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New faces at the College of Veterinary Medicine

We are fortunate to have several talented new faculty members at K-State. They are here to accept referrals and discuss cases of interest, as well as teach and conduct research. Please make them feel welcome as they become part of the K-State veterinary family.

Laurie Beard is an associate professor in equine internal medicine. She received a D.V.M. degree from Washington State University and master's from Ohio State. She has also worked at the Ohio State University, Columbus State University, C. Richard Nelson University, the University of Georgia, and Washington State University veterinary schools. She is a diplomate in the American College of Veterinary Internal Medicine. Her primary clinical interests include endocrinology and geriatric equine medicine.

Laurie Beard

Warren Beard is a professor of equine surgery. He received bachelor's and D.V.M. degrees from Texas A&M University, and a master's degree in equine surgery from Ohio State University. He remained on faculty at Ohio State as assistant professor and associate professor before coming to K-State. He is a diplomate in the American College of Veterinary Surgeons. His clinical interests include all aspects of equine surgery with a special emphasis on upper respiratory surgery, head and sinus surgery, and gastrointestinal surgery.

Warren Beard

Melanie Boileau is an assistant professor in agricultural practices. She received a D.V.M. degree from the University of Montreal in Quebec, Canada, and a master's degree from Oklahoma State University. She is a diplomate in the American College of Veterinary Internal Medicine. Her interests include food animal neonatology, bovine obstetrics and clinical pharmacology.

Melanie Boileau

Shane DeWitt, assistant professor, equine field service, received a D.V.M. degree from the Atlantic Veterinary College at the University of Prince Edward Island. He is a diplomate in the American College of Veterinary Internal Medicine. His areas of interest involve equine neonatology/critical care and preventative medicine, with specific interests in respiratory diseases and fluid balance in critically ill patients.

Shane DeWitt

Paul Jenson is an assistant professor in equine emergency care. He received a bachelor's degree from Utah State University, a D.V.M. degree from Washington State University, and a master's degree in equine surgery from K-State. He was previously in private practice in Utah before coming to K-State. His interests include all aspects of equine emergency health management.

Paul Jenson

Jill Lurve, assistant professor, small animal medicine, is a graduate of the University of Minnesota College of Veterinary Medicine and received a master's from Auburn Univer-

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Also in this issue

Thank you to the Pfizer Animal Health Group, Livestock Division, Cattle Products Group, for financial assistance in publishing this newsletter.

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sity. She did an internship in small animal internal medicine and surgery at the University of Illinois and was a visiting assistant professor and research fellow at Auburn University. She is a diplomate in the American College of Veterinary Internal Medicine. Her primary interests are in endocrinology. Specific interests include diabetes mellitus, adrenal and thyroid diseases, and endocrine tumors of dogs and cats.

Jill Lurve

Eric Moore recently joined the agricultural practices section with the Department of Clinical Sciences as a clinical instructor/postgraduate trainee. Moore is a 1994 graduate of the K-State College of Veterinary Medicine and has practiced in Oberlin and Great Bend, Kan. In addition to his clinical teaching and service duties, Moore will complete a master's degree program.

Eric Moore

Daniel Thomson, assistant professor, production medicine, is a third-generation veterinarian. He received a bachelor's degree from Iowa State University, a master's degree from South Dakota State University, a Ph.D. from Texas Tech University, and D.V.M. degree from Iowa State University. He worked internationally with neonatal and newly weaned bovine and swine nutrition for the American Protein Corporation. He was an associate veterinarian with Veterinary Research and Consulting Services in Greeley, Colo. He then supervised animal health at 10 commercial feedlots and directed animal health research at Cactus Feeders in Amarillo, Texas. His interests include the interactions between production management, environment and nutrition on the health and performance of beef cattle.

Daniel Thomson

Beware of winter health hazards to pets

This article was modified from "Winter and Holiday Health Hazards for Animals" by Pam Wilson RVT, published in the November 2004 issue of "Zoonosis News from Health Service Region 1" from the Texas Department of State Health Services, Canyon, Texas.

