Published as Issel, C.J. and Sadlier, M. 2007. Reducing the risks of infection in veterinary practices: Recent lessons learned with Equine Infectious Anemia (EIA). Hagyard Bluegrass Equine Symposium 2007 Proceedings: 89-102.

Reducing the risks of infection in veterinary practices: Recent lessons learned with Equine Infectious Anemia (EIA)

Charles J. Issel, DVM, PhD, DACVM[a] *and*
Michael Sadlier, MVB, CertES(Orth), CertESM, MACVSc(EqSurg), MRCVS[b]

[a] University of Kentucky, Department of Veterinary Science, Gluck Equine Research Center, Lexington, KY 40546-0099 and [b] Troytown Veterinary Hospital, Green Road, Kildare Town, County Kildare, Ireland

Introduction

The subject of infection control has been high profile recently with numerous teaching hospital closures, racetrack and other facility quarantines, newly developed AAEP infection control guidelines, and published monographs.[1-3] Despite our increased attention to the subject, we have yet to develop a meaningful set of guidelines based on appropriate science which quantitatively assesses the risks associated with horses shedding influenza, herpesvirus, strangles, EIA, etc. In most cases we rely on total facility shutdown or limit the quarantine to a single barn if prior experience has proven that to be sufficient. Fortunately, many agents shed by horses into the environment have limited survival outside the host and are in relatively low concentrations, especially when compared to the viremia of West Nile virus in the blood of many bird species (often in excess of 10,000,000,000 median infective doses per ml.)

This deficit of knowledge is not unique to veterinary medicine. The same dearth of data is available to estimate the risk of epidemic diseases of man. For example, the recent case of Andrew Speaker, with extreme drug resistant tuberculosis, demonstrated clearly the inability of the medical community to accurately assess risk and institute appropriate actions to avert possible disaster. The result of this lack of knowledge/action is often extreme overreaction to the real risks, but in many cases there are no viable options. Too much is at risk economically and/or politically to take other actions.

The fact remains that there is a crying need to develop quantitative models to assess risks of infectious disease transmission and spread within animal and human populations. Further, when our inability to control human movement and behavior is factored into the equation, chaos is an expected result.

A worst case scenario (or is it a daily or expected scenario?):

An acutely febrile horse presents to your clinic/hospital with no other signs. You admit the horse, and after a day of uninformative workups and lack of other observed clinical pathology abnormalities, the horse begins to bleed passively from the nostrils as it is taken to the stocks in the center of your building. You then collect samples from the horse and find a low platelet count (<20,000). You are unsuccessful in maintaining hemostasis and euthanize the horse.

Questions to be asked:
How do you proceed?
What samples would you test for what?
How do you clean and disinfect the stalls and building?
What risks of infectious agents have you likely encountered?
Would you handle this case differently if it presented without fever but with history of elevated liver enzymes, suggesting a hepatopathy?

And even more basic questions deserve attention:
What tests do you require before admitting patients?
What isolation/quarantine restrictions do you place on new patients?
What facilities/SOPs do you have to break transmission of infectious agents in your practice?
How do you clean up blood spills in your practice?

Now you know:

This horse has equine infectious anemia (EIA). For purposes of this exercise assume that the quantity of virus exceeds 1,000,000 horse median infective doses per ml of blood, an expected titer during acute episodes with virulent strains. How many of your staff have become mechanical carriers/transmitters of the virus since the horse was admitted? What is the chance that when you collected a blood sample for analysis that you became contaminated with blood and transferred that to another horse? How much EIA virus would be on your ungloved finger if you wiped the blood spot on the neck of the horse after withdrawing the vacutainer? If you cleaned and disinfected the hospital as you usually do, how much infective EIA virus did you put into the air?

Although you may view this as a theoretical exercise today, if you had been practicing in Ireland before the first historical cases were documented in 2006 it could have been *your* reality and *your* worst nightmare. This paper is written to present what we know about the outbreak of EIA in Ireland in 2006 and to place the current risks of EIA in the United States in perspective.

As we present this snapshot questions/thoughts will be introduced that in our opinion should be discussed and from which we can improve our chances of learning the same lessons painlessly.

