

U.S. Epizootic of Equine Influenza, 1963

Epizootiology

Robert G. Scholtens, D.V.M., M.S.,
James H. Steele, D.V.M., M.P.H.

IN MAY 1963 it became apparent that horses in the United States were suffering from a widespread epidemic of respiratory illness. An influenza virus was suspected to be the cause, and the problem appeared to be of public health as well as veterinary significance. Although epidemics of human illness have been followed during the course of their occurrence, inherent problems make effective studies of such phenomena difficult, and no similar study of an extensive epizootic has been possible.

During last year's epidemic illness in horses, many persons representing public health, university, veterinary, and racing interests cooperated closely over a period of several months, and this report represents the results of their efforts. Their frequent and timely contributions of information on the progress of the epidemic provided this early example of a nationwide cooperative investigation of animal illness. The role played by the Communicable Disease Center was to provide laboratory evaluation and epidemiologic interpretation and to distribute the accumulated information on the developing epidemic.

Background

Historical records referring to epidemics of "influenza" date from the 10th century. Fleming, in 1871, documented numerous outbreaks of disease in horses, some of which he described sufficiently to allow a retrospective

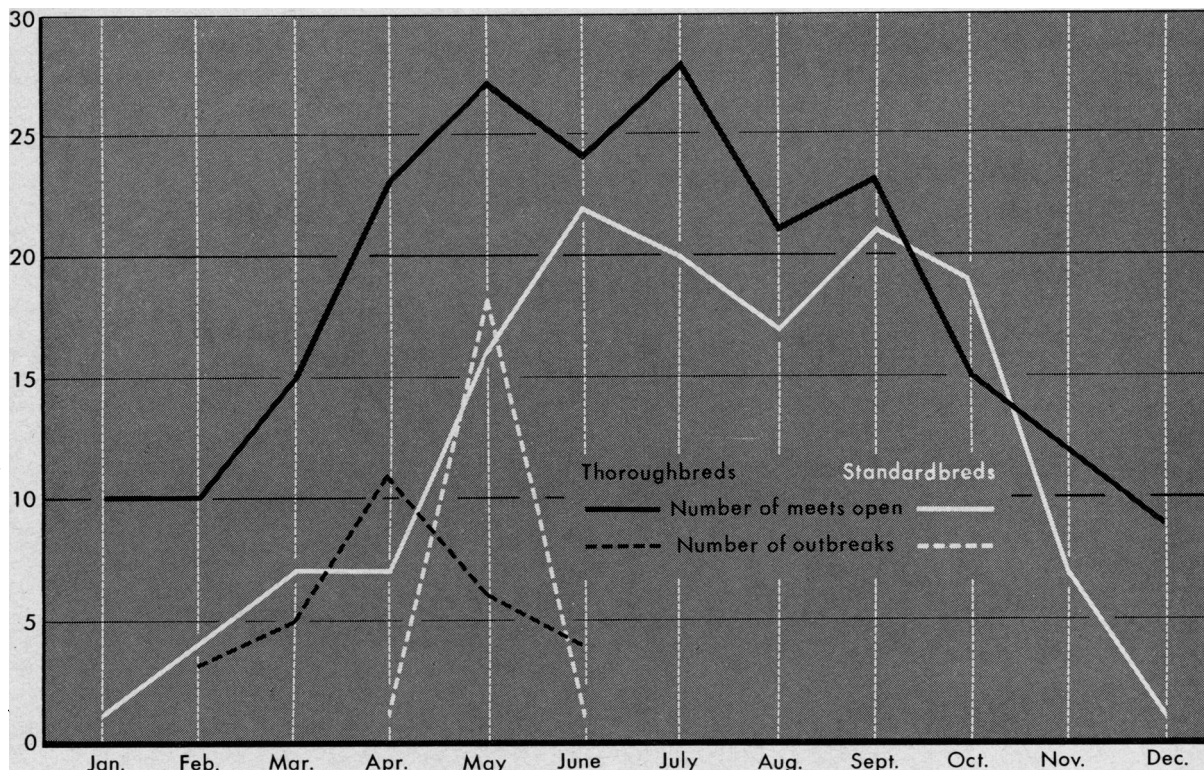
diagnosis (1). From the years 1173 to 1800 he recorded 15 epidemics of influenza in man and described 18 like-named epidemics in horses. Of these 18 equine epidemics, at least 4 were more serious illnesses than can now be ascribed to infection with influenza virus.

