Evaluation of an outbreak of West Nile virus infection in horses: 569 cases (2002)

Larry A. Schuler, DVM; Margaret L. Khaitsa, BVM, PhD; Neil W. Dyer, DVM, MS, DACVP; Charles L. Stoltenow, DVM, DACVPM

Objective—To characterize an outbreak of West Nile virus (WNV) infection in horses in North Dakota in 2002, evaluate vaccine effectiveness, and determine horse characteristics and clinical signs associated with infection.

Design—Retrospective study.

Animals—569 horses.

Procedure—Data were obtained from veterinary laboratory records, and a questionnaire was mailed to veterinarians of affected horses.

Results—Affected horses were defined as horses with typical clinical signs and seroconversion or positive results of virus isolation; affected horses were detected in 52 of the 53 counties and concentrated in the eastern and northeastern regions of the state. Among affected horses, 27% (n = 152) were vaccinated against WNV, 54% (309) were not, and 19% (108) had unknown vaccination status; 61% (345) recovered, 22% (126) died, and 17% (98) had unknown outcome. The odds of death among nonvaccinated horses were 3 and 16 times the odds among horses that received only 1 or 2 doses of vaccine and horses that were vaccinated according to manufacturer's recommendations, respectively. Horses with recumbency, caudal paresis, and age > 5 years had higher odds of death, whereas horses with incoordination had lower odds of death, compared with affected horses without these characteristics.

Conclusions and Clinical Relevance—Vaccination appears to have beneficial effects regarding infection and death caused by WNV. (*J Am Vet Med Assoc* 2004;225:1084–1089)

West Nile virus (WNV) is a flavivirus endemic in Africa, West Asia, and the Middle East and maintained in nature by a bird-mosquito cycle.[1] Since its introduction to the Eastern United States in 1999,[2,3] WNV has spread across the country. In 2002, there were more than 15,257 laboratory-confirmed cases of WNV infection or seroconversion in horses reported in 43 states.[4] Spread by mosquitoes,[5] WNV mainly causes disease in birds, equids (horses, donkeys, mules, and ponies), and humans.[6] The 2002 WNV epidemic in the United States was the largest arboviral meningoencephalitis epidemic reported in the western hemisphere.[7] The epidemic was most intense in the central United States, with the region's highest numbers of equine cases (700 to 1,100 cases/state) in Illinois, Indiana, Iowa, Kansas, Missouri, and Nebraska.[4] In North Dakota, WNV infection was first reported in 2002 and 569 equine cases were detected.[8] There was no evidence of WNV in North Dakota in 2001.[8] West Nile virus was detected in birds in Wisconsin and Iowa in late 2001, although neither Wisconsin nor Iowa reported any infected horses in 2001. Active surveillance of WNV in birds in North Dakota began in June 2002, and WNV was identified in North Dakota approximately 2 to 3 weeks before it was identified in Minnesota, the adjacent state to the east. In 2002, Canadian health authorities also reported WNV in 5 provinces.[9]

A few detailed reports[6,10-13] were made of WNV encephalomyelitis as it first occurred in immunologically naïve horses in the United States. Data suggest that approximately 40% of infections result in death of the horse and that most horses recover from the infection.[6,10-13] A WNV vaccine provisionally licensed by the USDA first became available for use in horses in August 2001 and is now used to prevent viremia in horses exposed to WNV.[4] However, the effectiveness of this WNV vaccine in protecting horses in an epidemic is unknown. The manufacturer recommends that adult horses receive 2 vaccinations 3 to 6 weeks apart, foals receive 3 vaccinations during the same interval, and the full series be completed prior to the vector (mosquito) season.[4] Annual boosters are recommended prior to the mosquito season.[4] The objectives of the study reported here were to describe the initial North Dakota outbreak of WNV infection in horses in 2002, determine the characteristics of the disease, estimate the effectiveness of the available vaccine, and determine which horse characteristics and clinical signs were associated with outcome.

