On Tuesday evening, April 28, 2009, Darrell Galloway was alone in his condo in Alexandria, Virginia, watching television and trying to unwind after work. His wife was in southern Utah, where they have a house, and where they hoped to retire soon. Galloway was a senior official at the Pentagon’s Defense Threat Reduction Agency, and for days he had been going to meetings about a new strain of influenza from Mexico that was spreading fast. The strain, which combined genes from humans, swine, and birds, had become known as swine flu. Earlier that month, two children in Southern California had caught it. Then the virus swept through a high school in Queens; more than a hundred students with symptoms were sent home. The Obama Administration had declared a national public-health emergency. That night, Galloway watched news reports from Mexico City about overcrowded hospitals and closed schools; an estimated hundred and fifty people had died. He telephoned his eldest son and urged him not to make a planned trip to Mexico.

Galloway is sixty-four years old. He is a short, athletic man with a welcoming but serious manner, like that of an amiable high-school baseball coach. The son of an intelligence officer, he was inspired to become a scientist by the launch of Sputnik and the space race that followed. In his spare time, he is an amateur astronomer, and he has built a small observatory in his back yard in Utah. He and a group of friends love to tinker with three old Soviet MIG fighter jets that they keep in a hangar nearby.

A former professor at Ohio State, Galloway is a microbiologist, and knew the grim history of influenza, a virus that often mutates faster than the body’s immune system can respond to it. The pandemic of 1918 infected a third of the world’s population and may have killed as many as fifty million people. In 2003, a strain of avian influenza emerged in Asia that was particularly lethal to humans, and the possibility that it could cause a human pandemic was a source of constant worry. But the virus did not spread between humans and remained confined largely to birds. Swine flu was a new, similarly threatening strain.

The Defense Threat Reduction Agency was created after the Cold War to protect the United States from weapons of mass destruction and to help other countries deal with the dangers of loose nuclear, chemical, and biological weapons. Galloway was authorized by the military to work on a specific set of threatening diseases that were considered potential weapons in war or in terrorism, including anthrax, smallpox, tularemia, plague, and the Ebola and Marburg hemorrhagic fevers. Influenza, Galloway said, “was outside my lane.” But countering it would test the government’s ability to respond quickly to a biological threat. That night in April, he resolved to do something about the looming pandemic.

The next morning, when Galloway arrived at work, he summoned his staff and announced that they were to begin work immediately on creating a new antiviral drug to combat the swine flu. “I said, ‘What are we waiting for?’ ” Galloway recalled. “This is about as real as it is going to get.”

A day later, at a meeting in the Pentagon, Galloway ran into stiff objections. Several officials said that it was a mistake for the military to get involved in the swine-flu outbreak. Galloway felt that the government was reacting too slowly to the spread of the pandemic. “I finally got fed up and blew my stack,” Galloway told me. “I said, ‘I didn’t come here to ask anybody’s permission to do this. I have done it.’ ” He got up and left, and the meeting broke up. Afterward, no one tried to stop him.

The Biological Weapons Convention of 1975 outlawed germ warfare. But in the nineteen-nineties two events unnerved the Pentagon. It was revealed that the Soviet Union had built a vast, illicit germ-warfare program, and that a Japanese cult, Aum Shinrikyo, had experimented with anthrax. The September 11th attacks increased the fear that terrorists could acquire dangerous pathogens; the anthrax letters in the weeks that followed raised the alarm. Former President George W. Bush, in his memoir, writes that, in October of 2001, while he was travelling in China, a White House pathogen detector went off, indicating the presence of deadly botulinum toxin. Vice-President Dick Cheney, his face pale, spoke with Bush in a video conference to inform him, saying, “The
chances are we’ve all been exposed.” It turned out to be a false alarm, but, Bush writes, “at the time, the threats were urgent and real.”

Between 2001 and 2010, Congress approved fifty billion dollars to protect against biological threats. In addition, a special reserve fund of $5.6 billion, known as Project BioShield, was created in 2004 to help build a national stockpile. But after several years it became clear that money was not solving the problem.

David Franz, the former commander of the United States Army Medical Research Institute of Infectious Diseases, told me, “We can’t afford it. We realize now how much it costs to make one vaccine for one pathogen. It is enormous, especially when you don’t know if you are ever going to need it.”

