Aspects of Equine Infectious Disease Control
From the United Kingdom Perspective

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Differences in approaches to the control of infectious diseases of horses exist between the United Kingdom and North America. Valuable lessons can be mutually learned to better understand respective approaches. Adoption of novel methods of prevention and control that have proved useful elsewhere in the world may have significant impacts on horse welfare. The examples of mandatory influenza virus vaccination, use of voluntary Codes of Practice for infectious venereal diseases, increasing acceptance of the significance of the carrier state for strangles and parallel development of differential diagnostics with the introduction of a new modified live strangles vaccine are used to illustrate a variety of different mechanisms by which improvements in equine disease control have developed in the United Kingdom. Author's address: Centre for Preventive Medicine, Animal Health Trust, Lanwades Park, Kentford, Newmarket, Suffolk CB8 7UU, United Kingdom; e-mail: richard.newton@aht.org.uk. © 2007 AAEP.

1. Introduction
The United Kingdom has periodically encountered significant equine-disease outbreaks, which have in themselves led to paradigm shifts in methods of control. A few notable examples are provided and discussed below.

- The introduction of mandatory vaccination against equine influenza virus among Thoroughbred racehorses attending race meetings.
- The introduction and evolution of the voluntary codes of practice for equine infectious venereal diseases.
- The increasing acceptance of the significance of the carrier state in the endemic persistence of equine strangles.
- The introduction of the first modified live equine vaccine to Europe for the control of equine strangles. This was followed by the occurrence of clinical signs of strangles in a small number of horses in the period after vaccination, which presented a novel diagnostic challenge.

The examples provided here illustrate apparent geographical and possibly cultural differences in approaches to equine infectious disease problems between the United Kingdom and the United States. However, with ever broadening cross linkages between the equine and veterinary industries on both sides of the Atlantic, it is hoped that the approaches outlined here will be of interest to and be adopted by a North American audience.

2. Mandatory Equine Influenza Vaccination
Among naive horses, equine influenza is a highly contagious respiratory disease that is characterized by pyrexia, associated depression and anorexia,
harsh dry cough, nasal discharge, and secondary bacterial respiratory infection. A novel H3N8 equine influenza A virus subtype, which first emerged in Miami, FL in 1963, initiated a worldwide pandemic of equine respiratory disease and was the stimulus for the development of multivalent, adjuvanted influenza vaccines for horses. This early work, based on experience from human vaccines, led to the development of the now broadly standardized schedules for equine influenza vaccination. These schedules recommend that a primary course of two doses of vaccines be given ~4–6 wk apart followed by a booster vaccination 6 mo after the end of the primary course; annual boosters are recommended thereafter. The same schedules are still used today for the product datasheet recommendations for the latest inactivated virus vaccines and are the basis for the regulatory rules for most international equine competitions.

It was recognized during several influenza outbreaks in the United Kingdom during the 1970s, especially in the 1979 outbreak, that vaccinated horses generally suffered less severe disease than those that were unvaccinated. Influenza vaccination of Thoroughbred racehorses in Great Britain became mandatory under the Jockey Club Rules of Racing at the start of the flat racing season in March 1981, and it was implemented soon after in Ireland and France.

3. Jockey Club Rules on Influenza Vaccination

The first vaccination of the primary course is given on day 0. The second vaccination of the primary course is given between 21 and 92 days (i.e., 3 wk–3 mo) after the first vaccination. The third vaccination of primary course (“6-mo booster”) is given between 150 and 215 days after the second injection (i.e., 5–7 mo). Subsequent booster vaccinations are given at intervals of no more than 12 mo (“annual booster”). No horse is permitted to race unless vaccinated 7 or more days previously, and the primary course must be completed.

