious Disease Modeling into Routine Public Health Practice, 23 EMERGING INFECTIOUS DISEASES e1 (2017) (providing a more up to date assessment of how the tool can be better used — incorporating real time data).

165. Which coincidentally also heralds the inception of mass use of antibiotics.

166. FENNER ET AL., supra note 150, at 1070.

167. Id.

168. Id. at 1073.

169. See, e.g., id. at 4, 327, 1070 (showing that case-fatality rates varied substantially by country, though whether due to hardiness of the strain or the population’s genetics or both is difficult to gauge, and that in post-WWII Spain and Portugal, “the overall case-fatality rate was only 3.1%”).


171. Id. at 163.
three-quarters of the outbreaks lasted no longer than two generations.\textsuperscript{172} The longest epidemics were caused by the weaker variola minor,\textsuperscript{173} corroborating the counterintuitive view that the more lethal a microbe is, the shorter the epidemic, and sustaining the converse: the less severe the strain, the longer the epidemic.

Mack also found that delays in diagnosis, ranging from one to twenty missed case identifications, occurred because European physicians lacked familiarity with the clinical features of smallpox. Nevertheless, he notes that this factor did not result in more spread or longer generations of transmission.\textsuperscript{174} To the contrary, “the larger the unrecognized generation (the more prominent was the spread of the disease in hospitals) the fewer additional generations occurred.”\textsuperscript{175}

Bhatnagar et al. performed another detailed analysis, studying fifty-one post-WWII outbreaks, identified from 1389 publications. The group found that

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\text{the median initial reproduction rate (R) across all 51 outbreaks was 2 with a range of 0 to 38. About half had an initial R of 1 or less, and over two-thirds had an initial R of 3 or less. The median duration, as measured by number of generations, was 1 with a range of 0 to 9. About a third did not extend beyond the index generation, and nearly three quarters lasted for 3 or fewer generations. The median outbreak size, as measured by the total number of cases, was 4 with a range of 1 to 134 cases. About half involved 3 or fewer cases, and two-thirds involved 15 or fewer cases. The median number of deaths was 1 with a range of 0 to 26. About two-fifths involved no deaths, and three quarters involved 3 or fewer deaths.}\textsuperscript{176}
\]

Bhatnagar’s findings discredit the high replication rates DW used,\textsuperscript{177} which were based on epidemics where the disease was endemic, and modern health practices are non-existent. In fact, the Bhatnagar group specifically did not examine data from nineteenth-century European smallpox outbreaks because the group assessed these were inapplicable to modern times; i.e., the replication rate prior to 1945 was significantly higher than it would be today due to modern medical practice, and hence irrelevant to

\textsuperscript{172} See id.
\textsuperscript{173} Id. at 163, 166.
\textsuperscript{174} See id. at 163.
\textsuperscript{175} Id. at 163.
\textsuperscript{176} Vibha Bhatnagar et al., \textit{Transmission patterns of smallpox: systematic review of natural outbreaks in Europe and North America since World War II}, 6 BMC PUB. HEALTH 1, 4 (2006) (internal citations omitted).
\textsuperscript{177} See id. at 4.
modeling a modern epidemic. Bhatnagar’s projected replication rates, as are Meltzer’s, are vastly incongruous with—and substantially lower than—DW’s. Specifically, Fenner, among the leading authorities on smallpox, unequivocally refuted DW’s outcomes, stating that

[t]he comparatively slow spread of smallpox[ ] . . . is attributed to the fact that infection is almost invariably transmitted during face-to-face contact with a patient after the rash has begun to develop. Patients with classical smallpox, unmodified by vaccination, are usually confined to bed during this period and are therefore not normally in contact with many susceptible persons. While previously vaccinated persons may experience a very mild, highly modified form of smallpox and remain ambulatory, such persons excrete fewer organisms and are less efficient transmitters of infection.

B. Research Plan and Methodology

For some reason, the data set forth in the studies described above do not etch themselves into the collective consciousness of policymakers. For example, an Israeli governmental body conducted a study for the purposes of developing policy and literally lifted assumptions from DW or used tertiary references cited therein, completely ignoring Fenner, Mack,

178. See id. (“The recent literature contains several estimates for the reproductive rate of smallpox. . . . Two of the studies do not use contemporary western outbreaks. One estimates the basic reproductive rate, (R)0, a theoretical parameter defined as the expected number of new infected hosts that an infectious host will produce . . . rather than effective reproduction rate.”); see also Eichner & Dietz, supra note 73, at 110-117 (estimating the maximum likelihood of the spread of smallpox in a 1967 epidemic in Abakaliki, Nigeria, in a group that refused vaccination and finding that their “results support the widely held belief that smallpox spreads slowly, mainly among close contacts, and that infectivity before the onset of rash was negligible.”).

179. See generally Bhatnagar et al., supra note 176 (describing the various reproductive rates in epidemics she studied, none of which support DW’s projections).

180. P. F. Wehrle et al., An Airborne Outbreak of Smallpox in a German Hospital and its Significance with Respect to Other Recent Outbreaks in Europe, 43 BULL. WORLD HEALTH ORG. 669, 677 (1970) (emphasis added) (citation omitted) (writing with D.A. Henderson).

181. Paul E. Slater et al., Preparation for an Outbreak of Smallpox in Israel, 4 ISRAEL MED. ASSOC. J. 508, 508 (July 2002) (“In an uncontrolled smallpox outbreak, one contagious case typically generates 10–20 secondary cases—a community transmission potential almost as high as that of measles and chickenpox. The secondary attack rate in susceptible household members is 50%. As in most infectious diseases, lighter cases of smallpox may be the most dangerous transmitters because the victims will be ambulatory for longer and will have many more contacts than those cases that result in early collapse.” (internal citations omitted)).
Meltzer, Bhatnagar and other world-class scientists’ compelling and competing data.

Notwithstanding the multiple studies corroborating Fenner’s view, DW’s extreme and hysteria-laden assertions are repeated and ingrained into the collective societal psyche, not just by the popular press, but by scientists—a question that begs for investigation, understanding and a “setting straight” of the record—and by prominent influence makers.182

I suggest that the mindless repetition of DW’s erroneous assumptions occurs because the DW fable resulted from “poetic failure,”183 causing an emotional entanglement in its viewers and readers with which statistical data in scholarly articles cannot compete. It may be that statistical evidence is ineffective in achieving much-needed policy change regarding biothreat modeling and response because it “can’t get the message out” to non-scientists. The DW episode also falsely portrays the exigencies of modern life—air travel, increased susceptibility to smallpox, and vaccine shortages—as new, epidemic-provocative issues, leading to the false conclusion that epidemics of the last century offer no guide or insights that could be helpful in planning a potential terrorist attack.

Parenthetically this approach suggests that a story format or narrative may be more appropriate to policy planning by the lay policy-maker. To effectuate this alternative approach of reviewing post-WWII smallpox epidemics in industrialized countries via a real life review, I reviewed all accessible historical records from these outbreaks, focusing on facts not commonly cited.

C. Factors of Concern

For ease of reference I reiterate DW’s key assumptions from which apocalyptic predictions derive: (1) because modern doctors are unfamiliar with smallpox, diagnosis would be delayed, allowing missed cases to infect others; (2) today’s population is largely unvaccinated and therefore more susceptible to infection; (3) increased air travel will disseminate smallpox faster, making tracing difficult; (4) smallpox is highly infective, more so than common diseases such as flu and measles; (5) airborne transmission is a viable means of infecting large populations with a viral disease; (6) there will be at least four generations of spread; (7) the replication rate will be ten to twenty throughout the four generations of spread; (8) the case-

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182. See, e.g., Perring, supra note 47 (noting even the well-respected Bill Gates promulgates DW’s false assertions).

183. Shulamit Almog, Creating Representations of Justice in the Third Millenium: Legal Poetics in Digital Times, 31 Rutgers Computer & Tech. L.J. 183, 211 (2006) (defining poetic failure and explaining that “even a poetically flawless articulation might fail to convey its intended meaning because of the addressee’s inability to receive it.”).
fatality rate will be thirty percent;\textsuperscript{184} (9) there will be an insufficient supply of vaccine for mass vaccination, the preferred means of response, leading to mass hysteria; and (10) it will take three or more weeks to get enough vaccine.\textsuperscript{185}

V. The Case-Studies

A. New York City in 1947: The Vaccine-Shortage Scenario

The New York City (“NYC”) epidemic began in 1947 when an expatriate named Eugene LeBar brought smallpox from Mexico to NYC.\textsuperscript{186} This incident occurred seventy years ago, but embodies many of the nightmare features of an imagined bioterrorist attack: NYC physicians were unfamiliar with smallpox, clinical and laboratory diagnosis were delayed, the index case traveled thousands of miles while infected, and the exposed population was largely unvaccinated.\textsuperscript{187}

On February 24, 1947, Mr. LeBar, a forty-seven-year-old rug merchant living in Mexico embarked on a bus trip to New York with his wife.\textsuperscript{188} That evening he suffered a headache and complained of a backache and two days later, en route, he developed a fine rash.\textsuperscript{189} The disease is transmissible once a rash becomes visible, and from this point on it is virtually certain he exposed others.\textsuperscript{190}

Over its six-day trip, the bus on which LeBar traveled stopped in seven major population centers: Monterrey, Mexico; Laredo, Texas; Dallas, Texas; St. Louis, Missouri; Cincinnati, Ohio; Pittsburgh, Pennsylvania, culminating

\textsuperscript{184} Enserink, supra note 85 (asserting that “O’Toole and her co-authors selected six [largely unidentified] smallpox importations into Europe after World War II that they thought might be typical for a 20th century attack . . . They settled on a[ clearly unsubstantiated reproductive number] of 10—although they think that may be on the low side,” even though it proved to be notoriously on the high side and that “[i]n one famous and ‘particularly instructive’ case, they wrote in a paper, a patient who returned to Yugoslavia from a trip to Iraq in 1972 infected 11 others, who in turn caused 140 ‘second generation’ cases”); see also Donald A. Henderson, The Looming Threat of Bioterrorism, 283 SCIENCE 1279 (showing that Henderson, a consultant on DW embraced this assumption; in other papers, including in Fenner et al., supra note 150, at 4, which he co-authored, he supports a case-fatality rate as low as five percent.).

\textsuperscript{185} O’Toole et al., supra note 104, at 973.

\textsuperscript{186} See Israel Weinstein, An Outbreak of Smallpox in New York City, 37 AM. J. PUB. HEALTH 1376, 1376 (1947).

\textsuperscript{187} Id.

\textsuperscript{188} Id.

\textsuperscript{189} Id.

\textsuperscript{190} NEW YORK DEP’T HEATH, Smallpox Questions and Answers: The Disease and the Vaccine (Apr. 2003), https://www.health.ny.gov/publications/7004/ (“[T]he person becomes most contagious with the onset of rash.”)
in NYC. The bus passengers, in turn, dispersed to over twenty-nine states, exposing hundreds more.

The LeBars arrived in NYC six days after they left Mexico, checked into a hotel, did some sightseeing and walked through a large department store. By March 5, 1947, LeBar developed a fever, the rash had worsened, and he had become sick enough to be admitted to Bellevue Hospital. On March 8, he was transferred to Willard Parker Hospital, a communicable-diseases hospital, where doctors ruled out smallpox because he claimed he was never exposed. He died two days later. An autopsy revealed hemorrhagic disease, the most dangerous and potentially contagious form of the disease, but the diagnosis was not made. Not until formal lab tests were returned several weeks later and the autopsy was re-evaluated was smallpox diagnosed—over a month after LeBar’s arrival in NYC.

