The AETA, the Nebraska Funeral Directors Association (“NFDA”) and the Nebraska Veterinary Medical Association (“NVMA”) have settled the litigation that they brought against GMO, Don Ellerbee, and Theresa Kehoe. As these cases progressed, it became clear that GMO, Ellerbee, and Kehoe would declare bankruptcy as a result of the judgments that the AETA, NFDA, and NVMA expected to obtain. Following several months of negotiations, the AETA, NFDA, and NVMA obtained a settlement offer that requires the defendants to contribute more to the settlement than would be available if the defendants sought bankruptcy protection. As a result, the AETA will recover more from the settlement than we would have obtained had we continued with the litigation, obtained a judgment for our losses, and then attempted to collect the judgment through the bankruptcy court. On Monday, July 11, 2005, the AETA Board of Directors accepted the settlement proposal that requires GMO, Ellerbee, and Kehoe to pay a total of $165,000 to the AETA, NFDA, and NVMA. The settlement funds will be apportioned among the AETA, NFDA, and NVMA based upon a formula which requires each entity to share in the cost of legal fees incurred and distributes the remainder based upon the pro rata share of losses suffered by each entity. The AETA will receive the largest portion of the settlement and recover $63,833.33. The Board and the GMO Resolution Committee voted unanimously to accept this proposal. Although the settlement does not fully reimburse the AETA for its losses, it does maximize our recovery from GMO, Ellerbee, and Kehoe. The AETA has now focused its efforts on the claims that it has against the financial institutions involved in allowing Kehoe to make unauthorized transfers of the AETA’s funds. Summerlin and the GMO Resolution Committee hope to have these claims resolved before our annual business meeting this fall. A full report on the settlement will also be available at that meeting.

Turning to meeting activities, now you should have received the registration information for the convention in Minneapolis. Pat Richards and David Duxbury have assembled some outstanding speakers and have been very busy planning an excellent meeting. The general theme this year is practical, useful bovine and equine embryo transfer techniques. Bovine and equine 101 seminars will be offered as well as informative discussions on disease control, donor management, and embryo freezing. For the participant that is looking for some real ‘take home’ material, this meeting has a great deal to offer. The companion tours look very entertaining, and again this year we are offering a fishing outing for the non-golfers. All registered members and a companion are invited to the pre-conference social. There will not be a charge for this event this year. Please take a minute to register for this meeting and join us in Minneapolis.

The Board of Directors agreed in April that the AETA should explore the feasibility of meeting with the Society for Theriogenology in 2007. The meeting is scheduled for August 7–11, 2007 in Monterey, California. The Board felt that this was an opportunity to bring together three organizations, the AETA, CETA, and SFT, which have similar interests and mission statements. Although most of the details are yet to be worked out, we felt this was a progressive step that may benefit all of our memberships. It may improve our attendance and financial situation as well. Each organization, SFT and AETA/CETA, will have its own program, but participants can attend any session they would like. In 2001, we met with CETA in Nashville, and that has been a very positive move for both organizations.

It has been relatively smooth sailing for the AETA the last few months. The Board considered co-sponsoring a special interest seminar with FASS prior to the fall meeting. We were unable to work out all of the details this year but are exploring the possibility of a seminar sometime next year. It looks as though we may be nearing the end of the GMO situation. I hope we can have a final resolution by our meeting in Minne-
I would like to thank Randall and his committee for all of their time and hard work.

John Hasler underwent open heart surgery in May to repair his mitral valve. The surgery was successful, and John is well on his way to recovery. He is feeling good enough that he agreed to speak to us in Minneapolis. Good luck, John.

I hope all of you will consider attending our annual meeting in September. It promises to be another great time with our Canadian colleagues.

### A Closer Look: Advertising Rates for 2005

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17890 WCR 5
Berthoud, CO 80513
PHONE: (303) 859-2439
FAX: (970) 535-0788
E-mail: tgeneticwest@aol.com

Vice President
Dr. Pat Richards
Pat Richards DVM
1215 E. 2000 S.
Bliss, ID 83314
PHONE: (208) 539-3076
FAX: (970) 355-0788
E-mail: drpat@richards.myrf.net

Secretary-Treasurer
Dr. Ron Kling
New Vision Transplants
456 Springs Road
Grantville, MD 21536
PHONE: (301) 895-5232
FAX: (301) 895-5237
E-mail: godsmusic7@verizon.net

Immediate Past President
Dr. Larry Kennel
Cornerstone Genetics
1489 Grandview Road
Mount Joy, PA 17552
PHONE: (717) 653-4825
FAX: (717) 653-6966
E-mail: lkdvm@juno.com

Directors
Dr. Todd Bickett
Bickett Genetics, Inc.
455 Brotherton Lane
Chickamauga, GA 30707
PHONE: (706) 375-6586
FAX: (715) 268-9900
E-mail: tjbickett@aol.com

Dr. David Duxbury
Midwest Embryo Transfer Service
1299 South Shore Drive
Amery, WI 54001
PHONE: (715) 268-9900
FAX: (715) 268-2691
E-mail: etmets@amerytel.net

Dr. Steve Hughes
7732 Garnett Street
Lenexa, KS 66214
PHONE: (913) 961-6666
FAX: (913) 961-6666
E-mail: shb2897@yahoo.com

Dr. Cheryl F. Nelson
Nelson Reproductive Service
1735 Pinckard Pike
Versailles, KY 40383
PHONE: (859) 873-7319
E-mail: cnl79@qx.net

Dr. Richard O. Whitaker
New England Genetics Corporation
10 Business Park Way
Turner, ME 04282
PHONE: (207) 225-2722
FAX: (207) 225-3883
E-mail: lkdvm@juno.com

AETA Committees

AUDIT COMMITTEE
Daniel Horncnickel, DVM, Chair
Sunshine Genetics Inc.
W7782 Hwy 12
Whitewater, WI 53190
PHONE: (262) 473-9095
FAX: (262) 473-3660
E-mail: Dan@sunshinegenetics.com

Committee Members:
Edwin Robertson, DVM
Richard Whitaker, DVM

CERTIFICATION COMMITTEE
Stephen Malin, DVM, Chair
Malin Embryo Transfer
N5402 Highway 151
Fond du Lac, WI 54937
E-mail: malin@spiritusa.net

Committee Members:
David C. Faber, DVM
Larry Horstman, DVM
Joseph M. Wright, DVM
James K. West, DVM

CONVENTION EXHIBIT COMMITTEE
David B. Duxbury, DVM, Chair
Midwest Embryo Transfer Service
1299 South Shore Drive
Amery, WI 54001
PHONE: (715) 268-9900
FAX: (715) 268-2691
E-mail: etmets@amerytel.net

Committee Members:
Phil Buhanan, DVM
Dan Horncnickel, DVM
Walter North, DVM

CONVENTION/PROGRAM PLANNING COMMITTEE
Patrick M. Richards, DVM, Chair
1215 East 2000 South
Bliss, ID 83314
PHONE: (208) 539-3076
FAX: (208) 352-1934
E-mail: etmets@amerytel.net