In 2006, the Pentagon ordered an unusual five-year research initiative to counter germs being used as weapons of war or terror, and assigned Galloway to launch it. Instead of targeting pathogens one by one, an approach known as “one bug, one drug,” the initiative would seek to invent therapeutic drugs and vaccines that could counter multiple germs. They would also develop new processes that could be used to quickly create drugs and vaccines to fight previously unknown pathogens. Galloway and another official called it the Transformational Medical Technologies Initiative.

T.M.T.I. set extraordinary expectations for itself. An official description said that it would “spark another medical revolution,” similar to the mass production of penicillin in the Second World War, and declared that it might create new drugs and vaccines “within days.” As Galloway envisaged it, T.M.T.I. would start with basic research and go as far as possible toward developing a new drug or vaccine. No other single government agency was trying to do anything quite so ambitious.

Galloway faced huge obstacles. The Defense Threat Reduction Agency had plenty of nuclear experts on its staff, but there were few people there with experience in microbiology or biotechnology. Critics argued that the program had overstated its capabilities. Michael T. Osterholm, the director of the Center for Infectious Disease Research and Policy at the University of Minnesota, told me that T.M.T.I.’s plans to create drugs rapidly were the result of “wishful thinking,” and were “like trying a moon shot in ten minutes.” Bringing a new commercial drug or vaccine from laboratory to market in the United States can take ten to fifteen years and cost more than a billion dollars.

The process of winning Food and Drug Administration approval, as the Pentagon has pledged to do with any drug or vaccine given to troops, involves preclinical testing in laboratory animals and three phases of clinical trials with human volunteers.

Nonetheless, Galloway pressed ahead. According to Patrick J. Scannon, the founder of a biotech firm called XOMA, who was an adviser to the military on biological issues during the Clinton and George W. Bush Administrations, “The Defense Department was creating a drug company.”

While Galloway was setting up the program, concern about biological war and terror waned slightly. No weapons of mass destruction were found in Iraq, and although Al Qaeda had attempted to work with anthrax before September 11th, it had not got very far. There hadn’t been a terrorist attack using biological agents. But there were numerous dangerous outbreaks of naturally occurring infectious diseases around the world. Between late 2002 and mid-2003, a virus that causes severe acute respiratory syndrome, or SARS, spread from southern China to twenty-eight countries and killed nearly eight hundred people. Then came avian influenza and swine flu, also known as 2009 H1N1.

There are two medical ways to deal with influenza: vaccines, which are given to healthy people before they are infected; and antiviral drugs, which can suppress the virus after infection, and give the body’s immune system time to regroup and recover. Galloway focused on drugs because the existing antivirals had a limited impact. Tamiflu, one of the leading products, can shorten the duration of flu by only a day or two, and the swine–flu strain was already resistant to two other antivirals, developed in earlier years. It was possible that it would become resistant to Tamiflu as well.

Galloway had assembled a technical staff for the T.M.T.I. program, and he had an early success with a drug to fight the Ebola and Marburg viruses. He saw swine flu as an opportunity to do more. “I wanted to prove that the program worked,” he told me. He also wanted to accomplish something tangible, if not strictly about war or terrorism. “How would it look if the government had a way to do this and we just sat on our hands?” he said. “If my job is to build a capability to respond to any unknown virus, how about this one?”

Another small team of scientists and medical experts within the Defense Department shared this sense of urgency. They had been trying for several years to modernize the way vaccines are made, and, during the pandemic, they decided to try to build a swine–flu vaccine using an entirely new method. Galloway was focussed on treatment; this group pursued prevention, under a program they called Blue Angel.
Vaccines are potentially the most powerful tool for preventing widespread illness and death from a virus. But they can be very difficult to create. Since the nineteen-fifties, there has been one F.D.A.-approved way to manufacture flu vaccines: inserting weak forms of the virus into chicken eggs. The egg-based vaccine depends on six discrete steps, and takes at least six months, or longer, to produce. The Centers for Disease Control and Prevention started preparations for a new vaccine in April, right after swine flu entered the United States. But a second wave of influenza would likely begin in four months, at the end of the summer.

The White House was concerned that the vaccine wouldn’t be ready in time for a pandemic. President Obama had just taken office, and aides worried about the prospect of a public-health disaster in his first year. Was there an alternative way to get a vaccine? The White House Homeland Security Council and the Office of Science and Technology Policy sent out a series of e-mail queries to government scientists in late April. One of them went to Michael Callahan, a physician specializing in infectious diseases and rapid response who works at Massachusetts General Hospital and at the Defense Advanced Research Projects Agency, or DARPA.

