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EXPERIMENTAL INFECTION AND HORIZONTAL TRANSMISSION OF MODOC VIRUS IN DEER MICE (*PEROMYSCUS MANICULATUS*)

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ABSTRACT: Deer mice (*Peromyscus maniculatus*) were inoculated with a sublethal dose of a field strain of Modoc virus to determine patterns of viral persistence, shedding, and transmission. Blood, serum, urine, fecal, and oral swab samples were collected at selected intervals until 63 days postinoculation (PI) after which lung, liver, spleen, kidney, and salivary glands were explanted. Viral assays were conducted by intracranial inoculations of suckling mice and antibody titers were determined by the micro-complement-fixation test. Viremias lasted for up to 4 days PI. Antibody titers were present by day 8 PI, peaked at day 13–20 PI, and persisted until day 63 PI. There was no evidence of viral shedding in urine, fecal, or oral swab samples. Virus was detected in explanted lungs only. In a separate experiment, deer mice were inoculated with virus and lungs were removed from five mice per wk for 10 wk. Indirect fluorescent antibody (IFA) techniques were used to determine the location of virus in lung tissue and to examine fixed tissue for lesions. IFA showed virus in lung parenchymal cells beginning 42 days PI and persisting at least 70 days PI. No histopathologic changes were seen. Horizontal transmission of the virus was studied by placing uninoculated mice with inoculated mice for 42 days and determining if the test animals developed antibodies or had virus in their lungs. Fifty-percent of the uninoculated mice developed antibody. One of these animals had virus in its lungs. Therefore, Modoc virus may be transmitted by direct contact.

Key words: Modoc virus, deer mice, *Peromyscus maniculatus*, arbovirus, experimental.

INTRODUCTION

Modoc (MOD) virus is a rodent-associated virus that, like many other such viruses, has the potential to cause disease in humans. It has been considered responsible for a case of aseptic meningitis in a boy in California (Davis and Hardy, 1973) and neutralizing antibody was found in sera collected from a group of American Indians in Alberta, Canada (Zarnke and Yuill, 1985). To date, MOD virus has been isolated only from deer mice (*Peromyscus maniculatus*) in California (Johnson, 1967), Colorado, Oregon, Montana (Berge, 1975), and Alberta, Canada (Zarnke and Yuill, 1985). However, the deer mouse is distributed widely in North America, from Mexico to the Canadian tundra excluding the southeastern United States (Hooper, 1968), and it is possible that the virus also could be found throughout this entire region. Although deer mice frequently live in close association with humans, not enough is known about the transmission of MOD virus to assess its risk to public health.

MOD virus is classified taxonomically as a flavivirus (Berge, 1975). However, it has no known vector and attempts to infect mosquitoes and ticks (Johnson, 1967) or cultures of mosquito (Main et al., 1977; Cahoon et al., 1979; Varelas-Wesley and Calisher, 1982) or tick cell lines (Yunker et al., 1981) have been unsuccessful. Other investigators have suggested that the virus may be transmitted vertically or horizontally, since the original isolate came from the mammary gland of a deer mouse (Johnson, 1967) and subsequent laboratory studies with hamsters (Davis and Hardy, 1974) and white mice (Johnson, 1967) demonstrated viral persistence in the kidney and shedding in urine. A previous investigation (Davis et al., 1974) of MOD virus infection in deer mice using prototype virus that had been passaged twice in hamsters showed that the virus persisted

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in lung and mammary tissue, but was not transmitted horizontally and had limited capacity for vertical transmission. Our report describes the persistent infection of deer mice with a field strain of MOD virus and suggests a mode of horizontal transmission.