There are special health hazards for animals in the winter and during holidays, and some are fatal. To keep pets safe protect them from contact with or ingestion of these items:

Antifreeze — This mixture contains ethylene glycol, a product that can cause metabolic acidosis (accumulation of acid in the blood and body tissues) and lethal kidney failure if ingested. It has a sweet taste that attracts animals and is toxic in small doses (1 to 2 tablespoons produces toxicity in a medium-sized dog). Antifreeze is toxic even when diluted in water. At least one brand of antifreeze is available that uses propylene glycol as an alternative to ethylene glycol. Larger quantities of the propylene glycol-based antifreeze have to be swallowed to produce toxicity compared to ethylene glycol-based antifreeze. Propylene glycol-based antifreeze does not metabolize in the animal's system to form products that cause kidney damage, but it can still cause illness and death from metabolic acidosis. Antidotes are available for antifreeze poisoning, but early recognition of ingestion and immediate intensive treatment are necessary for survival. Prevent animals from consuming this toxic substance by having antifreeze changed by a professional who knows how to properly dispose of it. Individuals changing their own antifreeze should not drain it into sewers or leave it setting in open containers, because it takes only a few seconds for an animal to ingest a toxic amount.

Baking chocolate — This particular chocolate product contains a higher concentration of the stimulant in cocoa (theobromine) than other forms of chocolate. One-fourth pound can be toxic if eaten by a small dog, such as a poodle. The vomiting this chocolate causes can create quite a mess.

Mistletoe — The berry of this plant is the most toxic component, especially if it is chewed instead of swallowed whole. If enough berries are ingested, they cause gastrointestinal and neurological effects, including convulsions.

Poinsettia — The toxicity of this plant has been debated for years. The most recent findings are that while it contains no severely toxic chemicals, it can induce vomiting and diarrhea (a protective mechanism to help eliminate foreign substances from the body), similar to any plant that an animal is not supposed to eat. Animals are attracted to poinsettias, so keep these plants out of reach.

Ivy — This plant is not acutely toxic, but can cause gastrointestinal upset if ingested. Many common household plants are attractive to animals, especially cats, and casual consumption may cause vomiting and moderate gastric upset.

Christmas cactus — This plant is nontoxic, but can cause vomiting and transient diarrhea in household pets that consume portions of it.

Tinsel — Cats are attracted to playing with Christmas tree tinsel. When swallowed, it can cause intestinal blockage or intussusception. If indoor cats are in the household, avoid using strands of tinsel on the tree and to place any breakable ornaments at the top of the tree. Shatterproof ornaments might also reduce risk.

Glow jewelry — Dibutyl phthalate is used in glow-in-the-dark jewelry, which is popular at holiday festivities. Although the chemical has the potential to cause death from respiratory paralysis, cats generally will not consume much because of its unpleasant taste and the small amount present in the jewelry. Cats biting into the jewelry develop heavy salivation, hyperactivity and aggressive behavior, but typically recover within minutes. Immediately after ingesting this chemical, feeding the animal a small amount of milk, canned moist food or tuna juice dilutes any chemical left in its mouth. Any chemical on the cat's coat should be washed off, and cat's eyes should be flushed with water if ocular exposure occurred. There is no known antidote for dibutyl phthalate, so if large quantities were ingested the animal should be closely monitored for several hours and be given supportive care if necessary.

Rodent control agents — A variety of these products may be around the home and, in the excitement of the holidays, some become accessible to pets. Because the chemicals in these formulations are intended to attract and control mice and rats, pets may also be attracted and consume them. Potentially seri-

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ous problems can result if sufficient amounts are ingested, including bleeding, seizures or damage to kidneys and other vital organs. Contacting an animal poison control center (see contact information at the end of this article) will determine what to do if significant exposure has occurred.