Callahan, who is forty-eight years old, thrives on practicing medicine under austere conditions in forbidding places. In earlier years, he served as an expedition doctor: climbing mountains and slogging through jungles with teams of explorers. One of his current projects is to help acclimate U.S. soldiers to the mountains of Afghanistan. “My thing is altitude and disasters,” he told me. When we met, he had just finished a three-hour stint as a doctor in the Afghan mountains, where he helped treat soldiers to the mountains of Afghanistan. “My thing is altitude and disasters,” he told me. When we met, he had just finished a night of hospital duty and was running on three hours of sleep. He repeatedly jabbed the button on a coffee machine, gulped down three cups of espresso, and chewed on candy-size tabs of an experimental nutritional supplement that could detect who would or would not become sick, days before symptoms arrived. Another, known as MIMIC, could model the human immune response in a test tube, creating a swift way to check the efficacy of vaccines. A third was SAVE, a relatively inexpensive ventilator designed for the battlefield, which was the size of two large paperbacks and could be widely used by civilians in places such as school gyms if hospitals were overcrowded.

But the centerpiece of the program was an effort to modernize and speed up the production of vaccines. Instead of using chicken eggs, Callahan wanted to insert genetic code into specially grown tobacco plants. The code would cause the plants to generate viral proteins, and these could then be made into the active component of a vaccine. Tobacco is fast-growing and easy to manipulate genetically; in theory, once you have inserted the genetic code of the virus, the plants can quickly make pure and safe proteins in huge quantities. No one had yet made a vaccine for the public this way, nor had there been human clinical trials to determine if the vaccine could induce immunity in large numbers of people. But the Obama Administration was looking at all possibilities.

The White House contacted Callahan on April 28th. According to his records, officials requested a timeline with worst-case, medium, and optimistic projections for using the tobacco-plant method to manufacture hundreds of millions of doses of vaccine. Just then, Callahan was planning a “live-fire exercise,” an experiment in which he would use tobacco plants to try to make the active components of an avian-flu vaccine. He quickly substituted swine flu. The experiment was to be carried out by the Center for Molecular Biotechnology, in Newark, Delaware, a nonprofit branch of Fraunhofer U.S.A., a subsidiary of the large German applied-research and technology organization. Callahan sent a fragment of the swine-flu genetic code by e-mail to the executive director of the center. The center, using the tobacco plants, produced a purified protein in twenty-one days. This wasn’t a finished vaccine, but it suggested that the process of making one could be rapidly accelerated.

By contrast, it proved hard to grow the weakened strain of swine flu that could be placed in eggs. When it was finally shipped to the manufacturers, they found that the growth in the eggs wasn’t optimal for large-scale production. The manufacturers tweaked the strain, but not until the end of June were they ready to begin mass production.

On June 11th, the World Health Organization raised the alert level to six, meaning that a full-blown global pandemic had begun.

Galloway kept an eye on Callahan’s work, but he stayed focused on building a drug, not a vaccine. The first step was to acquire a full genetic blueprint of the swine-flu virus, and, to do this, he turned to Ian Lipkin, one of the leading detectives in the viral and bacterial world. A professor at Columbia University and the director of the Center for Infection and Immunity at the Mailman School of Public Health, Lipkin scrutinizes hundreds of pathogens every week, sifting the genetic codes for clues to their origins, behavior, structure, and identity.

Viruses are barely life forms. They infect a cell, hijack its machinery to replicate themselves, and then escape to infect new cells. The genes of the influenza virus are carried in RNA, or ribonucleic acid. Unlike many other viruses and organisms, the influenza virus does not correct genetic errors when it replicates, so it produces offspring that are not identical. The slightly different versions of the viral genome collectively resemble a swarm. To sequence the virus’s genes, Lipkin needed to scan as many versions as possible. Then he lined up the data to create a “consensus” snapshot of the swarm.

On April 30th, Lipkin and his staff acquired a specimen of the virus taken from the school outbreak in Queens. On May 1st, they began to sequence it. Every hour or so, they telephoned or e-mailed a progress report to Paula Imbro, a geneticist at the Tauri Group, in Alexandria, Virginia, who had been providing advice to Galloway’s T.M.T.I. program for more than a year. Lipkin’s staff sent Imbro the swine-flu sequence when it was finished. It looked like a piece of fine embroidery—tiny dots of green, blue, yellow, red, and purple. Lipkin had sequenced the virus in thirty-one hours.

