ABSTRACT: Contagious equine metritis (CEM) has given rise to international concern since it was first recognized as a novel venereal disease of equids in 1977 and the etiologic agent was identified as a previously undescribed bacterium, *Taylorella equigenitalis*. Horse industry concerns over CEM centered on the ease with which this bacterium could be disseminated, the significance of *T. equigenitalis* as a cause of short-term infertility in the mare, and the existence of the carrier state in the stallion and the mare. The first known outbreak of CEM in the United States was in Kentucky in 1978. The economic impact on the Thoroughbred industry in the state was substantial. Before 2008, additional small-scale outbreaks occurred in Missouri in 1979, Kentucky in 1982, and Wisconsin in 2006, nearly all attributed to the importation of carrier animals. On each occasion, appropriate measures were taken to eliminate the infection, resulting in the United States regaining its CEM-free status. With the exception of the 1978 occurrence in Kentucky, none of the subsequent outbreaks significantly affected the horse industry. That changed dramatically in 2008, however, after the discovery of a Quarter horse stallion in Kentucky that cultured positive. Subsequent investigations turned up 23 carrier stallions and 5 carrier mares belonging to 11 breeds and located in 8 states. Shipment of infective semen and indirect venereal contact in stallion collection centers through the use of contaminated fomites were major factors in the spread of *T. equigenitalis*. Trace-back investigations of some 1,005 exposed and carrier stallions and mares in 48 states have failed to identify the origin of this latest CEM event. Neither clinical evidence of CEM nor decreased pregnancy rates were reportedly a feature in infected or exposed mares. In light of these findings, there was some question of whether or not the considerable expense incurred in investigating the latest CEM occurrence was warranted. Regaining CEM-free status for the United States will present considerable challenges.

Key words: contagious equine metritis, *Taylorella equigenitalis*, 2008 disease event, venereally transmissible

INTRODUCTION

Contagious equine metritis (CEM) is a bacterial disease of the reproductive tract of equids that caused consternation internationally within the Thoroughbred industry after its initial discovery in the United Kingdom and Ireland in 1977 (Crowhurst, 1977; Platt et al., 1977; Ricketts et al., 1977; Timoney et al., 1977b). Initial fears of the industry were prompted by the dramatic clinical features of CEM and by its highly contagious nature (Powell, 1978). These concerns were subsequently reinforced by the ease with which the disease spread internationally through the movement of carrier stallions or mares (Powell, 1981; Timoney and Powell, 1988). Of additional importance was the economic impact that CEM could have on previously unexposed equine breeding populations, as exemplified by the major disease outbreaks in Newmarket, United Kingdom, and Ireland in 1977 (Powell, 1981), and in Kentucky in 1978 (Kristula and Smith, 2004). As a consequence, many countries imposed rigorous requirements on horses imported for breeding purposes from countries in which CEM was known or was suspected to occur. This resulted in the disease becoming one of...
the most regulated, in terms of international trade in equids and equine germplasm, a situation that remains essentially unchanged to this day (Timoney and Powell, 1988; Timoney, 2000).

Contagious equine metritis is a nonsystemic disease that is restricted to the reproductive tract (Platt and Taylor, 1982). Clinical signs are only manifest in the mare and not in the stallion. In the mare, the infection is principally characterized by an acute endometritis, cervicitis, and vaginitis of variable severity together with a mucopurulent vaginal discharge, ranging from copious to minimal in amount (Powell, 1981; Platt and Taylor, 1982). Salpingitis was also demonstrated in a significant percentage of mares infected in one experimental study (Acland and Kenney, 1983). Contagious equine metritis very rarely results in abortion (Nakashiro et al., 1981).

The causal agent of CEM is *Taylorrella equigenitalis*, a bacterium not known to exist, much less taxonomically classified, before its discovery in 1977 (Platt et al., 1977; Ricketts et al., 1977; Timoney et al., 1977b; Platt and Taylor, 1982; Sugimoto et al., 1983). Initially, there was some confusion over the etiology of the disease in that there were individuals who contended that CEM was the result of infection with *Proteus mirabilis* (O’Driscoll et al., 1977). This was dispelled, however, once *T. equigenitalis* was successfully isolated from typically affected mares using enriched bacteriological culture media and microaerophilic conditions of incubation (Timoney et al., 1977a; Platt and Taylor, 1982).