Between LeBar’s arrival in NYC on March 1 and his hospitalization on March 6, he had direct contact with hundreds of people, plus potentially exposing three thousand additional guests staying at his hotel. Eventually, all of LeBar’s direct and indirect contacts were located and vaccinated; some were sequestered to prevent further spread; some were located as late as April 4, 1947, nearly a month after he arrived. None of LeBar’s direct contacts developed smallpox. A few weeks after LeBar’s death, however, two patients who had been hospitalized at Willard Parker at the same time as LeBar were diagnosed with smallpox—a baby with croup and a hospital worker admitted for mumps. Only after these diagnoses were made and LeBar’s autopsy results were re-evaluated did the

191. Weinstein, supra note 186, at 1378.
192. Id.
193. Id. at 1376.
194. Id.
195. Daniel Okrent, Daniel Okrent on the 1940s, The War, and the Great Smallpox Immunization of New York, VANITY FAIR, (Oct. 2013), http://www.vanityfair.com/culture/2013/10/daniel-okrent-on-the-1940s (“It took until the morning of April 4 for health officials to determine the cause of death: smallpox. New York had recorded only nine instances of the disease in the previous 25 years, none at all in the previous 8.”).
197. Okrent, supra note 195.
198. Roueché, supra note 196, at 70.
199. Okrent, supra note 195.
201. Weinstein, supra note 186, at 1379.
hospital vaccinate its staff and notify the NYC Health Department and the U.S. Public Health Service. ("USPHS").

At this point, the USPHS notified the bus passengers’ ultimate destination cities (all 29 of them) along with every passenger on board the bus and advised them to get vaccinated. Given the seven to seventeen-day incubation period, it would be safe to assume by the time LeBar’s contacts were identified, if they had sufficient exposure, they would have already fallen ill. None did.

The hospital worker LeBar infected later infected his wife and three other patients. One patient was a young boy treated for whooping cough who was diagnosed on March 17. The second patient, a baby, did not infect anyone, not even her parents. The third patient was discharged to a convalescent nursing facility before he was diagnosed. There he infected three others, two children and a sixty-two-year-old nun. Though neither patient had direct contact with LeBar, the hospital does not appear to have considered the possibility of airborne transmission; instead, doctors seem to have assumed it was transmitted via intermediary contact.

Mirroring current concerns, “[f]ew physicians in the city had ever seen smallpox, either on a microscope slide or in the appearance of its murderous excrescences on a doomed victim.” As a result, “[d]efinitive diagnosis had had to wait until laboratory studies could be conducted in Washington” about a month later.

Once LeBar’s diagnosis was confirmed, NYC’s mayor, William O’Dwyer, announced plans to vaccinate everyone in the city. Vaccine shortage was an initial concern; the Health Department had 250,000 doses of vaccine that could be immediately distributed and 400,000 doses that were not yet

202. Id.; see also Roueche, supra note 196, at 70.

203. See Smallpox Strikes Seaman from Here—Mess Steward Removed from an Army Transport to a Hospital in Germany. N.Y. TIMES, Apr. 19, 1947, at 13 (noting that two additional cases of smallpox were transmitted in the New York City area at that same time—a merchant seaman who temporarily lived with relatives in Manhattan from March 4 to 15, and R.C. Smith, who lived in Trenton, N.J. and died nearby in Camden, on April 17, 1947; neither had known contact with Le Bar or any cases traced to him).

204. Weinstein, supra note 186, at 1379.

205. Id. at 1379.


207. Okrent, supra note 195.

208. Id.

209. Weinstein, supra note 186, at 1381.

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ready for use.\textsuperscript{210} The Army and Navy sent 780,000 doses from all over the country.\textsuperscript{211} Within a week, “despite a shortage of vaccine, more than 2,500,000 of the city’s 7,500,000 had been vaccinated;”\textsuperscript{212} by months’ end over six million people were vaccinated.\textsuperscript{213} While some question these figures on the basis of vaccine availability,\textsuperscript{214} O’Dwyer had presciently received a commitment from several vaccine companies to provide nearly six million doses of smallpox vaccine and the Health Commissioner had put in place the mechanics of implementation.\textsuperscript{215}

The Health Department had “organized a corps of employees: 1,100 outside physicians, 242 clerks, and 3,000 civilian volunteers to vaccinate city residents [and] police were assigned to supervise the snaking lines of people waiting to be vaccinated at clinics around the city.”\textsuperscript{216} NYC residents did not become hysterical as DW might have prophesized; Time Magazine notes that Manhattan residents were calm, “queuing up by the thousands, sometimes in lines five blocks long[] for vaccination.”\textsuperscript{217} Still the public stayed calm.

The Health Commissioner, Israel Weinstein, took to the radio to encourage vaccination in a model example of how to convey information without creating hysteria.\textsuperscript{218} Even when the media published fearmongering articles and when “at the height of the program[] the vaccine

\textsuperscript{211} Okrent, \textit{ supra} note 195.
\textsuperscript{212} \textit{The Smallpox Scare, supra} note 200.
\textsuperscript{213} Roueché, \textit{ supra} note 196 (“Within the space of 28 days a total of at least six million three hundred and fifty thousand people had been vaccinated in the city.”); Weinstein, \textit{ supra} note 186, at 1381.
\textsuperscript{214} DAVID L. STREINER ET AL., \textit{PDQ PUBLIC HEALTH} 202 (2011).
\textsuperscript{215} Weinstein, \textit{ supra} note 186, at 1381.
\textsuperscript{216} N.Y. CITY DEPT. HEALTH & MENTAL HYGIENE, \textit{PROTECTING PUBLIC HEALTH IN NEW YORK CITY: 200 YEARS OF LEADERSHIP} 43 (2005) [hereinafter 200 Years of Leadership].
\textsuperscript{218} Israel Weinstein, Commissioner, New York City Health Department, Dr. Israel Weinstein Smallpox Message (Apr. 4, 1947), available at http://www.wnyc.org/story/dr-israel-weinstein-smallpox-message/.
supply vanished,” the havoc DW predicts never materialized. Perhaps it was because O’Dwyer downplayed the risk of waiting until more vaccine arrived. Perhaps it was because Weinstein’s radio announcements successfully dampened hysteria. Or perhaps DW exaggerates the likelihood of a hysterical reaction?

By May 3, 1947, all NYC vaccine clinics had closed and the outbreak was deemed contained. Only twelve people had contracted smallpox and only two had died. At least three people died from the vaccine. Three more people died of direct, vaccine-related complications, including septicemia and generalized vaccinia, and at least six more probably died of vaccine-

219. Id. ("In the 1947 campaign, trouble began on April 16, when (no longer on page 1), the Times announced, 'Vaccinations Stop; Drug Supply Gone; Thousands Turned Away'. With little warning, and at the height of the program, the vaccine supply vanished, something that was never explained . . . . During the shortage, the Times noted, "hundreds of eager men, women, and children queued up at Bellevue Hospital at dawn, although vaccinations were not scheduled to begin until 10 a.m. At some stations, the crowds did not take kindly to the news that the doctors had run out of vaccine and the police had a little difficulty dispersing a crowd of several hundred" outside one vaccine station."); see also Dale Smith, Let’s make new mistakes: planning for public health and civil defense, in BIOTERRORISM: PSYCHOLOGICAL AND PUBLIC HEALTH INTERVENTIONS 30 (Robert J. Ursano eds., 2004) (describing a situation in which panic did break out in neighboring states, serving as a point of comparison to New York City’s handling of the outbreak without any panic. But even with a seemingly calm public in NYC, the next day a million doses arrived, and 500,000 more people were vaccinated.).

220. Kent A. Sepkowitz, Letter to the Editor, The 1947 Smallpox Vaccination Campaign in New York City, Revisited, 10 EMERGING INFECTIOUS DISEASES 960, 960 (2004) (showing that on April 16, 1947 the New York Times ran the headline “Vaccinations Stop; Drug Supply Gone; Thousands Turned Away.”)

221. See Dale Smith, Let’s make new mistakes: planning for public health and civil defense, in BIOTERRORISM: PSYCHOLOGICAL AND PUBLIC HEALTH INTERVENTIONS 30 (Robert J. Ursano eds., 2004) (noting that when “[a] man died of smallpox in Camden, New Jersey . . . panic broke out in Pennsylvania, Delaware, and southern New Jersey[,]” perhaps indicating that New York City was indeed doing something right).

222. Sepkowitz, supra note 220, at 960.

223. 200 Years of Leadership, supra note 216, at 43.

224. Weinstein, supra note 186, at 1381-82.

225. See Smith, supra note 221, at 30 (describing a situation in which panic did break out and serving as a point of comparison to New York City’s handling of the outbreak without any panic, suggesting that perhaps New York City’s outbreak management was indeed done correctly).

226. Weinstein, supra note 186, at 1381.

227. Id. at 1382, 1379.
related encephalitis.\textsuperscript{228} The epidemic extended over two generations with a replication rate of 2.75,\textsuperscript{229} and a case-fatality rate of sixteen percent.\textsuperscript{230}

Even though LeBar contracted the most virulent form of smallpox, the hemorrhagic type, the attack rate was miniscule. Especially considering that he came into direct contact with hundreds of people, from hospital personnel who were exposed to him for long periods of time, to transient meetings with tourists and hotel guests, to intermediate exposures to fellow bus travelers. Even with factors primed to generate explosive spread and legitimize bioapocalyptic predictions, calamity was averted. Long before social media existed to get the word out, nearly six million people were vaccinated in a month.\textsuperscript{231} In the face of the unknown, an untried health department’s prompt and efficient response actions resulted in rapid containment of the disease as well as prevention of hysteria.