Committee Members:
Cheryl Nelson, DVM
Dave Duxbury, DVM
Larry Lanzon, DVM

COORDINATING COMMITTEE
Scott W. Armbrust, DVM, Chair
Paradoxxs Embryo Transfer Inc.
121 Packerland Drive
Green Bay, WI 54303
PHONE: (920) 498-8262
FAX: (920) 498-8181
E-mail: pdocset@sbglobal.net

Committee Members:
Darrel DeGroff, DVM
James West, DVM
Richard S. Castleberry, DVM
Byron Williams, DVM

GMO RESOLUTION COMMITTEE
Randall H. Hinshaw, DVM, Chair
Ashby Embryos/Ashby Herd Health Services Inc.
2420 Grace Chapel Road
Harrisonburg, VA 22801
PHONE: (540) 433-0430
FAX: (540) 433-0452
E-mail: RHinshaw@rica.net

Committee Members:
Darrel DeGroff, DVM
Dan Horncnickel, DVM
Steve Malin, DVM

GOVERNMENT LIASON & ANIMAL HEALTH & REGULATIONS COMMITTEE
Richard O. Whitaker, DVM, Chair
New England Genetics Corporation
10 Business Park Way
Turner, ME 04282
PHONE: (207) 225-2722
FAX: (207) 225-3883
E-mail: moocod@meaglink.net

Committee Members:
David Duxbury, DVM
Chuck Gue, DVM

MANUALS, PROMOTION, AND MEMBERSHIP COMMITTEE
Thomas A. Borum, DVM, Chair
Borum’s Veterinary Embryonics
145 East Franklin Street
Natchez, MS 39120
PHONE: (601) 442-5523
E-mail: aeta@borum.com

Committee Members:
Richard Zimmkas, DVM
Ronald Kling, DVM
Stanley Huels, DVM

MEMBERSHIP COMMITTEE
Cheryl F. Nelson, DVM, Chair
Nelson Reproductive Service
1735 Pinckard Pike
Versailles, KY 40383
PHONE: (859) 873-7319
E-mail: cnl79@qx.net

Committee Members:
Robert Zimmkas, DVM
Kathy Creighton Smith
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Identifying Reservoirs of Infection: A Conceptual and Practical Challenge

Daniel T. Haydon,* Sarah Cleaveland,* Louise H. Taylor,* and M. Karen Laurenson*

*University of Edinburgh, Roslin, U.K.


Many infectious agents, especially those that cause emerging diseases, infect more than one host species. Managing reservoirs of multihost pathogens often plays a crucial role in effective disease control. However, reservoirs remain variously and loosely defined. We propose that reservoirs can only be understood with reference to defined target populations. Therefore, we define a reservoir as one or more epidemiologically connected populations or environments in which the pathogen can be permanently maintained and from which infection is transmitted to the defined target population. Existence of a reservoir is confirmed when infection within the target population cannot be sustained after all transmission between target and nontarget populations has been eliminated. When disease can be controlled solely by interventions within target populations, little knowledge of potentially complex reservoir infection dynamics is necessary for effective control. We discuss the practical value of different approaches that may be used to identify reservoirs in the field.

Infectious agents that can infect more than one host species are ubiquitous. Indeed, 62% of all human pathogens are classified as zoonoses (1), and 77% of livestock pathogens and 91% of domestic carnivore pathogens infect multiple hosts. Fifty seven of the 70 animal diseases considered to be of greatest international importance infect multiple hosts (2). The ability of pathogens to infect a wide range of hosts has been demonstrated as a risk factor for disease emergence in both humans (1) and domestic animals (2). Virtually all recent outbreaks of disease in endangered wildlife have been caused by pathogens that can infect other, more abundant host species (3,4).

Pathogens that infect more than one host species are by definition likely to be encountered in several host populations, some of which may constitute infection reservoirs. Therefore, a key issue in the design of control measures for multihost pathogens is defining what is meant by reservoirs of infection and developing guidelines for their identification.

Although many emerging diseases of human, domestic animal, and wildlife populations are assumed to be maintained in reservoir hosts (4), these reservoirs are rarely identified. In recent years, several emerging infectious disease threats to human and animal health have been managed through large-scale measures directed at suspected reservoirs of infection. Sometimes action arises from a clearly perceived notion of where infection resides. For example, approximately 1 million pigs were slaughtered in Malaysia in 1999 to control Nipah virus (5); several million chickens were slaughtered in Hong Kong in 1998 and 2001 to prevent a projected pandemic of Influenza A virus (6); and several million cows were slaughtered in Britain to curtail the epidemic of bovine spongiform encephalopathy, and its possible transmission to humans (7). However, many situations exist in which the role of reservoirs is less clear; for example, the reservoirs that harbor emerging viruses such as Ebola and Marburg remain unknown. For Mycobacterium bovis in the United Kingdom, a complex reservoir system seems most likely, and identification of the most important source of infection for cattle remains highly controversial (8). Incomplete understanding of reservoirs has hampered control of many diseases in Africa, such as Ebola virus infection, Buruli ulcer, and rabies (9–13).

Many different and often contradictory definitions of reservoirs exist. Studies stress different characteristics of reservoirs, namely, that infections in reservoir hosts are always nonpathogenic; any natural host is a reservoir host; the reservoir must be a different species; reservoirs are economically unimportant hosts; or reservoirs may be primary or secondary hosts (14–18). Some definitions imply that a reservoir comprises only one species; other definitions suggest that an ecologic system may act as a reservoir (16,18). Confusing, conflicting, and often incomplete concepts of what constitutes a disease reservoir result. We propose a conceptual framework for defining and identifying reservoirs and discuss the practical value of different approaches that may be used to identify reservoirs in the field.
Proposed Framework

We propose the following approach, which can be applied to any disease system, for understanding the role of potentially relevant reservoirs in that system. Figure 1 illustrates how this framework might be applied to various systems.

Suggested Terminology

The target population is the population of concern or interest to us. All other potentially susceptible host populations that are epidemiologically connected directly or indirectly to the target population are nontarget populations and could potentially constitute all or part of the reservoir. If we are interested in protecting humans (the target species) from cryptosporidiosis, for example, the wide range of domestic and wild animal species in the environment in which Cryptosporidium parvum occurs is the nontarget population, and those species constitute potential reservoir hosts.

In epidemiologic theory, the critical community size is the minimum size of a closed population within which a pathogen can persist indefinitely. In smaller populations the number or density of infected hosts frequently falls to low levels, random extinction (fadeout) becomes inevitable, and the pathogen cannot persist. Populations smaller than the critical community size, or those rendered effectively smaller than that critical size through control measures, we term nonmaintenance populations. Pathogens will persist in populations larger than the critical community size, and these populations we term maintenance populations. In complex systems, pathogen transmission between a number of nonmaintenance populations could constitute a maintenance community. Any population that transmits infection directly to the target population, we define as a source population. Source populations may themselves be maintenance populations or, alternatively, may constitute all or part of a transmission link from a maintenance population to the target population.

