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IN THIS ISSUE

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LLOYD'S

COMMENTARY

In March of this year, a group of more than 300 equine researchers from 50 countries made a scientific pilgrimage to Dubai for the 8th *International Conference on Equine Infectious Diseases (ICEID)*. In the now settled aftermath of that eventful week, the following commentary is offered.

It is appropriate, in retrospect, to acknowledge the enormous debt owed to Drs. John Bryans and Heinz Gerber who, more than 30 years ago, conceived and bequeathed to us the idea of a continuing series of international meetings where equine infectious disease specialists could gather and exchange new information and views, and where young scientists and promising students could be exposed to the spirit and technology of the discipline.

A meeting of such proportions as that in Dubai required long and careful preparation and was made possible only by the unstinting dedication of a formidable team of organizers and staff. To those upon whom this burden so heavily fell, we are deeply appreciative. Their quiet, collective efforts produced a scientific program of intellectual high adventure that covered all themes of substance in a balanced fashion. Emerging, importantly, from the four-day symposium was a synthesis of priorities of the equine infectious disease problems requiring immediate attention and funding, in the solution of which researchers of all specialities can play a part.

Much of the scientific "gathering" took place as casual, hallway sessions in the form of spontaneous discussions and free exchanges that, to our loss, were not recorded stenographically for all to share. We are delighted that the thoughts expressed more formally by the 120 invited speakers have been judged worthy of being saved from similar oblivion and will be published in a printed Proceedings of the conference.

A special feature of the program was the recognition, with the prestigious Dubai Equine Award, of the career achievements of two individuals, Drs. Jenny Mumford (England) and Michael Studdert (Australia), who have "writ their names large" in equine infectious diseases research and whose heritage of work in equine virology has left indelible traces on the thinking and experimental work of us all.

A popular highlight of the Conference was the series of organized social programs, during which delegates were spirited nightly to exotic destinations and, for the good only of body and soul, catered with food and entertainment almost too fantastic for belief. Looking back at all those exquisite social accompaniments, played out on the panoramic backdrop of the Arabian sea and desert dunes, is an unforgettable remembrance still.

The holding of such a meeting required major funding. Here we were particularly fortunate in the generosity shown by our host and sponsor, Sheikh Mohammed bin Rashid al Maktoum, Crown Prince of Dubai, who believed enough in the importance of the horse to provide full financial support for the Conference. Our gratitude to him is boundless.

The aggregate of these reflective thoughts is that, from all accounts, the "1998 Dubai Equine Conference" will take its place among the classics. For it was, I believe, unrivaled in its impact and, from this point forward, the expectations for future *ICEID* meetings have been raised.

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First Quarter 1998

The International Collating Centre, Newmarket and other sources provided the following information.

Cases of African Horse Sickness were reported from South Africa. Contagious Equine Metritis was reported from Japan, Netherlands, Sweden, and Switzerland. Respiratory disease attributable to equine herpes virus (EHV) was reported from France and Ireland with cases of paralysis caused by the virus reported from Ireland, Netherlands, and the United States. Abortions caused by EHV-1 were reported from Ireland, Italy, Japan, Netherlands, and the United States. Cases of influenza were confirmed in France, Ireland and the United Kingdom.

Salmonella abortus equi was reported from Japan and strangles cases were reported from Australia, Ireland, Sweden, and Switzerland. Further to the reports of the isolation of a "CEM-like" organism from donkeys and mares in California and Kentucky as described in the April 1998 issue of the *Quarterly*, no additional cases have been reported. ***Correction:*** In the April issue, we did not acknowledge that during the investigation in Kentucky, samples were also examined by the Breathitt Veterinary Center of Murray State University, Hopkinsville, KY.

Toward the end of May, the State Veterinarian of New Mexico confirmed that Vesicular Stomatitis had been confirmed in two horses resident in the state.

USDA Funds Genome Project

The horse genome effort received a major boost in April 1998 when the United States Department of Agriculture (USDA) approved renewal of an initiative of the National Animal Genome Project which, for the first time, includes support for construction of the horse gene map ($45,000 per year for 5 years).