Polyurethane adhesives — Accidental ingestions of expanding wood glues (Gorilla Glue®, Elmer's ProBond®) produce large gastric foreign bodies as they absorb water in the moist stomach and swell to several times their initial volume. Small amounts licked from floors or towels generally produce mild digestive effects, but larger quantities cause varying-sized foreign bodies that require surgical removal. Abdominal radiographs several hours after ingestion will visualize the presence of the occluding mass and confirm the need for prompt treatment.

Grapes — While many dogs safely consume small amounts of grapes and raisins, some are over indulged by sympathetic owners and develop effects that vary from mild gastrointestinal signs to renal failure. Vomiting develops within six hours and may proceed to anorexia, depression and overt evidence of kidney dysfunction that require fluid diuresis and careful monitoring and symptomatic care.

Drugs and illicit compounds — The occasional unintended ingestion of stimulants, recreational drugs or human prescription compounds offer challenges for veterinary diagnosis and therapy. Compounds such as phenylpropanolamine, amphetamines, pseudoephedrine, herbal mixtures, marijuana, cocaine and other drugs can induce varied and confusing effects in household animals. It is especially difficult when the ingestion is not observed or volunteered as part of the clinical circumstances or history, and the veterinarian is presented with a patient showing vague, unexplained signs that progress to a life-threatening clinical situation. In the absence of large-scale ingestions, careful monitoring of the syndrome's progression and appropriate symptomatic and supportive therapy can be rewarded with patient stabilization and gradual recovery over the subsequent 36 to 72 hours.

Cold temperatures — The U.S. Animal and Plant Health Inspection Service's Animal Welfare Act recommends that ambient temperatures should not drop below 50F, especially when sick, aged or young animals are involved. All animals, particularly those confined in outdoor enclosures, should always be provided with adequate protection and shelter from wind, rain or snow.

If you suspect an animal has been placed in any of these hazardous situations or has ingested these or other items of concern, immediately consult a poison control center (toll-free national number 800-222-1222) or the K-State College of Veterinary Medicine 24-hour toxicology hotline (785-432-5679).

Avian influenza threatens animals, humans

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Background

Avian influenza has been in the news since 1997 when a H5N1 strain of high pathogenic avian influenza (HPAI) caused an outbreak in domestic poultry in Hong Kong and spread to people, killing six of 18 infected humans. This was the first known case of direct transmission of avian influenza virus from birds to humans. Since then there have been outbreaks of HPAI in poultry in the Netherlands, the US, and Canada with at least 90 human infections and two deaths. Currently, an outbreak in Asia of H5N1 HPAI in birds has caused at least 32 human deaths and shows no sign of coming under control in the near future. In the past year, avian influenza virus was isolated from the lungs of domestic cats, leopards, tigers and dogs with pneumonia, which are species considered not susceptible to influenza. Health officials are concerned because influenza viruses that had recently acquired genes from avian viruses have caused all human influenza pandemics in the 20th century. In addition, this year's supply of human influenza vaccine in the United States is about half of what was expected. This is a good time to review and update about influenza information.

Introduction[1,2,3]

Influenza is an acute, highly contagious disease caused by viruses of the genus *Influenzavirus* in the family *Orthomyxoviridae*. In humans, there are occasional devastating worldwide outbreaks of influenza. In the 20th century major pandemics occurred in 1918, 1957 and 1968 (some sources include 1977 as a pandemic year). The 1918-19 pandemic was the most deadly human disease event in history and killed an estimated 20 to 50 million people, including more than 500,000 in the United States. Concurrent with the 1918 pandemic, influenza suddenly appeared in swine in the United States and there is evidence that both outbreaks were caused by the same virus. It is not known if influenza was transmitted from swine to humans or the reverse. Recent molecular analysis of influenza virus RNA from archival samples from people dying in the 1918 outbreak indicate that the virus contained avian and mammalian viral genes, and genes from an unidentified source, and that the virus had been circulating only a short time in the human population.[4] Although they caused significantly less loss of life, the 1957 and 1968 pandemics resulted in more than 100,000 deaths in the United States alone. The viruses that caused the 1957, 1968 and 1977 outbreaks all contained genes that had been acquired from avian viruses.