Fleming's description of ocular involvement, edema of the legs, icterus, and rather high mortality is more compatible with the equine viral arteritis entity described by Doll (2). Other accounts, such as the following one of an epidemic occurring in 1732, are accurate descriptions of what was observed in the United States early in 1963: "Horses were suddenly seized with a vehement, dry-sounding cough" which, "though in no ways mortal, yet was so very catching that horses on either hand of them were generally infected as soon as they began to run at the nose."

Influenza virus was first recovered from horses by Sovinova and associates (3) in 1956. It was a type A virus, which is now classified as A1-equine/Prague/56. This virus caused epidemic illness in horses in Sweden and eastern Europe in 1955 and 1956. Serologic evidence indicated that this virus was present in the United States as early as 1957 (2), but neither this nor the other viruses recovered from horses in

All the authors are with the Communicable Disease Center, Public Health Service. Dr. Scholtens and Dr. Steele are with the Veterinary Section. Dr. Dowdle, Miss Yarbrough, and Dr. Robinson are with the Respirovirus Unit, Virology Section, and the World Health Organization International Influenza Center for the Americas. These papers were presented at the 91st annual meeting of the American Public Health Association, Kansas City, Mo., November 14, 1963.

Figure 2. Monthly incidence of equine influenza outbreaks at racetracks, January through June 1963



63 virus were made in the United States in the following months. A similar virus has recently been isolated from horses infected during an epidemic of respiratory disease in Uruguay in September 1963 (personal communications from Dr. H. G. Pereira, World Influenza Center, London, December 11, 1963, and January 6, 1964).

The usual clinical signs seen during this and subsequent outbreaks were coughing, fever of 102° to 105° F., and anorexia. The uncomplicated illness lasted from 3 to 10 days. Morbidity rates reached 90 percent at some tracks with mortality rates at or near 0 percent. Four deaths of horses ill with influenza were reported; in no case was influenza thought to be the sole cause of death.

Spread of the Epidemic

The spread of the epidemic was followed by surveillance of equine morbidity at racetracks. Numerous groups, including State health departments, veterinary practitioners, universities, racing officials, and the U.S. Department of Agriculture, contributed to the investigation.

The apparent routes of spread were derived from the surveillance data (fig. 1). From Florida, the epidemic spread to Louisiana, Kentucky, and Maryland. From these sites, it spread to the northeastern and midwestern States. Horses from the midwest carried the virus west to Nebraska and from there to Colorado and California. An outbreak of influenza near San Francisco infected numerous animals which spread the virus to other western States. The disease was reported from even the most widely separated parts of the country 5 months after its introduction.

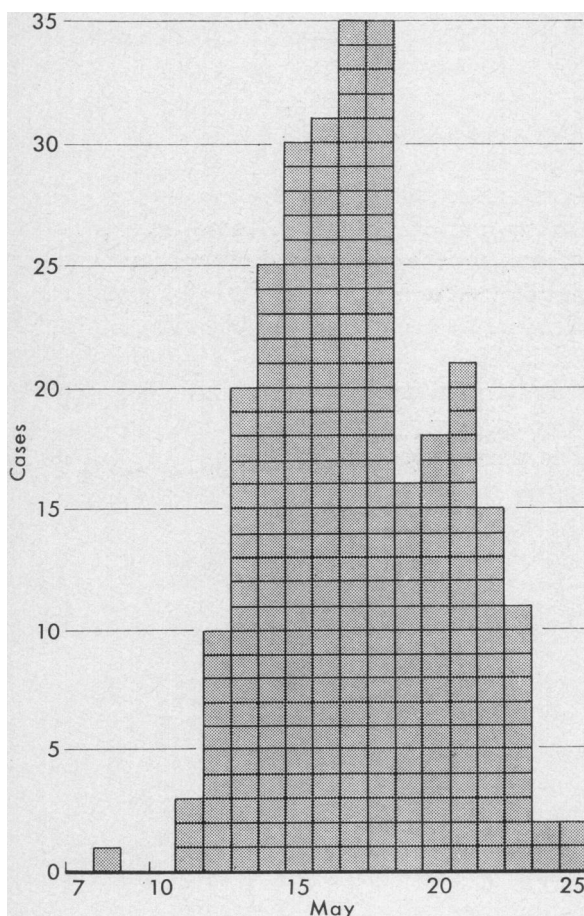
Twenty-five of the 48 contiguous States have horseracing, and among them there are 96 tracks which scheduled more than 10 days of racing in 1963. Some of these tracks had several meets during the year. A total of 153 meets were scheduled, 91 for thoroughbreds and 62 for standardbreds (harness racers). Eighty-eight of these meets were held during the surveillance period, January to July 1963, 56 for thoroughbreds and 32 for standardbreds.