EIA in Ireland

The exact details of the outbreak of EIA in Ireland remain undefined to this point and may remain somewhat shrouded in mystery until the official inquiry is completed. The reader is directed to the Irish Department of Agriculture and Food website for the official version.[4] Community thought on the outbreak leads to the following tentative scenario.

"Freedom from EIA":

Until June 15, 2006 equine infectious anemia had never been officially diagnosed in Ireland. Veterinarians and owners had never tested their horses per national, tripartite and/or EU requirements. Veterinarians thought they had the luxury of never having to worry about EIA as it was not present in Ireland. If this sounds like your practice situation in central Kentucky in 2007, we are thinking on the same wavelength. Under such conditions of freedom, real or perceived, complacency often is an unwanted consequence.

Source of EIA virus:

Plasma is used routinely for foals in Ireland with treatments at birth and 30 days of age. Plasma for use in horses in Ireland was expensive and had to be imported from the United Kingdom by special license (AR16), issued by the Department of Agriculture and Food (DAF). In at least one case (Farm 1), it is thought that the owner brought with him from his native country, Italy, a commercial plasma product, possibly collected from adult slaughter horses, for use in foals.

Discovery of the first case:

A mare from Farm 1 was admitted to the main barn of a veterinary hospital on June 12 with what was interpreted as a liver disease of unknown etiology. Treatments and diagnostic tests performed in hospital were unsuccessful and uninformative. On the afternoon of June 13, the mare began bleeding from the nostrils, had labored breathing and was found to have a low platelet count. Attempts to stem the flow were unsuccessful and during the night the mare continued to deteriorate and was euthanized about 5AM. The total estimate of blood loss was 20-30 Liters with the vast majority in the stall and at the door to the stall, with a low amount in the hall as the mare had been led outside for euthanasia. The barn was cleaned and disinfected per SOPs.

Later in the morning of June 14, the veterinarians at the hospital were notified by the veterinarians at Farm 1 that they had heard rumors of cases of EIA in Italy, possibly traced to the same plasma source used by them on foals in Ireland. This prompted the hospital veterinarians to contact the Irish Equine Centre and request tests for EIA on samples collected from the mare. On June 15 the sample was positive for antibodies to EIA virus, confirmed in the agar gel immunodiffusion (AGID or Coggins) test, and the hospital was notified.

Retrospective analysis:

Samples from other horses from Farm 1 had been submitted earlier in 2006 to the Irish Equine Centre or had been collected from their foals with postmortem exams performed at the Centre. Several had been saved and were tested; a total of 3 horses from Farm 1 were found to be positive for EIA.

Prospective analyses:

1. Additional samples from horses on Farm 1 proved to be positive.
2. Additional horses treated by veterinarians from Farm 1 proved to be positive.
3. Exposed horses from the hospital were released to home farms in a number of counties in Ireland before the diagnosis was made or before strict restrictions on movements were imposed.
4. All adult horses (14/14) but only 2 of the 7 foals in the main barn the night of June 13 became infected.
5. High infection rate in horses in the hospital suggest a common source/route of infection.
6. Few additional secondary and no tertiary cases of EIA were defined through extensive testing from June 2006 through March 2007, when restrictions were lifted.
7. After rigorous epidemiologic investigations were completed, the Irish Department of Agriculture has estimated that 25 of the 28 cases of EIA defined in Ireland in 2006 were in some way mediated by man; only 3 were thought to be transferred by insects.
8. More than 1200 horses were restricted on 53 premises as a result of this outbreak.
9. Over 50,000 samples had been tested for EIA at the Irish Equine Centre or the Department of Agriculture and Food's Central Veterinary Research Laboratory since July 2006. Many were tested in response to a voluntary EIA recommendation of the Irish Thoroughbred Breeders' Association (ITBA) in their Codes of Practice for 2007.

Making the best informed guesses

Data clearly support that the outbreak began on Farm 1 and was associated with administration of plasma to foals with subsequent transmission to mares. As EIA (and other blood-borne agents) was not suspected and never before encountered by veterinarians in Ireland, it may be reasonable to assume that

contamination of man and materials by blood from infected horses was more likely to occur than in areas of the world where EIA and Piroplasmosis are endemic. In Ireland it is not standard practice to wear personal protective equipment when working with horses or to wear gloves and change them between horses. Likewise, internationally veterinary medicine has not adopted as standard practice the "standard precautions" of human medicine, i.e., where all blood and bodily fluids from all patients are assumed to be infectious.