Criteria for Selection of Cases

A horse with WNV infection was defined as a horse with typical clinical signs and seroconversion against WNV, as measured by use of **IgM antibody capture (MAC)**-ELISA, or isolation of the virus.

Procedures

Data for affected horses were retrieved from medical records for 2002 that were obtained from the North Dakota State University **Veterinary Diagnostic Laboratory (VDL)**. Diagnosis of WNV infection in all horses was performed by the same staff at the VDL, thus minimizing diagnostic bias. Additional data were obtained by use of a questionnaire mailed to the veterinarians who treated the horses.

From the North Dakota State Board of Animal Health, 600 E Boulevard Ave, Dept 602, Bismarck, ND 58505-0020 (Schuler); and the Departments of Veterinary and Microbiological Sciences (Khaitsa), Veterinary Diagnostic Services (Dyer), and Animal and Range Sciences (Stoltenow), College of Agriculture, North Dakota State University, Fargo, ND 58105-5406.
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Address correspondence to Dr. Schuler.

Laboratory methods—Specimens (serum, blood, CSF, brain, and spinal cord tissue) from horses with clinical signs and suspected of having WNV infection were submitted to the VDL from veterinarians across the state. No laboratory fees were charged, which may have been an incentive for submission of samples. A MAC-ELISA developed by the USDA Animal and Plant Health Inspection Service[12] and modeled after an eastern equine encephalitis MAC-ELISA[14] was used to detect seroconversion. Confirmatory diagnosis obtained from specimens from the initial 85 horses in this study was accomplished by use of virus isolation, polymerase chain reaction assay, or plaque-reduction neutralization tests performed at **National Veterinary Services Laboratory (NVSL)** in Ames, Iowa. In addition, NVSL also performed MAC-ELISAs on the serum samples. There was 100% agreement between the VDL and NVSL results. Confirmatory testing at NVSL was discontinued at the laboratory's request because of the excessive national demand for testing.

Statistical analyses—Descriptive statistics of horses that tested positive for WNV infection were computed by use of software.[15] Characteristics of vaccinated and nonvaccinated horses were compared by use of χ^2 tests of independence. Software[a] was used to display the spatial distribution of all horses and infected horses in the state by county. Because of difficulty resulting from the large extent of the study area and temporal and resource restrictions, geocoding of individual addresses was not done on the available data to determine specific coordinates for locations of infected horses. Administrative base maps were obtained,[b] and the main thematic data layers were assembled by use of a software program.[c] The association between time-since-vaccination and outcome was investigated. Time-since-vaccination was computed as time in days from the last vaccination to onset of clinical signs or time when the veterinarian was contacted.

Logistic regression analysis as described by Hosmer and Lemeshow[16] was used to identify horse characteristics and clinical signs that were associated with outcome. Outcome (death) was used as the dependent variable. Independent variables included 10 clinical signs (present vs absent) and certain demographic variables, including age (≤ 5 vs > 5 years), sex (male vs female), breed (Quarter Horse vs other), horse type (stock vs other), and vaccination history (no vaccination, 1 or 2 vaccinations not following manufacturer's recommendations, or 2 vaccinations following manufacturer's recommendations). Several selection methods were evaluated along with an assessment of goodness-of-fit statistics (as described by Hosmer and Lemeshow[16]) and the stability of the parameter estimates during the modeling process to determine the final multiple logistic regression model. Vaccination history was incorporated into the model with a reference cell approach, with the nonvaccination group as the reference group. Collinearity among the independent variables (especially the clinical signs) was evaluated and not problematic. Simple Pearson correlation coefficients among the clinical signs were low ($r < 0.25$), which supported the collinearity results. For all final comparisons, a value of $P < 0.05$ was considered significant.