\textbf{B. Bradford, England, in 1961-62: High Case-Fatality Rate}

The worst of the modern outbreaks in terms of case-fatality rate occurred in Bradford, England, in 1962, with a rate of forty-two percent!\textsuperscript{232} This case presents another example of a potential nightmare scenario that failed to generate the anticipated mass epidemic. Like the NYC outbreak, the Bradford outbreak involved the dangerous hemorrhagic form of smallpox.\textsuperscript{233} A large proportion of the hospital staff was unvaccinated; the smallpox diagnosis was difficult given the hospital staff’s lack of experience with the disease—and hence not timely made.\textsuperscript{234} Even after the index case died and her body was flown back to Pakistan for burial, physicians did not

\begin{itemize}
  \item \textsuperscript{228} \textit{Id.} at 1383 (Some 46 cases of encephalitis were reported in the month following the mass vaccinations; eight died. Encephalitis has several causes— including smallpox vaccination. Vaccine causation could be ruled out in only two of the deaths. Six cases were equivocal. Notwithstanding, they were not considered as associated with the vaccine because “the Health Department has had no proof of any death due to post-vaccinal encephalitis in its vaccination campaign.” \textit{Id.} While epidemiology in 1947 was not as robust as it is now, one would be hard pressed to believe that New York City normally reported a monthly background level of six encephalitis deaths and 44 encephalitis cases with could not otherwise be accounted for.).
  \item \textsuperscript{229} \textit{Id.} (providing that the index case infected two people and that the three-person second generation infected eight people, leading to an average replication rate of 2.75); cf. O’Toole, \textit{supra} note 104 (projecting a replication rate of ten).
  \item \textsuperscript{230} \textit{Id.} (showing that two of twelve people died, resulting in a fatality rate of 16.6 percent).
  \item \textsuperscript{231} \textit{STREINER ET AL.}, \textit{supra} note 214.
  \item \textsuperscript{232} Derrick Tovey, \textit{The Bradford smallpox outbreak in 1962: A personal account}, 97 J. ROYAL SOC’Y MED. 244, 246 (2004) (showing 14 cases of smallpox resulting in 7 deaths, one of which is attributed to cerebral thrombosis).
  \item \textsuperscript{234} \textit{FENNER ET AL.}, \textit{supra} note 150, at 1079.
\end{itemize}
consider smallpox as the cause of death.\textsuperscript{235} The replication rate was astronomical; the index case infected ten others prior to the diagnosis—i.e., in the first generation.\textsuperscript{236} Yet, by the end of the epidemic, which lasted two generations, only fourteen people were infected and only six died from the disease.\textsuperscript{237}

A supervising pathologist later wrote that “if ever a smallpox epidemic arose again, it might well present itself to the clinicians not as a textbook case but as PUO [pustules of unknown origin] as in the Bradford outbreak or as a haematological disorder or some masking disease.”\textsuperscript{238}

The health authorities’ first inkling of disease occurred on December 25, 1961, when a twenty-four-year-old man arrived at Heathrow Airport. Health authorities suspected he had smallpox, but physicians diagnosed ten more cases before they identified the index case—a nine-year-old Pakistani girl.\textsuperscript{239} The girl had been vaccinated on December 5, 1961, prior to departing Karachi, arriving in London by air eleven days later. After her arrival in London, she and her parents took a train to Bradford, England, arriving the next day.\textsuperscript{240} On December 23, 1961, she was admitted to Bradford Children’s Hospital and diagnosed with malaria.\textsuperscript{241} On December 27, her condition worsened. Three days later she developed facial lesions and died shortly thereafter.\textsuperscript{242} A post-mortem examination attributed her death to septicemia and malaria. At the time physicians did not suspect she had contracted smallpox.\textsuperscript{243}

Between January 11 and 13, 1962, physicians discovered ten first-generation cases, each contracted in a hospital setting.\textsuperscript{244} Only three second-generation cases occurred, leading to a total of fourteen cases.

\textsuperscript{235} \textit{Id.}

\textsuperscript{236} \textit{Id.} at 246.

\textsuperscript{237} \textit{Fenner et al., supra} note 150, at 1079. \textit{See also} Benn, \textit{supra} note 233, at 343.

\textsuperscript{238} Tovey, \textit{supra} note 232, at 246 (Tovey, a physician on the scene, observed that physicians’ “immediate difficulty was that we would not be able to obtain laboratory confirmation of smallpox for at least 48 hours. At that time electron microscopy had not been established to obtain a speedy laboratory diagnosis. Specimens from the two deceased were dispatched by taxi to the local Public Health Laboratory where . . . the samples [were tested] by . . . complement fixation and egg culture. A quickly convened ‘council of war’ was held by the regional medical officer, the chief medical officer . . . , his deputy, the regional infectious diseases consultant[,] and myself.”).

\textsuperscript{239} \textit{Id.} at 245.

\textsuperscript{240} \textit{Fenner et al., supra} note 150, at 1079.

\textsuperscript{241} Benn, \textit{supra} note 233, at 343.

\textsuperscript{242} \textit{Fenner et al., supra} note 150, at 1079.

\textsuperscript{243} \textit{Id.}

\textsuperscript{244} Tovey, \textit{supra} note 232, at 245-46 (noting that the first-generation cases were a hospital visitor, a cook, a nurse, a pathologist, and six patients).
Seven patients died, six from smallpox, and the seventh from an unrelated cause.\textsuperscript{245} Identifying, tracing, surveilling, and vaccinating the Pakistani child’s more than 1400 contacts was “expensive, difficult, and time-consuming.”\textsuperscript{246} Although health authorities never contemplated country-wide mass vaccination, they opened vaccination clinics because so many people had been exposed to the disease.\textsuperscript{247} Health authorities also quarantined staff members from the four hospitals involved and surveilled nine hundred contacts.\textsuperscript{248} Nearly the entire population of Bradford—numbering nearly 250,000—was vaccinated in five days.\textsuperscript{249} In a month, the outbreak was over.\textsuperscript{250} While six people died from the smallpox epidemic, vaccination complications were not \textit{de minimis}.\textsuperscript{251} At least six people had post-vaccine symptoms requiring hospitalization, one adult died, and three children died of post-vaccinal encephalitis.\textsuperscript{252} The mechanism through which smallpox spread remained unclear because no direct contact occurred between the index patient and those she infected.\textsuperscript{253} Despite this, health authorities discounted aerial spread.\textsuperscript{254} Since the outbreak was already rather large by the time it was recognized, and there were numerous opportunities for transmission, both within the hospital and in the general community, health authorities concluded that an undiagnosed member of the medical or nursing staff likely transmitted the infection.\textsuperscript{255} Many of DW’s assumptions matched the facts of Bradford, but DW’s projections did not materialize, most significantly because the anticipated replication rate existed only for the first generation; for the second, the
replication rate dropped to 0.3, and after that, the epidemic died out, as do all epidemics with a replication rate less than one.

C. Canada in 1962: Limited Transnational Transmission

Because international air traffic increased in the 1960s and 1970s, more people traveled to Canada and the United States from countries with indigenous smallpox than in previous years. It would be reasonable, then, to expect more international transmission into North America. However, the facts do not bear out this forecast.

On August 10, 1962, a fifteen-year-old Canadian boy and his missionary family left Brazil by air, arriving in NYC, a trip that would have taken at least ten hours. The boy was feverish and ill on embarkation. After a six-hour hiatus in NYC, the family boarded a train to Canada, arriving in Toronto two days later. Soon after their arrival, the boy developed the characteristic centrifugal rash, but the smallpox diagnosis was delayed until two days after he was hospitalized.

Authorities identified, vaccinated, and surveilled every passenger on the plane from Brazil to New York, as well as the members of his family and other known close contacts in Toronto. The media advised those who had traveled on the train from New York or been at the station to seek vaccination, and those who did so were placed under surveillance. No one contracted the disease from the boy.

D. Stockholm in 1963: Spread by Close Contact

In 1963, sixteen years after NYC successfully dealt with its smallpox crisis and a year after the Bradford epidemic, a smallpox epidemic broke out in Stockholm. Health authorities did not recognize this outbreak until

256. See FENNER ET AL., supra note 150, at 1079 (indicating that there were three second-generation cases infected by the ten first-generation cases).

257. Id. (noting the pattern of a significant drop of replication rate after an initial peak in the first generation occurs in virtually every epidemic evaluated; when it reaches one, the epidemic dies out)

258. FENNER ET AL., supra note 150, at 1081.

259. See generally id.

260. Id.

261. Id.

262. Id.

263. Id.

264. Id.

265. Id.

266. Id.

long after its onset—after the index case became infectious and the seventh case was diagnosed, which was after the second generation had begun. 268

This case began with a twenty-four-year-old seaman returning to Sweden after a two-week sojourn in Australia. 269 The young man left Darwin, Australia, on March 22, 1963, and arrived in Stockholm two days later. 270 The initial leg of the flight stopped in five countries with layovers of fifty minutes or less: Jakarta, Indonesia; Singapore; Yangon, Myanmar; Calcutta, India; Karachi, Pakistan; Tehran, Iran; and Damascus, Syria, before arrival at its final destination—Zurich, Switzerland. 271 Once in Zurich, the seaman spent the night in a hotel. The following day, March 24, he boarded a flight to Stockholm. On April 6, 1963, he developed a “moderate fever and a mild rash,” the first signs of transmissible infection. According to the CDC, he was infected en route, “either at a terminal or on the plane,” and it was presumed he acquired the disease while in transit throughout Southeast Asia. 272 However, because the disease’s incubation period ranges from seven to seventeen days, 273 it is possible the infection began before the seaman began his voyage.

Once he fell ill, he remained at his grandmother’s house, where he infected her and three others with whom he had direct contact. 274 The grandmother infected three more women, and two others infected their partners, beginning the second generation. 275 Despite Sweden’s efforts to vaccinate all hospital personnel, eight hospital staff members and patients contracted smallpox, 276 some of which occurred without direct contact, constituting the beginning of the second generation. 277 The other cases contracted smallpox through face-to-face contact in the homes of patients. 278 It is possible that a nurse who treated these patients and later became infected served as the intermediary for the third generation. The virus also spread from a patient’s laundry, and possibly “by remote airborne

(1996) [hereinafter Stockholm CDC MMWR], available at https://www.cdc.gov/mmwr/preview/mmwrhtml/00042757.htm

268. Id.
269. Id.
270. Id. (flying on BOAC Flight #709).
271. Id.
272. Id.
274. Stockholm CDC MMWR, supra note 267.
275. Id.
276. Id.
277. See id.
278. Id.
exposure,” 279 although the health department did not seriously consider the latter.

One early case suffered an acute hemorrhagic form of the disease and died; the diagnosis was made post-mortem, leaving open the possibility that she exposed others. 280 Another case involved a woman who shared an apartment with her daughter in a boarding house that housed one hundred people. 281 The woman developed a rash, and the local hospital’s dermatology clinic evaluated her. Before a physician diagnosed her with smallpox, she had exposed at least 450 people; having spent four hours in the hospital in two crowded waiting rooms, and previously exposing all the women in the boarding house. 282 The source of the woman’s smallpox was traced to her daughter, a mortician who had prepared a body from one of the epidemic cases. 283 The CDC warned that these contacts “established an additional large group of contacts in which cases may yet occur.” 284 Yet none did.

Approximately eight thousand people lived in the initial case’s neighborhood, and 300,000 people in Stockholm took advantage of the offered vaccine, suggesting that a huge number of people feared exposure. 285 The CDC feared that the United States would be exposed, asserting that “at the time, there was justifiable concern about possible spread of infection from Sweden to the United States.” 286 The fear was illusory. The disease did not spread beyond the locality. At the conclusion of the outbreak, only twenty-five people became ill, and only four had died. 287

In the Stockholm outbreak, there was again an asymmetrical and non-redundant distribution of replication rates. The index case infected four others for a replication rate of four. The second generation infected seven others, indicating a replication rate of 1.75. The third generation bore a replication rate of two. The epidemic died out when the average replication rate would have been 2.58, confounding even conventional expectations. The virus spread both in the hospital and in the community. 288 But at the
conclusion of the outbreak, only twenty-five people became ill, and only four had died.  

E. *Germany in 1970: Airborne Transmission(?)*

The 1970 outbreak in Meschede, Germany, is the principal incident used to support the claim that airborne transmission of smallpox is possible. Airborne transmission would eliminate the need (time and proximity) for smallpox spread through direct person-to-person contact and could—theoretically—render smallpox into a realistic bioterrorist agent—if it is a realistic possibility.