**GEOGRAPHIC DISTRIBUTION**

In the course of the past 33 yr since CEM was first recognized as an emergent disease of equids, the causal agent, *T. equigenitalis*, has become more geographically widespread as a result of the shipment of carrier stallions and mares both within and between countries worldwide (Powell, 1981; Timoney and Powell, 1988). The extent to which fresh-cooled or frozen semen from carrier stallions has contributed to dissemination of the bacterium has yet to be more clearly defined.

Since 1977, CEM has been confirmed at one time or another in the equid populations of 29 countries, including (but not necessarily exclusive of) different countries in Europe, North America, and South America, as well as Japan and Australia (Timoney, 1996). The infection is believed to be endemic in non-Thoroughbred breeds in many countries in which it is currently known to occur. There is little doubt, however, that the global distribution of *T. equigenitalis* is more widespread than the foregoing would indicate. For various reasons, logistical and other, it has not been possible to carry out reliable surveillance for CEM in equid populations in other countries and regions of the world.

There have been several known incursions of CEM into the United States since initial discovery of the disease in 1977. The first reported outbreak involved the Thoroughbred breeding industry in Kentucky in 1978 (Holden, 1978; Swerczek, 1978). This outbreak resulted from the importation of 2 stallions from France in the fall of 1977. Although the occurrence was relatively short-term in duration, it was particularly disruptive for the Thoroughbred breeding industry in the state (Kristula and Smith, 2004). Lost income from stallion breeding fees, failure to get mares in foal, movement bans, and associated losses were estimated at $1,000,000 for every day that mares were not bred and movement restrictions were in force (Herbert, 1998). A conservative estimate of the economic loss from that occurrence was $13.55 million (Knowles, 1978; Kristula and Smith, 2004). Subsequently, additional small-scale outbreaks of CEM occurred in Trakehners in Missouri in 1979 (Fales et al., 1979), in Thoroughbreds in Kentucky in 1982 (USDA, 1982), and in Lipizzaners in Wisconsin in 2006 (Hayna et al., 2008). Imported carrier animals were implicated as the source of infection in almost all instances (Fales et al., 1979; Hayna et al., 2008). Thankfully, none of these outbreaks had a significant financial impact on the equine industries in the respective states. In December 2008 (USDA, APHIS, 2010), CEM was rediscovered in the United States, on this occasion in a Quarter horse breeding stallion in Kentucky (Clifford, 2008). This occurrence turned out to be much more extensive than initially thought, involving carrier stallions and mares belonging to 11 different breeds of horses located in 8 states (USDA, APHIS, 2010). A more comprehensive description of this most recent CEM event will be discussed subsequently, after review of selected features of the disease and its etiologic agent.

**FEATURES OF THE ETIOLOGIC AGENT**

As previously mentioned, CEM is caused by *T. equigenitalis*, which is a coccobacillary or bacillary gram-negative nonmotile bacterium (Platt et al., 1977; Ricketts et al., 1977; Timoney et al., 1977b; Taylor et al., 1978; Platt and Taylor, 1982; Sugimoto et al., 1983). It is frequently pleomorphic in appearance on isolation from carrier stallions or mares (Timoney et al., 1978b). Strains of *T. equigenitalis* are either sensitive or resistant to streptomycin (Swerczek, 1978; Powell, 1981; Platt and Taylor, 1982). Assessment of isolates of *T. equigenitalis* made to date has confirmed their sensitivity to a very broad range of antimicrobials (Taylor et al., 1978; Platt and Taylor, 1982). Sensitivity patterns of strains of the bacterium isolated over the years have remained essentially unchanged.

