

AIR SAMPLER MAY SPEED DIAGNOSIS OF BIRD FLU

Researchers at the California Animal Health and Food Safety (CAHFS) Laboratory working with colleagues at the Lawrence Livermore National Laboratory are testing air sampling technology to speed up accurate detection of avian influenza, a viral disease that has spread from Southeast Asia to the Middle East, Europe and Africa, resulting in the deaths of millions of birds and at least 127 humans.

The scientists have tested the Smart Air Sampler System, a portable, lightweight air sampler about the size of a household coffee pot, to collect airborne viruses. The equipment, manufactured by Research International Inc. of Monroe, Washington, eliminates the need to catch and swab individual birds for biological samples. Diagnosticians then screen the samples using a rapid DNA test. Experimenting with the air sampler and rapid screening, Dr. Sharon Hietala of the CAHFS and her colleagues have detected two different bird viruses.

This technology—which can trim diagnostic time down from the current 3–12 days to only six hours—could be advantageous in veterinary diagnostics by minimizing human contact with potentially infected flocks, and reducing both the time and cost of collecting and testing samples from individual birds.

The air sampling units, if placed in high-risk environments such as poultry farms or live bird markets, could make a huge difference in animal disease detection and even public health response, says school dean Bennie Osburn. “If global health officials take the next steps to fund training and place the tool at strategic sites, this diagnostic technology may help us battle avian influenza worldwide.”



Scientists hope, using the Smart Air Sampler System, to be able to sample hundreds to thousands of birds in as little as 10 minutes without handling or disturbing the flocks.

Researchers Identify Feline Gene Mutation That Causes Heart Disease

A spontaneous gene mutation responsible for a devastating heart disease in cats was identified in Maine coon cats and reported last October by a team of researchers at UC Davis, The Ohio State University and Baylor College of Medicine.

The disease, hypertrophic cardiomyopathy, also a leading cause of sudden death in young human athletes (as in the case of Boston Celtics’ Reggie Lewis in 1993 and Loyola Marymount University basketball player Hank Gathers in 1990), causes an excessive thickening of the muscle of the left ventricle of the heart. Hypertrophic cardiomyopathy in humans and Maine coon cats appears microscopically as poorly aligned muscle fibers, thought to interfere with the electrical activity of the heart. The disease occurs in one out of every 500 humans and is the most common heart disease in domestic cats. Hypertrophic cardiomyopathy is most frequently diagnosed in non-purebred middle-aged cats, although it has been diagnosed in cats ranging from 1 to 13 years of age.

“The prognosis for severely affected cats is often poor,” says veterinary cardiologist Mark Kittleson, a co-author of the study. “Preventing the disease from occurring by identifying affected cats before they are bred can save a lot of heartache.”

Dr. Kittleson says the finding paves the way toward developing a screening test to help breeders rid Maine coon cats of the gene mutation, and may provide a valuable model for investigators in both veterinary and human medicine. The study began when Maine coon cat owner Marcia Munro notified Dr. Kittleson that she knew of several cats related to her cat that also had the disease. The study was supported by the Winn Foundation and its Ricky Fund, the Center for Companion Animal Health and private donors.

Toxicology Study Links Thimerosal to Immune Dysfunction in Mice

Collaborating cell biologists, toxicologists, pathologists and molecular bioscientists at UC Davis published a study in March that links immune system dysfunction in mice with thimerosal—a cheap and effective mercury-based preservative. Potential effects on embryonic neuron development led to the removal of thimerosal from many pediatric vaccines, but it is still used in influenza, diphtheria and tetanus vaccines, blood products and many over-the-counter pharmaceuticals.

The study shows how communication between calcium channels in mouse dendritic cells is dramatically garbled when exposed to thimerosal, reducing the immune system’s ability to respond to external factors.

“Dendritic cells play pivotal roles in overcoming viral and bacterial invaders by coordinating the immune system’s overall combat response,” says senior author of the study Isaac Pessah, toxicologist with the UC Davis School of Veterinary Medicine and director of the Children’s Center for Environmental Health and Disease Prevention. One dendritic cell can activate as many as 300 T-cells—white blood cells that help find and kill external agents that attack the immune system.

At concentrations as low as 20 parts per billion, thimerosal initiates inflammatory responses, and at concentrations of 200 parts per billion causes death of dendritic cells. Affected dendritic cells can quickly become “rogue,” producing misinformation that could activate aberrant and harmful immune responses. “Even one rogue dendritic cell can activate many inappropriate immune responses,” says Dr. Pessah.

“Our findings do not directly implicate thimerosal as a single causative agent for triggering neurodevelopmental disorders such as autism,” he says. “There is growing evidence that autism is several disorders that we now refer to as just one, and that some children with autism have unique immune cell composition and responses to antigens. The results of our work provide a framework to test the hypothesis that the genetic background of some individuals may render them especially susceptible to thimerosal.” Dr. Pessah will next study dendritic cells in humans with and without autism. The mouse study was funded by the National Institute of Environmental Health Sciences and the UC Davis M.I.N.D. Institute.