Current Topics

Déjà Flu—Round 2: Key Scientist Points to H5N1 Misperceptions

Regarding claims about a modified H5N1 influenza virus that could move via airborne droplets from ferrets to infect others nearby, “there is a misperception that it would spread like wildfire,” says Ron Fouchier of the Erasmus Medical Center in the Netherlands. “We don’t know the efficiency of its spread.” Further, the modified virus is lethal for ferrets only when a “lot” is administered, he says. “If received via the aerosol route, we do not see severe disease [in ferrets]. We don’t think it will be a virus that will kill half the world’s [human] population.” He and several others who are debating H5N1 risks spoke during a special session convened as part of the 10th ASM Biodefense and Emerging Diseases Research Meeting, held last February in Washington, D.C.

Some experts say this new information deserves careful review, and others greet it with skepticism. “There was a perception, however it got there, that all ferrets [infected with the modified H5N1] died,” says Anthony Fauci, Director of the National Institute of Allergy and Infectious Diseases (NIAID) and another special session participant. “Ron [Fouchier] says that’s not what happens.” A strict confidentiality agreement makes the task of describing or interpreting the combination of Fouchier’s older but “clarified” and “new” data “difficult.”

Several months ago, the members of the U.S. National Science Advisory Board for Biosecurity (NSABB) painstakingly reviewed the Fouchier findings along with those from Yoshihiro Kawaoka of the University of Wisconsin, Madison, and his collaborators. “We spent hundreds of hours and gave [the research] an in-depth review,” says NSABB member Michael Osterholm of the University of Minnesota, Minneapolis. That review led many NSABB members to conclude that the modified flu viruses were both lethal and highly contagious among ferrets—and, potentially, a major or even catastrophic public health risk for humans.

In light of the now-expanded findings and also in the wake of a consensus statement from a committee of the World Health Organization (WHO) calling for the Fouchier and Kawaoka research reports to be published in full (Microbe, April 2012, p. xxx), NSABB will likely be reconvened. “We want to give NSABB members the same [materials] we had in Geneva [at the WHO committee meeting],” Fauci says. Other plans call for reviewing other potentially dual use research supported by the National Institutes of Health.

For now, there is “great uncertainty, and the parameters are not well enough defined to make an ultimate decision” about publishing the Fouchier and Kawaoka papers in full form or resuming the research, says NSABB acting chair Paul Keim, who is from Northern Arizona University in Flagstaff. “It’s time to have a broad discussion.”

“It is critical to understand how viruses go from one species to another,” Fauci says, noting that this issue was part of the “rationale for why these studies were done.” However, scientists remain sharply divided on pursuing this research. Some argue for a halt, others argue for a screening system to identify and stop such research at the review proposal stage, while others are seeking a way to continue experiments while safeguarding public health.

Jeffrey L. Fox
Jeffrey L. Fox is the Microbe Current Topics and Features Editor.

Sticky Extracellular Matrix Helps Biofilms Grow up, Collapse, Spread outward

The extracellular matrix (ECM) plays an unexpectedly important role in the spread of biofilms, according to mathematician Michael Brenner and his collaborators, including microbiologists and physicists at Harvard University in Cambridge, Mass. In particular, the exopolysaccharide (EPS) component of the ECM creates osmotic pressure that forces biofilms to swell upward and sprawl outward, they say. Further, Bacillus subtilis mutants that cannot secrete EPS mirror predictions in their mathematical model.

Because the ECM is a mesh of sticky proteins and sugars, many researchers thought it likely slows biofilm growth whereas bacterial flagella seem designed to propel biofilm expansions. However, B. subtilis mutants lacking flagella spread at about the same rate as cells with active flagella, when viewed through time-lapse microscopy. This expansion is so powerful that it increases the radius of some biofilms fivefold within 24 hours. In contrast, the growth of B. subtilis mu-