Therefore, if the first foals with EIA were handled and treated for their initial clinical illnesses with no extra precautions taken to prevent transfer of blood/secretions between horses, it is likely that EIA virus (EIAV) could have been transmitted via human contact. Several examples are listed below to give a sense of how humans can become contaminated and inadvertently transmit EIAV between horses.

Field studies on transmission of EIAV with inapparent carriers indicate that a high percentage (>90%) of foals from positive dams can be raised free of the infection, even when allowed to remain at risk with high blood-feeding vector populations in pasture situations.[5,6] By contrast, we have heard of numerous cases where foals from positive mares have been manipulated/intensively managed in isolation facilities with a low percentage of foals being raised free of the infection. In our opinion some of the observed extra risks faced by these foals include human contact and inadvertent exposure to blood during procedures to monitor the status of the foal.

A review of the expected level of EIAV in blood of horses and the potential of human contamination with a common practice will suffice to place the risk in perspective. Blood collections from horses for diagnostic purposes are performed using a range of techniques: from syringes and needles to closed systems with evacuated glass tubes using multi-sample needles with covers over the needles designed to prevent blood contamination of the tube. The latter system is preferred because the likelihood of contamination of the person collecting the sample is reduced to near zero. However, we have all collected blood samples and appropriately removed the needle from the jugular vein after relieving manual pressure, only to find a drop of blood visible at the needle entry site. What is the usual human reaction to the spot of blood? Wipe it off and rough up the neck in the process. If the horse being bled is infected with EIAV, there may be in excess of 100,000 infective doses of EIAV in that one drop of blood, which by now may be on your ungloved finger. **Enough virus to infect every horse in Ireland.** That is the potential. It is rarely realized, but the math is sound.

Spread within the hospital

Once the index mare was hospitalized in Ireland with suspected liver disease, similar inadvertent contamination of people would be expected to occur, especially with the massive blood loss. The number of cases of EIA in the hospital, however, did not fit the usual human vector scenarios, as some foals handled by people during the night did not become infected and some adult horses not handled became infected. In one case, a mare and her foal were brought into the barn for only 4 hours (5PM-9PM); both became infected even though the mare was not treated. Epidemiologic analyses suggest a common route/source of infection. In the absence of evidence of intentional transfer of blood by hospital staff, other potential means of transmission were entertained. The most likely candidate is considered to be air-borne transmission. Although there is no evidence for contagious spread of EIAV, EIAV should be expected to be present in all secretions and excretions during periods of high viremia loads, especially since contamination with blood cannot be ruled out. The most likely material for spread in the main barn of the hospital appears to be blood, with two probable sources. The first source is the index mare, which may have placed EIAV into the air during her labored breathing/snorting because of blood in the airways. The second source could have been the mass of the mare's blood contaminating the barn. These two are presented below with mathematical models based on a number of assumptions.

First let us consider the mare. The expected volume of expired air over 11 hours (5PM June 13 to 4AM June 14) was 39600 liters (6liters/breath x 10breaths/min x 60min/hr x 11h). As she was having breathing problems and as her platelet count was low and clotting was delayed, it is reasonable to assume that some blood was aerosolized during the 11 hours. If we assume that only 0.0001% of the air in each breath is plasma (0.006ml) and the viremia is 10^6 median horse infective doses per ml, the expected virus content of each breath is 1,666 HID_{50}; **over 11 hours that would be a total of 10,995,600 HID_{50} placed into the air of the barn.**

Next let us consider the environmental contamination in the barn. As stated earlier, the hallway of the barn was cleaned and disinfected per standard hospital procedures after the mare was led out for euthanasia. Review of the procedures indicates that power washing of the hallway was performed before Virkon S was applied to disinfect the hallway. Did this power washing put the other horses at risk? Again, let us apply a series of assumptions to gain a sense of the potential involved.