Using the number of meets to indicate the approximate number of equine populations at risk,

we found that 29 (52 percent) of the 56 thoroughbred meets had outbreaks of equine influenza. At 10 meets (18 percent) no outbreak was indicated, and for 17 meets (30 percent) we received no report. Of the standardbred meets, outbreaks occurred at 20 (62 percent), 4 (12 percent) had no outbreaks, and for 8 (25 percent) we received no report.

The epidemic curves, showing outbreaks of equine influenza at standardbred and thoroughbred racing meets during the first 6 months of 1963, are plotted in figure 2. The upper line represents the number of meets open during each month and indicates the approximate number of equine populations at risk. This is only a rough index because the meets frequently last for several months during which time the horse population is constantly changing, perhaps from a population of susceptibles to one of im-

Figure 3. Cases of equine influenza among 450 horses at racetrack A, New York State, May 1963



munes. The number of outbreaks indicated for any given month are those which began in that month, even though they may have continued into succeeding months. It was possible to have more outbreaks than meets in May because influenza struck at several tracks before they were due to open.

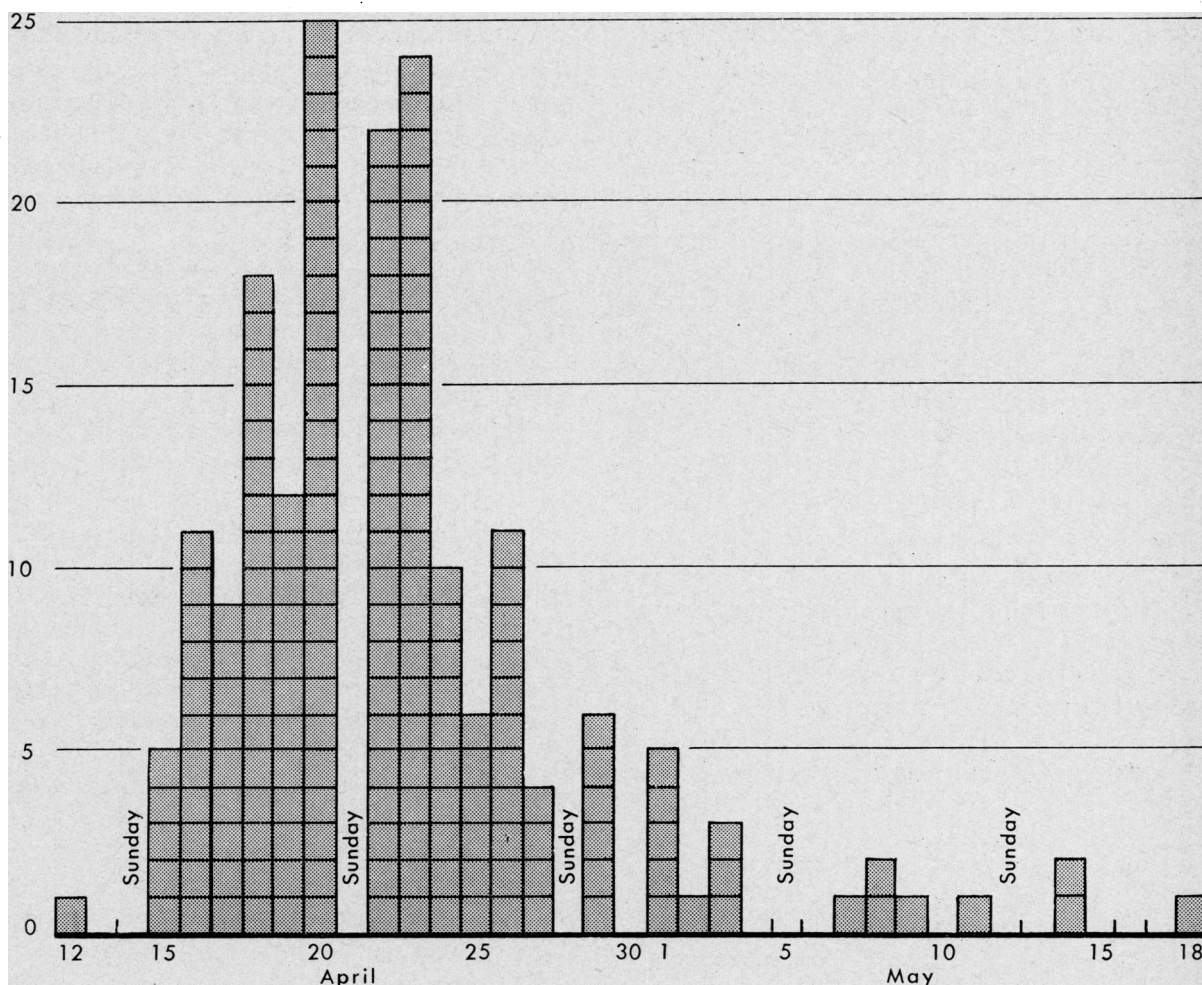
The epidemic began earlier in thoroughbred horses, peaked earlier, and persisted over a longer period of time. The epidemic in harness horses was briefer and more explosive. This is probably because there is harness racing in only 15 States and 13 of these are in the northeastern section of the United States. The confinement of the host population to a smaller area, permitting more frequent and rapid movement of horses, contributed to a more rapid spread of the virus in these animals. Some of the racing meets had closed before the epidemic began; therefore, horses at these meets were not really at risk. Also, there was considerable reluctance by some officials to recognize or make known the fact that the horses quartered at their tracks were experiencing an outbreak of disease. These factors tended to minimize the severity of the recorded epidemic.