There is strong circumstantial evidence to indicate that CEM existed in Ireland 2 yr before its recognition as a novel, venereally transmissible disease of equids in 1977 (Powell, 1981; Timoney and Powell, 1988). Failure to identify the etiologic agent at the time was attributable to a total lack of awareness among veterinarians and diagnosticians of the existence of *T. equigenitalis*, much less of its fastidious growth requirements, especially the dependency of the bacterium on enriched
bacteriologic media and microaerophilic conditions of incubation for its cultivation (Taylor et al., 1978; Platt and Taylor, 1982). Up until the widespread occurrences of CEM in the United Kingdom and Ireland in 1977, established practice in Ireland and elsewhere was to culture endometrial swabs from mares on blood agar plates incubated aerobically for no more than 2 to 3 d. As subsequent events proved, such laboratory conditions would not have enabled the detection of T. equigenitalis. Isolation of T. equigenitalis requires enriched media, such as Eugon chocolate agar, containing a selection of antimicrobials to control most of the gram-positive and gram-negative bacterial contaminants that frequently colonize the sites of persistence of this bacterium in the stallion and the mare (Heath and Timoney, 2008). Of additional importance in the successful cultivation of T. equigenitalis is the need to maintain inoculated cultures under microaerophilic conditions of incubation for at least 7 d; strains of the bacterium are slow-growing, and colonies may not be visibly detectable with shorter incubation times (Ward et al., 1984). In terms of its biochemical properties, T. equigenitalis is relatively unreactive. It is nonfermentative and nonproteolytic but positive for cytochrome oxidase, catalase, and alkaline phosphatase (Platt and Taylor, 1982; Heath and Timoney, 2008).

Survival of T. equigenitalis outside the body is short-term (Timoney et al., 1978a). It is rapidly killed by a range of commonly used disinfectants and by exposure to UV rays, high temperatures, and reduced humidity. Under favorable environmental conditions, however, the bacterium can survive for a variable period as a surface contaminant on different fomites used in the breeding of mares and collection of stallions. Unless adequately decontaminated between usages, equipment or objects such as artificial vaginas, wash buckets, or a phantom mare can serve as a means of dissemination of infection in a breeding shed or in a semen-collection center (Powell, 1981; Timoney, 1996).

Evidence would indicate that T. equigenitalis is a natural pathogen of equids, particularly horses. Under experimental conditions, the organism has been successfully transmitted to donkeys but not to cattle, sheep, swine, or cats (Timoney et al., 1978c, 1984, 1985a). Whereas certain congenic strains of mice can be infected with T. equigenitalis, infections are relatively short-lived and not associated with overt signs of disease (Timoney et al., 1985b). There is no evidence to indicate that the bacterium is transmissible to humans.

For many years, it has been known that strains of T. equigenitalis can vary with respect to their ability to cause clinical signs of reproductive tract disease in the mare. This was based on field observations of naturally occurring outbreaks of CEM and also on the outcome of early experimental studies with the etiologic agent (Timoney et al., 1978b, 1979). Clinical disease in the mare was reproduced after intrauterine challenge with large colonial variants but not with small colonial variants of the bacterium (Kanemaru et al., 1988). Aside from variation in virulence phenotype, molecular analysis of strains of T. equigenitalis from different countries and regions of the world has revealed the existence of multiple genotypes, some more commonly encountered than others (Matsuda and Moore, 2003). At this point, no attempt has been made to establish if there is any correlation between specific genotype(s) and ability to induce clinical infection in the mare.

**CLINICAL OUTCOME**

The outcome of exposure to T. equigenitalis in the stallion is in sharp contrast to that in the mare. Strictly speaking, stallions do not become infected with the bacterium, which merely exists as a commensal, colonizing specific sites on their external genitalia (Platt et al., 1978; Powell, 1981; Platt and Taylor, 1982). Its presence does not result in clinical signs or any evidence of a local or systemic inflammatory reaction, or development of a detectable serum antibody response. There has been 1 recorded case, however, describing isolation of T. equigenitalis from the testis, epididymis, seminal vesicles, and urethra of an experimental stallion on necropsy examination (Schluter et al., 1991). Although this is an isolated case, it would suggest that on very rare occasions, T. equigenitalis may give rise to an ascending infection of the reproductive tract in the stallion.