The incident in Meschede began on December 31, 1969, during the peak of holiday traffic, when a man flew from Karachi, Pakistan, to Düsseldorf, Germany—at least an eleven-hour flight. From Düsseldorf, he took a three-hour train ride to his home in Meschede, arriving New Year’s Day. He had been vaccinated shortly before his arrival in Germany like the index cases in several of the previously discussed outbreaks. Ten days after his arrival, the young man developed a fever. Because his doctors suspected he had contracted typhoid, he was hospitalized in an infectious-disease isolation unit and confined to a private room on the ground floor. Four days later, on January 14, 1970, doctors noticed a rash. On January 16, 1970, he was diagnosed with smallpox.

Even though “the original smallpox patient had not left his room during his hospitalization, all hospital patients and personnel were immunized” and quarantined, although WHO actively discouraged Germany from mounting an intensive vaccination campaign, this feature suggests that public-health officials in Germany seriously believed that hospital personnel were at risk, even though at the same time they discounted anything but miniscule contact between hospital personnel and the index patient. At the

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289. *Id.*

290. See *generally* Wehrle et al., supra note 180.

291. See *id.* at 677 (“Extensive studies in both endemic and non-endemic countries have clearly indicated the necessity for close, and often prolonged, personal contact before transmission of infection occurs. A single patient rarely infects more than a few persons, and only infrequently have cases been reported in which there was no direct or ‘face-to-face’ contact with an earlier case.” Thus, “the Meschede outbreak in which the index patient infected 17 others is clearly an exception.”).

292. *Id.* at 670.

293. *Id.*

294. *Id.*

295. *Id.; see also* FENNER ET AL., supra note 150, at 1072 (“Germany had considered mass vaccination . . . but had been persuaded by WHO not to implement this operation, on the grounds that it was unnecessary and would be costly and likely to result in many complications.”).
inception of the outbreak no one considered the possibility that airborne transmission might have been involved.

Following the diagnosis of smallpox, the building remained closed to visitors, after having been previously closed due to an influenza outbreak.\footnote{Wehrle et al., supra note 180, at 670.} Paul Wehrle, a WHO researcher tasked with monitoring the smallpox outbreak in Germany, published a seminal report shortly after the epidemic ended. Wehrle did not arrive on the scene immediately and wrote the initial chronology from hearsay reports. However, it is possible he may not have fully understood due to his lack of expertise in the local language and missed significant clues negating airborne transmission, such as the likelihood the nurses and doctors who treated the index case transmitted the disease elsewhere, thereby serving as intermediaries to other patients.\footnote{PRESTON, supra note 149, at 41.} These concerns were never seriously investigated heretofore, as we shall see. Wehrle’s report notes that the index patient had direct contact with a single visitor who later contracted the disease, triggering the first generation of cases.\footnote{Wehrle et al., supra note 180, at 672.} This contention contradicts other reports that the visitor did not enter the patient-care areas nor the isolation unit corridor.\footnote{Id. at 674.} It also fails to account for the fact that the night nurse had contact with the patient and could have transmitted smallpox to other wards, claiming her contact with the index patient was brief.\footnote{FENNER ET AL., supra note 150, at 189 (indicating contact with one nurse, and only for brief periods).} Finally, Wehrle’s report omits discussing significant contacts with other nursing and medical personnel, which will be discussed \textit{infra}. The index patient was responsible for sixteen additional cases on three floors of the hospital unit.\footnote{Wehrle et al., supra note 180, at 669.}

Because the WHO could not identify an obvious human or inanimate mechanism for spread, it assumed that air currents carried the virus.\footnote{FENNER ET AL., supra note 150, at 1115 (explaining that in the few suspected cases of airborne infection, the infected person’s clothing and bedlinens were heavily contaminated with viral secretions that may have served as a secondary source of infection); see also \textit{id.} at 669, 677 (alleging that “seventeen cases were infected by virus particles disseminated by air over a considerable distance within a single hospital building,” showing that Wehrle did not consider the patient’s bronchitis and cough, noting that “patients with more serious smallpox infections transmit infection more effectively than those with a mild or modified illness,” especially when “accentuated by coughing,” and discounting the possibility of person-to-person transmission via the nurses and object-to-person transmission via linens).} Their assumption was based on a smoke test performed several months after the outbreak that was interpreted as consistent with airborne
transmission because the smoke spread along the same pattern as the cases. Wehrle may have misinterpreted the results, however. The smoke plume, used as a surrogate for viral spread, dissipated as it rose, thereby reducing the concentration of smallpox particles as it spread. This would have rendered the patients on the third floor less likely to contract the disease than those closest to the index patient on the first floor, because the third-floor patients would have had less viral exposure. In actuality, more cases occurred on the third floor than the second, and more on the second than the first, which is scientifically contra-factual.

The smoke test—which was performed in April—also relies on the assumption that the index patient’s window was open. In fact, it had been, but just a little. Whenever the patient could sneak a cigarette, he opened the window a crack; when the nurses entered the room, they ordered him to keep the window closed. Whether this was sufficient to allow enough contaminated air to escape and infect patients on floors above is questionable. For the smoke to have entered the patients’ rooms on the second and third floors, however, their windows would have needed to be open as well. There is no evidence to support this; indeed, since the outbreak occurred in January, one might presume their windows were kept shut.

303. Wehrle et al., supra note 180, at 674 (Smoke tests use smoke as a surrogate for viral spread, unleashing the smoke, and monitoring where it meanders, based on the assumption that a virus spreads in the same manner and location. Using smoke as a surrogate for a virus is problematic. For one thing, particle deposition differs; for example, smoke and its particles rise, while particles such as viruses or asbestos tend to fall to the ground in wet conditions).

304. MICHAEL J. HODGSON ET AL., ASHRAE POSITION DOCUMENT ON AIRBORNE INFECTIOUS DISEASES (2009) (offering that it “is useful for understanding the impact of increasing the volume of fresh or disinfected air on airborne infection. Increasing [the volume of fresh air] decreases exposure by diluting air containing infectious particles with infectious-particle free air.”). Hodgson’s analysis would indicate that patients on the third floor would be less likely than patients on the second to have contracted the disease.

305. Wehrle et al., supra note 180, at 674.

306. PRESTON, supra note 149, at 32.

307. Wehrle et al., supra note 180, at 674.

308. PRESTON, supra note 149, at 32.
Wehrle interpreted the results of the smoke test described earlier as consistent with airborne transmission, because the pattern of smoke dissemination was the same as where the cases were found. However, the smoke was used as a surrogate for viral spread and hence, the concentration of smallpox particles should have diluted as the plume rose, rendering the patients on the third floor less likely to contract the disease because they would have had less exposure as the plume dissipated when in fact, as noted earlier, the contrary occurred.

Experimental attempts to replicate Wehrle’s assertions that airborne transmission occurred in Meschede have been unsuccessful. In 1961, “attempts to recover smallpox virus by air sampling in the [smallpox] wards and in close proximity to the patient’s mouth were practically all unsuccessful.” Further, examination of the air samples indicates that virus collected from in front of the patient’s mouth was present in relatively large droplets or particles . . . [but e]ven when talking and coughing, patients with lesions in the mouth seem to eject very little virus in small droplets of the aerosol type.

The frequent failure to find virus in the air samples collected in the impinger was rather surprising. Even air sampled with the impinger held near the mouths of patients who had obvious mouth lesions and who talked or coughed during the period of collection was usually

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309. The above chart accounts for only thirteen patients of the first generation’s seventeen. There may have been greater dissipation on the second floor, but the plume would have been less concentrated and, hence, less infectious. This data is drawn from Wehrle et al., supra note 180. Compare Hodgson et al., supra note 304.

310. Id.

311. Id.

312. A.W. Downie et al., The Recovery of Smallpox Virus from Patients and Their Environment in a Smallpox Hospital, 33 BULL. WORLD HEALTH ORG. 615, 615 (1965).

313. Id. at 617.
negative. In many of these patients, swabs from circum-oral skin and pillow yielded virus. . . . [O]ur results indicate that very little virus is discharged from the mouth of the patient in droplets or droplet nuclei of this order of size when he is breathing, talking, or coughing.314

In virtually all the cases in which health authorities allegedly observed airborne transmission in hospitals, transmission could have occurred through non-human sources, like contaminated laundry and clothing,315 or via carriers such as doctors and nurses. Wehrle ruled out this possibility, asserting that there were no cases in other parts of the hospital “despite the sharing of food-preparing and laundry facilities and frequent movement of staff between the first and second floors of the isolation building and the main hospital building.”316 However, this observation does not eliminate the possibility that inanimate objects or indirect transmission were responsible for infection within the unit.

While Fenner notes that infection “rarely[] seemed to be airborne over a considerable distance.”317 Fenner and Wehrle are adamant that the index case’s contact with the nursing staff was insignificant.318 However, it appears that in addition to the night nurse,319 who Wehrle rejected as a transmission vector because her contact with the index patient was brief—though he did not quantify how brief—two nurses were assigned to him during the day,320 and their cumulative exposure may not have been so minimal. The patient was on antibiotics that were administered multiple times a day by the nurses. He was forbidden to use the bathroom and made to use a bedpan, which meant that nurses entered his room several times a day to empty it.321 Nurses also brought food carts and removed them from the patient’s room several times a day,322 all suggesting that the nurses’ cumulative exposure may not have been as minimal as heretofore assumed. Further, the index patient had a cough which might have accounted for infecting people at a short distance.323 Even Wehrle notes that

314. Id. at 621.
315. W.H. Bradley, Smallpox in England and Wales 1962, 56 J. ROYAL SOC’Y MED. 335, 336 (1963); Downie et al., supra note 312.
316. Wehrle et al., supra note 180, at 673.
317. FENNER ET AL., supra note 150, at 190.
318. Wehrle et al., supra note 180, at 673.
319. Id.
320. Id.; FENNER ET AL., supra note 150, at 192.
321. Wehrle et al., supra note 180, at 670.
322. See id. at 673.
323. Id. at 677.
patients with more serious smallpox infections transmit infection more effectively than those with a mild or modified illness. This is attributed to the fact that such patients are likely to have a greater number of lesions on the mucous membranes, thus shedding larger quantities of virus into the saliva and subsequently into the air. At Meschede, virus dissemination was undoubtedly accentuated by coughing and the virus particles undoubtedly survived in the air for unusually long periods since the relative humidity in the hospital was very low.  

Another overlooked possible mode of transmission is the doctor who examined the index case on admission, Dr. Deiter Enste, and who saw him on at least one occasion thereafter. Later when the patient’s conditioned worsened a number of new doctors examined him. When the patient manifested a rash, the doctors punctured a pustule on his skin, drained the pus onto a cotton swab, and put it in a test tube. A state official then took the test tube to the regional smallpox expert, Karl Heinz Richter, at the state health department in Düsseldorf. This procedure involved both personnel and equipment, both of which might have been infection conduits.

After making the diagnosis, Richter spoke to Wehrle, and Wehrle’s first hand reports only begin at this time. Richter ordered that the patient be transferred to a special isolation-unit hospital thirty miles away. To move the patient, “a squad of attendants dressed in plastic biohazard suits and with masks over their faces ran inside the building and wrapped [the patient] in a plastic biocontainment bag that had breathing holes in it.” But no one can be sure the biohazard suits were secure, and if not, a pinhole or a lapse in safety protocol when removing protective biohazard garb can create an opportunity for infection and transmission.

Even with the claimed and feared airborne transmission, a case-fatality rate of twenty-five percent, and a reproductive rate of seventeen—for the first generation, which went down to two for the second—a total of twenty cases and four deaths occurred in Meschede; a far cry from any of the pandemic projections related to DW.