If a target population is smaller than the critical community size and thus cannot maintain a pathogen, completely isolating the target population from any transmission from outside (ring-fencing) will cause the pathogen to become extinct in the target population. A reservoir is present if the pathogen repeatedly appears in such a nonmaintenance target population. For example, completely preventing tick transmission of Borrelia spirochetes to humans from other spe-

Figure 1. Examples of simple and more complex target-reservoir systems.
cies would result in Lyme disease’s disappearance from humans; thus, a reservoir must exist. This procedure for identifying reservoirs will not apply to maintenance target populations. However, in practical terms reservoirs generally only become of concern when disease control within the target population reduces transmission within a target population to a very low level relative to transmission from nontarget to target populations. For example, Foot-and-mouth disease virus (FMDV) is maintained in unvaccinated cattle populations in many parts of Africa. The identification of wildlife reservoirs (e.g., buffalo) generally only becomes important once vaccinated cattle can no longer maintain infection at the population level, as is the case, for example, in parts of southern Africa (21).

We propose that a reservoir be defined as one or more epidemiologically connected populations or environments in which the pathogen can be permanently maintained and from which infection is transmitted to the defined target population. Populations in a reservoir may be the same or a different species as the target and may include vector species. As long as a reservoir constitutes a maintenance community and all populations within the maintenance community are directly or indirectly connected to each other, the size of the reservoir has no upper limit.

**Previous Concepts of Reservoirs**

Previous reservoir definitions often required that the relevant infectious agent be nonpathogenic to the reservoir host species (14,15). However, pathogenicity, per se, has little bearing on the persistence of infectious agents in populations. Excluding the possibility of a reservoir solely because the infectious agent was pathogenic to a nontarget host—as is the case with pathogens such as Nipah, Hendra, and rabies viruses and with bovine spongiform encephalopathy—would clearly be a mistake.

Cleaveland and Dye (12) proposed criteria to identify reservoir hosts but did not take into account multihost aspects of reservoirs. Swinton et al. (16) used the terms reservoir and satellite to describe the dynamics of Phocine distemper virus in the North Sea population of harbor seals (Phoca vitulina). Infection from a satellite population effectively induces persistence of infection in the reservoir population (17). Neither population constitutes a maintenance population, but infection can be maintained in a coupled system (illustrated in Figure 1B). Both satellite and reservoir populations would be components of our reservoir.

In an insightful paper, Ashford recognized many of the problems in the simplistic use of the term reservoir and proposed a consistent definition of a reservoir as an “ecological system in which the infectious agent survives indefinitely” (18). This definition differs from ours in that it does not reference a target population and thus does not require that a reservoir be a source of infection for a target population. Ashford defined reservoir hosts as those essential to maintenance of the pathogen. We, however, argue that reservoirs may include nonessential hosts. Excluding nonessential hosts from a reservoir causes two problems. First, populations harboring infection may be nonessential to maintenance yet play a major role in transmitting the pathogen to the target population. For example, FMDV persists indefinitely in African buffalo herds; yet impala may constitute an important source of infection for the cattle target population (22) (e.g., population Z in Figure 1C). Second, as Ashford recognized, the definition of reservoir membership becomes ultimately intractable if each constituent population in the reservoir is considered nonmaintenance. Under these circumstances, a reservoir could be composed of subsets of nonmaintenance populations in a variety of ways (Figure 1E). Although a minimal definition of a reservoir is clear, a fully inclusive definition is much less so. In Figure 1D, population V is not an essential host; nonetheless, this population must be considered a component of the reservoir because, if infection is eliminated in some other parts of the reservoir, eradication would not be achieved. For the same reason, our concept of a reservoir differs from the notion of a critical species assemblage, which is defined as the minimum set of host communities in which a parasite can persist (16).

**Control of Infection**

Practical disease control requires answers to two questions: 1) Can an acceptable level of control be accomplished without consideration of a reservoir? 2) If not, what populations constitute the reservoir? Given a target-reservoir system, policies to manage infection may contain elements of three broadly different tactics: 1) target control: directing efforts within the target population with no reference to the reservoir (e.g., human vaccination against yellow fever [23]); 2) blocking tactics: directing control efforts at blocking transmission between source and target populations (e.g., game fences to control FMDV in cattle); and 3) reservoir control: controlling infection within the reservoir (e.g., culling programs, vaccination, or treatment of reservoirs). These three approaches require progressively increased levels of understanding of reservoir structure and function.

Target control has the important advantage of requiring no knowledge of potentially complex reservoir dynamics. A complete understanding of infection dynamics within the reservoir is also not necessary to implement blocking tactics, although identifying source populations in the reservoir is essential. The more precisely that source populations can be identified and the more quantitative data that are available on their relative contribution to transmission, the more efficient the allocation of resources is for disease control. Reservoir control tactics require a much more complete understanding of the structure and transmission processes that occur within the reservoir. For example, efforts directed at control-
ling infection in nonmaintenance components of a reservoir are unlikely to be effective if infection in the maintenance component of the reservoir remains uncontrolled.

The practical problem of identifying reservoirs of rabies for humans in Zimbabwe provides a useful illustration of some issues involved. After a rise in the incidence of jackal and dog rabies in the 1990s, debate has centered on whether jackals (Canis adustus) are reservoirs of this disease, an issue that has important implications for formulating national rabies-control programs (10,11). In Zimbabwe, domestic dogs are a maintenance and source population of rabies for humans. However, jackals account for >25% of all confirmed rabies cases in animals and are also an important source of infection for humans (10,11). Jackals may be important components of the reservoir as a maintenance or nonmaintenance population (Figure 2). Because rabies can be maintained in dogs without jackals, jackals are not an essential constituent population of the reservoir. But can infection persist in jackals without dogs (Figure 2B)? Jackals may constitute part of a maintenance community in conjunction with an assemblage of other wild carnivores (Figure 2A). The question is important because if dogs are the only maintenance population in the reservoir, effective vaccination campaigns targeted at dogs should successfully eliminate human rabies from Zimbabwe. If, however, jackals comprise all or part of a maintenance community independent of dogs, eliminating rabies will only be successful if jackal rabies were also controlled (10,11).

The recent high incidence of jackal rabies in Zimbabwe might suggest that jackals are maintenance populations. A high incidence of disease alone is neither necessary nor sufficient evidence for this claim, particularly when wide fluctuations in disease incidence occur (as with jackal rabies). Mathematical models suggest that jackals are probably unable to support infection without frequent reintroductions from outside sources (24). However, Bingham et al. (11) argue that spatial patterns are critical and that jackal epidemics may be sustained independently within key geographic areas. The issue can be resolved unequivocally through implementation of a mass dog vaccination campaign, which would be a logical first phase of a national program. If jackal rabies persists in the absence of dog rabies, an effective program for rabies elimination will likely need to include oral vaccination of jackals.

Rabies also provides an example of the need to identify a target population when defining reservoirs. In the Serengeti Plain in Tanzania, a distinct strain of rabies appears to be maintained independently in spotted hyenas, without causing them any clinical disease, and with no evidence of spillover infection or disease occurring in any other species (within the limits of current knowledge) (25). By our definition, unless this strain is identified as the cause of disease in another species (i.e., a target population), hyenas in the Serengeti cannot be considered as a reservoir of rabies.