Primary exposure to T. equigenitalis in the mare results in a non-life-threatening infection that is restricted to the reproductive tract (Powell, 1981; Platt and Taylor, 1982). The outcome can range from overt disease of variable clinical severity to asymptomatic infection (Timoney 1978, 1979). Unlike the stallion, mares infected with the bacterium develop a humoral antibody response that is usually moderately short-lived (Benson et al., 1978; Croxton-Smith et al., 1978; Bryans et al., 1979). There is no definitive evidence to support the view that horse mares are more susceptible to this infection than pony mares.

As previously indicated, after an incubation period of 2 to 13 d, mares typically affected with CEM present with an odorless grayish-white mucopurulent vaginal discharge of uterine origin (Platt et al., 1978; Timoney et al., 1978b). The discharge, which can vary greatly in volume, may persist for 2 wk or longer in untreated cases. It is accompanied by an endometritis, cervicitis, and vaginitis of variable severity (Crowhurst, 1977; Platt et al., 1977, 1978; Ricketts et al., 1977; Timoney et al., 1977b, 1978b; Powell, 1981). Intrauterine accumulation of fluid can often be detected on ultrasonographic examination. In such cases, discharge can be observed seeping between the folds of the external os of the cervix and accumulating on the floor of the vagina. Most mares fail to conceive upon primary exposure to T. equigenitalis and return to estrus after a shortened diestrous period (Powell, 1981; Platt and Taylor, 1982). Infertility is short-term, however, and no long-term adverse effects have been documented in infected mares.
Experimental re-exposure of mares previously affected with CEM was associated with minimal or zero clinical signs of disease (Timoney et al., 1979; Fernie et al., 1980; Sahu et al., 1980).

*Taylorllera equigenitalis* infection in the pregnant mare may be symptomatic or asymptomatic. Symptomatically infected mares may or may not present with an intermittent vaginal discharge over the course of pregnancy. Some such cases may develop a low-grade placentitis (Ricketts et al., 1977; Powell and Whitwell, 1979). Abortion at approximately 7 to 8 mo of gestation is a very rare sequel to infection (Nakashiro et al., 1979). It is important to emphasize that persistence of *T. equigenitalis* in the reproductive tract of the pregnant mare does not appear to compromise the health of the unborn foal (Timoney et al., 1978d; Powell and Whitwell, 1979).

**EPIDEMIOLOGY**

A thorough understanding of the various factors involved in the epidemiology of CEM is critical to the development of effective strategies for its prevention and control. Among the most important factors are the following: modes of transmission of *T. equigenitalis*; variation in pathogenicity and other phenotypic characteristics among strains of the bacterium; occurrence of the carrier state in the stallion and the mare; breeding shed management practices; and industry trends influencing the national and international movement of stallions and mares for breeding and shipment of semen.

**Transmission**

Contagious equine metritis is a venereally transmissible disease that can be spread by direct or indirect genital contact between an infected mare and a stallion or vice versa (Crowhurst, 1977; Platt et al., 1977; Ricketts et al., 1977; Timoney et al., 1977b; Powell, 1981; Platt and Taylor, 1982). Transmission is optimized when mares are bred by natural service in that it allows for maximal physical contact between the sites of persistence of *T. equigenitalis* in the mare or in the stallion, whichever is the source of the organism. There is evidence to indicate that transmission of infection can also take place by AI through the use of freshly cooled or extended semen from a carrier stallion, even where appropriate antibiotics are included in the extender (USDA, APHIS, 2010). The risk of transmission by this means is considerably less, however, compared with exposure by natural service. The attendant risk associated with using cryopreserved semen from a carrier stallion, although believed to be minimal, has not yet been established.