324. Id. at 677-78.
325. PRESTON, supra note 149, at 31.
326. See id. at 32.
327. Id. at 37.
328. Id. at 38.
329. Id. at 16.
330. See FENNER ET AL., supra note 150, at 192.
331. PRESTON, supra note 149, at 43.
332. Wehrle et al., supra note 180, at 671.
F. Yugoslavia in 1972: Instant Access to Eighteen Million Doses of Vaccine

The most recent smallpox epidemic in an industrialized country occurred in Yugoslavia in 1972. Again, the events mimicked the worst nightmare of DW’s epidemic planners. The outbreak’s unusual and explosive initial presentation resulted in part from the hemorrhagic smallpox suffered by an early case, and the sizeable number of susceptible individuals in the population. The index case infected almost a dozen people before the disease was diagnosed, and he exposed hundreds of others.\(^{333}\) Delayed diagnosis was prevalent, hospital transmission was rampant, unsuccessful revaccinations were common, and communication problems in tracing contacts were considerable.\(^{334}\) The Yugoslav epidemic also had an extraordinarily high replication rate of thirty-eight cases spread by one person during the first of the three generations of transmission,\(^{335}\) leading to an artificial average replication of 12.8.\(^{336}\) In actuality one person in the second generation infected thirty-eight others, while others infected none,\(^{337}\) hence the 12.8 number is a rank artificial average. Conventional mathematical modeling predicted that twenty-one thousand people would be infected and that about seven thousand would die. The facts, however, indicate otherwise. When the epidemic ended, only 175 people had been affected and only thirty-five had died.\(^{338}\)

The epidemic began in early February. The index case, a Muslim cleric, Ibrahim Hoti, who was vaccinated two months earlier, contracted smallpox en route from Mecca to his home in Kosovo, Yugoslavia.\(^{339}\) The cleric was leading a group of twenty-four people on a pilgrimage for the Hajj,\(^{340}\) which was attended by approximately 688,000 people that year,\(^{341}\) about 2700

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335. *Id.*


337. *Id.*


339. *Ctrs. Disease Control & Prevention, International Notes: Follow-Up on Smallpox—Yugoslavia, 21 Morbidity & Mortality Weekly Rep.* 136, 136 (1972) (documenting that Hoti had been vaccinated four times, most recently a year before the incident.).

340. See *Fenner et al.*, *supra* note 150, at 1091.

from Kosovo alone.\textsuperscript{342} In addition to visiting Mecca, the group traveled by bus to Baghdad, which was suffering its own smallpox epidemic, and spent three nights in a hotel there, although Hoti denied having contact with smallpox patients. Hoti also visited a market and dervish sanctuaries in nearby Basra,\textsuperscript{343} where the virus was also registered. While most reports attribute Hoti’s infection to his sojourn in Iraq, it is equally possible that he contracted the disease in Mecca, perhaps from a co-pilgrim.

On February 6, 1972, Hoti and his party returned to his native village of Danjani in Kosovo.\textsuperscript{344} A few days later he manifested signs of illness, but there was limited evidence of rash, and relatives, friends, and acquaintances visited him.\textsuperscript{345} Eleven visitors contracted smallpox between March 1 and 7. None of his twenty-one immediate family members became ill, including his three unvaccinated children.\textsuperscript{346} No one suspected smallpox until March 14, by which time sixteen more people had contracted the disease. They, in turn, infected others. Social conditions undoubtedly contributed to the epidemic’s development.\textsuperscript{347} Specifically, “[f]amilies in which variola was present were usually very poor and lived in hard residential conditions. There was the custom that everyone ate and drank from the same dish and slept together in a bed littered at the floor without boarding.”\textsuperscript{348}

On March 24, Yugoslave health officials notified the WHO, but between March 15 and 30, an additional one hundred cases were diagnosed, constituting the disease’s second generation.\textsuperscript{349} Between April 1 and 11, fourteen more cases were recorded in Kosovo, constituting the third generation.\textsuperscript{350} Between mid-March and mid-April, 124 people were affected and twenty-six died.\textsuperscript{351} One of the cases Hoti infected was a thirty-year-old teacher named Ljatif Muzza, who contracted an especially virulent
form of hemorrhagic smallpox. Muzza was seen in no fewer than four medical institutions, exposing dozens of medical-care personnel with whom he came in close and prolonged contact, although no one suspected he had smallpox. On March 8, Muzza was transferred from Caceck Hospital via ambulance in the company of an unidentified pregnant woman who did not become infected. Retrospective evaluation confirmed the diagnosis, indicating that he had infected a total of thirty-eight patients, eight of whom died. Muzza died two days after admission, on March 10.

Because Muzza had thousands of difficult-to-trace contacts, the government undertook a mass vaccination campaign, which later was extended to the entire country. Within three weeks, eighteen million of the country’s population of twenty-one million were vaccinated. While medical personnel should have been vaccinated previously, this policy had not been implemented, leaving unprotected medical personnel and first responders who had refused vaccination out of fear of the dangers of vaccination.

It is not difficult to understand how a communist country could use martial law to enforce quarantine. The government imposed isolation directives, roadblocks, and other drastic measures, including detaining suspected carriers under armed guard, forcing cancellations of events at which large numbers of people might collect, and compelling mass vaccination. The Yugoslav incident, however, occurred in a population in which language differences impaired communications between the

353. Litvinjenko et al., supra note 338, at 2.
354. Fenner et al., supra note 150, at 1092.
355. Id.
356. Id. at 1094.
357. Lane, supra note 346, at 13.
358. Id. (“The press and medical authorities readily acknowledged that this policy has not been vigorously implemented. Some medical personnel were apparently afraid of vaccination, having received most of their ideas about vaccination from the writings of Herrlich. Magazine and newspaper articles commented on the practice of selling certificates of exemptions from vaccination, and named names of physicians who refused vaccination themselves . . . . The CDC teams performed several surveys of take rates in villages in and around the infected communes of Kosovo. These surveys indicated that from 22 to 30 percent of the population had primary reactions and were thus either previously immunized, or in some instances were aged people with remote primary vaccinations.” This is not as Neanderthal a reaction as might seem. Following the 2001-2 bioterrorist scare, when vaccine was finally released, CDC’s own physicians refused to be vaccinated, and medical personnel and first responders across the nation followed suit.).
359. Guillemin, supra note 333, at 275.
affected population and medical and governmental personnel, a factor that
could have—but did not—negatively impacted results.
Yugoslavia procured more than eighteen million doses of vaccine in less
than two weeks; the country itself had one million doses on hand. WHO
supplied only 500,000 doses as demand was especially acute that year and
it had to supply other countries as well.\footnote{Fenner et al., supra note 150, at 567 (noting that Iran, Iraq, and Syria were also
suffering from outbreaks at that time, and they required seventeen million doses).} A total of an additional 13.5
million doses came from other nations, (see Appendix figure 2) for a grand
total of fifteen million doses.\footnote{Lane, supra note 346, at 30 (showing that Yugoslavia received three million doses
from the former Soviet Union, 2.325 million from Switzerland, 3.5 million from
China, plus additional doses from Canada, the United States, and others; it is
feasible that those same countries continue to keep similar stockpiles to this day,
a possibility that DW does not consider).} From where did Marshal Tito, a Yugoslavian
leader, obtain these and the missing three million doses? It is likely that at
least some of the missing doses were created by diluting existing
vaccines.\footnote{Sharon E. Frey et al., Clinical Responses to Undiluted and Diluted Smallpox Vaccine,
346 NEW ENG. J. MED. 1265, 1265 (2002) (explaining the vaccine is still useful with
1:5 and 1:10 dilutions).} The authors of the DW exercise did not include the possibility
of either dilution or importation of stocks from other countries in their war
game, nor was it raised in any of the post-exercise literature produced.

By the time health officials officially declared the epidemic over on May
9, 1972, 175 cases and thirty-five deaths had been reported.\footnote{Fenner et al., supra note 150, at 1094.} Eight
percent of those who died had previously been vaccinated;\footnote{Litvinjenko et al., supra note 338, at 5.} ten percent
of the cases were the hemorrhagic type, but the overall case-fatality rate
did not exceed twenty percent.\footnote{Id. at 279.} The statistics were attributed to three
factors: first, the frequent and close contact between families;\footnote{Guillemin, supra note 333, at 278 (explaining that the virus spreads among
families because of “the frequent, intimate contact of large extended families in a
rural setting, a demographic feature not typical in the United States, where many
people live alone, but common among immigrants, minorities, and the poor.”).} second,
the multiple hospitalizations fostering in-hospital contact;\footnote{Id. at 279.} and third, the
low rate of vaccination among hospital and medical workers.\footnote{Lane, supra note 346, at 13.}

G. 1973 and 1978: Laboratory Outbreaks

Some questions have been raised regarding whether risks associated
with vaccination outweigh the risk of infection. A secondary risk associated
with vaccination involves dangers inherent in experimentation,\textsuperscript{369} including failure to properly dispose of research specimens, potentially endangering laboratory workers. It must be recognized that such danger cannot be totally eliminated.\textsuperscript{370} In 1973 and 1978, these risks came to fruition in the United Kingdom, causing six cases and three deaths.\textsuperscript{371}

The 1978 laboratory accident further illustrates smallpox's poor capacity for transmission. Following the index case's infection, health authorities identified approximately three hundred people who might have had contact with the patient during her illness, including her family members and relatives, patients of the hospital, and her colleagues at the medical school.\textsuperscript{372} None became ill.\textsuperscript{373}

VI. Analysis

A. Modeling Revisited

In a word, the value of mathematical modeling is limited. As one expert said:

There are efforts to predict the best anti-epidemic measures on the base of mathematic modeling . . . . The quality and predictive value [of mathematical modeling is] limited and depend[s] very much on the inclusion of a sufficient number of necessary and correct parameters. A slight change in a parameter can lead to exaggerated effects that do not follow the common sense experience. A lot of decisive factors can only be roughly estimated, like transmission rate, population immunity or the effectiveness of post-exposure vaccination.\textsuperscript{374}

Factors that vary from epidemic to epidemic affect the pattern, speed, and duration of an outbreak. Using past epidemics to predict future epidemics’ outcomes is thus fraught with problems and danger. Reproductive rates estimated from historical data and outbreaks in developing countries are inapt measure in today’s modern society.\textsuperscript{375} Different release scenarios “yield different results in part because the

\begin{itemize}
\item \textsuperscript{369} See, e.g., \textit{Fenner et al.}, \textit{supra} note 150, at 1095.
\item \textsuperscript{370} See \textit{Jen Christensen, CDC: Smallpox found in NIH storage room is alive}, CNN (July 11, 2014), http://www.cnn.com/2014/07/11/health/smallpox-found-nih-alive/ (explaining how employees found two viable anthrax samples when they were preparing to move a lab to a different location).
\item \textsuperscript{371} \textit{Fenner et al.}, \textit{supra} note 150, at 1097.
\item \textsuperscript{372} \textit{Id.}
\item \textsuperscript{373} \textit{Id.}
\item \textsuperscript{374} Ammon et al., \textit{supra} note 143, at 413.
\item \textsuperscript{375} Milton, \textit{supra} note 90, at 1.
\end{itemize}
parameters and modified natural history of the outbreak itself will vary with the scenarios,\textsuperscript{376} and therefore would prove futile in projecting new estimates. Moreover, it is difficult and expensive to model how quickly smallpox would spread in a developed nation.