Results

In 2002, specimens from 1,028 horses were submitted to the VDL. Of these, 769 (75%) were from

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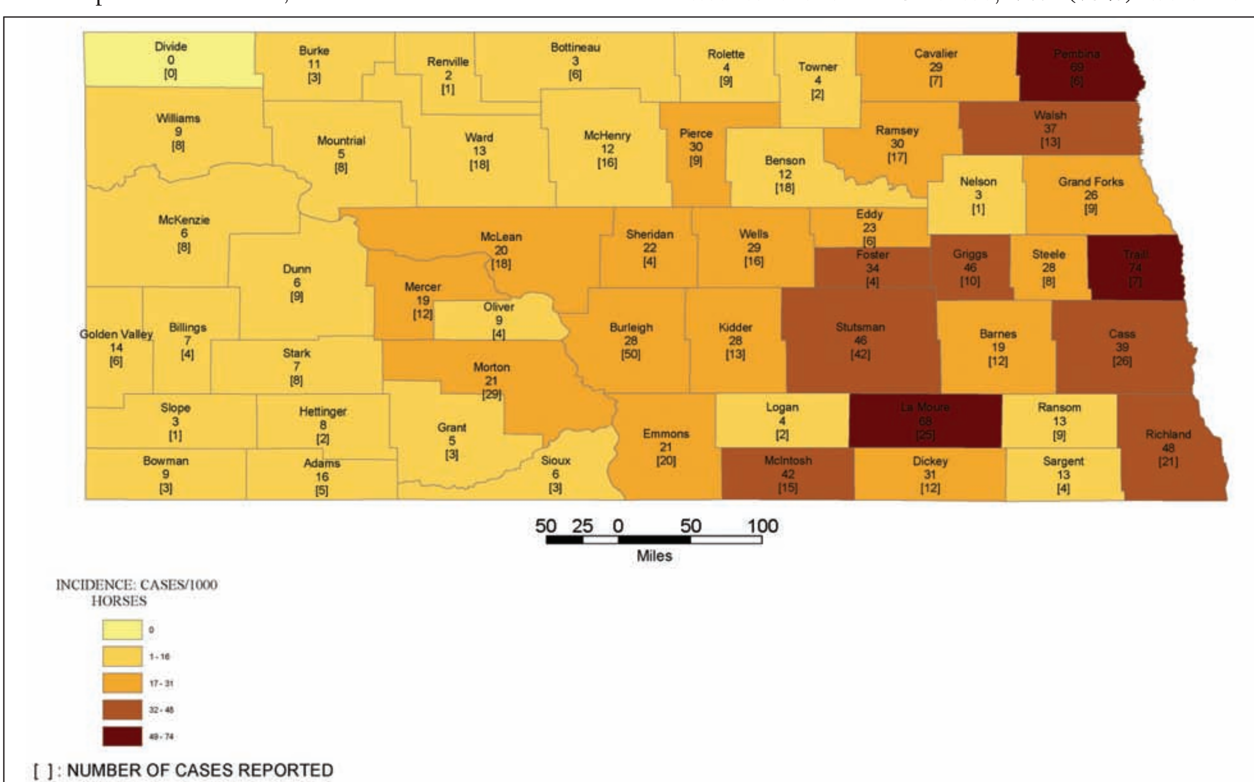

Figure 1—Geographic distribution by county (number of cases/1,000 horses [number of cases reported]) of 569 horses with West Nile virus (WNV) infection in North Dakota, 2002.

horses that met the case definition for this study. Of the 769 cases, 569 were from North Dakota and were included in the study. Questionnaires were mailed to veterinarians of the 569 affected horses, and all questionnaires were returned. Of the 569 horses, 345 (61%) recovered, 126 (22%) died, and 98 (17%) had an unknown outcome. Among horses that died, 77 (61%) were euthanatized and 49 (39%) died from disease. Also, 152 of the 569 (27%) horses were vaccinated, 309 (54%) were not vaccinated, and 108 (19%) had unknown vaccination history.