If only 2ml of blood with an infectivity of 10^6 HID_{50}/ml (a total of 2,000,000 HID_{50}) was mechanically added to the air in the barn by power washing, what is the virus load expected in the air? If the infective air is evenly distributed in the barn, what is the expected virus load inhaled by each horse within 10 minutes of the event? We used a 10 minute window as this enveloped lentivirus would be expected to have a relatively short half life in the air. We also had to make assumptions about the percentage of virus that would be present in large droplets or in droplet nuclei. Large droplets probably were the predominant species and virus survival for transmission in large droplets would be expected to be lower than in droplet nuclei. For this exercise, we estimated that 90% of virus was in large droplets and virus survival within them was only 1 minute. By contrast, droplet nuclei are smaller, have a greater potential to reach lower regions of the lung, and would be expected to have greater potential for transmission by being able to contact alveolar macrophages. For this exercise, we estimated that only 10% of the virus was contained within droplet nuclei and that virus survived in them for 10 minutes. A reasonable air volume for the lower half of the barn where the water would be expected to be distributed was calculated at 2,800,000 liters. Thus there could have been 2,000,000 HID_{50} in 2,800,000 liters, or 0.7 HID_{50} per liter of air. If we assume each adult horse moved 60 liters of air per minute, **the calculated inhaled virus dose per horse is 42 HID_{50} in the first minute and a total of 80 HID_{50} within 10 minutes of the power washing event**. The expected dose for foals would be proportionally lower due to the lower volume of air moved per minute.

Blood in the air?

Thus it is theoretically possible that if the mare had infective EIAV in relatively high concentrations in her blood that every horse in the main barn of the hospital could have been exposed to EIAV by merely breathing the air. To date, there is no evidence for contagious spread of EIAV. Probably the best way of thinking of the potentials listed above is transmission with **blood in air**, as there is no evidence for true aerosol transmission, such as with influenza virus after virus replication in respiratory epithelium or with foot and mouth disease virus after nasopharyngeal replication.

In this case one is compelled to believe that the potential was realized. Unfortunately, blood from this case was not archived to analyze the level of infectivity and, to date, isolates of EIAV from this outbreak have not been studied in critical pathogenesis studies to determine if virus levels would be expected in abnormally high concentrations in unusual sites. One recent report, however, with samples from this outbreak suggests virus presence in secretions in the absence of virus in plasma samples.[7] These data should be viewed with interest and a level of skepticism. Unfortunately, most laboratories today do not adopt techniques to assure that the presence of an isolate or a given sequence (as in the recent report) is from the sample in question.[8] Methods to help make those assurances were perfected in arbovirus research laboratories in past years and still are relevant when analyzing contemporary data. Because of constraints of time and space in the laboratory, and

because of the need for rapid dissemination of results, there is often undue haste and failure to use the controls needed to make the types of assurances which highly qualified arbovirologists had the reputation for. The methods included: Samples from the field are divided in the field and brought to facilities where no agents or their nucleic acid are stored: the split sample vessels permit reisolation from the same source. The samples are then analyzed in facilities where there is complete separation of personnel by time and space.

The bottom line is this. In contemporary medical research, few, if any, laboratories have the luxury of time and space to be able to make isolates and assure 100% proof of their origin. The confidence in contemporary results goes down even further when the desired endpoint is amplification of minute quantities of nucleic acid (PCR, RT-PCR). In those cases, as it is virtually impossible to apply the gold standards of arbovirologists, the potential risks for dissemination of false data increase exponentially. Therefore, the data on amplification of sequences from samples collected during this outbreak are interesting, provoke thought and deserve confirmation/verification in additional controlled studies. Without such confirmation they remain curiosities.

Collection and use of blood products from horses

Commercially available plasma from horses is used widely in equine practices, most frequently for treatment of foals for failure of passive transfer (FPT) or for prophylaxis against *Rhodococcus equi* infection. In many cases these products are collected from horses in closed herds, certified free of certain infectious diseases and hyperimmunized against selected agents. In other cases these products may be from less desirable sources such as from random source horses passing through slaughter facilities. In all cases the safety of the products is subject to testing before packaging and sale to veterinarians. In many cases, however, the standards may not be sufficient to give the needed assurances for either safety or efficacy. For example, a test for antibodies against EIAV might suggest presence of EIAV in the product, but analysis of specific lots free of antibodies against EIAV, even by sensitive PCR methods, might not be sufficient to detect infective EIAV. In another example the lack serum antibodies against EIAV in a random horse whose background/clinical status is not known may be misleading; the sample may contain significant quantities of EIAV because of a recent exposure. Additionally, there are unknown numbers of infectious agents which may be infecting horses today for which we have no suitable methods of detection. Suffice it to say that there is risk in administering plasma products to the horse, and the risk level can be lowered, but not totally removed, if the source of the plasma is a certified herd. Because of these risks, plasma should only be used when indicated.