The importance of indirect genital contact with fomites contaminated with *T. equigenitalis* in the transmission and dissemination of CEM cannot be overemphasized (Powell, 1981). Contaminated vaginal specula, forceps, tail bandages, obstetrical sleeves, and so on, used in the examination of the reproductive tract of the mare or when performing AI, have been incriminated in the spread of the disease. Disregard for adequate standards of breeding shed management or failure to follow appropriate standard operating procedures when handling mares or stallions at time of breeding has facilitated the transmission of *T. equigenitalis* in past outbreaks of CEM. There is considerable evidence to indicate that the vast majority of the carrier stallions involved in the 2008 to 2009 CEM event in the United States were exposed to the organism by direct or indirect genital contact with contaminated fomites (e.g., an artificial vagina, wash bucket, or phantom mare) in various semen-collection centers.

Although considerably less significant in the epidemiology of CEM, a small percentage of pregnant mares can continue to harbor *T. equigenitalis* in utero throughout pregnancy (Timoney et al., 1978d; Powell and Whitwell, 1979). This may result in congenital infection of the fetus that very rarely can result in abortion (Nakashiro et al., 1981). A much more likely outcome is that the foal is born alive and healthy though culture-positive for *T. equigenitalis* on its external genitalia. Exposure of foals out of carrier mares could also occur through contact of their external genitalia with an infected placenta, placental fluids, or contaminated clitoral area of the mare at time of foaling (Timoney and Powell, 1982). It is also possible that exposure of the foal takes place in the immediate postpartum period through genital contact with bedding or pasture contaminated with infective vaginal discharge or placental fluids. A final mode of indirect venereal transmission of *T. equigenitalis*, which has been postulated but not yet proven, involves the use of sponges or wash cloths inadvertently contaminated with the bacterium, between colts or stallions (Powell, 1981; Timoney and Powell, 1982). This could explain the infrequently encountered cases of colts or stallions found culture positive while in training without any history of being bred, or being born on a known CEM-affected premises, or out of a carrier mare.

**Strain Variation**

Variation exists among strains of *T. equigenitalis* with respect to their ability to cause clinical signs of CEM in previously exposed mares. This was first recognized in late 1977 when certain outbreaks of the disease were characterized by very few cases of symptomatic infection (Timoney, 1978; Day et al., 1979). The majority of at-risk mares were asymptomatically infected with *T. equigenitalis*. This trend continues up to the present, especially in countries or breeds in which CEM is endemic. It is not known whether the carrier stallion or mare or both are responsible for genomic modulation among strains of *T. equigenitalis* and the emergence of genotypes of the organism of reduced pathogenicity for the mare.


**Carrier State**

Of major importance in the epidemiology of CEM is occurrence of the carrier state in the stallion and the mare (Platt et al., 1978; Simpson and Eaton-Evans, 1978; Powell, 1981). The international spread of CEM has, on repeated occasions, been the result of importation of a carrier animal, usually a stallion, in some instances with significant repercussions for the equine industry in the importing country (Holden, 1978; Timoney, 2000).

Stallions can become inapparent carriers of *T. equigenitalis*, with the bacterium capable of persisting on the external genitalia of some untreated stallions for an extended period of time, at least several years (Platt and Taylor, 1982). Preferential sites for detection of *T. equigenitalis* in the stallion include the urethral fossa, urethral sinus, distal urethra, and the external surface of the penis and prepuce (Powell, 1981; Heath and Timoney, 2008). The transmission rate of the bacterium to mares bred by natural service can vary between stallions and over time in the same stallion (Bryans and Hendricks, 1979). The rate of transmission is believed to be influenced primarily by the concentration of *T. equigenitalis* on the external genitalia at time of breeding and the level of innate susceptibility or resistance of the mares exposed to infection. As already indicated, the frequency of transmission of CEM to mares bred by AI with fresh-cooled or extended semen from a carrier stallion is considerably less than if natural service had been used (C. Klein, University of Kentucky, Lexington, personal communication; USDA, APHIS, 2010). To this point, it has not been possible to estimate the corresponding risk of spread of *T. equigenitalis* through cryopreserved semen. It may be equivalent to or perhaps even less than in the case of fresh-cooled semen. Klein and coworkers (C. Klein, University of Kentucky, Lexington, personal communication) failed to achieve transmission of infection to a limited number of mares artificially inseminated with cryopreserved semen from a culture-positive stallion.