Cases of WNV infection in horses were reported from 52 of the 53 counties of North Dakota (no affected horses were reported from Divide County), and the number of affected horses ranged from 1 to 50, with the most reported from the central and southeastern counties **(Figure 1)**. Counties with the highest incidence of WNV infection and counties with the highest number of horses did not necessarily match. After accounting for the total number of horses at risk per county, the incidence of equine cases (No. of equine cases/1,000 horses) was higher in the eastern and northeastern parts of the state. Ninety-eight veterinarians participated in the study, and the median number of cases per veterinarian was 5 (range, 1 to 18; mean, 5.8). The majority (84%) of the veterinarians evaluated 1 to 10 affected horses; only 16% evaluated more than 10 affected horses. Affected horses had a range of clinical signs that were mainly attributable to encephalomyelitis, with incoordination (69%) reported most frequently **(Table 1)**.

Cases were reported from June to October 2002 with the majority (363/481 [76%]) occurring in August 2002 **(Figure 2)**. Recovery times varied from 1 day to 3 weeks with the majority (94%) ranging from 1 to 14 days (median, 7.2 days). The majority (334/569 [59%]) of horses were Quarter Horses with at least 15 horse breeds reported, and the predominant horse type was stock (352/420 [84%]); the remainder were light breeds (8%), draft horses (5%), and ponies (2%). Age of affected horses ranged from 3 months to 30 years with a median of 8 years. Age-specific case fatality rate was skewed toward older horses **(Figure 3)**. Of the 152 vaccinated horses, data on time-since-vaccination were available for 113 (74%) of the horses. For horses that died from disease (n = 7), this mean value was 5.4 days (median, 7 days; range, 1 to 9 days); for horses that were euthanatized (12), this mean value was 15.7 days (median, 7 days; range, 1 to 67 days); and for horses that recovered (94), this mean value was 10.7 days (median, 12 days; range, 0 to 44 days). There was a significant ($P = 0.049$) association between time-since-vaccination and outcome of dying, being euthanatized, or recovering. Horses with a relatively shorter time-since-vaccination interval were more likely to die or be euthanatized than those with a longer time-since-vaccination interval.

The case fatality rate among horses that were vaccinated (22/152 [14%]), compared with those that were not vaccinated (104/309 [34%]), was found to be significantly ($P < 0.001$) different by use of the χ^2 test. A significant ($P < 0.01$) difference in case fatality rate was detected between horses that were not vaccinated (case fatality rate, 34%) and those that were vaccinated with only 1 dose or 2 doses that were not administered as per manufacturer's recommendations (case fatality rate, 16%). Also, a significant ($P < 0.01$) difference in case fatality rate was detected between horses that were not vaccinated (case fatality rate, 34%) and those that received 2 doses administered according to manufacturer's recommendations (4%). However, the difference in case fatality rate between horses that received vaccination as per manufacturer's instructions and those that received vaccination but not per manufacturer's instructions was not significant. A Bonferonni adjustment was made to the pairwise comparisons of case fatality rate among the 3 vaccination categories to correct for multiple testing.

A significant difference was also detected via univariate analysis in outcome to WNV infection (death or

Table 1—Distribution (number [%]) of horses with West Nile virus infection in which various clinical signs were evident.

Clinical sign	No. (%) of horses
Incoordination	330 (69)
Muscle tremors, twitching face or muzzle	248 (52)
Weakness or paralysis of limbs	182 (38)
Caudal paresis	141 (29)
Recumbency, difficulty rising, or both	111 (23)
Lip droop	101 (21)
Teeth grinding	40 (8)
Fever	36 (7)
Circling	29 (6)
Blindness	16 (3)