Indications for plasma use in FPT are clear and proven effective. For example, monitoring passive antibody absorption in the foal is common practice and plasma indicated when levels of IgG fall below 800mg/dl.

It has almost become standard practice in areas of the world where *Rhodococcus equi* is endemic to administer 1 liter of plasma to foals at birth and again 30-45 days later in attempts to reduce the impact of this organism in foal morbidity and mortality. Although there are reports in the literature stating protection by administration of plasma, there are few controlled studies documenting efficacy.[9-13] In fact, protective specific immunity against *R equi* is thought to be mediated through/by cellular, not humoral, immune effectors.[14-18] As most commercial plasma preparations are devoid of cells, there appears to be a logical disconnect between use of antibody rich plasma and protection. The presence of non-immunoglobulin and non-specific factors contained in plasma must be considered potentially protective as similar effects have been seen using plasma products from random donors or from hyperimmunized donors.[19] Additionally, the potential of plasma transfusions to activate or suppress the immune responses of the recipient would be dependent on the content of non-antibody factors (e.g., interleukins or other cytokines) in the plasma, none of which are currently

monitored. Despite the somewhat conflicting evidence for its effectiveness, the almost routine use of plasma is likely to continue until research efforts demonstrate effective immunization or other prophylactic methods which are proven to be more effective than current approaches. The frustrations practitioners have with Rhodococcus pneumonia are shared by researchers and this disease remains a top priority for research funding.

Observations of field transmission of EIAV

The transmission of EIAV is generally thought to be effected by the mechanical transfer of blood from an infected horse, with no evidence of agent replication in insect vectors. Man is considered to be the vector with the greatest potential for transmission because of the high volumes of blood transferred by transfusion, by use of contaminated needles and syringes and by inadvertent transfer of blood between horses by contaminated personnel and equipment. The best insect vectors appear to be the biting flies of the Family Tabanidae, because their bites inflict pain which tend to elicit host defensive behavior (e.g., a muscle twitch) which interrupts the blood feeding. Once interrupted the insect often seeks an alternate blood source, such as another horse in close proximity, in an attempt to feed to repletion.

Ireland has no indigenous snakes, was free of EIA, and has rather limited distribution of blood-feeding insects. On closer inspection, there appear to be at least 5 tabanid species in Ireland (3 horsefly species and 2 deer fly species, according to the National Biodiversity Network website[20]) and the stable fly (*Stomoxys calcitrans*), all of which are presumed (tabanid) or proven (stable fly) efficient mechanical vectors of EIAV.[21] It is of interest to note that during late July 2006, tabanid populations were noted on several of the restricted farms with active cases of EIA. Tabanids are also affectionately called beasties in Ireland and are recognized nuisances by trekkers and horseback riders.

Despite the fact that the major spread of EIA in Ireland occurred during the summer vector season and that a number of cases were found after the horses had been at pasture in close proximity to other horses, only 3 secondary cases were thought to have been acquired through vector transfer. How could facilities and procedures in Ireland have contributed to a lower number of cases than expected?

Data indicate that if a horsefly is interrupted during blood-feeding the chance of a second horse being the blood source for additional feeding decreases in a linear fashion as distance from the initial horse increases.[22-23] These data suggest that the chance of transmission from an active case would likely be dramatically reduced (perhaps in excess of 50%) by separating the horses by a double fence (<5 meter separation), a standard on many breeding farms. Frankly we were surprised by the low number of secondary cases attributed to vector feeding. It would be of interest to know the exact conditions where all restricted horses were kept to study the possible effects from double fencing and other factors which may have contributed to the low transmission rate observed. We await official publication of the extensive DAF findings once they have released legal constraints.