Galloway had solved the first major problem in developing his drug. To deal with the next, he chose Patrick Iversen,
a scientist at AVI BioPharma, a small bio-tech company in Corvallis, Oregon. They had worked together on the Ebola and Marburg viruses. On May 3rd, the swine-flu sequence arrived in Iversen’s e-mail in-box. The son of a national-park ranger, he is a hefty man, fifty-five years old, with a handlebar mustache and a gentle voice. He earned a Ph.D. in pharmacology at the University of Utah, and later became an assistant professor at the University of Nebraska. From his early days in science, Iversen was fascinated by the possibility that a chemical substance could target a precise location in genetic material, such as that of a virus or a tumor, and change its behavior.

Iversen’s main research involved a technology known as “antisense,” which was first discovered in the nineteen-eighties. The process involved chemically synthesizing a short strand of DNA or RNA that could precisely interlock with a sequence found in a natural virus, like one Lego block attached to another. The natural strand was known as the sense strand, and the synthetic one as the “antisense” strand. If the antisense strand could attach tightly enough and in the right place, it would become a wrench in the gears of the genetic machinery, and stop the virus from replicating. The strand, in theory, could thus be turned into a powerful antiviral drug.

In the nineteen-eighties and nineties, Iversen wrestled with the forbidding obstacles in antisense technology. One of the most difficult was to deliver the antisense strand to the right place at the right moment, after the virus had penetrated the cell, but before it had replicated and escaped to infect other cells. To accomplish this, the synthetic strand must be non-toxic, and it must not interfere with other genetic processes in the body. It must be potent enough to be effective and strong enough to resist rapid degradation. It must bind tightly to the invading virus. Each step in the process is complicated. Some of the early hopes for antisense technology were later dashed, and one scientific paper in the late nineties declared, “The technology remains in its infancy.” According to Cy Stein, of the Albert Einstein-Montefiore Cancer Center, in the Bronx, who also began working with antisense in the eighties, “The concept is the best idea since the hole in the toilet seat. But, in making this happen, there is one barrier after another that nature puts up to prevent you from doing what you want to do.”

In 1997, Iversen left Nebraska and joined AVI BioPharma, which had pioneered antisense chemistry. In his first years there, he tried to figure out how to use antisense to combat major diseases such as AIDS and cancer. But after September 11, 2001, he became preoccupied with viruses and terrorism. He had been planning to fly to New Jersey that day, but his flight was cancelled and the drive home from the airport took three hours. Along the way, he thought about the potential use of viruses: “I just thought, you know, flying a plane into a building—for a sort of low cost, you create a very high-cost event. If I were a terrorist, I would do a virus. This came to me as I was driving home, thinking, Things are a lot scarier if you could take a dog with some zoonotic virus and let him go in some neighborhood and the next thing you know people are tying up the whole medical system.”

Iversen’s new focus soon led him to obtain a patent on using antisense to target four major virus families. In some virus families, certain parts of genetic code appear the same across several species. These locations are known as “highly conserved regions,” meaning that they do not change from one strain of influenza virus to another. If he could target them, he thought, antisense technology could knock out our different strains.

In 2002, the West Nile virus infected two dozen Humboldt penguins at the Milwaukee County Zoo, killing eleven. Iversen called Roberta Wallace, the senior staff veterinarian at the zoo, offering to synthesize an antisense compound against West Nile virus if she would give it to the remaining sick penguins. When Wallace agreed, he took the sequence of the virus from a database, designed the compound, and sent it to her in a vial. She injected it into three sick penguins. The birds survived the infection.

The success of the injection provided only anecdotal evidence that antisense could work, and Iversen was eager to find a more difficult challenge. On February 11, 2004, he made a presentation to the U.S. Army Medical Research Institute of Infectious Diseases, at Fort Detrick, Maryland, the Army’s premier laboratory for biodefense research. Hours later, a researcher at Fort Detrick accidentally stuck herself in the thumb with a needle while injecting mice with the Ebola virus. Ebola has gruesome symptoms that often cause the victim to bleed to death; there is no licensed vaccine or therapeutic drug to stop it.

While the terrified researcher was put in isolation, in a complex known as the Slammer, two cinder-block patient rooms that were hermetically sealed and filled with monitoring equipment, laboratory officials called Iversen. They wanted to know how rapidly he could synthesize an antisense compound against the Ebola virus. He quickly designed compounds based on the genetic sequence. Chemists worked for two days to synthesize it. In a telephone conference call, the F.D.A. gave emergency approval for use of the untested drug. The president of AVI BioPharma flew to the East Coast, carrying the vial. In the end, the researcher did not come down with Ebola, and she did not need Iversen’s drug. But the rapid response persuaded everyone involved, including Iversen and the Army laboratory, to launch a major new research effort into antisense and viruses.