Models may have a limited role in deciding appropriate response actions, but mindless attention to modeled data is counterproductive. Even D.A. Henderson cautions against accepting models without scrutiny because they could ultimately create policies that “take a perfectly manageable epidemic and turn it into a national disaster.”\textsuperscript{377}

B. Replication Rate

Mainstream scientists today agree that smallpox’s replication rate is nowhere near the ten to twenty that DW assumes.\textsuperscript{378} Even the CDC strategy is only “based on the assumption that each infected person will infect only two or three others.”\textsuperscript{379} In fact, one patient rarely infects more than a few others, and very rarely without direct or face-to-face contact. Only models or scenarios that rely on outliers, like the Meschede and Yugoslavia outbreaks, disagree.\textsuperscript{380} However, even in those extraordinary cases high replication rates only existed for one generation.\textsuperscript{381}

Moreover, in every real-life epidemic in the last century the reproduction rate decreased sharply after the second generation. Furthermore, in no epidemic was the replication rate linear; after a peak replication rate was reached—generally during the first generation—the replication rate sharply declined. A model’s use of the same replication rate throughout all generations of the outbreak is, therefore, biologically invalid.

The results that the DW fable predicts are therefore a function of arbitrary mathematics, not biology or actual historical events viewed in their entirety.\textsuperscript{382} DW relies on “constantly upward revisions [that] are driven by a cruel epidemiologic statistic—the highest reproductive rate of disease, or average number of new infections each case creates,” selected from the generation with the highest number.\textsuperscript{383} It disregards variations among

\begin{itemize}
\item \textsuperscript{376} Bhatnagar et al., \textit{supra} note 176, at 10.
\item \textsuperscript{377} STANLEY M. LEMON ET AL., ETHICAL AND LEGAL CONSIDERATIONS IN MITIGATING PANDEMIC DISEASE 16 (2007).
\item \textsuperscript{378} See, \textit{e.g.} Weinstein et al., \textit{supra} note 186; Tovey, \textit{supra} note 232.
\item \textsuperscript{379} Bicknell, \textit{supra} note 65.
\item \textsuperscript{380} Guillemin, \textit{supra} note 333, at 278 (“The extraordinarily high transmission rate of 1:12 for the Yugoslavian smallpox outbreak is an unreliable basis for an imagined United States attack scenario, although the extrapolation is commonly made.”).
\item \textsuperscript{381} Bhatnagar et al., \textit{supra} note 176 (noting that the Nigerian epidemic’s high replication rate, 6.9, was due entirely to close household contacts).
\item \textsuperscript{382} See \textit{generally} Dark Winter, \textit{supra} note 86.
\item \textsuperscript{383} Michaud & Kates, \textit{supra} note 35.
\end{itemize}
different epidemics, variations within an epidemic, and other key factors. By selecting the highest reproductive rates available without regard to biological plausibility or historical accuracy, it is easy to game the system, which is exactly what DW did.

C. Delay in Diagnosis

One dreaded concern fueling the DW statistics is an anticipated delay in diagnosis resulting from physicians’ unfamiliarity with the disease, thereby allowing contagious cases to go unnoticed, and poised to launch an infection-avalanche—an alarmist claim suggesting that second or third-generation cases would move about unfettered, thereby exposing others.

Delayed diagnosis, however, would not be a new phenomenon and is not predictive of a runaway epidemic. In historical examples, physicians frequently failed to diagnose smallpox for two weeks or more, and in at least three instances, more than four weeks from presentation of the index case elapse prior to diagnosis. In fact, although it was not unusual to detect the index case until two or three generations of cases occurred, the epidemics were nevertheless rapidly and effectively contained—even when doctors were unfamiliar with the disease.

In Yugoslavia, even though the index case occurred in “one of the country’s least-developed areas,” involved an undiagnosed man who moved from hospital to hospital, and took a month to be diagnosed, the government was still able to effectively contain the outbreak. The same situation occurred in New York. And once diagnosis was made, laboratory confirmation was often delayed taking between weeks, as in Germany, and a month, as in NYC, the epidemic was rapidly contained. Even once clinical diagnosis was made, laboratory delays stalled action. Nevertheless, the epidemics were rapidly contained. Some of these delays in reporting to government officials, which occurred because of delayed laboratory confirmation—sometimes taking upwards of a month, would not occur today, because the turn-around time in modern lab analysis is often less than twenty-four or forty-eight hours.

384. Dark Winter, supra note 86.
385. Wehrle et al., supra note 180, at 676.
386. D. A. Henderson, Importations of Smallpox into Europe 1961-1972, WORLD HEALTH ORG., WHO/SE/74/62,
http://apps.who.int/iris/bitstream/10665/67627/1/WHO_SE_74.62.pdf; see Litvinjenko et al., supra note 338, at 7. (The physicians had no personal experience in its diagnosis, epidemiology, or control. Thus, there was a delay before the correct diagnosis was made even though the clinic picture of the first generation of cases was typical.).
387. FENNER ET AL., supra note 150, at 1077.
388. See, e.g., Leonard F. Peruski, Jr. & Anne Harwood Peruski, Rapid diagnostic assays in the genomic biology era: detection and identification of infectious disease and
In summary, because it takes weeks to infect a substantial number of people, even delays in diagnosis should not prevent effective containment. And delays in diagnosis would be somewhat compensated by the rapidity of today’s laboratory assessments, which was unavailable in the past.

D. Infectivity and Spread

The recent interest in smallpox bioterrorism has provoked discussion about the parameters that govern the virus’s transmissibility. Using historical data from a 1967 epidemic in a previously unvaccinated group in Nigeria, one set of prominent researchers found that these data supported “the widely held belief that smallpox spreads slowly, mainly among close contacts, and that infectivity before the onset of rash was negligible.”

“Extensive studies in both endemic and non-endemic countries” that show that close, prolonged, and personal contact are necessary predicates to smallpox transmission. These studies “clearly indicated the necessity for close, and often prolonged, personal contact before transmission occurs.” The few cases of transmission without direct contact are the rare exception, rather than the rule.

As noted earlier, whether an individual contracts smallpox depends on whether she has had enough exposure to reach the threshold dose, which helps explain why brief contact usually fails to induce illness. Indeed, exposure to low doses of smallpox, the practice of variolation widely practiced in the eighteenth and nineteenth centuries, was once the preferred means of preventing the disease.


389. Id. at 677.
390. Eichner & Dietz, supra note 73 (internal quotations omitted).
392. Id. at 2.
393. Id.
394. Heiner et al., supra note 119, at 1, 5.
395. Id. at 5 (“Contacts whose duration of exposure was seven days or more showed significantly higher attack rates than persons exposed for less than seven days.”).
397. See David A. Freedman, On types of scientific inquiry: The role of qualitative reasoning, Statistics at UC Berkeley, at 2, http://www.stat.berkeley.edu/~census/anomaly.pdf (“As a preventive measure, patients could be deliberately infected (through scratches on the skin) with minute quantities of material taken from smallpox pustules, the idea being to induce a mild case of the disease that would confer immunity later.”) (last visited Apr. 1, 2017).
Even in the Yugoslavia incident, secondary cases originated from a single source because the patient had long and intimate contact with susceptible people.\textsuperscript{398} As Fenner states: “virtually all cases in the last century arose in the context of hospital/medical treatment or intimate contact with cases in affected households.”\textsuperscript{399}

It is critical to keep in mind that contrary to popular belief, smallpox is far less infectious than measles and various strains of flu.\textsuperscript{400} Most smallpox experts believe that “smallpox is a barely contagious and very slow-spreading infection.”\textsuperscript{401} A former director of the smallpox-eradication unit at the CDC, Michael Lane, argues that “the way it spread in Dark Winter was ‘silly’” and that “[t]here’s no way that’s going to happen.”\textsuperscript{402}

\textbf{E. Susceptibility}

Another concern is population susceptibility, leading to the fear that Americans would be easy prey. In the outbreaks studied, poor population immunity did not result in run-away epidemics.\textsuperscript{403} Moreover, counterbalancing factors of modern industrialized life-styles would likely compensate, such as better medical care, intervention and nutrition.\textsuperscript{404} The existence of

\begin{itemize}
\item [m]icro-organisms are necessary but not sufficient in the causation of infectious disease and other causal factors are required for infection to occur . . . . Indeed, the decline in infectious diseases in high-income countries is more readily attributed to increased host
\end{itemize}
resistance from better nutrition than to a reduction in the virulence of the relevant micro-organisms.\textsuperscript{405}

And while “[m]ost people in the United States today lack immunity, which could make matters much worse than in the past. On the other hand, they don’t live in crowded and squalid conditions, like many of the disease’s . . . victims, which could reduce transmission rates.”\textsuperscript{406}

\section*{F. Case-Fatality Rates}

Major texts assert that smallpox has a worldwide case-fatality of twenty percent,\textsuperscript{407} although this figure is an average, and based on historical data without specification of which years and which locales are being considered. While in the past centuries in unindustrialized countries without modern medicine smallpox was known to produce a case-fatality rate of thirty percent, using that figure for pandemic planning today is plainly inappropriate. In point of fact, in post-WWII non-endemic countries the case-fatality drops to sixteen percent or less.\textsuperscript{408}

The United States’ experience with smallpox is comparatively benign, even before the age of modern medicine. In the Boston epidemic of 1721, 844 of 5980 infected people died, producing a case-fatality rate of fourteen percent.\textsuperscript{409} According to the CDC, in 1900 there were more than 21,000 cases, with a case-fatality rate of four percent.\textsuperscript{410} Between 1900 and 1904, smallpox caused an average of 48,164 cases and 1528 deaths,\textsuperscript{411} producing a case-fatality rate of three percent. The high case-fatality rate DW assumes is not in line with historical epidemics.

\section*{G. Spread by Air Travel}

One of the most feared means of international dissemination is international air transport, a mode of transportation that has increased in

\begin{itemize}
\item 405. Roberts, supra note 155, at 660.
\item 406. Enserink, supra note 85, 1593.
\item 408. See, e.g., FENNER ET AL., supra note 150, at 1077.
\item 411. Id.
\end{itemize}
use dramatically over the last decades. Some public-health practitioners assume that international air travel would increase the likelihood of a pandemic because of the high number of potential contacts in an airport setting or on an airplane. They claim those who are exposed might continue to spread the disease upon arrival at their respective destinations and to casual contacts along the way. In fact, the data points otherwise. Though air travel has considerably increased in recent years, outbreaks have been far less frequent than in the past.

Contrary to DW’s assumptions, air travel is not a new development:

[i]n the early nineteen fifties airports were concerned with only hundreds of thousands of passengers each year; whereas in 1961 more than 100 million passengers used the international routes, and at London Airport there were 6 million passenger movements.

The longer plane trips of the 1960s and 1970s would have been more conducive to effective transmission than today’s shorter, more frequent trips. As more people traveled between North America and places where smallpox was still endemic, some believed that “there was a constant and increasing risk of importing smallpox into North America . . . [but o]nly 1 importation of smallpox into North America was reported—a single case of variola minor imported from Brazil into Canada via the USA.”