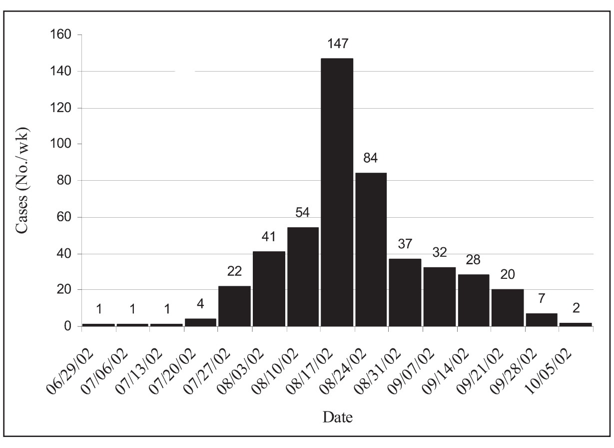

Figure 2—Temporal distribution of 481 horses with WNV infection in North Dakota, 2002.

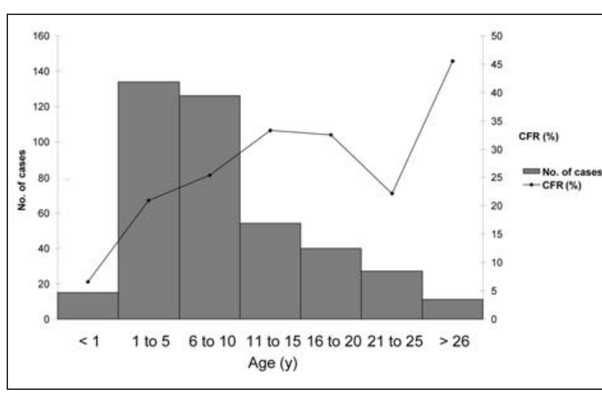

Figure 3—Age distribution (bars; n = 569) and corresponding case fatality rate (CFR; diamonds; 471) of horses with WNV infection in North Dakota, 2002.

recovery) depending on whether horses had specific clinical signs. Recumbency, caudal paresis, teeth grinding, and weakness or paralysis were associated with increased odds of death, whereas incoordination and lip droop were associated with decreased odds of death, compared with affected horses that did not have these characteristics (**Table 2**).

Vaccinated versus nonvaccinated horses differed significantly only in regard to proportion with recumbency ($P = 0.018$) and lip droop ($P = 0.017$). Fewer vaccinated horses were recumbent and more vaccinated horses had lip droop, compared with nonvaccinated horses.

The final multivariate logistic regression model included the variables incoordination, caudal paresis, recumbency, age, and vaccination history. Horses with caudal paresis (**odds ratio [OR]**, 2.6; 95% **confidence interval [CI]**, 1.4 to 5.1), recumbency (OR, 27.2; 95% CI, 13.9 to 53.2), and age > 5 years (OR, 2.3; 95% CI, 1.2 to 4.4) were more likely to die than those without these signs or characteristics. Horses with incoordination (OR, 0.3; 95% CI, 0.1 to 0.5) were less likely to die than those without this clinical sign. Horses that received 1 or 2 doses of vaccine that were not given as per the manufacturer's recommendation (OR, 0.32; 95% CI, 0.15 to 0.68) and those that were vaccinated as per manufacturer's recommendations (OR, 0.062; 95% CI, 0.007 to 0.58) were less likely to die than those that were not vaccinated. The sample size available for the final logistic regression model was 389, concordance was 88.8%, actual misclassification rate was 11.8%, and the Hosmer and Lemeshow goodness-of-fit test suggested that the model was adequate ($P = 0.748$).

Table 2—Univariate analysis of associations between death rate and various clinical signs, characteristics, and vaccination history variables among 471 horses with West Nile virus infection.