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Etiology[1,2,3]

Influenza viruses are grouped into types A, B or C based on antigenic characteristics. Groups B and C viruses are pathogens of humans and do not cause disease in animals. Group A viruses cause disease in humans, birds, swine, horses, mink and sea mammals (and have crossed over to dogs and domestic and nondomestic cats in the past year). In humans, clinical disease from group A viruses is more severe than disease caused by groups B and C viruses. Major pandemics are caused by group A viruses.

The genome of influenza A viruses contains eight gene segments of single-stranded RNA. The two most important surface proteins are the hemagglutinin (HA) and neuraminidase (NA) proteins, which are used in viral classification. The HA protein is the dominant surface protein and is responsible for binding viruses to host cells. It is the most important target of the host's immune system. Antibodies

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to a homologous HA will prevent infection. The NA protein is the second major surface protein and it is necessary for progeny viruses to escape infected cells. Anti-NA antibodies do not prevent infection, but they limit viral spread. There are 15 HA and nine NA types, both genes have a high mutation rate and the different types do not cross-react serologically. Despite all the possible HA and NA combinations, only HA types 1, 2 and 3, and NA types 1 and 2 have circulated widely in humans during the past century. Currently, H1N1, H3N2 and H1N2 viruses circulate in humans. The H1N2 virus is a reassortment progeny of the H1N1 and H3N2 viruses. Classical swine influenza is caused by a H1N1 virus, and H7N7 and H3N8 viruses cause equine influenza. However, all HA and NA subtypes exist in wild bird populations, especially waterfowl and shorebirds.

Emergence of new influenza strains

Like all RNA viruses, influenza viruses lack a proofreading mechanism to insure that there is accurate reproduction of viral RNA. There is a high mutation rate, especially in the HA and NA genes that results in small changes in antigenicity. This is referred to as antigenic drift and it is an important reason why yearly flu vaccinations are required. Normally the same basic virus subtypes circulate in the human population from year to year. After exposure, we are immune to the infecting HA type, but because of antigenic drift, the immunity does not remain complete. However, because of our partial immunity, the severity and duration of clinical symptoms are lessened. Cells infected by multiple group A viruses randomly incorporate genes from each virus into the progeny, which creates viruses with gene segments from multiple-parent viruses. This is known as genetic reassortment and it can produce viruses to which a population has no immunity. This antigenic shift and it is the cause of major pandemics.

The role of birds and pigs in human pandemics

Although only a few HA and NA subtypes of influenza A viruses circulate in humans and other mammals, all subtypes circulate in wild aquatic birds in which infection is usually subclinical. But infected birds excrete large quantities of virus in feces and respiratory secretions. Domestic birds, such as poultry, are very susceptible and once infected, poultry are a source of large quantities of virus for poultry, other domestic animals and humans. Normally, humans are not susceptible to avian influenza viruses because we lack the cellular receptor to which the viruses bind. However, pigs are susceptible to both avian and human influenza viruses because they have both types of receptors. If simultaneously infected with multiple influenza viruses, pigs can produce reassortment progeny that contain genes from multiple viruses. Thus, pigs have been considered "mixing pots" for emergence of new reassortment influenza viruses that might cause pandemics. In all pandemics in the last century, the causative viruses have been reassortment viruses with avian and human influenza virus genes, including an avian HA gene not previously circulating in humans.

Avian influenza in humans

Until 1997, it was thought that transmission of avian viruses to humans required production of reassortment avian-human viruses in pigs.[1,3,5,6] In 1997, 18 people in Hong Kong were infected with a H5N1 HPAI virus and six died. Infections were acquired directly from infected poultry, but the virus did not spread person-to-person and the outbreak was stopped by eradication of the entire poultry population in and around Hong Kong. This event caused worldwide concern, because it was the first known instance of direct transmission of avian influenza from birds to humans. Between 1997 and 2004 there were at least 90 additional human cases of avian influenza in Hong Kong, Canada and the Netherlands. Two of these cases were fatal.