**Practical Indicators To Identify Reservoirs**

Newly emerging diseases usually originate from reservoirs of infection in other host species. When such diseases first appear, only rapid, accurate identification of the reservoir will enable appraisal of the full range of disease-control options. Ring-fencing is clearly impractical when no knowledge of the reservoir populations exists, but other steps can be taken to acquire progressively more detailed information about the reservoir structure.

**Epidemiologic Evidence of Association**

Accumulating epidemiologic evidence is often the best first step in identifying a reservoir. Initially, such analyses are often based on sparse data and are rarely published. Links be-
between target and reservoir may be particularly elusive when transmission from reservoir to target is rare or sporadic, as, for example, occurs with Ebola virus or Marburg virus (26).

Quantitative data on risk factors for infection can be obtained through more formal epidemiologic research, such as case-control and cohort studies. For example, a case-control study of Borna disease in cats indicated that hunting mice was a risk factor and that rodents might be virus reservoirs (27). Case-control studies have identified badgers as risk factors for *M. bovis* infection in cattle in some parts of the United Kingdom (28). In other cases, putative reservoirs have been ruled out. For example, a risk factor analysis of *Helicobacter pylori* infection in young children showed that household pets were not incriminated (29). Although such associations may suggest a link between reservoir and target populations, further evidence is required to establish the identity of a reservoir.

**Evidence of Natural Infection in Nontarget Populations**

Identifying natural infection is a useful step towards determining natural hosts that may constitute potential reservoirs. Natural infection may be determined in two ways: by identifying previous infection through antibody detection or by identifying current infection through isolating the infectious agent or its genes from the host. The appropriate approach depends on the longevity of the infection in the host and the resources available. For example, very large sample sizes might be required to isolate a virus from a reservoir population; a serologic survey might be less expensive and more feasible. In a number of studies, demonstration of natural infection has been considered strong evidence that hosts are reservoirs, e.g., *Leishmania* in small mammals in Iran (30) and hantavirus in rodents in the Americas (31).

Seropositivity indicates that infection has occurred. However, not all natural hosts are reservoir hosts, and to include a nontarget population in a reservoir, evidence of transmission to the target population, direct or indirect, must exist. Furthermore, the level of seroprevalence does not provide information as to whether a nontarget population is a maintenance host. High seroprevalence at a single point in time may simply indicate an outbreak in the host population, rather than pathogen persistence (32). Low seroprevalence may arise when case-mortality rates are high in the reservoir (as in rabies infections), during an interepidemic trough, or when a pathogen persists at a stable but low prevalence, particularly when the duration of the infectious period is high (e.g., as in carrier animals). The critical issue is the persistence of infection in the reservoir, which can only be determined through longitudinal studies.

Similar guidelines apply to data based on demonstration of the pathogen within a host. For example, detection of *Trypanosoma brucei gambiense* in wild ruminants and primates in West Africa has been taken as evidence of an animal reservoir for Gambian sleeping sickness (33). However, as animal-to-human transmission has never been demonstrated, wildlife remain classified as potential reservoir hosts, and disease control relies on treatment of people. In contrast, for Rhodesian sleeping sickness, isolation of *T. brucei rhodesiense* from a single bushbuck in the 1950s (34) led to the assumption that wildlife was the principal reservoir for human disease and resulted in widespread culling of wildlife for disease control. Only in 1966 were cattle identified as reservoir hosts (35). Current strategies focus on treating cattle with trypanocidal drugs (36).

Detecting a pathogen, particularly its transmission stage, in secretions or tissues provides supportive, but not unequivocal, evidence that transmission to the target population can occur. Even where experiments demonstrate that transmission is possible, it may not occur in nature for a variety of behavioral or social reasons, because the population is below critical community size or because of constraints of pathogen life history.

**Genetic/Antigenic Characteristics**

Genetic and antigenic characterization of pathogens isolated from different populations provides a more powerful tool for identifying key components of reservoirs. Antigenic and genetic variation of pathogens isolated from the target population within the range observed in the reservoir is consistent with reservoir-target transmission. This pattern can be demonstrated by applying phylogenetic methods to sequence, random amplified polymorphic DNA, or restriction fragment length polymorphism data, or by using serum cross-reactivity studies. Such methods have also been used to rule out important animal reservoirs of human disease in studies of *Ascaris* in Guatemala (37) and *Cryptosporidium* in Australia (38).

**Intervention Studies**

Complete ring-fencing of target populations is the ultimate step in identifying the existence and structure of reservoirs. In practice, however, ring-fencing has rarely been achieved and, as a result, even those reservoirs we consider to be most fully understood are not usually incontrovertibly proven. Despite this, once a potential reservoir is identified, intervention studies can permit incidental but powerful inferences about the dynamics of infection in target-reservoir systems. In many cases, disease-control programs can effectively act as intervention studies.

Control in a reservoir host population may be achieved by reducing host or vector density (e.g., culling possums to control tuberculosis in New Zealand [39], mosquito control for West Nile fever, or sandfly control for cutaneous leishmaniasis [40]). Alternatively, control measures may focus more directly on preventing transmission from the reservoir, e.g.,
separation of cattle and wildebeest to prevent transmission of malignant catarrhal fever in East Africa (41). The success of such interventions often provides reasonable confirmation of the original assumptions concerning transmission and maintenance of infection in the target-reservoir system.

Conclusions

We have a poorer understanding of the epidemiology of multihost pathogens than simpler single-host systems. This dearth of understanding is a particular problem with emerging diseases, since most emerging human, domestic animal, and wildlife diseases infect multiple hosts. Reservoirs must be defined with reference to particular target populations. Disappearance of the pathogen in the target population after ring-fencing provides categorical evidence of the existence of a reservoir and its possible identity. However, exhaustive identification of all constituent populations of a reservoir may be difficult. This identification need not be a management priority if disease control is directed at the target population or at blocking transmission between reservoir and target. For infection to be eliminated, however, disease-control measures must be directed at the reservoir. Thus, an understanding of reservoir infection dynamics is essential.

When the risks and costs of control are low, circumstantial evidence may be sufficient to justify implementing control measures. Specifically designed intervention studies have ultimately been required to determine whether a particular species is a maintenance host, a source of infection, or one that has been infected incidentally. Control measures are likely to be ineffective if they are directed at components of the reservoir that are neither maintenance hosts nor transmitters of the pathogen to the target population.

Acknowledgments

We thank Paul Coleman, Scott McEwen, Eric Fevre, Mark Woolhouse, and three anonymous referees for commenting on versions of this manuscript, the rest of the epidemiology group at the Center for Tropical Veterinary Medicine for valuable discussions during its gestation, and Kevin Bown, whose thesis sparked the question.

Dan Haydon, Louise Taylor, and Karen Laurendon were supported by the Wellcome Trust during this work. Sarah Cleaveland was supported by the United Kingdom Department for International Development.

Dr. Haydon is currently conducting postdoctoral research at the University of Guelph. His interests include the evolutionary dynamics of picornaviruses, epidemiologic and ecological modeling, and landscape ecology.