Variable	Category	N	Dead (%)	*P* value
Recumbency*	Present	110	79	0.001
Incoordination†	Present	318	18	0.001
Caudal paresis*	Present	139	41	0.001
Lip droop†	Present	100	15	0.002
Weakness or paralysis*	Present	178	34	0.003
Teeth grinding*	Present	39	44	0.016
Horse type	Stock	352	24	0.004
	Other*	68	41	
Breed*	QH	334	23	0.006
	Other*	111	37	
Age (y)	> 5	153	23	0.076
	≤ 5	245	31	
Sex	Male	217	24	0.085
	Female	210	32	
Vaccination history	None*	309	33	0.001
	CM + NCM	152	14	
Vaccination regimen	None*	309	33	0.001
	CM + NCM	127	16	
Vaccination regimen	None*	309	33	0.016
	CM	25	4	
Vaccination regimen	NCM	127	16	0.150
	CM	25	4	

*Variables significantly ($P < 0.05$) associated with higher death rate, compared with horses without those characteristics. †Variables significantly ($P < 0.05$) associated with lower death rate, compared with horses without those characteristics.

N = Number of horses. CM = Vaccination in compliance with manufacturer's recommendations. NCM = Vaccination not in compliance with manufacturer's recommendations.

Discussion

In this WNV epidemic, 73% (345/471 horses with known outcome) of horses with WNV infection recovered from the disease. This agrees with data in the literature[1,13] that suggest that most affected horses recover. Also, the percentage of horses with muscle tremors or fasciculations (52%), lip droop (21%), teeth grinding (8%), and blindness (3%) was similar to that of horses reported with WNV infection in 2000 from 33 states.[12] The clinical signs we observed shared similarities with those reported in other epidemics.[17-23] It is possible that the number of horses reported as dead or even those that recovered was underestimated, given that our sample included only specimens or horses that were tested at the VDL. Also, the outcome of a fairly large proportion (18%) of the affected horses was unknown. Increasing age was generally associated with higher case fatality rate, although this was not true for horses that were 21 to 25 years old.

The wide age range (4 months to 38 years) was similar to that reported in 33 other states[12] and elsewhere,[21-23] suggesting lack of previous exposure to WNV in the horses. Clinically affected horses and those that died in the US outbreaks of 1999 and 2000 were generally older than the horses reported here (median age 14.5 years in 1999 and 16 years in 2000).[10,12] Another study[10] reported a median age of 8 years, similar to ours, for horses that were euthanatized.

A variety of horse breeds have been affected by WNV elsewhere,[12] which is similar to our findings. The spatial distribution of cases in North Dakota closely matched that of known areas of concentration of migratory birds in the state. For several reasons, migratory birds have long been suspected as the principal hosts for introduction of WNV into new regions. Outbreaks of the virus in temperate regions generally occur during late summer or early fall. There may be a temporal relationship between the spring hatching of migratory birds and a subsequent increase in the vector population. These outbreaks also occur in humans living in or near wetlands where high concentrations of birds come into contact with large numbers of mosquitoes, and antibodies to WNV virus and the virus itself have been detected in some species of actively migrating birds.[24] In 2002, seropositive wild-caught birds were reported from 21 North Dakota counties,[25] which included 5 of the 7 counties with most equine cases (≥ 19), and the counties with most cases were located across the migratory routes for wild birds.[26] Elsewhere in the United States in 2002, 144 seropositive wild-caught birds and 366 seropositive captive sentinel birds were reported from 72 counties in 11 states (Florida, Indiana, Iowa, Kansas, Louisiana, Nebraska, New York, North Carolina, Ohio, Pennsylvania, and Texas).[27] A possible association between case sites and proximity to communal bird roosts or waterfowl congregations was reported in a case-control survey conducted by the USDA.[11]

The highest incidence of equine cases was in the eastern and northeastern parts of North Dakota, which

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are the wet regions of the state.[28] It is possible that the eastern part of the state provided better conditions for mosquitoes to breed, compared with other parts of the state, thus creating favorable conditions for WNV transmission to horses. Because most WNV infections in horses do not cause disease, all infected horses may not have been identified.