Since 1981, British racing has not been cancelled because of equine influenza, but there have been continued seasonal peaks of infection among unvaccinated non-Thoroughbred horses that are associated with increased mixing at shows in the summer months. Because of the absence of systematic, consistent, and long-term surveillance data, it is not possible to provide absolutely conclusive evidence of the true impact of mandatory influenza vaccination on reducing the incidence of influenza virus infection and associated disease. However, it is the widely held belief of many that this is indeed the case. There were markedly different financial and welfare consequences of significant influenza outbreaks in Thoroughbreds in 2003 in Great Britain and South Africa in which mandatory vaccination was and was not, respectively, adopted. This highlights the benefits of vaccination in horses at risk of exposure to this highly contagious disease agent. Details of these outbreaks of influenza that occurred in 2003 will be described later in this paper.

With changes in equine H3N8 influenza A viruses caused by antigenic drift, influenza outbreaks have caused periodic disruption to the training schedules of vaccinated Thoroughbreds in individual yards or training centers in the United Kingdom, which brings mandatory vaccination under periodic scrutiny. However, investigations of these largely clinically mild and geographically limited outbreaks have permitted closer assessment of factors associated with disease occurrence in vaccinated populations.

Outbreaks of influenza virus infection among racehorses vaccinated according to Jockey Club Rules were investigated in Newmarket in 1995 and 1998 to establish reasons for vaccine failure. Investigations showed that, in 1995, horses were protected from infection if they had antibody levels above a threshold equivalent to that observed in experimental-challenge infections using nebulised aerosol. Investigations in 1998 showed no such protective threshold. Subsequent characterization showed that the 1998 H3N8 virus was antigenically distinct from the 1995 virus and those contained in available vaccines. These outbreaks highlighted the need for potent vaccines to maintain protective antibody and inclusion of epidemiologically relevant viral strains in vaccines.

Subsequent field vaccine studies were conducted to determine what factors might lead to inadequate antibody levels in Thoroughbreds. Investigations showed that yearlings receiving their third vaccine dose were protected for only a few months. To avoid infection during the high-risk period of the autumn sales, they would require further strategic boosters. Subsequent studies confirmed that “time since last vaccination,” “number of previous doses,” “age first vaccinated,” and “previous vaccine types” were all important antibody-level determinants in young racehorses entering training. Also, delaying the interval between two doses of a new primary vaccine course in previously primed animals provided more sustainable levels of protective antibody. These findings have important implications for optimal control of influenza by vaccination and are being used to inform strategic vaccination policies.

However, a more recent outbreak in racehorses in the United Kingdom did serve to confound some of the widely held beliefs regarding vaccine breakdown in young horses, and it again highlighted the benefits of detailed epidemiological and microbiological investigation.

Between March and May 2003, equine influenza virus infection was confirmed as the cause of clinical respiratory disease among both vaccinated and nonvaccinated horses in at least 12 locations in the United Kingdom. In the largest outbreak, at least 21 Thoroughbred training yards in Newmarket, including >1300 racehorses, were variously affected;
horses showed signs of coughing and nasal discharge during a 9-wk period (Fig. 1).

An American sublineage H3N8 equine influenza virus, previously not identified in the United Kingdom, was responsible for the outbreak. Many infected horses had been vaccinated within the previous 3 mo with a vaccine that contained representatives from both the American and European lineages. On the basis of accepted criteria, there did not seem to be significant antigenic differences between the infecting virus and Newmarket/1/93, the American lineage virus representative in the most widely used vaccine. Two-year-old horses were apparently less susceptible to infection than 3-yr-olds and older animals, despite broadly equivalent single radial hemolysis (SRH) antibody levels. This was consistent with qualitative rather than quantitative difference in the immunity conveyed by vaccination.

However, multivariable analyses comparing infected and non-infected animals showed that the apparently counterintuitive inverse age effect was explained by elements in the vaccine history of these animals. Among factors in the vaccine history associated with influenza infection, there was significantly increased risk of infection associated with male gender, a primary course interval >92 days, and a period >3 mo since the last vaccination. There was a significantly decreased risk of infection associated with increasing pre-infection antibody level and first vaccinating after 6 mo of age. There was also significant variation in risk according to both the first and last type of vaccine administered. Findings highlight the benefits of relating vaccine history with risk of infection among vaccinated horses suffering influenza infection.