In December 2003, the Republic of Korea announced an outbreak of H5N1 HPAI in poultry.[5,6] The virus quickly spread to seven more Asian countries and more than 100 million birds died or were destroyed during the first two months of the outbreak.[6] However, the outbreak was not contained and the World Health Organization (WHO) has little hope that it will be contained any time soon. Since January 2004, Thailand and Vietnam have 44 confirmed human cases of H5N1 virus infection, of which 32 (73 percent) were fatal. The WHO does not believe that all human cases have been identified and that the case fatality rate is actually 73 percent, but it is still incredibly high for influenza. The only good thing about the outbreak is that given the tremendous opportunity for bird-to-human transmission, there have been few cases and there has been only one report of human-to-human transmission. The WHO is concerned about the possibility that the H5N1 virus could mutate or acquire human influenza virus genes and become highly transmissible between people. If this occurred, the possibility of a new pandemic would increase because there is no existing immunity to H5 viruses, there are no human H5 vaccines, and four to six months would be required to produce sufficient quantities of vaccine. The 1918 influenza virus spread to all continents in the world in five months at a time when transoceanic travel was by ship. In areas infected with avian H5N1 virus, the WHO is encouraging vaccination of as many people as possible against the currently circulating subtypes of human influenza virus, especially people in contact with poultry. The purpose is to reduce the likelihood that a person who becomes infected with H5N1 influenza would be simultaneously infected with a human strain, which would provide the opportunity for emergence of human-avian influenza reassortment viruses. To further complicate the situation, the H5N1 avian virus was isolated from pigs in China in August.[5]

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H5N1 HPAI infection in domestic cats, leopards, and tigers

Scientist at Thailand's Kasetsart University isolated H5N1 virus from two of three domestic cats from a household where 14 of 15 cats died.[7] They then exposed cats by intratracheal (IT) instillation of H5N1 virus, placing cats in contact with the IT-infected cats, and by feeding a two-day-old chick infected the day before with H5N1 virus. All cats developed fever, respiratory distress and conjunctivitis, and H5N1 influenza virus was isolated from their respiratory tract secretions from three-days post-inoculation (PI) until the end of the study at seven-days PI. One cat died at six days PI. At necropsy, all cats had pneumonia with histologic lesions of diffuse alveolar damage that resembled the lesions in humans dying of H5N1 virus.

Also in Thailand, H5N1 virus was identified in lung tissues from two leopards and

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two tigers with pulmonary consolidation and hemorrhages in multiple organs.[8] Clinical signs included high fever, respiratory distress and unexpected death. The tigers and leopards were housed at the same zoo and fed fresh chicken carcasses from a local slaughterhouse in an area where large numbers of chickens were dying of H5N1 influenza virus. The Web site of the Food Animal Organization of the United Nations reports that since September 2004, 147 of 441 tigers at the Sriracha Tiger Zoo in Thailand have died or were culled because of H5N1 avian influenza.

Influenza virus has never been isolated from naturally infected domestic or non-domestic cats. There have been attempts to experimentally infect domestic cats with human influenza viruses, but the cats remained healthy and there was only a mild increase in body temperature and transient virus excretion. The fact that the H5N1 virus spread horizontally between domestic cats raises the possibility that cats could be sources of infection for humans.