References

Evaluation of Risks that Bovine Embryos Arising from Fertilization with Virus-Infected Semen Will Transmit Infection to Recipients

Proposed new EU legislation prompted a scientific literature review. From studies in laboratory animals, humans, and horses, it is apparent that viruses may sometimes attach to, or be integrated into, spermatozoa; although in domestic livestock, including cattle, this seems to be a rare phenomenon. Carriage of viruses through the zona pellucida into the oocyte by fertilizing sperm has never been described in these species.

Four specific viruses—enzootic bovine leukosis virus (BLV), bovine herpesvirus-1 (BHV-1), bovine viral diarrhoea virus (BVDV), and bluetongue virus (BTV)—all of which tend to cause subclinical infections in cattle, but which can occur in bovine semen, were reviewed with regard to the risks that use of infected semen might lead to production of infected embryos.

With regard to in vivo-derived embryos, when internationally approved embryo processing protocols are used, the risks from BLV- and BTV-infected semen appear to be negligible, and the same is almost certainly true for BHV-1, if the embryos are also treated with trypsin. This would apply especially to bulls that are not proven to be BHV-1 negative. For BVDV, there is insufficient data on how the virus is carried in semen and how different BVDV strains can interact with sperm, oocytes, and embryos. There is a potential, at least, that in vivo-derived embryos resulting from virus-infected semen might carry BVDV, although field studies so far suggest this is very unlikely.

With regard to in vitro-produced embryos, use of semen infected with any of the four viruses, with the probable exception of BLV, will often lead to contaminated embryos, and virus removal from IVF embryos is difficult even when the internationally approved embryo processing protocols are used. However, it has never been demonstrated that such embryos have resulted in transmission of infection to recipients or offspring.

IETS HASAC Research Subcommittee
7 January 2005
Embryo analysis in field MOET: High success rate after overnight culture of microblade-biopsied cattle embryos

K. Kananen-Anttila, K. Vartia, A. Hyvönen, J. Virta, J. Peippo, and M. Halmekytö

1Institute of Applied Biotechniques, University of Kuopio, 70211 Kuopio, Finland; 2ProAgria Osuoskunta Jalostuspalvelu, 90100 Oulu, Finland; 3MTT Agrifood Research Finland, Animal Production Research, 31600 Jakionen, Finland; and 4Finnish Animal Breeding Association, 01301 Vantaa, Finland.

Embryo storage before embryo transfer is a necessity in field MOET to enable complex embryo analysis. Cryopreservation of biopsied embryos may reduce pregnancy rates to unacceptable levels, below 40% (Shea BF 1999 Theriogenology 51, 841-854). Our primary objective was to investigate the effect of short-term storage in overnight culture on the pregnancy rate of microblade-biopsied and sexed cattle embryos. A specific aim was to apply embryo storage in culture to marker-assisted selection of MOET embryos.

Day 6.5 embryos were produced using standard superovulatory, AI, and embryo flushing procedures. Embryo donors were lactating cows of top genetic merit. Embryos were transported in straws in Holding medium (ICPbio, Auckland, New Zealand) to the laboratory and individually biopsied by a microblade.

Biopsied embryos were cultured overnight in individual oil-overlaid 20-µl drops of Medium 199 with glutamax-1 (GIBCO™, Baisley, UK) containing 0.25 mM sodium pyruvate, antibiotics, and 4 mg mL⁻¹ fatty acid-free albumin. The biopsies were lysed in proteinase K and analyzed for sex with the BOV-Y/kappa-casein PCR method (Peura et al. 1991 Theriogenology 35, 547-555). Overnight-cultured grade I-II female embryos were transferred into the uteri of heat-synchronized recipients (Day 7.5). Pregnancy was confirmed by rectal palpation at 2-3 months after ET. Grade I-II male embryos as well as embryos of unknown sex were frozen in ethylene glycol, stored in liquid nitrogen, thawed, and cultured overnight for estimation of re-expansion rate and cell counts.

In total, 74 embryos of eight donors were overnight-cultured and sexed. The success rate of the sexing method was 95%. Of the successfully analyzed embryos, 41% (29 of 70) were females and 59% (59 of 70) males. The survival rate of microblade-biopsied overnight-cultured embryos was 99% (73 of 74); 73% of the surviving embryos were of grade I, 23% of grade II, and 4% of grade III. Sixteen of the 27 (59%) grade I-II female embryos transferred resulted in pregnancy. Forty-two grade I-II embryos were frozen and 36 (86%) re-expanded after thawing and overnight culture. Twenty-four of the re-expanded embryos (67%) were of grade I. The re-expanded embryos had on average 73 cells (range 43-114).

In conclusion, overnight culture of microblade-biopsied cattle embryos does not compromise embryo viability, resulting in high pregnancy rate and post-thaw re-expansion rate. The method can be utilized as a short-term embryo storage in the field MOET scheme and it will be applied in marker-assisted selection of MOET embryos for genotypes associated with milk production.

The study was supported by the HAKA-Top breeding animals from North-Savo-project.
News Release
Texas Animal Health Commission
Box 12966 * Austin, Texas 78711 * (800) 550-8242 * FAX (512) 719-0719
Bob Hillman, DVM * Executive Director
For info, contact Carla Everett, information officer, at 1-800-550-8242, ext. 710
or ceverett@tahc.state.tx.us

For immediate release—

State-Federal Team Responds to Texas BSE Case

The US Department of Agriculture announced June 29 that genetic testing has verified that a 12-year-old cow that tested positive for Bovine Spongiform Encephalopathy or BSE originated from a Texas beef cattle herd. Tissues for laboratory testing were initially collected from the animal in November 2004, and the carcass was incinerated and did not enter the human food, animal feed or fertilizer supply system. While tests in November indicated the animal did not have BSE, retesting in England in June confirmed the animal had the disease. The Texas Animal Health Commission (TAHC), the state’s livestock and poultry health regulatory agency, and USDA have jointly assigned a state-federal team to conduct the epidemiological investigation and response.

“The TAHC and US Department of Agriculture’s Veterinary Services are working with a complement of experts from federal and state animal health, food safety, public health and feed regulatory agencies to ensure the continued safety and wholesomeness of our meat supply,” said Dr. Bob Hillman, Texas state veterinarian and executive director of the TAHC, the state’s livestock and poultry health regulatory agency.

“Epidemiological investigations are thorough and focus on verifying the herd of origin, and when, where and how the animal and potentially, any herd mates, were exposed to the abnormal prion, or disease agent, that causes BSE. Additionally, epidemiology investigations trace the infected animal’s movement and herd mates. Animals potentially exposed to the disease will be depopulated, with proper disposal. The animals will not be introduced into the human or animal food chain.”

The USDA’s BSE testing protocol requires testing of emaciated or injured cattle, cattle that exhibit central nervous system disorder, cattle unable to rise or to walk normally, and cattle that die of unknown causes. Since June 1, 2004, brain tissue samples from more than 394,000 cattle have been tested in the U.S. and were negative for BSE. Of those, 38,320 were tested in Texas, Dr. Hillman noted. BSE surveillance has been conducted in the U.S. since 1990.