One of the most enthusiastic participants was the researcher who had had the accident. She joined Iversen, and others in the lab, to create, test, and modify antisense compounds to counter viruses, including Ebola and Marburg. The first generation wasn’t potent enough; the second generation had problems with toxicity. With the third generation, the scientists had something to boast about. In 2006, they published the results of a trial in which seventy-five per cent of the monkeys given an antisense compound survived infection with the Ebola virus.

Galloway’s programs had funded some of the Ebola and Marburg research, just as T.M.T.I. was getting started. He told me he knew that the antisense technology was working against those exotic viruses, and he felt confident that it would work against others. In the first hour after he launched the swine-flu effort, Galloway instructed his staff to call Iversen and invite him to join them. Iversen immediately agreed. On May 5th, the T.M.T.I. program, on Galloway’s orders, rushed a $4.1-million contract to AVI BioPharma. This kind of speed is almost unheard of in defense contracting. An accompanying memo from T.M.T.I. said it was possible that the experimental drug could be designed and found effective
“in four to six weeks.” After that, “millions of doses” could be produced in time for the fall wave of swine flu. The memo warned that a pandemic could cripple military deployments, but it said nothing about the time required for testing by the F.D.A.

Iversen puzzled over the genetic sequence in the first days after he received it. He could not find the precise site he needed in order to attach the antisense compound. Lipkin had produced a richly detailed genetic blueprint, but the highly conserved region that Iversen needed was cloudy, as if covered with translucent tape.

Within days, Imbro, the geneticist, had helped Iversen find a trove of swine-flu genetic sequences in a European database. They worked together on the phone—Iversen in Oregon and Imbro in Virginia—scrutinizing more than a thousand sequences each. Finally, Iversen recognized the precise place where he could attach his compound.

With the chemists, he attempted to create a synthetic compound that would latch on to the viral RNA in the right place, and stay there. On May 14th, he phoned the T.M.T.I. program: he and the chemists had succeeded. Afterward, Iversen held the substance in a small glass vial. It was fluffy and white. On closer examination, you could see that it was made up of spindly rods that seemed clean and pure.

Randall Kincaid, a biologist and entrepreneur who that week had become the scientific director of T.M.T.I., described Iversen’s work: “He distilled the background of influenza, the intricacies of the virus itself, and the types and strategies that would likely work or not work. I thought, Man, this is a guy who either has thought about this for a long time or is incredibly smart and in a few days came up with this. Either way, it is great.”

Engineering the compound did not mean that Iversen and Galloway had found a workable drug. The next step was to test the substance in laboratory animals, starting with mice and moving on to ferrets, which are extremely susceptible to influenza infection and develop some of the symptoms seen in humans. After the blistering pace of events in May, the animal tests were delayed, week after week. There weren’t enough ferrets, and reams of paperwork were needed to obtain permissions from special committees, which sometimes meet only once a month.

In July, the influenza project faced a worrisome new crisis. In Argentina, the mortality rate among patients with swine flu began to soar. It was nine times as high as elsewhere. If the virus had mutated into a much more dangerous strain, it would make the egg-based vaccine useless, and it could also force Iversen to start over. Most people who had come down with swine flu in the spring had survived; a mutation could mean that the death rate would be much higher when the virus returned in full force in the fall. Several specimens from Argentina were rushed to Lipkin’s laboratory, at Columbia University, and the first analysis was carried out over the July 4th weekend.

The researchers, entering the lab for dangerous pathogens, donned heavy protective suits. It turned out, however, that people in Argentina were being infected not only by the swine-flu virus but, at the same time, by streptococcus, a bacteria.

In August, Iversen got the laboratory-mice tests under way, but the first weeks were frustrating. The mice were set out in groups of ten and infected with a strain of influenza. Then one group was given the antisense compound, a second group Tamiflu, and a third a saline solution. Some mice in the early tests died unexpectedly because they were being given too much of the compound too fast. In the fourth round of tests, the scientists made the compound more concentrated but the doses less frequent, and the results looked promising. The treated animals had lower concentrations of virus and did not lose weight, as the animals usually do when they’re sick. “By the time we had finished with the mice, there was a high degree of optimism,” Kincaid told me.

Subsequent tests, in September, showed that infected ferrets that got Iversen’s compound had dramatically lower levels of swine flu than those who didn’t get it. The antisense compound also outperformed Tamiflu. “You know, ferrets sneeze, ferrets’ eyes get all runny, their noses get stuffy—they look like they’ve got the flu!” Iversen told me. “The first ferrets we treated were perfect. We had no sneezing, no runny nose, no stuff in the eyes, activity scores were perfect, and the sick guys were sick. So, you know, pretty cool.”