This initiative, titled National Research Sponsored Project number 8 (NRSP-8), funds collaborative efforts among American laboratories working on cattle, sheep, pigs, chickens, and horses. The American laboratories working on the horse genome are located at the University of California, Davis, University of Kentucky, Texas A&M University, Cornell University, University of Minnesota, Tufts University, Shelterwood Labs in Carthage, Texas and Applied Biosystems of Foster City, California.

NRSP-8 is part of a larger international collaboration including laboratories in the United States, United Kingdom, Australia, New Zealand, Japan, France, Sweden, Norway, Denmark, Germany, Czech Republic, and The Netherlands working together to construct a gene map for the horse. The international workshop began in October 1995 and is being conducted under the auspices of the Dorothy Russell Havemeyer Foundation. The group met in January 1998 to discuss construction of the first linkage map for the horse; this map, including over 150 markers, will be reported on later this year.

A gene map for the horse will be useful to investigate the hereditary basis of behavior, performance and diseases. Already geneticists are using the map to study: the hereditary basis of muscle diseases such as tying up; developmental bone diseases such as *Osteochondrosis dessicans*; and allergic diseases such as culicoides hypersensitivity. Likewise, this tool could provide greater insight into genetic predispositions to laminitis, cryptorchidism, conformation defects as well as metabolic and immunological diseases.

Another application will be to investigate genetically determined responses to infectious diseases. Some horses appear resistant to certain infectious diseases and knowing the basis for disease resistance will suggest effective methods to prevent or treat the diseases and allow scientists to be more effective in evaluating trial vaccines.

In summary, this research will help veterinarians and horse owners make better management and treatment decisions and understand the contributions made by genetics, environment, and chance to complex traits and diseases.

The horse gene map is being constructed using a variety of tools. Several laboratories are developing genetic markers, called microsatellite DNA markers, which form the framework for the map; 5 laboratories are conducting cytogenetic studies to map genes to

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chromosomes; 20 laboratories are conducting family studies to determine the genetic distance between markers and to further localize the position of the markers.

The map is quickly taking form and will be a valuable tool for scientists. If you are interested in seeing the status of the gene map you can visit the gene map database at the following web sites:

Roslin, Scotland http://www.ri.bbsrc.ac.uk/cgi-bin/arkdb/browsers/browser.sh?species=horse

Jouy-en-Josas, France http://locus.jouy.inra.fr/cgi-bin/horsemap/Horsemap/main.pl.

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N A T I O N A L

Are Race Times Improving?

Periodically, usually just before the Kentucky Derby, at least one member of the media raises the question why Thoroughbred horses are not running faster now than formerly. This is thought to be particularly puzzling since record times in human track events continue to fall.

Quite apart from the Derby and the media's need for color stories, many people both within and without the horse industry raise the same concern.

Following a suggestion by Dr. Steve Kamerling of Louisiana State University, I looked at the race times for the Triple Crown races over a period of years, beginning when the present distances were established. The results are shown in the accompanying graphs.

It is apparent in **Figure 1** that rate times fluctuate but show a generally declining trend. In **Figure 2** a statistical tool, linear regression, was used to best fit the race time data to a straight line. It is clear that race times for these races have been decreasing; *i.e.*, the winning horses in these races are going faster.

The times for the Belmont, the longest race, have declined the least, the Derby somewhat more, and the Preakness the most.

The concern for lack of improvement as measured by race times for these three races seems, then, to be misplaced. The times are decreasing, the best three year olds are running faster. Will that continue? It is not possible to say.