The temporal distribution of equine cases in North Dakota in 2002 matched that of cases in the 10 states from which data were available,[7] with onset of illness from June or July to November and peaking in August or September. In North Dakota in 2002, equine cases preceded avian cases. It is possible that avian cases preceded equine cases, as has been reported elsewhere,[7,22] but were not observed earlier because passive bird surveillance in the state started in June. The 17 human cases (2 deaths) that were reported during 2002 in North Dakota occurred simultaneously with the equine cases, as was reported in other midwestern and north-central states,[7] although the human cases occurred somewhat later in the season than the equine ones. Nationally, the epidemic peak of human WNV-associated illness during 2002 occurred in late August[7]; human cases in southern states preceded those in northern states by approximately 1 month. In New York in 1999 and 2000, it was reported that equine cases occurred after human cases had been identified.[22] In North Dakota, it is possible that aggressive vector control and public education efforts by state and local public health officials limited the number of human cases. Results of a study[29] indicate that differential mosquito abatement efforts are especially important risk factors regarding the occurrence of WNV infection in humans.

Fever was reported in only 6% of our affected horses, an indication that the viremia in horses may be short-lived or that many horses do not develop clinical signs, as has been reported.[17] A transient, low-level viremia has been detected in naturally and experimentally infected horses and donkeys.[30-32] Research suggests that birds are the only animals in which a viremia develops that is sufficient to infect mosquitoes and propagate the infective cycle.[33]

Significantly fewer vaccinated horses had recumbency, compared with nonvaccinated horses; therefore, vaccination may have reduced the odds of death because recumbency was the clinical sign most strongly associated with death. Interestingly, horses that had incoordination and lip droop were less likely to die than those that did not. It is possible that horses with incoordination and lip droop were in a stage of recovery and had remained alive long enough for these clinical signs to be noticed by the veterinarian and for them to receive supportive treatment, which further improved their chances of survival. Horses that did not have these clinical signs possibly died suddenly or progressed to recumbency quickly and were euthanatized as a result.

In another study,[34] it was also reported that lower odds of death were evident for horses that were vaccinated, compared with those that were not. In our study, even horses that received only 1 or 2 doses of vaccine not given as per manufacturer's recommendations had lower odds of death than nonvaccinated horses, suggesting that any vaccination is beneficial to survival. Horses vaccinated according to manufacturer's recommendations had even lower odds of death, compared with those that were not vaccinated, which further supports the beneficial effect of vaccination.

[a]Geographic Information Systems, ArcInfo 8, ESRI, Redlands, Calif.
[b]ESRI Data and Maps, 1999 edition, ESRI, Redlands, Calif.
[c]ArcInfo 8, ESRI, Redlands, Calif.

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Selected abstract for *JAVMA* readers from the *American Journal of Veterinary Research*

Evaluation of environmental and management-related risk factors associated with chronic mastitis in sows

Fredrik Hultén et al

Objective—To evaluate environmental and management-related risk factors associated with chronic mastitis in sows.

Animals—1,254 sows from 76 herds.

Procedure—Prevalence of chronic mastitis was determined by a veterinarian who performed clinical examinations at the time of weaning and approximately 1 week later in a sample of the sow population on each farm. Information concerning environmental factors and management practices was collected. In addition, the herd veterinarian made an assessment of the farmer's skills in swine production.

Results—Use of partly slatted floors in the farrowing pens, use of disinfectants between batches in the farrowing and breeding areas, feeding lactating sows whey, and avoiding cutting or grinding of the piglets' teeth were significantly associated with a decreased risk of chronic mastitis. A high hygienic standard on the farm, as determined by the herd veterinarian, was associated with a significant reduction in the prevalence of mastitis.

Conclusions and Clinical Relevance—Chronic mastitis in sows is a common disease that has a negative influence on productivity. Results indicate that certain management practices and environmental factors influenced the development of mastitis, which may contribute to the development of methods useful for controlling the disease. (*Am J Vet Res* 2004;65:1398–1403)

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