An Engineered Doomsday

Scientists have long worried that an influenza virus that has ravaged poultry and wild birds in Asia might evolve to pose a threat to humans. Now scientists financed by the National Institutes of Health have shown in a laboratory how that could happen. In the process they created a virus that could kill tens or hundreds of millions of people if it escaped confinement or was stolen by terrorists.

We nearly always champion unfettered scientific research and open publication of the results. In this case it looks like the research should never have been undertaken because the potential harm is so catastrophic and the potential benefits from studying the virus so speculative.

Unless the scientific community and health officials can provide more persuasive justifications than they have so far, the new virus, which is in the Netherlands, ought to be destroyed. Barring that, it should be put in a few government-controlled laboratories with the highest containment rating, known as biosafety level 4. That is how the United States and Russia contain samples of smallpox, which poses nowhere near the same danger of global devastation.

In the future, it is imperative that any such experiments be rigorously analyzed for potential dangers — preferably through an international review mechanism, but also by governmental funding agencies — before they are undertaken, not after the fact as is happening in this case.

The most frightening research was done by scientists at the Erasmus Medical Center in Rotterdam, who sought to discover how likely it is that the “bird flu” virus, designated A(H5N1), might mutate from a form that seldom infects or spreads among humans into a form highly transmissible by coughing or sneezing. Thus far the virus has infected close to 600 humans and killed more than half of them, a fatality rate that far exceeds the 2 percent rate in the 1918 influenza pandemic that killed as many as 100 million people.

Working with ferrets, the animal that is most like humans in responding to influenza, the researchers found that a mere five genetic mutations allowed the virus to spread through the air from one ferret to another while maintaining its lethality. A separate study at the University of Wisconsin, about which little is known publicly, produced a virus that is thought to be less virulent.
These findings led to an unprecedented request from an American federal advisory board that the researchers and the two scientific journals that plan to publish the studies omit any details that might help terrorists figure out how to unleash a devastating pandemic. That presumably includes details on how the engineered virus was made and details on the precise mutations that allowed it to go airborne.

We doubt that anything at all should be published, but it seems clear that something will be.

The two journals reviewing the papers seem inclined to follow the advisory board’s recommendations that the research be published in a redacted form, provided there is some way for researchers who need the information to gain access to the full details. The Erasmus team believes that more than 100 laboratories and perhaps 1,000 scientists around the world need to know the precise mutations to look for. That would spread the information far too widely. It should suffice to have a few of the most sophisticated laboratories do the analyses.

Defenders of the research in Rotterdam claim it will provide two major benefits for protecting global health. First, they say the findings could prove helpful in monitoring virus samples from infected birds and animals. If genetic analysis found a virus somewhere that was only one or two mutations away from going airborne, public health officials would then know to bear down aggressively in that area to limit human contact with infected poultry and ramp up supplies of vaccines and medicines.

But it is highly uncertain, even improbable, that the virus would mutate in nature along the pathways prodded in a laboratory environment, so the benefit of looking for these five mutations seems marginal.

A second postulated benefit is that the engineered virus can be used to test whether existing antiviral drugs and vaccines would be effective against it and, if they come up short, design new drugs and vaccines that can neutralize it. But genetic changes that affect transmissibility do not necessarily change the properties that make a virus susceptible to drugs or to the antibodies produced by a vaccine, so that approach may not yield much useful new information.

We cannot say there would be no benefits at all from studying the virus. We respect the researchers’ desire to protect public health. But the consequences, should the virus escape, are too devastating to risk.