4. Introduction of the Horserace Betting Levy Board Codes of Practice for Equine Infectious Venereal Diseases and Its Practical Application in Disease Control

The first Horserace Betting Levy Board (HBLB) Code of Practice was published for the 1978 breeding season after an outbreak of contagious equine metritis (CEM) that had first been seen in the United Kingdom the previous year. In the 1977 outbreak, large numbers of Thoroughbred mares showed profuse purulent vaginal discharge after mating, and many studs were eventually forced to stop breeding. However, the disease had already spread, and there was serious disruption and economic losses in the Thoroughbred breeding industry.

The first code was heralded a great success; despite the severity of the requirements, which included swabbing mares three times at weekly intervals before covering, high compliance was...
achieved. Only 7 cases were seen in 1978, and none were seen in the following season. This initial code emphasized several important factors that are still recognized today for successful infection control and maintenance. They emphasized the importance of careful hygiene measures when teasing, mating, and examining mares. The role of the veterinarian and stud handler in disease transmission was brought into sharp focus by this outbreak. The practice in Newmarket of “walking in” mares from boarding studs to stallion studs, which was believed to have effectively propagated CEM, highlighted the importance of honest and timely communications between all parties, particularly stud managers and veterinarians. The fastidious nature of the causative organism and the care required to successfully culture it led to the formation of a register of dedicated competent laboratories that subsequently came under the umbrella of the annual accreditation process. The phenomenon of the healthy carrier state in mares, stallions, and teasers became recognized, and the elimination of these animals from the population was achieved through application of the Codes of Practice.

The favorable impact of the first code for CEM, which also included disease caused by Klebsiella pneumoniae and Pseudomonas aeruginosa, subsequently resulted in inclusion of disease control on equine herpesvirus (EHV) and equine viral arteritis (EVA) and guidelines on strangles (Streptococcus equi). Since this time, CEM and EVA have become regulated diseases in the United Kingdom, and on diagnosis or suspicion, these cases are compulsorily referred to the national veterinary authorities, who take on the responsibility of implementing control. An expert subcommittee was also formed, which now meets each year to review and update the recommendations as needed in light of new scientific knowledge or practical experience like outbreaks. Each year, the codes are widely distributed to Thoroughbred and non-Thoroughbred breeders and veterinarians. They can also be downloaded in PDF format from the HBLB’s website (http://hblb.org.uk/sndFile.php?fileID=%203) and are freely available from the Board, the Thoroughbred Breeders’ Association, and the British Horse Society.

5. Codes of Practice and EVA in Europe

The annually updated Codes of Practice continue to be the practical means by which prevention of EVA is implemented in the United Kingdom, and it is done particularly but not exclusively by the Thoroughbred breeding industry. This is based on annual pre-breeding serological screening of both stallions and mares and uses a killed vaccine in stallions. This approach has to date been proven to control disease on at least two occasions when previously infected mares have been detected after sub-clinical outbreaks among Thoroughbreds in France in 2000 and Ireland in 2003. It highlights the importance of serosurveillance to detect infected animals in the absence of clinical signs. This has subsequently led to heightened control measures among imported animals on some U.K. Thoroughbred stud farms.

In the event that EVA is confirmed, the Code of Practice recommends that the local Divisional Veterinary Manager of the Department for the Environment, Food, and Rural Affairs (DEFRA) be immediately notified in accordance with the EVA Order 1995. In addition, all movements and breeding is stopped, all cases and contacts are traced, sampled, and isolated, and all other horses on the affected premises are screened and grouped according to infectious status. It is also important that good communication exists between interested parties including those that have received animals (and semen if relevant) from the infected stud, those that are planning to send animals, and those in the breeder’s association. Testing and screening should continue on all possible affected premises until the end of the outbreak; seropositive animals and pregnant mares should be isolated for 4 wk after first sampling, and stallions must have their shedding status investigated. Serosurveillance is used on stallions vaccinated using Artervac, the only killed virus vaccine for equine arteritis virus (EAV) available in Europe. This testing shows that achievement and maintenance of the immunity required to protect against developing semen shedding requires stallions to have several boosters in addition to the 2–3 dose primary course (Fig. 2). This indicates that many first-season sires are probably inadequately protected against EVA infection by use of killed vaccine. This could be overcome by vaccination and subsequent boosting of potential stallions while they are still racing.

