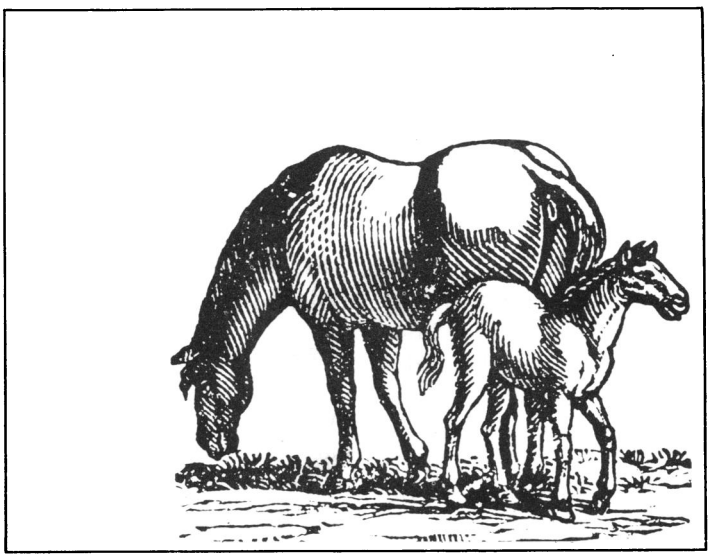
Venezuelan Equine Encephalitis Vaccination Survey in Arizona and New Mexico, 1972



ROSCOE M. MOORE, Jr., DVM, JAMES I. MOULTHROP, DVM, GLADYS E. SATHER, MPH, CHARLES L. HOLMES, BS, and RICHARD L. PARKER, DVM

EPIDEMIC VENEZUELAN EQUINE ENCEPHALITIS (VEE) entered Texas for the first time in late June 1971 from Mexico. The epidemic in Texas has been described in previous reports (*1–3*).

In 1972, there was no evidence of epidemic strain VEE activity in the United States although endemic VEE had been recognized as a cause of sporadic cases of human disease in southern Florida since 1963 (*4*). Vaccinating equines with a modified live virus (TC-83) vaccine against VEE during the 1971 Texas epidemic was one of several control measures used (*1*). From the public health standpoint and the viewpoint of horse owners, the best means of protecting the community against VEE is to insure that all equines have been vaccinated against VEE and that active programs of vaccination and surveillance are maintained.

In late July 1972, because of the possible threat of the reintroduction of VEE into the United States from Mexico (*5*), the Center for Disease Control (CDC) initiated field surveys in parts of Santa Cruz and Yuma Counties, Ariz., and Dona Ana County, N. Mex., to assess the immunization status of equines in these areas.

☐ *Dr. Moulthrop, Mr. Holmes, and Dr. Parker are with the Bureau of Epidemiology and Ms. Sather is with the Bureau of Laboratories, Center for Disease Control, Atlanta, Ga. At the time of this survey, Dr. Moore was also with the Bureau of Epidemiology. He is now chief, Technical Evaluation and Review Branch, Office of Extramural Coordination and Special Studies Branch, National Institute for Occupational Safety and Health.*

Tearsheet requests to Dr. Roscoe M. Moore, Jr., Park Bldg., Rm. 3-32, 5600 Fisher's Lane, Rockville, Md. 20857.

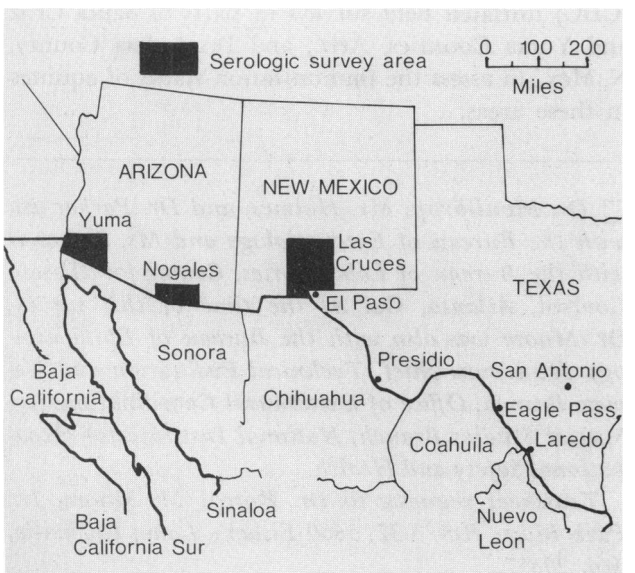
These counties are located on the Mexican border, and each is a potential route for spread of the VEE virus into the United States because of topography or because of highways and railways connecting the county with Mexico (see map). Dona Ana County is located on the Rio Grande across from the State of Chihuahua, Mex. A major railway connects Chihuahua with the United States at Ciudad Juarez, separated from Dona Ana County only by El Paso, Tex. The Rio Grande valley leads from the vicinity of the railhead north into Dona Ana County. Santa Cruz County is adjacent to the State of Sonora, Mex., and is linked to the south by a major highway and a busy railroad. Yuma County is bordered on the west by the Colorado River, which passes between the Mexican States of Sonora and Baja California as it flows to the Gulf of California.