The U.S. has taken preventive measures against the introduction of BSE since 1989, when prohibitions were placed on cattle and other ruminants from BSE-affected countries, noted Dr. Hillman. In 1997, the importation ban was extended to all of Europe.

Dr. Hillman said the US Food and Drug Administration (FDA) in 1997 banned the use of ruminant-derived protein (from animals such as cattle and sheep) in feed for cattle and other ruminants. There is no evidence that BSE spreads from live animal to animal in the herd, but cattle can be exposed by eating feed that contains rendered protein from infected animals. “These measures taken by the USDA and the FDA are safeguards that work to protect livestock, and ultimately, our meat supply,” he said.
WELLINGTON (Dow Jones)—An extortionist’s claim that he has released foot and mouth disease in New Zealand is very unlikely to be true, but the government has nonetheless moved quickly to limit the impact of the likely hoax on the country’s export markets.

By being open from the outset about the threat Tuesday to release the foot and mouth virus on Waiheke Island, off New Zealand’s North Island, the government is hoping that markets will respond to fact rather than rumor and innuendo.

So far, the response from international market has been fairly calm.

Japan and the European Union have requested that they receive no exports from Waiheke Island and Mexico said it will inspect each shipment of meat before allowing its import, a spokeswoman for Trade and Agriculture Minister Jim Sutton said, adding that there won’t be any exports from Waiheke at all for the time being.

Officials said Tuesday that an unknown person had written to Prime Minister Helen Clark earlier that day and had claimed to have released the virus on either Monday or Tuesday.

The extortionist demanded a sum of money and a change to the country’s taxation laws, and threatened to release the virus again in another part of the country.

New Zealand Ministry of Agriculture and Forestry’s director of biosecurity, Barry O’Neil, told reporters the threat is probably a hoax and it’s unlikely that the extortionist has the capacity to carry out the threat.

But New Zealand has never before had a confirmed case of foot and mouth disease and the government is justifiably concerned about anything that would tarnish the “clean, green” image that makes the country’s food exports so attractive in overseas markets.

Agriculture accounts for about 7% of New Zealand’s gross domestic product, while dairy and meat alone make up around a third of total goods exports.

It will be a nervous week or two for New Zealand’s farmers and government while they wait for the threat to be confirmed as a hoax.

In the meantime, the government has let trading partners know what has happened.

Overnight New Zealand trade officials briefed officials of 40 nations and the 15-member European Union on the scare.

The government is hoping that by having been open about the threat that the fallout from its international trading partners will be limited. So far that approach seems to be paying off.

**Effect Of Foot & Mouth Would Be Devastating**

Sutton himself said the international reaction has been good.

“I think the damage at this point would be very limited. The government’s decision to be open and informative has been vindicated,” he said.

“I don’t see it as a source of concern to the (agricultural) experts and the official bodies of our trading partners, but the consuming public may get a little worried even though it is not a disease that affects human beings.”

As part of the government’s response it is ensuring that no animals such as sheep, cattle and pigs, or risk materials such as fodder, meat, cheese, wool, and milk can be moved off Waiheke Island.

While it’s unlikely that foot and mouth disease has been released into New Zealand, its effects would be devastating.

In a study in 2003, the Reserve Bank of New Zealand found that even a “limited” release of the disease would cause the country’s gross domestic product to decline by 4% and push the New Zealand dollar down by 20%.

The Reserve Bank would be likely to cut interest rates by 200 to 250 basis points in each of the two quarters after the release, which would see 90-day interest rates fall to about 1% to 2%, according to the study.

Despite this gloomy scenario, reaction in financial markets has been limited after an initial knee-jerk reaction.

At 0345 GMT Wednesday, the New Zealand dollar was at US$0.7308, down slightly from the US$0.7323 before the threat became public, after dipping about 40 points soon after the news.

As Sutton said, this probably vindicates the government’s open stance, which has ensured that markets don’t have to rely on rumor or word of mouth.


If as is likely the threat is confirmed to be a hoax in a week or two, there’s unlikely to be any lasting fallout, Gibbs said.

“I would have thought it will all just disappear into the ether,” he said. “I wouldn’t be looking for any ongoing impact.”
You can be a crack shot and still never bag a trophy if you don’t have ammunition.

That pretty well describes the ability of cattle producers to hunt down an type of leptospirosis long thought to be the most common type. Until Pfizer’s recent debut of Spirovac®, there were no vaccines with the antigen to guard against hardjo-bovis specifically.

It’s not like hardjo-bovis has been hiding out; it’s everywhere. In a prevalence study by Texas A&M University’s (TAMU) Steve Wikse, samples containing hardjo-bovis equated to a national prevalence rate of 42%, with rates as high as 58% in some southern states.

Carol Bolin, an international leptospirosis expert at Michigan State University, analyzed the samples in the study and explains, “Hardjo-bovis is more prevalent in the U.S. than previously estimated. In fact, this study confirms hardjo-bovis is the most common type of leptospirosis in the U.S.”

Consequently, Wikse, and associate professor of large animal medicine in TAMU’s College of Veterinary Medicine, believes, “If you’re in the lower two-thirds of the U.S., and concerned about maximizing reproductive efficiency in your herd, I’d recommend you vaccinate for hardjo-bovis because it’s likely your herd is already infected or will become infected with the addition of new animals.

While testing is available, Wikse explains it’s so costly that vaccinating makes more economic sense. For perspective, testing protocol would include testing 15 head (basis a herd of 100). The blood and urine analysis cost $50/sample. Add in a veterinarian’s call charge and you’re talking around $1,000. Then, even if testing proves negative, it’s likely the herd will become exposed through additional purchase if they aren’t vaccinated.

Although hardjo-bovis is more prevalent in the South, Wikse believes even producers in the northern tier of states should discuss with their veterinarians the use of vaccination as a control program.

While that may sound like too general of a recommendation at first glance, add to the prevalence rate mentioned earlier the fact that the test used to identify infection is only 70% sensitive. That means 30% of the animals in the study determined to be negative for hardjo-bovis infection were actually false negatives. Thus, the prevalence rate runs even higher.

“I don’t think there’s any question about vaccinating for hardjo-bovis in all cattle because of the widespread prevalence of it in beef herds and its proven detrimental effect on fertility. Calves are just too valuable to risk losing,” Wikse says.

A delay here, an abortion there

Hardjo-bovis can cause the whole spectrum of maladies associated with reproductive pathogens, including early embryonic death, abortions, stillbirths, weak calves, delayed estrus and overall reductions in reproductive performance.

What makes it tough to determine hardjo-bovis’ exact cost is that, unlike other types of leptospirosis that producers have been able to vaccinate against for years, hardjo-bovis tends to cause sub-clinical symptoms. The other types are often associated with acute clinical signs.

According to researchers, the contrast has to do with the fact that hardjo-bovis produces what’s termed a “host-adapted infection” in cattle. That means the maintenance host animal lives with the infection, shows no overt symptoms, but serves as a reservoir to continue shedding the organism.

The infection resides in the kidneys and reproductive tract, whereby the pathogen is shed in both urine and reproductive fluids. An added challenge with hardjo-bovis is it can also infect embryos in utero so that a calf can be born as a maintenance host.

