RISK FACTORS ASSOCIATED WITH UPPER RESPIRATORY TRACT DISEASE DURING AN OUTBREAK OF INFLUENZA

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In August of 1990 an outbreak of upper respiratory tract disease occurred at the racetrack in Saskatoon, Saskatchewan, Canada. There were 450 thoroughbred racehorses at the racetrack stabled in 11 standard and 7 pole barns. The pole barns were more open to the environment and perhaps better ventilated than were the standard barns. Most horses remained at the track during the course of the outbreak.

The investigation began 8 days after the identification of the initial case. Its purpose was to identify the etiology of the epizootic and to examine the association of various risk factors with the occurrence of disease. Animals showing evidence of a cough and a nasal discharge or animals with one of these signs plus one other sign consistent with respiratory disease (fever, inappetence, depression or lymph node enlargement) were classified as "cases" of disease. Horses with only a cough or nasal discharge were classified as "suspect cases" and those with neither a cough nor nasal discharge were classified as "control" animals.

MATERIALS AND METHODS

Initially, all horses stabled at the facility were considered eligible for inclusion in the study. Clinical histories were obtained for 390 horses and the owners and trainers permitted examination and sampling of 350 animals. Our protocol included taking a history, performing a physical examination and obtaining a serum sample from each horse. This process was repeated three weeks later. During the outbreak, nasal swabs were also taken from a number of horses showing obvious signs of acute upper respiratory tract disease.

Laboratory tests included attempts to isolate viruses from the nasal swabs and serologic techniques to test for antibodies to equine herpesvirus (enzyme linked immunoabsorbent assay) and equine Influenza virus (serum radial haemolysis).

Prior to analysis, log transformation was performed on the antibody levels. Simple associations were examined by chi-square analysis and Student's t test. Conditional associations were examined using the Mantel-Haenszel technique, logistic regression

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Proceedings of the 6th International Symposium on Veterinary Epidemiology and Economics, 1991
Available at www.sciquest.org.nz

and least squares regression as appropriate.

RESULTS

By the end of the three week study 69 (19.7%) horses had been classified as cases, 40 (10.3%) as suspects and 281 (72.1%) as non-cases or control animals. Cases of respiratory disease occurred in 16 of the 18 barns. Nasal swabs were submitted from X animals showing clinical signs of disease. Equine Influenza A2 (Miami-like) was isolated from Y of these cases and was the only virus isolated from swabs taken during the outbreak. Comparison of acute and convalescent sera among case and control animals demonstrated a significant increase ($p=0.001$) in equine Influenza A2 antibody levels in affected animals. Changes in antibody levels to equine Influenza A1 were not significant and the Equine Herpesvirus serologic study has not yet been completed.

The significant simple and conditional associations of the various risk factors (age, sex, type of barn, vaccination status, antibody levels, and number of days at the track prior to the outbreak) with the occurrence of respiratory disease are presented in the Table 1. Age and initial antibody levels were simply and conditionally associated with disease occurrence.

No association was found between vaccination against equine Influenza A1 or A2 (within the past 3 to 6 months or within the past 3 months) and the occurrence of respiratory disease. However, age and vaccination status were simply and conditionally related to initial antibody levels.

DISCUSSION

The association of decreased age and low equine Influenza A2 antibody level with an increased risk of disease was expected. Conditionally, the risk of disease was 6 times greater in animals less than 4 years of age than in those 4 years of age and older and 12 times greater in animals with low A2 antibody than in those with high A2 antibody levels. The importance of age as a risk factor in equine upper respiratory tract disease has long been recognized. The association of high initial antibody levels to A/equine 2 virus with a decreased incidence of disease suggests that this antibody was protective. This is consistent with the fact that equine Influenza A2 virus was the only virus isolated in this study and also with the finding that case animals experienced a significantly greater rise in A2 antibody level than did non-cases.

The results of this study did not demonstrated a significant relationship between vaccination and the occurrence of respiratory disease in individual animals. However, it is recommended that for adequate protection against influenza, horses should be vaccinated every 3 months. Forty-six percent of horses in this study had been vaccinated within 3 to 6 months but only 8% had

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been vaccinated within 3 months of the outbreak. With only a few animals adequately vaccinated against the disease, any effect of vaccination may have been overwhelmed by the challenge of the outbreak. We believe that there was an insufficient number of adequately vaccinated horses examined in this study to provide a convincing test of vaccine effect.

An increase in the number of days stabled at the track was not associated with a decrease in the risk of disease. We had expected to find that an increase in the length of exposure to the track environment would lead to greater protection against disease. It is possible that a new strain of virus was introduced just prior to the outbreak or that an increase in pathogenicity of virus already present in the population may have occurred.

The simple and conditional associations of A1 antibody level with the risk of disease was not expected. Equine Influenza A1 (Prague strain) has not been isolated during outbreaks of disease in North America for a number of years, nor was there any indication through virus isolation or seroconversion that it was the cause of this outbreak. We have looked for evidence of cross-reactivity in our serologic tests for these antibodies and have found none. The commonly used vaccines contain A1 antigen. Logically, antibody to A1 developed in response to previous vaccination. We speculate that A1 antibody levels may have acted as a surrogate measure of overall immune function but we have not substantiated this.

Table 1. Factors associated with clinical signs of influenza in an outbreak among 350 thoroughbred horses

SIMPLE ASSOCIATIONS

variable	Chi-square	odds ratio	95% CL's
age (<4 yrs)	71.28	12.5	6.5-24.2
low A1 antibody	37.61	6.2	3.4-11.4
low A2 antibody	95.52	23.4	11.1-49.7

CONDITIONAL ASSOCIATIONS

model	variable	Chi-square (M-H)	odds ratio	95% CL's
1.	age (<4 yrs)	41.2	9.0	4.7-17.4
	low A1 antibody	18.9	4.4	2.3-8.5
2.	age (<4 yrs)	24.6	6.2	3.1-12.5
	low A2 antibody	38.5	11.8	5.5-25.2

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