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Figure 1. Triple Crown Races

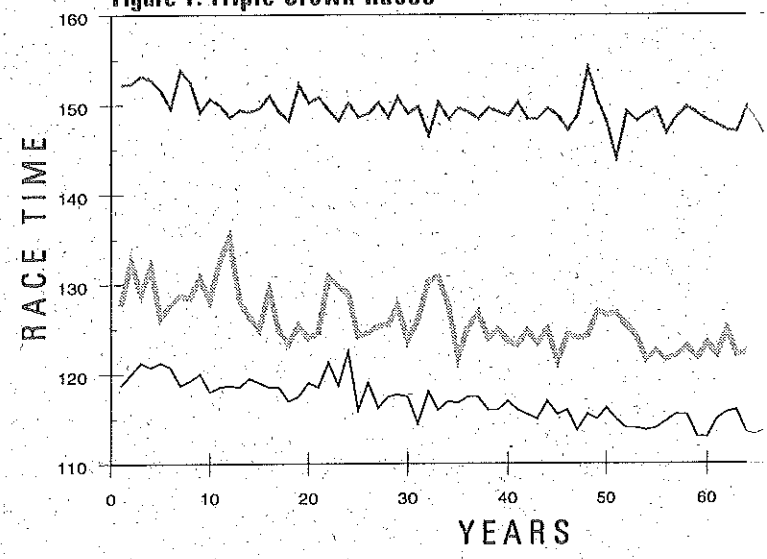
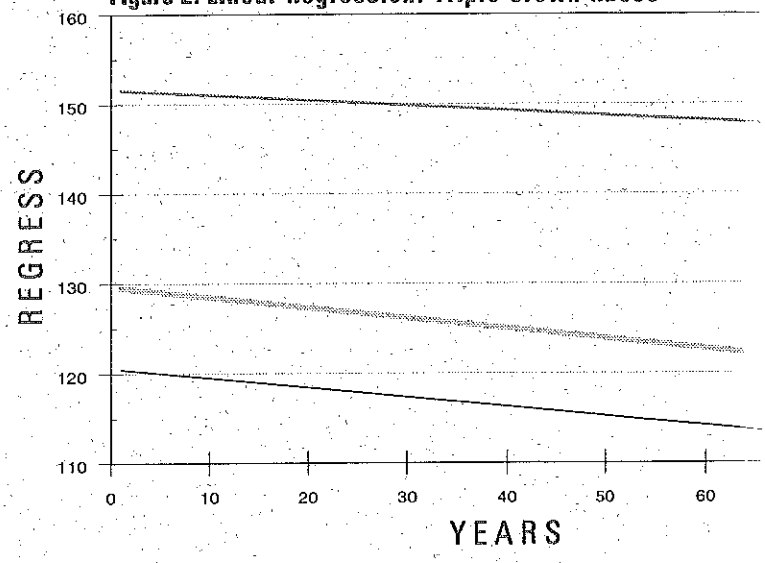


Figure 2. Linear Regression: Triple Crown Races



Placentitis in Mares

For several years a unique form of placentitis, referred to as "nocardioform" or "mucoid" placentitis, has been diagnosed at the University of Kentucky Livestock Disease Diagnostic Center. While the number of cases of mucoid placentitis has remained fairly constant over the preceding years, for unknown reasons during the current foaling season there has been a dramatic increase in the number of these cases.

This type of placentitis is unique due to the location of the infection, the type of material that accumulates on the placenta, and the bacteria that is usually involved. In a departure from other forms of placentitis, the infection is localized on the body of the placenta in the area of the junction of the body and horns. The affected area varies in size and appears to begin on the ventral aspect of the placenta, with extension cranially onto the horns, caudally on the body, and circumferentially around the placenta.

Typically there is a large accumulation of thick brown mucoid material on the surface of the placenta (chorionic surface). The underlying chorionic villi of the placenta are reduced in size and, in the central portion of the lesion, may be completely absent. Variable numbers of inflammatory cells invade the placental membrane. There is no communication of the placentitis with the cervical portion of the placenta and, therefore, it does not appear to be an ascending infection as usually is the case in mares with placentitis.

Associated with mucoid placentitis are gram-positive branching filamentous bacteria, which are often numerous in the exudate. These bacteria appear to be a heterogenous group, in that there are variations in morphology and *in vitro* growth characteristics between cases and even within the same case. At present these bacteria are unclassified. Invasion into the placental membranes by the bacteria and spread to the fetus are not observed; instead, the infection likely harms the fetus by causing placental insufficiency through disruption of the uterine-placental interface.