This was particularly highlighted in the 2003 breeding season when there were problems with availability of Artervac in Europe. The consequence of this was that first-season sires were left completely susceptible to EAV infection, whereas previously well-vaccinated stallions had good levels of residual immunity that was evidenced by high virus-neutralization (VN) antibody levels. The outbreak in Ireland resulted in infection of a first-season sire, which did not subsequently shed virus in its semen.

The most important aspect of control programs after an outbreak of EVA has been traced to a stud is to stop covering immediately. If a stallion is not infected, then attempts must be made to prevent infection of all stallions. If a stallion has already been infected, then the most efficient means of maintaining the spread of the infection around the stud must be used. The rates of infection on the index stud in the 1993 outbreak in the United Kingdom closely followed the rate of covering in the preceding week (Fig. 3).

All horse and semen movement should be stopped on and off the stud, and recently departed animals should be traced, placed in isolation, and tested.
The control of outbreaks of EVA can be based on the fact that virus is most unlikely to be present in the horses (other than stallions) for >3 wk after exposure. It is sometimes impossible to determine the precise day of infection during an outbreak. Thus, horses should be kept in isolation for at least 3 wk after their first positive blood test. By then, they are likely to be free from infection. Because EVA usually spreads poorly between animals through the respiratory route, it should be possible to stop the spread of infection around a stud after covering has ceased.

6. Codes of Practice and EHV in Europe

Although spontaneous abortion cannot be completely prevented because of EHV-1, there is much that can be done through sensible management, hygiene, and vaccination to reduce its incidence and to limit the spread of EHV-1. This will prevent so-called abortion storms and the dreaded paralytic form of the disease that, in the past, accompanied some severe abortion outbreaks; more recently, it has been affecting groups of non-pregnant sports horses. The HBLB’s Code of Practice outlines these recommendations for the prevention and control of EHV-1 disease on studs.

- Mares should be foaled away from the stallion stud wherever possible.
- If this is not possible, then pregnant mares should be kept in small groups and isolated from possible high-risk contact groups such as weanlings, yearlings, and horses out of training.
- Pregnant mares, especially in the later stages of pregnancy, should not be stressed or transported with other horses.
- Foster (nurse) mares should be isolated, especially from pregnant mares, until it is established that her own foal did not die from EHV-1 related disease.
- Sensible and regular hygiene practices should be adopted, preferably with pregnant mares having a dedicated staff.
- The adoption of a vaccination program against EHV-1 is strongly recommended, but it must be emphasized that this will not necessarily protect all vaccinated mares from abortion and is not a substitute for good management and hygiene.
- Vaccination at 5, 7, and 9 mo of gestation is recommended in accordance with the manufacturer’s recommendations to reduce (but not eliminate) the risk of abortion storms.

If disease occurs in the stud and EHV-1 is a possible cause (abortion, neonatal foal death, paralysis, etc.), then control measures need to be implemented immediately even before the diagnosis is confirmed.

- Veterinary advice should be sought.
- The mare (and foal) should be isolated.
- The fetus and placenta should be sent to the laboratory.
- Movements on and off the stud should be stopped immediately.
- Owners of mares planning to visit the premises should be notified.
Laboratory results are needed to confirm a diagnosis of EHV-1. If results confirm EHV-1, isolation and movement restrictions should be maintained for at least 1 mo after the last case of disease or until extensive laboratory testing shows absence of on-going active infection.

Mares in contact with the affected animals should be isolated in small groups until after they have foaled to limit the transmission of EHV-1 should further disease occur.

The breeder’s association and the owners of horses that have left, that are still resident, or that are planning to visit the premises should be notified.