Adopting "Standard Precautions"

In human medicine, standard precautions, which synthesize the major features of body substance isolation and universal precautions (initially designed to address bloodborne infections), are adopted to prevent transmission of a variety of organisms[24] Today we are fortunate that horses are infrequently infected with agents that are present in blood in sufficient quantities to pose a risk to man, but a number of horse-specific agents can be transferred by blood and other secretions and excretions free of visible blood. Thus we should be cautious and perhaps adopt the methods of containment used in the human medical field when dealing with contamination of facilities, e.g., the hemorrhages in this case. This is true even if pre-admission testing indicates freedom from selected agents; no testing can guarantee the individual is not shedding the agent in question without additional

safeguards of time and space. Additionally, agents such as EHV-1 are carried by most horses, and shedding can be precipitated by stress, i.e., absent today but present several days later. Although necessary and somewhat reassuring, negative results on testing do not indicate a total lack of risk and should not be overly relied upon, because a false sense of security can emerge.

The approved and effective blood spill actions include the following:

1. Use of absorbent materials to contain the spill
2. Use of effective disinfectants in sufficient concentrations and for sufficient time to inactivate the infective agents in the spill
3. Cleaning of the area of the spill
4. Thorough disinfection of the area (concentration and time are important here, too)

In veterinary medicine, the physical contamination of facilities by blood from a large animal with uncontrolled hemorrhage can be extensive. The strict adoption of the time-tested methods listed above can be problematic or virtually impossible for large animal facilities. Use of non-toxic area-wide decontamination treatments, e.g., by using hydrogen peroxide vapor generators, may be a practical option/adjunct. By adopting the principles of **"contain, disinfect, clean, disinfect"** rather than the methods learned by many of us of "cleaning and disinfection", we can do a better job of reducing iatrogenic/nosocomial risks.

Unfortunately, the majority of veterinary hospitals throughout the world were not designed to be high security isolation facilities to contain highly infective agents in individual enclosures. Veterinary medicine has been victimized in recent years by hospital outbreaks of a number of serious diseases; even the most careful and proactive programs have not been immune to these events. The best approach to minimize these outbreaks appears to include:

1. Establish and follow guidelines for monitoring and containing infective agents
2. Apprise clients of the potential risks of hospitalization and your control plan
3. Monitor individual patients for selected agents throughout their stay
4. Pray….and know that your faith in science is sound but realize that science is fallible

For more detailed information, including recommendations for specific organisms, the reader is referred to the Vet Clinics of NA monograph and AAEP web sites.[1,2] Additionally, standard precautions for veterinary practices have been recently promulgated by the National Association of State Public Health Veterinarians and are available on their web site.[3]

Unique strain of EIAV?

One handy explanation for the unique spread of EIA within the hospital in Ireland could be that the virus strain had mutated to a form that is more likely to be spread by aerosol. Although this is possible, the ease of spread appeared to be associated only with the hospital event of June 13-14. Few secondary cases of EIA were defined and no tertiary cases were found, suggesting that the increased risk at the hospital was a unique event not related to specifics of the virus strain.

Lessons learned and wisdom gained

The experience with EIA in Ireland in 2006 has taught us some valuable lessons:

1. International dialog on infectious diseases must remain open, honest and transparent.
2. Standards for production, use and safety of products from horses should be reexamined.
3. The potential for man to transmit EIA between horses cannot be overstated.
4. Transmission dynamics of EIA virus by blood-feeding insects is predictable.

5. SOPs for veterinary facilities should be reviewed.
6. We should develop a better understanding of all infectious diseases of horses.

Risks of acquiring EIA in the US: 2007

The Coggins test was accepted and used throughout the world by 1974. Since the late 1970's in the US the number of test positive horses found each year has decreased dramatically. Rather than the common occurrence of clinical EIA at racetracks in the early 1970's, today in the mobile tested population EIA is a rare occurrence. In 2006 the reported number of equids positive for EIA was 188 out of nearly 2 million samples tested (<0.01%). If the 86 cases that were found on 2 premises are discounted, the rate decreases even further.[25] Testing for over 30 years has reduced the likelihood of encountering EIA to such an extent that recommendations have been made to reduce the interval of testing from every 12 months to every 24-36 months in most areas of the country. The exceptions would be in states where history suggests a higher prevalence of infection in the untested reservoir population.

The challenge for "smarter testing" includes doing a better job of delivering testing to the equids who have never been tested. In states where it has been studied, a required test for change of ownership appears to be an effective mechanism for reaching the untested reservoir in a manner designed to protect the new owner while not being construed as intrusive.