While most mares that have this type of placentitis are normal in appearance, some exhibit early development of the mammary gland and lactate prematurely. Ultrasound examination of the reproductive tract at that time may reveal areas of separation of the placenta from the uterus. Usually one of the following four outcomes of this type of placentitis are seen: 1) some mares abort the fetus in late gestation; 2) other mares produce a stillborn foal at term; 3) have an alive but small, weak, malnourished appearing foal; or 4) have a normal, healthy appearing foal. Mares that have had this form of placentitis typically do not have problems with subsequent conception and pregnancy and are not at an increased risk of redeveloping placentitis.

The commonly accepted mechanisms by which placentitis occurs include infection of the placenta by bacteria or fungi that gain access to the uterus by ascension through the lower reproductive tract or by gaining access by spread through the blood stream. The consistent location of the placentitis in mucoid placentitis and the lack of communication with the cervical area suggests that a different pathogenesis may be involved in this type of placentitis.

Studies are in progress to help elucidate when and how this form of placentitis occurs and, consequently, if intervention may be possible.

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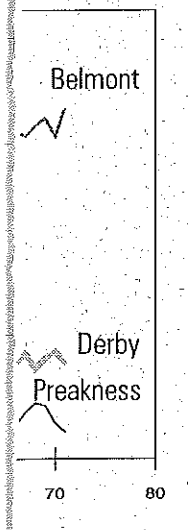
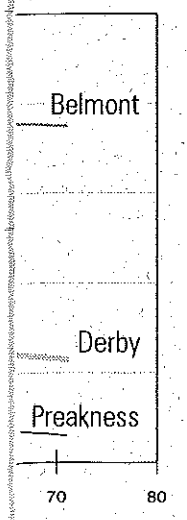




Rabies in Kentucky—1997

In 1997, the Division of Laboratory Services and the Breathitt Veterinary Center tested 1656 specimens for rabies; 29 (1.8%) tested positive (see **Figure 3**). The majority of cases (24) were wildlife (4 bats, 1 fox, 19 skunks) and 5 were domestic animals (1 dog, 2 cattle, 2 horses). As usual, skunks were the majority (65.5%) of rabid animals and also the highest percent positive (32.2%) for any given species.

The annual total of 29 cases is very similar to the preceding 5-year mean of 29.8 rabies cases. Interestingly, nearly as many rabid horses (10) were reported as rabid dogs (12) for the same 5-year period. Through

April 17, our laboratories have identified 2 rabid horses and no rabid dogs in 1998.