7. Successfully Managing Equine Strangles

Infectious diseases pose a considerable threat to the smooth running of any equine premises, and these risks are, in general, proportionally greater for large premises than for smaller operations. Of all equine infectious diseases, strangles can be particularly problematic, because it may lead to considerable long-term problems for infected premises. Additionally, the stigma associated with the disease may persist long after the infection has been eliminated. Strangles has the ability to become endemic, particularly on studs, and its regular recurrence over a number of years frequently coincides with the new crop of susceptible weaned offspring. It is important that horse owners and their veterinarians recognize strangles as early as possible and that appropriate means of control are implemented.

Strangles in horses is caused by infection with the bacterium Streptococcus equi, and typical signs are pyrexia, anorexia, soft cough, purulent nasal discharge, and swollen lymph nodes of the head that frequently abscessate and discharge pus. In some outbreaks, the largely pathognomonic sign of lymph-node abscessation may occur only in later cases or may not be evident at all. To maximize the chance of effective containment and control, it is important that a diagnosis of strangles is made as early as possible, even in horses that may not be showing classic signs. This is best achieved by culture and identification of the S. equi bacterium on appropriate samples by a specialist bacteriology laboratory that has experience in the typing of specific streptococcal species. The optimal chance of confirming the diagnosis may be achieved if samples of aspirated pus, swabs from discharging abscesses, and swabs of the nasopharynx are each submitted from as many suspect cases as possible. A negative result for one nasal swab from a single horse, for example, should not be taken as assurance that S. equi is not involved, and it is always wise to follow the general rule of thumb that “if it looks like strangles, then treat it as strangles.”

The cornerstone of control of any infectious disease is prevention of transmission of the causative organism from infectious to susceptible animals, and this can only be effectively achieved when control measures are based on a thorough understanding of the epidemiology of the disease. In strangles outbreaks, purulent discharges from clinical and recovering cases are an extremely important and easily visible source of infection. It is important that all measures are taken to prevent both direct and indirect transfer of these discharges between affected and susceptible horses. Direct transmission here refers to horse to horse contact, whereas indirect transmission can be achieved by sharing of water sources, feeding utensils, tack, equipment, and other less obvious transmitters such as handlers, farriers, and veterinarians as well as their hands, clothing, and equipment.

Because it is harder to identify sources of infection, transmission that originates from outwardly healthy horses is of greater importance. Categories of outwardly healthy horses that are potentially infectious for S. equi include those horses that are incubating the disease and go on to develop signs. It is still not clear for how long horses can be infectious while incubating the disease before they develop obvious outward signs, and it is assumed that normal nasal secretions are the source of infection in these animals. The other important category of asymptomatic horses that are potentially infectious to other animals are those recovered cases that continue to harbor the organism after clinical signs of disease have cleared up. There is evidence that a moderate proportion of horses continue to harbor S. equi for several weeks after clinical signs have disappeared, but in the majority of horses, the organism is no longer detectable 4–6 wk after total recovery. It is, therefore, sensible to consider that all recovered horses may be a potential source of infection for at least 6 wk after their purulent discharges have dried up. However, there is growing evidence that in a small but important proportion of recovered cases of strangles, carriage and periodic shedding of S. equi continues for many months and in some cases, even years after apparent full and uncomplicated recovery. These horses are referred to as long-term, asymptomatic strangles carriers, and there is strong anecdotal evidence that they can be a significant source of new outbreaks, even in well-managed groups of horses. If control measures are to be fully effective, there must be recognition of the importance of this category of animal, and appropriate detection and management of them should be adopted in a disease-control strategy. Although there have been periodic references in the scientific literature to the carrier state for strangles, it has really only been in the past 15 yr that this has been more widely accepted as the most fundamental factor in the endemic persistence of S. equi in equine populations. Only targeted identification and resolution of the infection in these animals can effectively act to bring previously recurrent disease under control.