Persistence of *T. equigenitalis* can also occur in a variable percentage of mares after symptomatic or asymptomatic CEM infection (Platt et al., 1978; Timoney et al., 1978b). The carrier state can occur in as many as 20 to 25% of mares, most of which are asymptptomatically infected (Wood et al., 2007). However, in a small percentage of cases, carriage of the bacterium may be associated with an intermittent vaginal discharge (Rick- etts et al., 1977; Powell and Whitwell, 1979). Whereas persistence of *T. equigenitalis* in the mare often does not last longer than several weeks or months after recovery from the acute phase of the infection, some individuals will remain carriers for many months or even years (Powell, 1981). The majority of carrier mares are considered clitoral carriers insofar as they harbor the CEM bacterium in the clitoral sinuses and clitoral fossa (Simpson and Eaton-Evans, 1978). On the other hand, a minority are uterine carriers in which *T. equigenit-

a minority are uterine carriers in which *T. equigenitalis* is sequestered in the uterus and not in the distal reproductive tract (Timoney and Powell, 1988). It is important to emphasize that there can be considerable variation in the shedding pattern of the bacterium by some carrier mares. Wide fluctuations in the number of organisms recovered on culture from sequential sets of swabs have been documented (Timoney et al., 1978e). This can present problems from a diagnostic viewpoint when attempting to assess the infectivity status of a particular mare or stallion.

Aside from the carrier state in the barren or open mare, *T. equigenitalis* can also persist in the pregnant mare. In a small percentage of such cases, the organism can set up a localized placentitis, which may lead to vertical transmission and congenital infection of the fetus in utero and, very rarely, abortion (Powell and Whitwell, 1979; Nakashiro et al., 1981). More often, it is believed responsible for contaminating the newborn foal either at time of parturition or in the immediate postpartum period, as has been described previously. Foals exposed to *T. equigenitalis* very early in life can continue to harbor the organism for an extended period, very probably into adulthood, when they could serve as a source of infection for future outbreaks of CEM (Timoney and Powell, 1982). Although major attention is paid to the significance of the breeding stallion or mare in the transmission of *T. equigenitalis*, a carrier teaser stallion should not be overlooked as a potential source of the bacterium (Dingle, 1977; Timoney and Powell, 1988).

**Breeding Management Practices**

Aside from the importance of natural service in the transmission of CEM, as has already been mentioned, the infection can also be efficiently spread through indirect venereal contact via contaminated fomites used in the breeding of stallions and mares (Powell, 1981). Lack of appropriate biosecurity safeguards has been responsible for transmission of *T. equigenitalis* among stallions in semen-collection centers.

**Industry Trends**

Over the past 40 to 50 yr, significant changes have taken place in the equine industries of most major horse breeding and performance countries around the world; these have served as the catalyst for growth in the volume of international trade in horses and equine germplasm (Timoney, 2000). Increased global movement of equids carries a greater inherent risk of the spread of a wide range of equine infectious diseases, not only CEM. Included among the industry changes is shuttling of stallions between the northern and southern hemispheres for breeding, and the national and international shipment of mares for breeding to specific sires or because of change of ownership. The potential...
also exists for the dissemination of CEM through the shipment of fresh-cooled and, possibly, cryopreserved semen from a carrier stallion.