Incidental infections (non-host adapted infections) on the other hand—caused by other common lepto types—can cause acute disease symptoms. But little of the responsible pathogen is shed in the urine, meaning there’s little animal-to-animal contamination.

Wikse is the first to point out a high prevalence of a disease organism doesn’t necessarily equate to a high level of production loss. But, in the case of hardjo-bovis, he says a growing body of evidence suggests controlling it pays more than the cost of control.

For instance, Wikse points to a study conducted in Cali...
fornia dairy heifers. Heifers infected with hardjo-bovis re-
quired 3.4 services/conception, compared to 2.1 for uninfected
heifers, and they took longer to conceive after first calving—
132 days vs. 95 days for the uninfected heifers. “You can
infer you’d see similar results with beef heifers,” Wikse says.

Vaccination and biosecurity

Availability of a vaccine to prevent hardjo-bovis is still new
enough that Wikse says there’s no industry-standard con-
trol program. But he suggests the Cadillac of programs would
be vaccinating the whole herd, including bulls, which can pass
the infectious agent along in semen as well as urine—
the agent enters through small lacerations in the skin, be-
tween the toes, etc. Vaccinating mature animals requires treat-
ing them with a long-lasting antibiotic first in order to clear
up any renal carriers (chronics). With that done, Wikse says
you’d vaccinate, booster them in 406 weeks, then plan on
boostering them every 12 months.

Where it’s not possible to treat the entire herd twice within
406 weeks, Wikse explains, “An option would be to apply
the same program, but only to replacement heifers, so that
over a period of years the entire herd, given the annual booster
vaccinations, should be protected from infection. If it’s a herd
already experiencing low levels of reproductive efficiency, I
wouldn’t recommend this slower option,” Wikse says.

Keep in mind the focus on replacement heifers also has to
do with the fact reproductive pathogens impact younger ani-
mals more severely than older ones. So the actual damage
can be largest in the younger population where the preva-
ience rate is actually lower.

Moreover, even though producers can identify hardjo-bovis
infection by means of collecting blood an urine samples from
a portion of the herd, then having diagnostic test run, Wikse
believes the high prevalence rate makes vaccination without
testing a wise economic strategy.

Ron Gill, a TAMU Extension beef specialist, says produc-
ers must understand the five-way leptospirosis vaccines
they’ve used in the past don’t contain the hardjo-bovis anti-
gen.

“To protect against hardjo-bovis, a vaccine must have the
hardjo-bovis antigen in it,” Gill says.

More specifically, traditional leptospirosis vaccines have
included protection against one type of the organism—hardjo-
prajitno—which has never been found in the U.S., while not
including an antigen for hardjo-bovis.

So, whether using a monovalent vaccine with the hardjo-
bovis antigen, along with a traditional five-way leptospirosis
vaccine, or by using a single multivalent vaccine that con-
tains the hardjo-bovis antigen, producers should make sure
they’re covering all the bases.

“Vaccination for hardjo-bovis is available, and it’s highly
effective,” Wikse emphasizes. “For a comprehensive lep-
tospirosis control program, producers should use a standard
five-way lepto vaccine, plus a vaccine that protects specifi-
cally against hardjo-bovis, or a multivalent vaccine that con-
tains it. You need both, since standard five-way lepto vac-
cines don’t have a hardjo-bovis component.”

Given the ease with which hardjo-bovis is transmitted,
Wikse points out effective control demands vaccination be
accompanied by strategic biosecurity. In addition to the age-
old wisdom of knowing some history on the source of new
additions and keeping new arrivals separate for observation,
Wikse says a growing number of producers are testing new
additions for infectious diseases before purchasing or intro-
ducing them to the herd. These infectious diseases include
bovine leukemia, neosporosis, Johne’s disease and persistently
infected BVD.

Again, because of the testing costs associated with hardjo-
bovis, Wikse repeats his advice to address hardjo-bovis by
assuming all new introductions are infected. That means treat-
ing them with a long-lasting antibiotic, then vaccinating and
boostering than for hardjo-bovis.

Whatever control strategy is chosen, Wikse emphasizes
the importance of committing to it over the long haul.

“Diseases are cyclical from year to year. In the case of
hardjo-bovis, it may not cause significant reproductive losses
every year inside infected herds, but then one year it hits you
hard,” he says.

With that in mind, Wikse believes hardjo-bovis control “is
part of a ranch management program aimed at enhancing pro-
duction. If your goal is to have as much protection as pos-
sible against disease that can potentially interfere with repro-
ductive efficiency, there is no doubt this kind of protection
more than pays for itself over time.

“Now that we have an effective vaccine against
hardjo0bovis, it’s time for producers to meet with their vet-

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General Information

Convention Registration

The registration form has been mailed to all members and posted on the AETA website.

- Early registration deadline is Monday, August 8, 2005.
- Student registration is for individuals enrolled in school full-time.
- Refunds—Early registration fee will be refunded if notification is received prior to August 17, 2005.
- There is a $50 fee for the pre-conference seminars being held on Thursday, September 8. Please indicate on the registration form the number of individuals who will be attending each session. There will be a limited number of participants for the seminars (first come–first served).

Accommodations

The 2005 AETA & CETA/ACTE Joint Convention will be held at the Marriott City Center, Minneapolis, Minnesota. Rooms are being held at a group rate of $119 per night for single/double occupancy. Reservations must be made prior to August 8, 2005. After this date, reservations will only be accepted upon availability and at the current rack rate. The convention rate will not apply for reservations after August 8, 2005. Please provide the hotel with the AETA/CETA group name when making a reservation.

***RESERVE YOUR ROOM NOW***
Marriott Minneapolis City Center
30 South 7th Street
Minneapolis, MN 55402
Tel: 800-266-9432 or 612-349-4000

Reservations can also be made thru the AETA website at www.aeta.org/05mtg.asp

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Pre-conference Events

Thursday, September 8

Golf Tournament 6:45 am – 1:30 pm  
Tee time: 7:30 am

Sponsored by Schering Animal Health Products-Makers of ESTRUMATE®

Description

Rush Creek Golf Club, a public course containing the golf amenities of a world-class resort course, stretches across 260 acres of rolling prairie and natural marshes. Located in Maple Grove, just 20 minutes west of downtown Minneapolis, Rush Creek Golf Club features an 18-hole championship course strategically designed to offer new and challenging experiences, an advanced-design teaching center, a full-service golf shop, a clubhouse, and service and hospitality generally reserved for members-only clubs. This 18-hole championship golf course has hosted many national events including three LPGA events. In addition, Rush Creek was voted “Best Public Golf Course” in 2001 and 2002 by Citysearch.com. Come out and play Rush Creek to find a true test of golf and to experience nature at its best. The golf tournament fee includes green and cart fees, use of driving range, transportation, and lunch.