As the rate of EIA in the US continues to decline, the natural tendency is to consider it an exotic agent, and adopt a "not present in" attitude. Once it attains that status, an inevitable consequence may be the Ireland scenario on our turf. We must not become complacent about EIA. We can, however, urge the adoption of smarter testing options to conserve some of the $50 million spent each year for EIA testing.[26] We can and must deliver better surveillance testing at lower cost to the industry.

Combating the stigma of EIA

Recently an online publication talked about the stigma associated with EIA and how it was misplaced on the infected horse, which can be effectively managed a safe distance from other horses.[27] The true stigma belongs on untested horses, which represent about 60% of the horses in the US. Collectively, we estimate, they pose the major risk for transmission. Their uncontrolled movement and commingling is calculated to be more than 10,000,000 times more important for transmission than from the safely (200 yard segregation) quarantined infected horse, even if the assumption is made that the rate of EIA is 1 in 10,000 in the untested horses.

Another feature about EIA that continues to surprise us is that the psychological fear of EIA greatly exceeds the risk it poses as a biologic agent of disease. The outbreak of EIA in Ireland, where the majority of cases were attributed to human contact/intervention, reminds us that we must take active measures to prevent iatrogenic/nosocomial transmission, and that can only be accomplished if EIA remains on our collective radar.

Action plan to maximize lessons learned from Ireland

(changes at Troytown are added parenthetically)

- **Test all hospital admissions**
- **Try to maintain greater separation of personnel and patients by time and space to reduce in-hospital transmission**
- **Where possible design and maintain strict isolation facilities for suspect cases until a diagnosis is reached** (Troytown has constructed two new isolation units with viewing stations and separate air spaces. All horses whose presenting condition has not yet been given a name enter the isolation unit initially until blood tests, etc., are completed. Troytown has also constructed a separate off-hospital

isolation facility (1 mile away) to house any horse with initial presenting symptoms suggestive of a notifiable disease.)

- **Insist on keeping detailed written records on admissions, treatments, personnel, and in-hospital location and movement** (This proved invaluable at Troytown.)
- **Consider adopting standard precautions for horses** (All horses at Troytown are now handled with gloved hands. Blood spills are contained and disinfected before cleaning.)
- **Demand a safety and efficacy review of equine plasma products from producers**
- **Work to help develop effective biologics against Rhodococcus pneumonia**
- **Continue surveillance testing for EIA and encourage testing by risk, not by regulation**

References

1. Advances In Diagnosis and Management of Infection. 2006. Vet Clinics of North America 22(2):279-661. Southwood, LL, editor. Elsevier Press.

2. Equine Infectious Disease Outbreak: AAEP Control Guidelines. 2006. *Developed by the AAEP Infectious Disease Task Force.* (available online to members at https://www.aaep.org/control_guidelines_intro.htm).

3. The National Association of State Public Health Veterinarians in 2006 published a Veterinary Standard Precautions Compendium and a Model Infection Control Plan for Veterinary Practices http://nasphv.org/documentsCompendia.html which are available on their web site.

4. The reader is directed to the Irish Department of Agriculture and Food website (http://www.agriculture.gov.ie/index.jsp?file=animal_health/EIA/index.xml) for the official version.

5. Issel, C.J., Adams, W.V. Jr., and Foil, L.D. 1985. Prospective study of progeny of inapparent equine carriers of equine infectious anemia virus. Am. J. Vet. Res. 46:1114-1116.

6. Issel, C.J. and McConnico, R.S. 2001. The risk of EIA in foals. Equine Disease Quarterly Newsletter 9(2): 3-4.

7. Quinlivan, M, Cook, RF, and Cullinane, A. 2007. Real-time quantitative RT-PCR and PCR assays for a novel European field isolate of equine infectious anaemia virus based on sequence determination of the gag gene**.** Vet Rec 60(18):611-618.

8. Hanson, PR, Spalatin, J, and Dickinson, EM. 1967. Criteria for determining the validity of a virus isolation. Avian Dis 11(3):508-514.

9. Martens, RJ, Martens, JG, and Fiske, RA. 1989. Rhodococcus equi foal pneumonia: Protective effects of immune plasma in experimentally infected foals. Equine Vet. J. 21(4):249-255.