Even though “[t]wenty-seven of the 29 importations [into Europe] from overseas for which data are available were associated with air travel,” no transmission occurred on an airplane. Between 1950 and 1973, there were at least fifty smallpox outbreaks in Europe that originated in ten different countries from the Indian subcontinent, Africa, and the Middle East. These outbreaks resulted in “1113 cases and 107 deaths . . . Twenty-four of the index patients from these outbreaks travelled by air, two by sea, and 1 by land. About one-half of these cases [generated in the epidemic] occurred by nosocomial transmission among hospital personnel and their contacts, hospital patients, and visitors.” In none of them did transmission occur en route.

412. Wehrle et al., supra note 180, at 676.
413. Id. at 669.
415. FENNER ET AL., supra note 150, at 1081.
416. Id. at 1074.
418. Id.
419. FENNER ET AL., supra note 150, at 1081 ("As international air traffic increased in the 1960s and 1970s, and many more people travelled from Canada and the USA to Africa, South American and Asia, in which smallpox was still endemic, there was a
In 1962, the United Kingdom, receiving increasing numbers of airline passengers, reported five smallpox outbreaks triggered by five travelers arriving from Karachi, Pakistan. At the time, “special charter flight arrangements were in vogue to move these people quickly, cheaply and in maximum numbers by air,” yet investigators found that “[t]here is no evidence that they infected any of their fellow travellers [sic].” In addition to exposing fellow passengers on the flight, other opportunities for exposure included taxi or train rides from the airport. There is no evidence that such transmission occurred.

In most countries during the post-WWII years, passengers from endemic areas were not allowed to disembark without a certificate proving they had received a smallpox vaccination. No epidemic or pandemic resulted, even though “[i]n 1960 just over 2 million passengers arrived at London Airport who were [without any documentation proving that they had been vaccinated].” The Ministry of Health stated that, in retrospect, one might remark upon the good fortune that these did not apparently infect anyone during flight; for the aircraft, carrying maximum pay-loads, were full and a first generation might well have been infected before arrival, if recent vaccination had been unsuccessful, and have subsequently dispersed widely over the country before their own, possibly vaccine-modified, smallpox came to light.

Even super-spreaders like the Yugoslavian patient did not spread the illness to any contacts on either his bus ride . . . or his ambulance ride. This keeps unblemished the European experience that smallpox has not been transmitted on a common carrier such as a bus, plane, or train. The contingency plans for a United States importation should consider such transmission highly unlikely.

constant and increasing risk of importing smallpox into North America . . . . [However, o]nly 1 important of smallpox into North America was reported—a single case of variola minor important from Brazil into Canada via the USA.”).
H. Airborne or Weaponized Transmission

DW-type projections depend on aerosol transmission via weaponized smallpox, obviating the need for long duration and close contact. Based on this possibility, one researcher promised that

[i]n&egrave;rted terrorists could expose people in several cities (and on public transportation between cities) before becoming visibly ill. Identifying such cases of exposure within the four-day period is logistically impossible. Furthermore, aerosolized dispersion of the virus could make the situation much worse . . . . A single case of smallpox in the Yugoslav outbreak required the administration of 18 million doses of vaccine to arrest the spread of disease in a population with substantial immunity.428

To date, fifteen years after DW, attempts to weaponize smallpox appear to have failed. Further, while it cannot be absolutely refuted that aerosol transmission may have a role in spread, that role, if it does exist, is minor.429 And the Meschede outbreak is the only one in “which a large group of cases at a considerable distance from the index patient appears to have been infected as a result of transmission of the virus by air”430 or by large particle or droplet infection.431 Even those who contend that airborne transmission is possible concede that transmission only occurs during active infection, i.e., when a rash is visible.432

In summary, an effective bioterrorist-instigated airborne transmission would require the confluence of two rare events: an initial release via airborne transmission and the consequent spread by patients without a visible rash. The statistical odds of two such unlikely events acting contemporaneously are highly remote.

428. Bicknell, supra note 65, at 1323.
429. Milton, supra note 90 (“Mack . . . emphasized that 85% of cases had clear-cut exposures to known cases. However, the remaining 15% had no obvious exposure suggesting that a small number of more distant or casual contacts transmitted infection as would be expected if smallpox were transmitted by dilute virus aerosols . . . . Some well-known hospital-associated outbreaks make it clear that airborne transmission at a distance of more than a few feet did occur occasionally. But, these examples were rare” (citations omitted)).
430. Wehrle et al., supra note 180, at 677.
431. Milton, supra note 90 (“[L]ead&egrave;ng authorities disagree regarding the relative importance of fine and large particle routes of transmission; some state that smallpox was transmitted primarily via airborne droplet nuclei, while others emphasize ‘face-to-face’ contact and state that, airborne transmission was rare” (citations omitted)).
432. NEW YORK DEP’T HEATH, supra note 190.
I. Hype, Hyperbole, Hysteria, and the Press

Notwithstanding historical data and experimental evidence demonstrating smallpox’s poor transmission potential, the hype continues. As Fenner writes, “[d]espite the prompt measures usually taken by the public health authorities, the deep-rooted if sometimes unfounded fears of the population were difficult to quell,” causing several countries to suffer considerably.\textsuperscript{433}

Hyperbolic media reports can worsen the public’s fear; the ramifications of inaccurate reporting and baseless opinions should be carefully considered in any public discussion—including wargames—especially when enacted for lay politicians and media types. The real problems of an epidemic, however, may not be related to the disease itself, but to its economic after-effects. Perhaps if doctors and scientists were included in the “DW experience,” they might have come up with more innovative response measures.

Dr. Derrick Tovey, who managed the Bradford incident, states that the most difficult part of his job was handling the press, because “[t]he national press was often irresponsible, printing such headlines as ‘City in Fear!’ ‘Keep Out Pakistanis’, [sic] but the local press, particularly the Telegraph & Argus, was helpful and reported accurately the local position which was of ‘refusal to panic’. [sic]”\textsuperscript{434} In the NYC episode, the press, following Weinstein’s lead, was exemplary: restrained, yet informative. At times the press can be the best antidote for addressing concerns and allaying hysteria; it can be used as an effective tool in motivating desired population behavior and conveying real-time information, or if used counter-productively, the worst adjuvant, creating or exacerbating infectivity and counter-productive behavior.

Thus, instead of the prudent calm that Tovey and Weinstein dispensed, today’s pundits, both scientific and political, seem to relish in hysteria-mongering. Cloaked in the do-goodism of motivating the government to be prepared, the press drives the hysteria by advising the government to obtain ill-advised vaccines for mass vaccination.\textsuperscript{435} Interestingly, Tara

\textsuperscript{433} FENNER ET AL., supra note 150, at 1077-78 (“[i]n the first few days after the recognition of the large outbreak in Yugoslavia, the country was in turmoil: people were afraid to visit public places until they and their families had been protected by vaccination; trucks carrying market-products from affected areas were turned back; tourist bookings were cancelled; and some countries closed their borders and advised their nationals not to visit Yugoslavia.”).

\textsuperscript{434} Tovey, supra note 232, at 246 (emphasis added); Eric Butterworth, The 1962 Smallpox Outbreak and the British Press, 7 RACE & CLASS 347, 351, 355 (2004) (noting that the kernel seeding the hysteria was not the disease itself, but legislation reform restricting immigration or visits of foreigners, sparked by the index case being Pakistani).

\textsuperscript{435} See, e.g., PRESTON supra note 149, at 58 (“It has taken the world twenty years to reach roughly fifty million cases of AIDS. Variola could reach that point in ten or twenty weeks. The outbreak grows not in a straight line but in an exponential rise,
O’Toole, the co-creator of DW, did accept some responsibility for hysteria-mongering, but she also asserts that the media, too, is responsible. At the end of the DW exercise, she asserted that “there were still fewer dead than at Pearl Harbor.”

J. Vaccine Availability

O’Toole, in explaining DW, stated that one reason the DW scenario “was so bleak . . . is that the scenario provided only 15.4 million doses of vaccine,” which she inaccurately believed was the U.S. stockpile at the time. In fact, DW tells participants there were only twelve million doses available. But the actual available doses should have been more than enough to quell the outbreak if used responsibly, which the audience could not have known.

DW also assumed that, in 2002, the United States government could not obtain more doses of the vaccine, even though—over forty years ago—Yugoslavia procured more than eighteen million doses in less than two weeks. In reality, in 2002, the DOD had between seventy-five and ninety million doses that were hiding in DOD’s closets—their existence was only revealed after the DW exercise and the allocation of government funding. O’Toole also failed to consider that dilution is effective, and would have rendered the entire exercise moot, because the United States had the capacity to produce enough diluted vaccine for every American at the time. But the actual available doses should have been more than expanding at a faster and faster rate. It begins as a flicker of something in the straw in a barn full of hay, easy to put out with a glass of water if it’s noticed right then. But it quickly gives way to branching chains of explosive transmissions of a lethal virus in a virgin population of non-immune hosts. It is a biological chain reaction.

436. Enserink, supra note 85, at 1593 (“‘We have to take some of the responsibility’ for giving smallpox an extremely scary reputation, concedes O’Toole.”).
437. Id. (citation omitted).
438. Id.
439. Dark Winter, supra note 86.
440. Id.
441. See Flight, supra note 352.
444. Id.
445. Interview by Victoria Harden with Dr. Anthony S. Fauci, Director, Nat’l Inst. Allergy & Infectious Diseases (Aug. 9, 2002),
enough to quell the outbreak if used responsibly through ring vaccination (see infra, next page), which the lay audience could not have known. Moreover, the dilution possibility was only tested after the new vaccines were ordered and found effective.

K. Vaccine Hysteria

To investigate the realism of tying hysteria to lack of vaccine, I pose the following four questions:

a. Would inadequate vaccine engender hysteria as Dark Winter predicts?

The answer depends on how the shortage is handled. When Health Commissioner Weinstein faced the problem in NYC in 1947, he was able to avoid the hysteria. Footage from a Germany epidemic demonstrates the same thing: no hysteria. To Today’s politicians and press could achieve the same result through hysteria-prevention tactics. In fact, taking lessons in hysteria prevention from the DW exercise might have been a better outcome than procuring 300 million doses of vaccine.

b. Was an approach other than mass vaccination available?

Mass vaccination is not the only option; there is another approach, ring vaccination, that was used to eradicate endemic smallpox in the 1970s. Ring vaccination targets only individuals in an area with suspected exposure, forming a ring around the outbreak, and effectively choking it off. Since the smallpox vaccine poses a real danger, ring vaccination would be a preferable approach, in that it results in fewer vaccinations.


448. Id.

449. WILLIAM CHARNLEY, EMERGING INFECTIOUS DISEASES AND THE THREAT TO OCCUPATIONAL HEALTH IN THE U.S. AND CANADA 242 (2006) (“When the Bush administration announced support for mass vaccinations, WHO did not change its position, but the CDC . . . decided to acquiesce . . . . Despite efforts to avoid vaccination of those who might be at elevated risk, the CDC reported that there were 145 serious adverse events . . . . Of these cases, at least three were deaths.”); see also Bozette et al., supra note 50, at 419 (confirming that ten simultaneous attacks at airports waged by skilled bioterrorists is one of the only scenarios that would produce enough risk to outweigh the dangers of vaccine).

450. Flight, supra note 352 (“Although mass vaccination calmed fears, it was not always the most medically efficient way to combat the disease. Henderson and his team developed a strategy of containment and surveillance. Every time there was an outbreak, a WHO team would arrive, vaccinate and isolate those who were ill and trace and vaccinate all their contacts. Effectively they ring-fenced the disease until
c. Could the United States have gotten more vaccine sooner?