Methods

Roads were selected at random in each county (from the populated portion of Yuma County only), and at least 450 horse owners in each county were contacted by door-to-door canvass and asked about the vaccination status of their animals. Canvass teams consisted of at least one veterinarian and one or more nonmedical persons from local health departments or the U.S. Department of Agriculture (USDA) field stations, or both, located near the survey area. A residence was revisited until someone was found who could give definitive equine vaccination histories. The owners of 1,490 equines were interviewed, and information was taken on 1,260 animals. In addition, blood specimens were obtained from every third equine, regardless of the vaccination status of the animal.

Serum neutralization antibody determinations on collected serums were made for VEE, western equine encephalomyelitis (WEE), and eastern equine encephalomyelitis (EEE)—all are antigenically related group A arboviruses. The plaque reduction neutralization test, using 75–100 plaque-forming units of each virus, was performed in duck embryo cell cultures on serums diluted 1 : 5 and 1 : 50. A 90 percent reduction in plaque count was considered significant. A small sample of the serums was also tested in the standard mouse neutralization test (*6*), using undiluted serum and serial virus dilutions. There appeared to be good agreement between these two techniques, and the tissue culture system was adapted as the test system.

Venezuelan equine encephalitis serologic survey, United States, 1972



Results

Of the 1,260 animals on which information was obtained, 1,181 (94 percent) had been vaccinated against VEE in 1971 (table 1).

Serum samples were collected from 446 equines in the 3-county area; only 226 (50.7 percent) had both a history of VEE vaccinaton in 1971 (including 20 revaccinated in 1972) and serum neutralization antibody against VEE. Of the remaining 220 with no detectable neutralization antibody to VEE, 197 (89.5 percent) had a history of VEE vaccination in 1971 (including 5 revaccinated in 1972), 15 (6.4 percent) had no history of vaccination, and 9 (4.1 percent) had an unknown vaccination status.

Of the 197 equines in the survey with a history of VEE vaccination and no VEE antibody, 160 (81.2 percent) had both WEE and EEE antibodies, 25 (12.7 percent) had WEE antibody only, 2 (1.0 percent) had EEE antibody only, and 10 (5.1 percent) had no detectable antibodies to VEE, WEE, or EEE.

Of the 226 equines with a VEE vaccination history and VEE antibody, 9 were not tested for WEE or EEE antibodies. Of the 217 remaining, 110 (50.7 percent) had both EEE and WEE antibodies, 34 (15.7 percent) had WEE antibody only, 8 (3.7 percent) had EEE antibody only, and 65 (30.0 percent) had no antibodies to WEE or EEE.

Discussion

In a preliminary CDC study of horses not known to have been vaccinated against either WEE or EEE but which had been vaccinated against VEE in 1971,

Table 1. Venezuelan equine encephalitis (VEE) vaccination survey in a three-country area, 1972

County	Number equines on premises	VEE vaccination history					
		Status known	1971 vaccinated	Percent vaccinated	Not vaccinated ≥ 1 year	Not vaccinated 1 year	Status unknown
Santa Cruz	454	395	386	98	2	7	59
Dona Ana	500	412	376	91	16	20	88
Yuma	536	453	419	92	19	15	83
Total	1,490	1,260	1,181	94	37	42	230

89.1 percent (41 of 46) had neutralization antibody against VEE after 11 months.

Some researchers (*5, 7–9*) and J. A. Ferguson and W. C. Reeves, in a paper, "Post-Vaccination Serologic Studies in Horses," presented at the Symposium on Venezuelan Equine Encephalitis in Kansas City, Mo., June 1972, have hypothesized that the presence of WEE or EEE antibody, or both, in horses before VEE vaccination may suppress the development of a detectable VEE antibody response.

WEE is endemic in the surveyed areas (*10*), but EEE activity in these areas has not been previously reported in equines or humans. Therefore, the finding of EEE antibody in serum samples collected in this survey apparently reflects the use of the bivalent, killed WEE-EEE vaccine in the surveyed area.