At one New York State racetrack, 275 cases of influenza were recorded among 450 horses during a 17-day period (fig. 3). The index case occurred in a horse that came from another track and was ill on arrival. These data indicate approximately a 3-day incubation period. The intensity of the outbreak illustrates the extreme susceptibility of these horses and supports other evidence that this influenza virus is a new parasite in this population. At another racetrack in New York State, data were obtained on the "scratches," or number of horses scheduled to race which failed to appear (fig. 4). (This is an indirect way of obtaining the epidemic curve and is analogous to absenteeism at schools or industrial plants.) At this racetrack between April 12 and May 22, 75 to 90 percent of the horses were infected with influenza, and the track was closed for a time in April.

Conclusions

Epidemic illness of horses in the United States due to the A2-equine/Miami/63 virus now appears to be ended. This virus, like the

Figure 4. "Scratches" among 600 horses during epidemic of equine influenza at racetrack B, New York State, April to May 1963



A1-equine/Prague/56 virus, seems likely to become an endemic parasite of the U.S. horse population. Its presence then will be characterized by sporadic cases of influenza in young susceptible horses. In man, during any one period of time, a single strain of type A influenza virus predominates. When a new strain becomes established the old one may disappear for long periods of time. If this successive pattern of influenza viruses in man is followed, the A2-equine virus will replace the A1-equine virus as the cause of equine influenza in this country.

Numerous reports of concurrent illness in man and horses were received during the course of the epidemic. All thorough investigations of these uncovered some other virus or failed

to fully incriminate the A2-equine virus as the cause of disease in man. The agents causing some of these illnesses were found to be Asian and parainfluenza viruses. Currently there is no evidence that either of these equine influenza viruses are a direct public health hazard to man. Whether they play a role in the evolution of the human influenza viruses or are an example of parallel parasitism in separate hosts remains to be determined.