10. Caston, SS, McClure, SR, Martens, RJ, Chaffin, MK, Miles, KG, Griffith, RW, and Cohen, ND. 2006. Effect of hyperimmune plasma on the severity of pneumonia caused by Rhodococcus equi in experimentally infected foals. Vet Ther. 7(4): 361-375.

11. Madigan, JE, Hietala, S, and Muller, N. 1991. Protection against naturally acquired Rhodococcus equi pneumonia in foals by administration of hyperimmune plasma. J. Reprod. Fert., Suppl. 44: 571-578.

12. Higuchi, T, Arakawa, T, Hashikura, S, Inui, T, Senba, H, and Takai, S. 1999. Effect of prophylactic administration of hyperimmune plasma to prevent Rhodococcus equi infection on foals from endemically affected farms. J. Vet. Med. B 46: 641-648.

13. Giguere, S, Gaskin, JM, Miller, C, and Bowman, JL. 2002. Evaluation of a commercially available hyperimmune plasma product for prevention of naturally acquired pneumonia caused by Rhodococcus equi in foals. JAVMA, 220(1): 59-63.

14. Patton, KM, McGuire, TC, Fraser, DG, and Hines, SA. 2004. Rhodococcus equi-infected macrophages are recognized and killed by CD8+ T lymphocytes in a major histocompatibility complex class I-unrestricted fashion. *Infect Immun* 72:7073-7083.

15. Kohler, AK, Stone, DM, Hines, MT, Byrne, BA, Alperin,DC, Norton, LK, and Hines, SA. 2003. Rhodococcus equi secreted antigens are immunogenic and stimulate a type 1 recall response in the lungs of horses immune to R. equi infection. *Infect Immun* 71:6329-6337.

16. Hines, SA, Stone, DM, Hines, MT, Alperin, DC, Knowles, DP, Norton, LK, Hamilton, MJ, Davis, WC, and McGuire, TC. 2003. Clearance of virulent but not avirulent Rhodococcus equi from the lungs of adult horses is associated with intracytoplasmic gamma interferon production by CD4+ and CD8+ T lymphocytes. *Clin Diagn Lab Immunol* 10:208-215.

17. Giguere, S., Wilkie, BN, and Prescott. JF. 1999. Modulation of cytokine response of pneumonic foals by virulent Rhodococcus equi. *Infect Immun* 67:5041-5047.

18. Hines, SA, Kanaly, ST, Byrne, BA, and Palmer, GH. 1997. Immunity to Rhodococcus equi. *Vet Microbiol* 56:177-185.

19. Perkins, GA, Yeager, A, Erb, HN, Nydam, DV, Divers, TJ, and Bowman, JL. 2001. Survival of foals with experimentally induced Rhodococcus equi infection given either hyperimmune plasma containing R. equi antibody or normal plasma. Vet Ther. 2(3): 334-346.

20. The National Biodiversity Network can be accessed at http://www.nbn.org.uk/ . From their gateway site, searching for species and their known locations/distributions can be accomplished.

21. Foil, LD, and Issel, CJ. 1991. Retrovirus transmission by arthropods. Annu Rev Entomol 36:355-381.

22. Foil, LD. 1983. A mark-recapture method for measuring effects of spatial separation of horses on tabanid (Diptera) movement between horses. J Med Entomol 20:301-305.

23. Barros, ATM, and Foil, LD. 2007. The influence of distance on movement of tabanids (Diptera: Tabanidae) between horses. Vet Parasitology 144:380-384.

24. A description of standard precautions and universal precautions can be found at the CDC website http://www.cdc.gov/ncidod/dhqp/bp_universal_precautions.html . The terms are often used interchangeably but standard precautions include agents where contact and/or aerosol transmission occurs.

25. Testing statistics compiled by the USDA from 1972 through 2006 can be accessed at http://imsprod.aphis.usda.gov/website/eia_grouped_toc/viewer.htm The data are updated each year in the fall.

26. Issel, C.J. and Anderson, G. 2007. The stigma of EIA is misplaced. Article#9038 on TheHorse.com: http://www.thehorse.com/ViewArticle.aspx?ID=9038).

27. Issel, C.J., Cordes, T.R., and Halstead, S. 2007. Control of EIA should take some new directions. Article#8787 on TheHorse.com: http://www.thehorse.com/ViewArticle.aspx?ID=8787).