The presence of WEE antibodies appears to reduce the proportion of equines that respond to VEE vaccination whether EEE antibody is present ($P < .05$) or absent ($P <. 0005$). When the effect of EEE antibody on all animals is controlled by combining the results in a weighted X^2 (*11*), the effect of WEE antibody remains strong ($P < 0.0001$), as shown in table 2. Although the number of animals having EEE antibody without WEE antibody is small, controlling WEE antibody by the use of the same weighted X^2 method suggests that the presence of EEE antibody also results in a reduced proportion of equines with antibody response to VEE vaccination (table 3). Note that the results in tables 2 and 3 are similar, which indicates interference between either WEE or EEE antibody and response to VEE vaccination in equines. It is not known which is the dominant factor or whether both WEE and EEE antibodies combine to produce a cumulative effect.

As a result of the survey conducted in the three-county area in July 1972 and of the presence of VEE in Mexico, USDA again recommended, in early September 1972, VEE vaccination of equines. As an added precaution, the USDA also suggested that equines in the threatened areas that were vaccinated in 1971 be revaccinated. Previous official recommendations had been that revaccination was unnecessary (*12*).

The current USDA recommendation is that all horses be vaccinated and that foals previously vaccinated at less than 6 months of age be revaccinated. USDA also recommends that annual revaccination be performed in high-risk areas (Arizona, California, New Mexico, and Texas) to increase protection.

Table 2. Presence or absence of Venezuelan equine encephalitis (VEE) and western equine encephalomyelitis (WEE) serum neutralizing antibodies in equines vaccinated against VEE, by status of eastern equine encephalomyelitis (EEE) antibodies

EEE status	VEE+ Number	VEE+ Percent	VEE−	Total
With EEE antibodies:				
WEE present	110	40.7	160	270
WEE absent	8	80.0	2	10
Total	118	42.1	162	280
	$X^2=4.592$ $df=1$ $P<0.05$			
Without EE antibodies:				
WEE present	34	57.6	25	59
WEE absent	65	86.7	10	75
Total	99	73.9	35	134
	$X^2=4.592$ $df-1$ $P<0.05$			
All animals:				
WEE present	144	43.8	185	329
WEE absent	73	85.9	12	85
Total	217	52.4	197	414
$X^2=46.368$ $df=1$	Weighted $X^2=20.568$ $df=1$ $P<.0001$			

NOTE: (+) = presence; (−) = absence

Table 3. Presence or absence of Venezuelan equine encephalitis (VEE) and eastern equine encephalomyelitis (EEE) serum neutralizing antibodies in equines vaccinated against VEE, by status of western equine encephalomyelitis (WEE) antibodies

WEE status	VEE+ Number	VEE+ Percent	VEE−	Total
With WEE antibodies:				
EEE present	110	40.7	160	270
EEE absent	34	57.6	25	59
Total	144	43.8	185	329
$V^2 = 4.945$ $df = 1$ $P < 0.05$				
Without WEE antibodies:				
EEE present	8	80.0	2	10
EEE absent	65	86.7	10	75
Total	73	85.9	12	85
$X^2 - 0.007$ $df = 1$ $P < 0.95$				
All animals:				
EEE present	118	42.1	162	280
EEE absent	99	73.9	35	134
Total	217	85.4	197	414
Weighted $X^2 = 5.927$ $df = 1$ $P < 0.5$				

NOTE: (+) = presence; (−) = absence

References

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SYNOPSIS

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Field studies were conducted in 1972 to determine the immunization status of equines along the Mexico, Arizona, and New Mexico borders. Interviews with horse owners were conducted along roads selected at random in the counties of Santa Cruz and Yuma, Ariz., and in Dona Ana County, N. Mex. At least 450 horse owners in each county were asked about the vaccination status of their animals, and information was taken on 1,260 animals. Blood specimens were obtained from every third equine, regardless of stated vaccination status, and tested for the presence of Venezuelan equine encephalitis (VEE), western equine encephalomyelitis (WEE), and eastern equine encephalomyelitis (EEE) neutralization antibodies.

Serum samples were collected from 446 equines in the 3-county area; only 227 (50.7 percent) had both a history of VEE vaccination in 1971 (including 20 revaccinated in 1972) and serum neutralization antibody against VEE. Of the remaining 220 with no detectable neutralization antibody to VEE, 197 (89.5 percent) had a history of VEE vaccination in 1971 (including 5 revaccinated in 1972), 14 (6.4 percent) had no history of vaccination, and 9 (4.1 percent) had an unknown vaccination status.

Eighty-two percent (160 of 197) of the equines with a history of VEE vaccination and presence of detectable WEE or EEE antibodies, or both, had no detectable levels of VEE antibody. Therefore, the results of this study suggest that the presence of WEE or EEE antibodies, or both, may suppress the development of detectable vaccine-induced VEE antibody response in the equine. As a result of this investigation, the U.S. Department of Agriculture, as an added precaution, recommended the revaccination of equines in areas of the United States bordering Mexico.