Summary

An epidemic of influenza in horses occurred in the United States during 1963. The etiologic agent was a previously unknown type A influenza virus. The disease was mild, character-

ized by fever and a cough. The epidemic was explosive. Within 5 months, it had spread to all parts of the country. Horses of all ages were susceptible, and attack rates were 70 to 90 percent in local outbreaks. Few deaths resulted.

This epidemic was apparently one of a series of like events which have afflicted horse populations over many generations. No cases of illness caused by this virus were uncovered in man.

Etiology

Walter R. Dowdle, Ph.D.,

Wilma B. Yarbrough,

Roslyn Q. Robinson, Ph.D.

WHEN equine influenza first appeared in epidemic proportions in Florida in the late winter of 1963, an agent was isolated from nasal washings from an infected horse. This agent was found to be an influenza virus (4). Subsequently, a similar virus was isolated from infected horses during an influenza outbreak in Lexington, Ky. (personal communication from Dr. J. T. Bryans and Dr. E. R. Doll).

As the epidemic continued to spread, epidemiologists from the Communicable Disease Center and elsewhere collected specimens for study in the CDC laboratory. Nasal swabs and washings were received from Ohio, New York, and New Mexico. Paired serum specimens were collected from infected horses at racetracks in New York, Ohio, Nebraska, and California. The original virus isolates were sent to us from Florida and Kentucky, and, as the epidemic progressed, we received virus strains isolated from horses at racetracks in Delaware, Maryland, Michigan, New Mexico, New York, Ohio, and Oregon.

Materials and Methods

Clinical material. Nasal swabs or nasal washings or both, were obtained from horses within 24 to 72 hours after onset of illness and frozen in dry ice for shipment to the laboratory. Acute phase serums were collected as soon as possible after onset, and convalescent specimens were collected 2 to 3 weeks later.

Virus isolation. Nasal specimens were treated with an equal volume of tryptose phosphate broth containing 0.5 percent gelatin, 400 units penicillin, 200 μ g streptomycin, and 200 units mycostatin per ml. Cell cultures of primary rhesus monkey kidney, human fetal kidney, and diploid human lung fibroblasts were each inoculated with 0.3 ml. of the treated specimens. Ten-day-old embryonated chicken eggs were inoculated with 0.1 ml. of the treated specimens by both the amniotic and allantoic routes. Cell culture tubes were incubated for 7 days for each passage at 33° C. and tested for the presence of virus by hemagglutination and hemadsorption with guinea pig erythrocytes (5). Eggs were incubated for 72 hours at 33° C. Amniotic and allantoic fluid harvests were tested for presence of hemagglutinins at dilutions of 1:2 in 0.4 ml. with equal volumes of 0.5 percent chick and 0.4 percent guinea pig erythrocytes at room temperature and at 4° C.

Biochemical and physical properties. Sensitivity of the virus to ethyl ether was determined by the method of Andrews and Horstmann (6) and acid lability by the method of Ketler and associates (7). A comparison of virus size with a known influenza A virus was made with graded millipore filters. A tissue culture adapted equine virus was mixed with A2/NC/1/63, a current human strain, and portions were passed through filters of 100 $m\mu$ and 220 $m\mu$ pore diameters. One portion of each filtrate was treated with antiserum specific for one of the viruses in the mixture and the resulting infectivity titer determined in monkey kidney. A second portion of each filtrate was treated in the same manner with antiserum against the second virus in the mixture. The identity of the virus yielding infectivity titers was reconfirmed upon completion of the tests.

Hemagglutinins were titrated in phosphate buffered saline (pH 7.2) with 0.5 ml. of virus dilutions and an equal volume of erythrocytes. All erythrocyte suspensions were adjusted to cell concentrations equivalent to that of a 0.5 percent (by volume) suspension of chick cells, that is, approximately 4×10^7 cells per ml.

Serology. Hemagglutination-inhibition and complement fixation tests were performed as previously reported by this laboratory (8).