**REDISCOVERY OF CEM IN 2008**

Rediscovery of CEM in the United States came to light mid-December 2008 with confirmation of the carrier state in a 16-yr-old Quarter horse stallion that was being routinely screened for *T. equigenitalis* before approval of his semen for export (Clifford, 2008). The stallion was located on a breeding farm in central Kentucky, where he had resided since February 2008, when he had been shipped in from Texas. He was one of 22 non-Thoroughbred stallions on the farm during the 2008 breeding season, all of which were nonnative to Kentucky and had been relocated to the state for the breeding season that year. The breeding of all the stallions was done artificially, and there was no history of natural service. An additional 3 stallions on the index premises were subsequently confirmed carriers of CEM. The intensive epidemiological investigations that followed failed to demonstrate spread of *T. equigenitalis* to other equine premises in Kentucky or contact, direct or indirect, with any of the Thoroughbred breeding farms in the state. Since discovery of the initial positive stallion, follow-up investigations revealed that upon conclusion of the 2008 breeding season, 13 of the original 22 stallions had been relocated to other states and 1 additional stallion had been moved to another farm in Kentucky. Four of the 13 stallions in this group, 3 in Indiana and 1 in Texas, were later confirmed carriers.

Many months of intensive epidemiological investigation by federal and state regulatory officials followed that entailed the identification, tracing, quarantining, and culturing of mares and stallions that had direct or indirect venereal contact with any of the known carrier stallions or had shared the same premises where they had been for a period of time. An exposed horse was defined as one bred naturally or by AI to a horse positive for *T. equigenitalis*, or one otherwise epidemiologically linked to a positive horse, as determined by state and federal animal health officials. To date, a total of 23 stallions, including one that is currently a gelding, have been confirmed positive for *T. equigenitalis* (USDA, APHIS, 2010). Infection was also detected in 5 mares. Of the 5 mares, 3 had been inseminated with fresh-cooled semen, 1 shared the same premises as 3 carrier stallions and had been bred by AI with semen from 2 of them, and the fifth mare had been bred by natural service to a known carrier stallion. The distribution of the carrier stallions and mares by state is presented in Figure 1. It became evident in the course of investigations into this event that cross-contamination between stallions had occurred on the index premises in Kentucky and also on 2 premises in Wisconsin and 1 in Illinois. After extensive analysis of their respective histories, none of the carriers of *T. equigenitalis* were considered the original source for the 2008 to 2009 CEM event.

Aside from the total of 28 carrier animals, an additional 722 mares and 255 stallions were considered potentially exposed to *T. equigenitalis*. These were located in 48 states; Hawaii and Rhode Island were the only states without a positive or exposed horse. The distribution of exposed stallions and mares by state is represented in Figure 2. Some 958 (95.3%) of the 1,005 positive or exposed horses have been confirmed free of *T. equigenitalis*. In the case of stallions, 253 or 91.0% have completed their testing and treatment protocols.
and been declared free of CEM. Of the 727 mares, 705 or 97.0% have satisfied all testing and treatment requirements and have also been confirmed free of *T. equigenitalis*.

Several significant epidemiological findings have emerged from this CEM event. Although 11 breeds were represented among the carrier stallions and mares, Quarter horses were very much in the majority. It is worth emphasizing that the greatest number of carrier animals (i.e., 82%) were stallions; this is consistent with the findings of earlier field investigative studies that underscored the greater frequency of the carrier state in the stallion, as well as the more extended duration of the carrier state in individual stallions compared with mares. This is also borne out by the results of postentry quarantine and testing of stallions and mares imported into the United States from CEM-affected countries. Over a 12-yr period, 23 of 31 (74%) confirmed carriers of *T. equigenitalis* were stallions and 8 (26%) were mares (P. J. Timoney, unpublished data). It should also be pointed out that the extended interval between breeding and eventual tracing and sampling of the mares identified in connection with the 2008 to 2009 CEM event may have militated against detection of a greater number of carrier animals. The latter figure might have been greater had this interval been of shorter duration. Early experimental studies have shown that the carrier state with *T. equigenitalis* in the mare is generally shorter than in many stallions, lasting only several weeks or months (Timoney et al., 1978b). This and the fact that exposure of the mares was by AI and not by natural service could have significantly influenced the small carrier rate detected.