COST: $95 per person

BASS Fishing Tournament 7:00 am – 2:00 pm

Sponsored by Pfizer

Description

Please do not miss this opportunity to fish one of Minnesota’s best bass lakes. Lake Minnetonka lies on the west side of the metro and provides more that 14,000 acres of water and 100 miles of shoreline for your fishing pleasure. You will fish with guides, provided by Big Dog Guide Service. There will be two people per boat, and you may pick your partner if you wish. Big Dog Guide Service will provide the rods/reels, tackle, and bait. The guides are local tournament fishermen who know the lake and can provide a good morning of fishing. Fish will be weighed and released (except for wall hangers), and the guides will track totals on weight sheets. We will fish from 7:30 am to 11:30 am and finish the morning with a lunch at Lord Fletchers on the lake. Awards will be presented for the best of the best.

FISHING LICENSES REQUIRED! You will need to buy your license before you arrive. Two options for this are

1. Phone 1-888-MNLICEN (665-4236) or
2. On line google Minnesota DNR or go to www.dnr.state.mn.us and click on Buy A License.

Please note that our date is Thursday, September 8. Your license will be mailed to you but I believe that you can fish on your confirmation number if time is short.

COST: $95 per person plus your license fee (approximately $10)

Evening at Nicollet Island Pavilion Pre-Conference Social 6:00 pm – 9:30 pm

Sponsored by Bioniche Animal Health USA and Bioniche Animal Health Canada

Description

Nicollet Island Pavilion is a beautiful building situated on an island in the Mississippi River. This unique park facility offers one of the best views of downtown Minneapolis, the historic Horseshoe Falls and the Stone Arch Bridge.

The evening will include dinner and music by the Paul Cherba Jazz Quartet. There will also be a cash bar. Transportation will be provided from the conference site to the Pavilion; however, the scenic walk (approximately 1 mile) is highly recommended (directions will be provided onsite). There will not be a charge for this evening, thanks to the very generous sponsorship of Bioniche Animal Health USA and Bioniche Animal Health Canada. This evening is limited to registered members and a companion. Children may attend if space is available. Registered exhibit representatives may attend if space permits. This event is limited to the capacity of the facility, so please send in your registration form early if you wish to attend.
**Program**

**Wednesday, September 7, 2005**
8:00 am – 10:30 am  CETA Certification Committee Meeting
9:00 am – 6:00 pm  AETA Board of Directors’ Meeting
11:00 am – 6:00 pm  CETA Board of Directors’ Meeting
1:30 pm – 5:00 pm  AETA Certification Exam
1:30 pm – 5:00 pm  CETA/ACTE Certification Exam

**Thursday, September 8, 2005**
7:00 am – 2:00 pm  Golf Tournament–“Rush Creek Golf Club”
7:00 am – 2:00 pm  Fishing Tournament– “Lake Minnetonka”
1:00 pm – 5:00 pm  Registration
12:00 pm – 5:00 pm  Exhibit Set-Up
3:00 pm – 5:00 pm  Bovine ET101 Seminar
                  Dr. Reuben Mapletoft, University of Saskatchewan, Saskatoon, SK, Canada
                  Dr. John Hasler, Bioniche, LaPorte, CO
3:00 pm – 5:00 pm  Equine ET101 Seminar
                  Structure, Management, and Techniques Used in a Commercial Equine Embryo Transfer Center
                  Dr. Phil Matthews, Peterson and Smith Equine Hospital, Ocala, FL
                  What to Expect when Superovulating Mares with eFSH: Equine Embryo Vitrification
                  Dr. Ed Squires, Colorado State University, Ft. Collins, CO

**Friday, September 9, 2005**
6:45 am – 7:45 am  Continental Breakfast in Exhibit Area
6:45 am – 7:00 pm  Exhibits Open
6:45 am – 5:00 pm  Registration
9:00 am – 4:00 pm  Companion Tour—Stillwater Shopping and Cooking Class
7:45 am – 8:00 am  Welcome and Introductory Comments
8:00 am – 8:30 am  USDA/APHIS Update
                  Dr. Sarah Kaman, National Center for Import and Export, Riverdale, MD
8:30 am – 9:30 am  Efficiency of Programs that Control Follicular Development and Ovulation for the Donor
                  Superovulation Without Estrus Detection
                  Dr. Gabriel Bo, IRAC, Cordoba, Argentina
9:30 am – 10:30 am  Management Considerations for Donor Cows
                  Dr. Cliff Lamb, University of Minnesota, Grand Rapids, MN
10:30 am – 11:00 am Coffee Break in Exhibit Area
11:00 am – 12:00 pm AETA Annual Business Meeting
11:00 am – 12:00 pm CETA/ACTE Annual General Meeting
12:00 pm – 1:30 pm CETA/ACTE New Board of Directors' Meeting
12:00 pm – 1:30 pm Lunch
1:30 pm – 2:30 pm  BVD Impact on Pregnancy and Control in ET Herds
                  Dr. Vic Cortese, Pfizer, Downington, PA
2:30 pm – 3:30 pm  Conventional Freezing Update
                  Dr. John Hasler, Bioniche, LaPorte, CO
3:30 pm – 4:00 pm  Coffee Break in Exhibit Area
4:00 pm – 5:00 pm  Management of the Post-Partum Mare
                  Dr. Phil Matthews, Peterson and Smith Equine Hospital, Ocala, FL
6:00 pm – 8:00 pm  Social “Hour” in Exhibit Area
8:00 pm  Banquet; MC will be Dr. Dan Hornickle, Sunshine Genetics, Whitewater, WI
Saturday, September 10, 2005

6:45 am – 8:00 am  AETA Past Presidents/Board of Directors’ Breakfast
7:00 am – 4:00 pm  Registration
7:00 am – 3:00 pm  Exhibits
7:00 am – 8:00 am  Continental Breakfast in Exhibit Area
10:00 am – 1:30 pm  Companion Tour—Walker Art Museum
8:00 am – 9:00 am  Application of Fixed-Time AI and Embryo Transfer Programs in Beef Cattle Operations
                  Dr. Gabriel Bo, IRAC, Cordoba, Argentina
9:00 am – 10:00 am  Leptospira borgpetersenii serovars hardjo-bovis—Impacts to Reproduction in Cattle Operations
                    Dr. Vic Cortese, Pfizer, Downingtown, PA
10:00 am – 10:30 am  Coffee Break in Exhibit Area
10:30 am – 11:30 am  AETA Certification Session
11:30 am – 12:30 pm  Embryonic Loss
                    Juan Romano, University of Minnesota, St. Paul, MN
12:30 pm – 2:00 pm  Lunch
12:30 pm – 2:00 pm  2006 Joint Convention Planning Committee
2:00 pm – 3:00 pm  Factors Affecting IVF Pregnancy Rates in Recipients
                   Dr. Cliff Lamb, University of Minnesota, Grand Rapids, MN
3:00 pm – 3:30 pm  Coffee Break in Exhibit Area
3:30 pm – 5:30 pm  Practitioners’ Forum—Moderator: Dr. Reuben Mapletoft, University of Saskatchewan, Saskatoon, SK, Canada
                   Panel: Dr. Gabriel Bo, IRAC, Cordoba, Argentina; Dr. Cliff Lamb, University of Minnesota, Grand Rapids, MN; Dr. Vic Cortese, Pfizer, Downingtown, PA; and Dr. Walter Johnson, OVC, Guelph, ON, Canada

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