Veterinary investigation of a strangles outbreak on affected premises should begin with a detailed
history through interview with the owners. This allows the full extent of the problem, which may actually extend back several years, to be evaluated. Review of the background to the outbreak should identify affected groups of horses and allow the geography of the premises and the management practices adopted to be examined. The next stage is to agree on and implement a practical disease-control strategy. The general aims and measures for such a strategy are outlined in Table 1.14 This outline strategy. The general aims and measures for such a strategy must be adapted to the individual circumstances of each specific premise and outbreak.

In summary, all movements of horses on and off the affected premises should be stopped, and parallel segregation and hygiene measures should be implemented immediately. Cases of strangles and their contacts should be maintained in well-demarcated “dirty” quarantine areas. The aim of the control strategy is to move horses from the “dirty” to “clean” areas where non-affected and non-infectious horses are maintained after bacteriological screening. Every care should be taken to maintain high hygiene standards throughout the premises and for the duration of the investigation. Screening of all convalescing cases and their healthy contacts should be conducted using nasopharyngeal swabbing or lavage. Swabs are taken at weekly intervals over several weeks and are tested for \textit{S. equi} by conventional culture and identification techniques. Sensitivity for detection of \textit{S. equi} is improved if washes or lavage samples are also tested by polymerase chain reaction (PCR) test.14 Because PCR has the ability to detect dead as well as living bacteria, positive PCR results should only be considered as provisional positives subject to further investigation. It has been found that the vast majority of asymptomatic, long-term strangles carriers harbor \textit{S. equi} in the guttural pouches in association with grossly visible pathology. Therefore, endoscopy of the upper respiratory tract and guttural pouches should be performed in all outwardly healthy horses in which \textit{S. equi} is detected, either by culture or PCR. Lavage samples from guttural pouches should be tested for \textit{S. equi} by culture and PCR. \textit{S. equi} infection and pathology, which range from slight empyema through to multiple chondroids and severe empyema in one or both guttural pouches, may be successfully eliminated without the need for referral or hospitalization,15 but the intensity and cost of treatment is dependent on the extent of pathology involved. Experience suggests that repeated use of an endoscopic helical retrieval basket in conjunction with

### Table 1. Summary of Aims and Measures to Control Transmission of \textit{S. equi} on the Stud

<table>
<thead>
<tr>
<th>Aim</th>
<th>Control Measure</th>
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</thead>
<tbody>
<tr>
<td>1 Prevent spread of infection to horses on other premises and to new arrivals on the affected stud farm.</td>
<td>All movement of horses on and off the stud should be stopped immediately until further notice.</td>
</tr>
<tr>
<td>2 Establish whether horses are infectious in the absence of clinical signs of strangles (asymptomatic carriers).</td>
<td>All recovered cases and contacts have at least three nasopharyngeal swabs taken at approximately weekly intervals and tested for \textit{S. equi} by culture and PCR.</td>
</tr>
<tr>
<td>3 Determine if horses are likely to be free from infection with \textit{S. equi} (non-infectious).</td>
<td>Three consecutive swabs are negative for \textit{S. equi} by culture and at least the third swab of the series is also negative by PCR.</td>
</tr>
<tr>
<td>4 Determine if horses are likely to be harbouring \textit{S. equi} (infectious).</td>
<td>\textit{S. equi} is cultured or detected by PCR on any of the screening swabs.</td>
</tr>
<tr>
<td>5 Isolation of infectious horses from those screened negative for \textit{S. equi}.</td>
<td>Infectious horses are maintained in so-called “dirty” isolation areas which are physically cordoned from the other “clean” areas of the stud where non-infectious horses are kept. Clustering of cases in groups may allow parts of the stud to be easily allocated as “dirty” and “clean” areas.</td>
</tr>
<tr>
<td>6 Prevent cross infection from horses in the “dirty” area to those in the “clean” area of the premises.</td>
<td>Potentially infectious horses in the “dirty” area are preferably looked after by dedicated staff or are dealt with after non-infectious horses in the “clean” area. Strict hygiene measures are introduced including provision of dedicated clothing and equipment for each area, disinfection facilities for personnel and thorough cleaning and disinfection methods.</td>
</tr>
<tr>
<td>7 Monitoring of horses in the “dirty” areas for persistence of \textit{S. equi} infection and establishment of sites of carriage.</td>
<td>Nasopharyngeal swabbing is continued with endoscopic examination of the upper respiratory tract including guttural pouches in those horses in which \textit{S. equi} was isolated after clinical signs had disappeared. Horses that satisfy the non-infectious criteria of measure 3 above are returned to the “clean” area.</td>
</tr>
<tr>
<td>8 Elimination of guttural pouch inflammation and \textit{S. equi} infection</td>
<td>Removal of pathology through a combination of flushing and aspiration of saline and removal of chondroids using endoscopically guided instruments. Topical and systemic administration of penicillin antimicrobial treatment to eliminate \textit{S. equi} infection.</td>
</tr>
</tbody>
</table>
topical and systemic administration of penicillin antimicrobial therapy is appropriate for even the most refractory cases.