It is especially noteworthy that clinical expression of CEM was virtually absent in mares bred with fresh-cooled semen from any of the carrier stallions. Only 2 of the 5 carrier mares that were inseminated with fresh-cooled semen were reported to have exhibited 1 or more clinical signs compatible with CEM postbreeding. In the face of widespread asymptomatic infection, there is significant risk of *T. equigenitalis* remaining undetected in equine breeding populations for months or even years. This is probably what transpired before the rediscovery of CEM in the United States in December 2008. It is believed, but not proven, that the original source for this latest occurrence of the disease was a particular warmblood stallion imported from Europe in 2000. In view of its insidious nature, CEM can be easily overlooked unless potential cases of infection are rigorously investigated and diagnostically evaluated for *T. equigenitalis*.

A finding of major epidemiologic significance to emerge from the 2008 to 2009 CEM event was the fact that most of the carrier stallions that were detected had apparently been exposed to *T. equigenitalis* through the use of contaminated fomites in semen-collection centers. There was clear-cut evidence that the standards of hygiene practiced on some of these premises when collecting semen was inadequate to prevent indirect transfer of *T. equigenitalis* between stallions. More rigorous attention must be paid to implementing appropriate biosecurity protocols in semen-collection centers if a similar situation is to be prevented from recurring in the future.

From a laboratory viewpoint, there are several features surrounding the 2008 to 2009 CEM event that de-
serve comment. The first concerns the very large isolation rate of *T. equigenitalis* on culture, with all 5 carrier mares, and 20 out of 23 stallions (87%) detected on pre-breeding examination, the vast majority of which were positive on the initial set of bacteriological swabs that were collected. Only 3 stallions required test breeding to confirm their carrier status. This contrasts markedly with the findings from testing imported stallions when only a very small percentage were detected on prebreeding cultural examination (P. J. Timoney, unpublished data). It is speculated that the difficulties experienced in culturing *T. equigenitalis* from the latter population may be due to the fact that many of them had probably undergone antimicrobial treatment before export. All of the strains of *T. equigenitalis* recovered in the 2008 to 2009 CEM event are streptomycin-resistant but sensitive to a wide array of other antibiotics (M. Erdman, personal communication). The strains have also been evaluated by pulsed-field gel electrophoresis, and they have been found to belong to a unique genotype based on their identical banding patterns, which has not been previously recorded.

**SUMMARY AND CONCLUSIONS**

For several reasons, not least of which is the facilitation of international trade in horses and equine germplasm, it is important that the United States strive to regain its CEM-free status. While this is a laudable goal, achieving it will present significant challenges, not only for federal and state animal health officials but also for the equine industry in the country. Regaining disease-free status for the country will be more difficult on this occasion than in the past. Previous episodes of CEM in the United States were much more limited with respect to breed, numbers of infected and exposed animals, and geographic distribution. The following represent a listing of the major challenges that confront both regulatory agencies and all sectors of the industry in attaining that goal: 1) the overall size and numerical representation by breed and activity of the estimated national population of 9.2 million equids (Deloitte, 2005), a very significant percentage of which are non-Thoroughbreds; 2) the absence of any nationally recognized official or voluntary industry control program for CEM; 3) the frequency of interstate movement of stallions and mares for breeding purposes; 4) growth in the volume of inter- and intrastate shipment of equine semen; 5) the insidious nature of CEM in the mare and the absence of clinical expression of the disease in the stallion; 6) the shortcomings of existing national monitoring, surveillance, and reporting programs for equine diseases; 7) the costliness of current diagnostic testing for CEM; and 8) the continuing risk of re-introduction of CEM through imported stallions and mares.

Regaining CEM-free status for the United States will require the concerted efforts of regulatory authorities, the equine industry, and veterinarians if it is to be successful. Major responsibility resides primarily with the equine industry, however, to ensure that the breeding stallion population in the country is effectively monitored for *T. equigenitalis* and that appropriate safeguards are put in place to prevent direct or indirect venereal transmission of CEM in breeding sheds or semen-collection centers. In time, if widely implemented, such measures will be instrumental in the country being able to re-affirm its freedom from this infection and to regain its CEM-free status.

**LITERATURE CITED**