Changes in influenza viruses isolated from U.S. pigs since 1998

During the 1968 pandemic, H3N2 virus appeared in European and Asian pigs, and in the late 1970s a completely avian H1N1 virus appeared in European pigs. These new subtypes were soon more common in European and Asian pigs than the classical H1N1 swine virus. In the United States, with one exception, all influenza viruses isolated from pigs before 1998 were the classical H1N1 swine strain. During the summer and fall of 1998 there were several widely separated outbreaks of influenza in US pigs caused by H3N2 viruses. Of four swine H3N2 viruses from four states, one virus was a reassortment between human H3N2 and swine H1N1 viruses, and the remaining three were reassortments between human H3N2, swine H1N1, and unknown avian viruses.[9] The HA and NA genes of all four viruses were derived from progeny of the 1968 pandemic virus that were circulating in people in 1995. A study in 2000 found that 20.5 percent and 8.3 percent of 4,382 swine serum samples from 23 states were positive for antibodies against the triple and double H3N2 reassortment viruses, respectively, whereas the seroprevalence as late as 1987-89 was less than 1.5 percent.[10] The same study also identified HA antigens from three distinct H3N2 humans viruses. Since then H1N2 viruses that are the result of reassortment of H1N1 and H3N2 viruses have also become common in U.S. pigs.[11] Currently most triple reassortment H3N2 viruses react poorly if at all with antisera to the original 1998 H3N2 triple reassortment viruses and there are at least three antigenic types of H1 swine viruses.[11] Since 1998, influenza virus in US swine has gone from a stable lineage to a complex, rapidly changing, and antigenically diverse pool with an affinity for reassortment.[11] All three swine influenza viruses are known to be able to inflect and replicate in humans, although because human viruses with H3 and H1 antigens circulate among people the current pig isolates are unlikely to cause a pandemic. But the high rate of reassortment makes it increasingly possible for emergence of influenza viruses with new avian HA genes to which people are readily susceptible and have no pre-existing immunity. Turkeys have long been known to be susceptible to classical H1N1 swine influenza and recently there have been reports of isolation of swine H3N2 and H1N2 influenza viruses from turkeys.[12] Turkeys are also very susceptible to avian influenza viruses and classical H1N1 swine influenza and provide another opportunity for mixing avian and mammalian influenza.

Equine influenza in dogs

As reported in the last Veterinary Quarterly, equine influenza virus was recently isolated from the lungs of greyhounds with hemorrhagic pneumonia.[13,14] The dogs were at a race track in Florida and there was an acute outbreak of severe tracheobronchitis and pneumonia. Eight of 24 affected dogs died. Like cats, dogs are not considered to be susceptible to influenza viruses and this is another example of influenza A virus jumping to a new species.

Conclusions

Most influenza experts think it is only a matter of time before the next influenza pandemic and it is important to continually monitor influenza in livestock, especially poultry and swine. It is also important to learn as much as we can about influenza and the conditions that give rise to influenza pandemics in order to reduce the effects of the next pandemic.

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Diagnostic Laboratory updates serology requirements

Equine infectious anemia: Federal guidelines for equine infectious anemia (EIA) testing have been tightened. All EIA forms must be filled out completely. The owner's name and address, and animal identification information including markings, must be provided. All forms must be signed, dated and marked with the desired test type: AGID with test results the next working day or ELISA with test results in one hour. If the above information is not included, we are not allowed to run the test. If you forget to sign the form, we cannot sign it for you. All test results are reported, so there is no such thing as an unofficial EIA test to determine the EIA status of a horse before running the official test. All EIA tests are official tests.

Johne's disease: We require a minimum of four samples to run the ELISA test. The ELISA is set up and completed on the same or the day after the sample is received. The AGID test is run daily and the results are read in 24 or 48 hours.

Canine export: Submission forms for canine export are available on our Web site at: *www.vet.ksu.edu/depts/dmp/service/serology/index.htm* Forms must be filled out completely because we can no longer accept changes.

Continuing Education

March 5
Veterinary Technicians Conference – *Movin' On Up*

March 6
13th Annual Small Animal Conference on Radiology

April 2 and 3
22nd Annual Frank W. Jordan Seminar – *Pavlov and Beyond: A Program So Sweet, It'll Make Your Mouth Water*

April 16
Bovine Conference on Production Management

April 17
Small Animal Medicine Lecture Series

June 5 – 8
67th Annual Conference for Veterinarians, Centennial Celebration and KVMA Vendor Trade Show

Brochures for these conferences will be available approximately two months before their scheduled date. This is the conference schedule as of Dec. 14, 2004. More conferences may be added.

For the most complete, up-to-date conference information visit our Web site at: *www.vet.ksu.edu* and click on Continuing Education, or contact: Linda M. Johnson, Ph.D., at 785-532-5696 or *johnson@vet.ksu.edu*

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