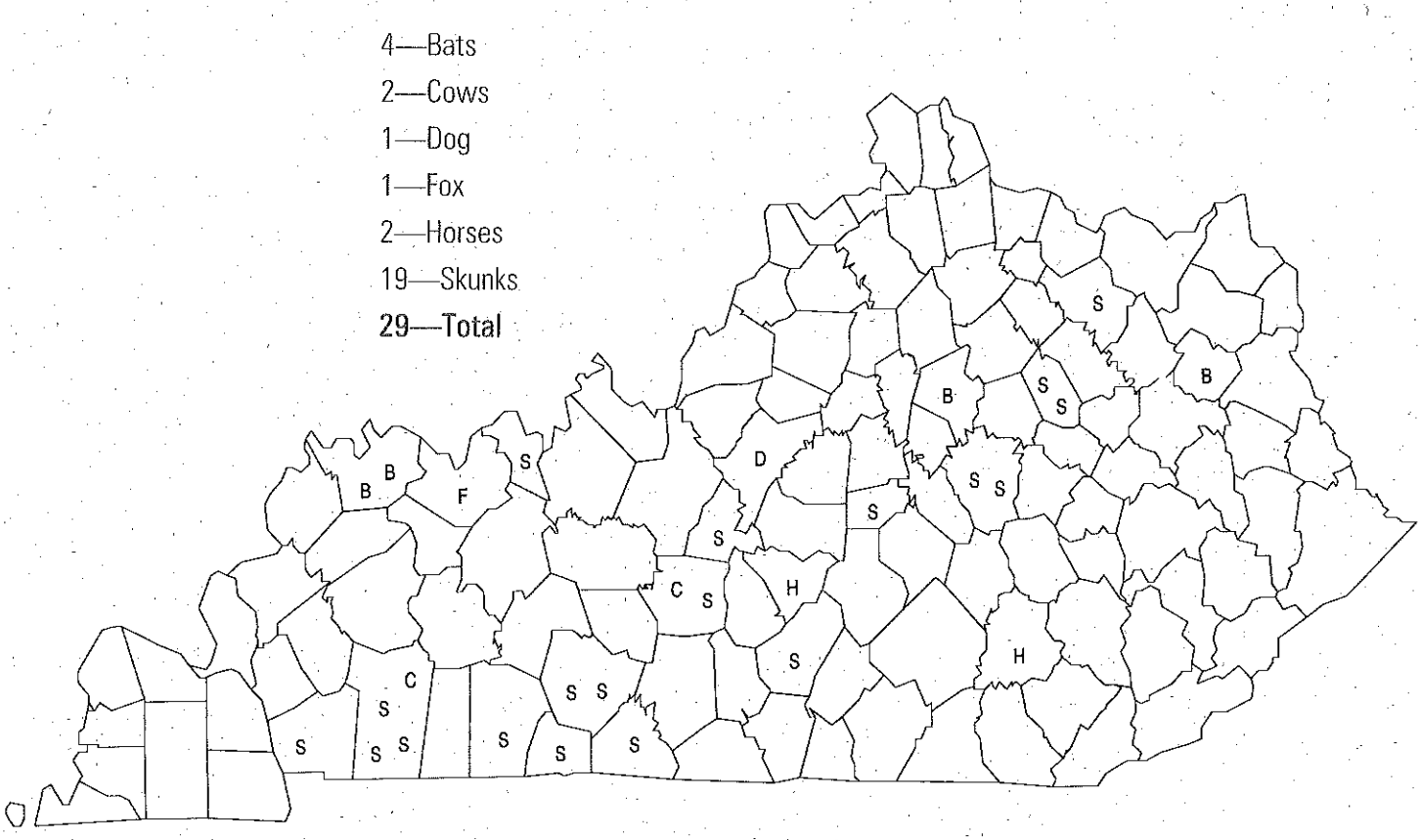
The skunk is the most common wildlife reservoir in Kentucky, but one of the rabies positive horses in 1998 was determined by monoclonal antibody techniques to have been infected by a bat rabies variant. At least one other horse in the preceding 5 years contracted rabies from a bat rabies variant.

Bats are becoming an important wildlife reservoir for rabies in humans and domestic animals. Kentucky has reported an average of 5 rabid bats per year for the last 5 years. However, there were 24 cases of human rabies acquired in the United States from 1980-1997, and 21 of the 24 cases were determined to be due to bat rabies variants. (This includes 1 case of human rabies in Kentucky in 1996.)

This pattern of human rabies infections has led to new recommendations by The Centers for Disease Control and Prevention (CDC) for postexposure prophylaxis (PEP) regarding bat incidents. The CDC recommended indications for human rabies post exposure treatment have been extended to anyone who may have had a bite, scratch, or mucous membrane exposure to a bat and cannot rule out rabies in the bat through testing it. This includes persons who may have been potentially exposed, but cannot give an accurate history of direct contact, *e.g.* finding a bat in the same room as a sleeping child or someone mentally or physically impaired. Bat bites can be quite innocuous and superficial and, therefore, nearly undetectable.

In spite of the increasing importance of bats as a source of rabies infection, certain wildlife conservation groups still recommend building bat houses to encourage bats to live in close proximity to humans and domestic animals. The usual argument for bat

Figure 3.
Rabies cases in Kentucky—1997



houses is that bats consume large quantities of insect pests including mosquitoes. Bats do play an important ecological role in reducing agricultural pests, but the actual consumption of mosquitoes as part of their diet is limited.

While no knowledgeable person condones the mass slaughter of bats, most public health officials discourage building bat houses that would encourage bats to live in closer proximity to humans.

It is also recommended that bats be excluded from attics, porches, barns, and other human or domestic animal dwellings. Vaccination of domestic animals for rabies is always appropriate, and personal protection from bites should always be taken when handling bats.

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