8. Investigating Strangles Post-Vaccination

The first strangles vaccine to be licensed in the United Kingdom was a modified-live strangles vaccine\(^\text{16b}\) that became available in 2004. Although clinical signs in horses apparent within several days of vaccination might seem like an adverse reaction to the vaccination, the possibility of concurrent natural infection with wild-type \textit{S. equi} also needs to be considered.

In response to several suspected adverse reactions, several differential diagnostic methods have been developed and successfully applied.\(^\text{17}\) These include (1) PCR to differentiate vaccine from field \textit{S. equi} strains (Fig. 4), (2) DNA sequencing to rule out possible reversion to virulence of the vaccine strain, and (3) DNA sequencing of M-protein regions to subtype and differentiate \textit{S. equi} strains.

One horse developed pyrexia and painful submandibular lymph-node swelling 7 days post-vaccination, and vaccine-strain \textit{S. equi} was isolated from the nose at that time. Field-strain \textit{S. equi} was subsequently isolated from the discharging lymph node. DNA sequencing confirmed that the field strain did not occur through recombination with \textit{S. zooepidemicus}, and the M protein was different from that of the vaccine strain. In another incident, a foal developed parotid lymph-node abscessation after vaccination, and \textit{S. equi} was isolated. Shortly afterwards, a nearby non-vaccinated horse also developed clinical signs of strangles, and \textit{S. equi} was isolated from mucopurulent nasal discharge. PCR and DNA sequencing applied to these isolates showed that they were both field strains that did not come from recombination with \textit{S. zooepidemicus}; again, these cases were not caused by infection with vaccine \textit{S. equi} strains. Further investigations subsequently identified an infected carrier from which field-strain \textit{S. equi} was isolated. M-protein sequencing showed that this “carrier” isolate was identical to that isolated from the non-vaccinated case with the nasal discharge. Other investigations have subsequently identified a few cases of strangles in which the vaccine strain has been isolated in pure culture from abscessed lymph nodes, and it was associated with administration of the vaccine in accordance with the manufacturer’s recommendations. Therefore, there is limited potential for the vaccine strain to cause disease.

This work highlights the discriminatory potential provided by modern molecular techniques. In assessing strangles-like clinical presentations post-vaccination, consideration should be given to two possibilities: modified live vaccines being responsible for clinical disease or carriers contributing to \textit{S. equi} persistence.

9. Concluding Remarks

The limited examples provided above are intended to illustrate the variety of different mechanisms by which improvements in disease control may be introduced and subsequently, evolve. This ranges from the introduction of a mandatory vaccination program, which within the defined sector of Thoroughbred racing has been able to be effectively policed through the use of equine passports, to the voluntary codes of practice, which through widespread compliance continue to achieve their intended aims. However, delayed recognition of the importance of other significant epidemiological features, such as strangles carriers, has consequently taken longer to bring improvements in disease-control measures, although there has been progress on this front. Finally, the application of sensitive molecular techniques in diagnostic applications is improving our understanding of disease investigation and control.

References and Footnotes


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