In 1972, Marshal Tito had eighteen million people vaccinated in about two weeks, obtaining the vaccine he needed from other sources: he had one million doses on hand; the WHO supplied an additional half million. But from where did Tito get the remaining 16.5 million doses so quickly? The answer: China, Switzerland, the United States, the U.S.S.R., the United Kingdom, Canada, Albania, Bulgaria, France, Greece, the Netherlands, West Germany, East Germany and Switzerland. If Tito could do this, isn’t it likely the United States could tap other governmental sources as well? It appears that none of the DW conference organizers or participants considered this.

To be sure, there are likely fewer available doses stockpiled around the world today, but collecting vaccine from other countries would be a start in the event of a smallpox outbreak, a possibility DW does not consider. And if O’Dwyer was able to convince seven vaccine manufacturers to produce six million doses in 1947, one is hard pressed to believe we would be unable to duplicate that feat today.

d. Is vaccine dilution feasible?

Some months after the hysteria of DW died down and after the government had already contracted for vaccine production, research concretely established that the smallpox vaccine can be effectively diluted. Even operating under DW’s erroneous assumption that there were only twelve million doses available at the time of the exercise, the vaccine could have been diluted enough to serve the American population. Using a five-to-one dilution factor means that seventy-five million doses would have been immediately available, which is more than necessary for ring vaccination; at a ten-to-one dilution factor, there would have been enough vaccine for two-thirds of all eligible individuals.

it had no way of moving on to its next victim.”); see also Henderson, supra note 149.


452. Lane, supra note 346, at Appendix (showing 500,000 doses came from WHO, 3 million from the USSR, 3.25 million form the USA, 2.3 million from Switzerland, 3.5 million from China, and several million more doses from the other countries). See infra Appendix.

453. Smallpox Vaccines, WORLD HEALTH ORG., http://www.who.int/csr/disease/smallpox/vaccines/en/ (last visited Apr. 4, 2017) (stating that there is a stockpile of about thirty-one million doses available worldwide that can be obtained on an emergency basis).

Even more significantly, data supporting the viability of dilution were available before the exercise. For example, in 1962, in the wake of the Bradford incident, all university students in Britain were offered vaccination, but there was not enough vaccine to inoculate everyone and, thirty-three percent of the students had never been vaccinated. To solve this problem, the vaccine was successfully diluted on a 1:30 basis, leading health officials to conclude that “a titer of 1:30 can be used effectively in an emergency.” Assuming that only fifteen million doses were available, titering by 1:30 would have furnished 450 million doses, enough for one-and-a-half times the American populace.

That O’Toole blames the hysteria on the roiling waters of vaccine insufficiency is problematic, worrisome and costly—costing over two billion dollars. Even when 500,000 doses were made available to first responders, the vaccines went unused because health professionals did not want to be inoculated. Fewer than eight percent of CDC eligible personnel participated, asserting that the vaccine involved too many adverse events.

L. Who makes decisions on the data to use?

A mass-casualty smallpox epidemic is unlikely in modern America. However, there is still some debate about whether bioweapons pose a threat—and if so, how much. Some academics believe that the threat is inevitable, while others “pose questions about the realistic capabilities of terrorists and if bioweapons truly fit their organizational goals.” Those who believe that a bioterrorist threat is unlikely “emphasize the technical barriers that prevent bioterrorism from being used as successfully as bombs and other traditional tactics.” However this may be “a question better answered by the Central Intelligence Agency, and not epidemiologists.”

455. See Douglas & Edgar, supra note 248, at 614.
456. Vaccination in a University, supra note 114, at 614.
457. Id.
458. Id; see also Douglas & Edgar, supra note 248, at 614.
459. See Charney, supra note 449, at 242 (“The vaccination campaign did not proceed as planned. Opposition arose on both safety and political grounds.”).
460. CDC Vaccine Acceptances, CTR. DISEASE CONTROL & PREVENTION (on file with author).
462. Id.
463. Enserink, supra note 85, at 1593.
Conclusion

In summary, the goal should be that once an outbreak begins, policy questions can be answered using real-time modeling,

not just ‘most likely’ or ‘worst case’ scenario modeling results are communicated but that a more detailed understanding of the sensitivity of predictions of predictions of outbreak size and policy optimality to model assumptions is conveyed, together with open acknowledgement of model or data weakness. Once an outbreak has begun, such questions may also be answered more precisely through the use of real-time modeling to refine parameters and better inform policy.464

Many experts believe that “bioterrorism preparedness programs have been a disaster for public health.”465 Currently, most epidemics from all agents are modeled identically, without regard to individual microbiological parameters for different agents, including both physical requirements and biological propensities, such as growth rate, survival, particle size, and transmission characteristics, which are species-dependent, and extrinsic factors such as weather conditions, public health capacity, and population health. In other words, biological and physical constraints are not incorporated into the models. Further, the worst-case-scenario model often used for pandemic planning is a mathematical construct that bears no relation to reality or to relevant epidemic history.

Models generating cataclysmic projections rely on seriously skewed and artificially manipulated data.466 The lure of personal gain for manipulating the data is huge, both financially and academically. Institutional bias also plays a role in determining policy. Federal spending on biodefense has increased and “the Department of Defense is currently at the center of initiatives to enhance federal capabilities to respond to biological terrorist threats.”467

Even where the modelers have no personal or institutional interest, choosing correct data is fraught with uncertainty. In the words of the head of the Department of Mathematical Biology at Imperial College, “[g]iven the many such uncertainties . . . no model can be truly predictive in the context of smallpox outbreak planning, and no one control method can be identified

464. Ferguson, supra note 91, at 685.
465. CHARNEY, supra note 449, at 245 (citation omitted).
466. It is not surprising, therefore, to find one consultant, the Chief Executive Officer of a risk assessment company working with both the DOD and the Federal Bureau of Investigation, stating that he “think[s] the chance is about eighty percent of terrorists obtaining smallpox.” He asserts that “experts” think that “the potential for a serious smallpox attack is frighteningly feasible.” Mary Jacoby, Terrorist expert cites smallpox risk, ST. PETERSBURG TIMES, Sept. 18, 2001, at 3.
467. Ajayi, supra note 52.
a priori as best.” Ferguson argues that, while the usefulness of models for precise prediction is limited, they should have a role in planning, assuming the modelers work with actual, regularly updated data, and relate the math to the experiences of the population. And “for models to have a meaningful role in influencing policy decisions,” it is critical that policymakers communicate “not just ‘most likely’ or ‘worst case’ scenario modelling [sic] results,” but also a “detailed understanding of the sensitivity of predictions of outbreak size and policy optimality to model assumptions . . . together with open acknowledgement of model or data weakness.”

While the usefulness of models for precise prediction is limited, models should have a role in planning, provided they work with real, up-to-date, and regularly updated data, and relate the math to the experiences of the population. Projections that grotesquely overestimate the incidence and severity of a potential epidemic also generate a climate of fear and suspicion regarding the government’s ability to accurately predict and prepare for bioweapon attacks, diverting resources from necessary but less glamorous quotidian public-health concerns.

It appears that to obtain precise or reliable information, we must wait until an epidemic begins. Contrary to many fears, this is not a tragedy. Smallpox’s reputation as catastrophic “seems unwarranted under modern conditions.” In reality, experts have said that “it has appeared as a plodding nuisance with more bark than bite.” A smallpox outbreak “would offer, regardless of precautions, fewer biological and administrative challenges to local workers than will . . . diseases such as polio or diphtheria.” An outbreak, even one as severe as the one envisioned by the DW gods, could have been, and could still be, more safely addressed.
Appendix

<table>
<thead>
<tr>
<th>Nation</th>
<th>Doses of Vaccine</th>
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<tbody>
<tr>
<td>Albania</td>
<td>200,000</td>
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<tr>
<td>Bulgaria</td>
<td>600,000</td>
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<tr>
<td>France</td>
<td>225,000</td>
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<tr>
<td>Greece</td>
<td>300,000</td>
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<tr>
<td>Netherlands</td>
<td>250,000</td>
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<tr>
<td>People’s Republic of China</td>
<td>3,500,000</td>
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<tr>
<td>West Germany</td>
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<tr>
<td>Eastern Germany</td>
<td>60,000</td>
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<tr>
<td>Switzerland</td>
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</tr>
<tr>
<td>USA</td>
<td>3,251,000</td>
</tr>
<tr>
<td>USSR</td>
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<tr>
<td>WHO</td>
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Figure 2. Vaccines Acquired by Yugoslavia from Other Nations

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<td>1</td>
<td>1</td>
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<td>1</td>
<td>20</td>
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<tr>
<td>Case fatality</td>
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<td>16.6%</td>
<td>23%</td>
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<td>30%</td>
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<td>3</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Max R gen 1</td>
<td>3</td>
<td>10</td>
<td>4</td>
<td>4 + 2 2 + 2</td>
<td>16</td>
<td>16 &gt; 17</td>
</tr>
<tr>
<td>Max R gen 2</td>
<td></td>
<td></td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>38 + (62) &gt; 100</td>
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<td>Max R gen 3</td>
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<td></td>
<td></td>
<td>1 + 1</td>
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<td>1 (47)</td>
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<tr>
<td>Average R</td>
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<td>-3</td>
<td>1</td>
<td>&lt; 1</td>
<td></td>
<td>7</td>
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<tr>
<td>Attack rate</td>
<td>.000002%</td>
<td>.00003%</td>
<td>??</td>
<td>.0001%</td>
<td>Uncontrollable</td>
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<tr>
<td>Source/Distance</td>
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<td>Karachi</td>
<td>Bugdud</td>
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<td></td>
<td></td>
<td>Asia</td>
<td>? Karachi</td>
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<td>Mexico?</td>
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<tr>
<td>Actually infected</td>
<td>12</td>
<td>14</td>
<td>25</td>
<td>19</td>
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<td>6</td>
<td>4</td>
<td>4</td>
<td>35</td>
<td>1,000,000</td>
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</table>

Figure 3. A statistical compilation of data from post-WWII epidemics. The last column is the DW model.

477. Lane, supra note 346, at Appendix.

478. Data compiled from actual statistics of post-WWII epidemics compared to the forecasted (projected data) of the Dark Winter Exercise.
Figure 2: U.S. Government Funding for Emerging Infectious Diseases, FY 2012 – FY 2015

![Bar chart showing funding for various infectious disease programs from FY 2012 to FY 2015.]

NOTES: Abbreviations mean Centers for Disease Control and Prevention (CDC), U.S. Agency for International Development (USAID), Department of Defense (DoD). "n/a" means the information is not publicly available currently. * indicates President’s Budget Request.

SOURCE: Kaiser Family Foundation analysis of data from congressional budget justifications, appropriations bills, the U.S. Foreign Assistance Dashboard [www.foreignassistance.gov], communication with GEIS staff, and AHHC 2013/14 Annual Report.

Figure 4. U.S. Government Funding for Emerging Infectious Diseases, FY 2012-2015

479. Figure 2: U.S. Government Funding for Emerging Infectious Disease, FY 2012 — FY 2015, KAISER FAMILY FOUND., https://kaiserfamilyfoundation.files.wordpress.com/2014/10/8514-03-figure-2.png.

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