In Washington, on September 28th, Kincaid appeared before more than thirty government officials involved in the swine-flu crisis, from both civilian and military agencies. At this point, the second wave of the pandemic had arrived, but the conventional egg-based vaccine was still weeks away from delivery, and the Blue Angel tobacco-plant-based vaccine remained untested. A White House report warned that the pandemic could place “enormous stress” on the public-health system and cause between thirty and ninety thousand deaths in the United States in the coming months.

In a windowless Pentagon conference room, Kincaid spoke extemporaneously. He acknowledged that Galloway and T.M.T.I. didn’t have a formal mandate to create a new antiviral drug, but “we saw an opportunity to see whether or not we could.” He described the good news from the four rounds of tests on mice. The exercise had shown “the potential for this capability to respond rapidly to an emerging threat,” Kincaid said, according to his notes. But he was taken aback at the first question: Can you make fifty million doses?

Kincaid insisted that it was just an exercise; the group was not ready to make fifty million doses. The officials pressed him about the remaining obstacles, and he told them that there were two main problems: how to give the drug to a large population, and how to get it approved by the F.D.A., which the group had not even consulted during the exercise. “People were energized,” Kincaid said. “They saw the spindly white compound in Iversen’s vial as a real response to a deepening crisis.”

The questions didn’t stop there. “We pushed it very hard,” said Andy Weber, the assistant to the Secretary of Defense for Nuclear and Chemical and Biological Defense Programs. Weber, who took office in May of 2009, told me that, if needed, the new drug and the new vaccine would have been taken immediately into human clinical trials. “We realized in dealing with this declared national emergency that we needed a Plan B and a Plan C,” he said.

In the year after the pandemic began, swine flu infected between fifteen and thirty per cent of the population of the United States. But it was not as lethal as many officials had feared it would be. As a result, neither Galloway nor Callahan got
that a rapid response to a biological emergency must be accompanied by an equally rapid process for testing and approval. “There is no sense having brilliant, rapid science without it,” he said. The F.D.A.’s existing system of approval for new drugs, one of the most rigorous in the world, does not move quickly. Emergency fast-track procedures exist, but they are cumbersome. In the case of the most dangerous agents, such as Ebola, it is neither feasible nor ethical to run clinical trials on humans. Licensing for drugs and vaccines against these deadly agents must be based on testing in laboratory animals, despite the limitations of such work. If the country were engulfed by a deadly pandemic or bioterrorism attack, there would be questions of what risks to take, and whether people would be better off with or without a new drug or vaccine that had not been tested in humans.

The Obama Administration’s strategy calls for improving “regulatory science,” which means finding new methods and tools that can help the F.D.A. reach judgments more quickly. One idea is to look for indicators, known as biomarkers—such as protein levels in blood—that can allow you to make an early diagnosis or accurately predict a drug or vaccine’s effectiveness. Another proposal is to create F.D.A. “action teams” of experts and have them start working early with scientists who are developing a high-priority drug or vaccine.

At the end of 2009, Galloway retired from the Defense Threat Reduction Agency, moved to Utah, and set up a small biodefense consulting company. This past December, AVI BioPharma submitted a formal request to the F.D.A. for approval to begin clinical trials of the antiviral. Meanwhile, there still is not a highly effective antiviral drug for dealing with swine flu. Over the past few months, a new wave of swine flu has hit Britain, sending more than seven hundred people to hospitals. Britain released 12.7 million doses of the pandemic vaccine for immediate use. Wide usage of the vaccine will stop the disease from spreading, but it isn’t going to do much for the people who are already sick. Their instructions are to stay hydrated and warm, and not to go to school or work. A similar outbreak occurred this winter in Cairo.

These outbreaks will probably be contained soon. But no one knows when the next deadly pathogen will show up and whether we’ll be able to respond rapidly to it. Patrick Scannon told me that Galloway was right to act when he did. “Where could you hide if the flu turned rogue, and was Tamiflu-resistant?” he said. “The only thing left is to grab masking tape and a bottle of water, and lock the door. What is the responsible federal official going to do? Say, ‘Let’s not rush into this? What if you were wrong?”

In the event of another crisis, Galloway’s gamble may have pointed the way toward a rapid response. “What has been missing is an example,” Scannon said. “This was the first.”