MATERIALS AND METHODS

Stock virus

The strain of MOD virus used in these experiments was isolated originally from blood clots taken from two deer mice in northeastern Alberta, Canada (Zarnke and Yuill, 1985). It was passaged three times in suckling white mouse brains prior to use. Stock virus was prepared as a clarified 10% suspension of suckling mouse brain in M199 media (Gibco Laboratories, Grand Island, New York 14072, USA) with 10% heat-inactivated fetal calf serum and titered 10^{11} suckling mouse intracranial lethal dose 50% ($SMICLD_{50}$) per ml. Suckling mice inoculated with the virus became obviously ill (lethargic, uncontrollable twitching, cessation of suckling) 1 to 2 days prior to death. Death occurred from day 7 to day 13 postinoculation (PI), depending upon the dilution used. Identity of the virus was confirmed by the suckling mouse neutralization test using reference antiserum supplied by the Centers for Disease Control (Atlanta, Georgia 30333, USA). Unless specified otherwise, the dose of virus used in all experiments was 0.5 ml of virus suspension containing $10^{7.6}$ $SMICLD_{50}$/ml inoculated intraperitoneally.

Mouse inoculations

Adult (≥3 mo old) female deer mice (*Peromyscus maniculatus bairdii*) were used in all experiments. Animals were obtained from an outbred colony maintained by our laboratory for 3 yr and had no serologic evidence of prior infection with MOD virus. Original breeding stock was obtained from colonies maintained by Dr. W. P. Porter, University of Wisconsin, Madison, Wisconsin 53706, USA, and Dr. C. R. Turman, College of William and Mary, Williamsburg, Virginia 23185, USA. Mice were housed in polypropylene boxes with stainless steel lids and provided with Purina® certified rodent chow and water ad lib.

Viremia and antibody patterns. Ten deer mice were inoculated intraperitoneally with the test dose of stock virus. Whole blood was collected daily from the retroorbital sinus for 7 days for viremia determination and again on day 46 PI to examine for persistent viremia. Blood was diluted 1:5 in M199 with 10% heat-inactivated fetal calf serum and stored at −70 C until tested. Serum samples were collected at selected intervals from 8 to 63 days PI and stored at −20 C until tested for antibody titers.

Virus shedding. Ten additional animals were inoculated intraperitoneally with 0.5 ml of virus suspension containing 10^{10} $SMICLD_{50}$/ml. Urine samples were taken on days 2, 6, 12, 16, 19, 23, 30, 37, and 50 PI to determine patterns of viral shedding. Fecal and oral swab samples were collected on day 37 PI. Urine and fecal samples were obtained by placing each mouse in a glass jar with a 1-cm^2 mesh steel screen over the top. The jars were inverted in glass funnels the stems of which were placed in 5 ml plastic test tubes sitting in wet ice. Mice were left in the jars for 2 to 3 hr. Urine accumulated in the test tubes and fecal material was collected from the screen.

Urine was diluted 1:2 in M199 containing twice the normal amount of penicillin and streptomycin prior to storage. The pH of urine diluted in M199 was approximately 7.0. The pH of undilute urine was 8.0. A 1:2 dilution using the stabilizing medium devised by Davis and Hardy (1974) (sodium borate plus bovine serum albumin) increased the pH to 9.0. Fecal samples also were suspended in M199 plus antibiotics in a 1:2 (weight : vol) ratio, centrifuged, and the supernatant stored at −70 C until assayed for virus. The pH of the fecal suspensions ranged from 6.8 to 8.0.

Oral swabs were taken using dacron tipped plastic applicators that were then placed in 0.5 ml of transport media and stored at −70 C until the media was assayed for virus. In a pilot experiment with swabs dipped in stock virus of known titer and then stored in M199, it was determined that dacron tipped plastic applicators did not reduce the titer of virus present, whereas calcium alginate (calgi) swabs decreased virus titer by 4.5 $\log_{10}$, and cotton tipped wooden applicators decreased virus titer by 3 $\log_{10}$.

Tissue tropism. The 10 mice that had been inoculated with virus to determine viremia and antibody patterns were divided into two equal groups and killed by CO_2 exposure 65 days PI. Lungs from mice within each group were removed, pooled, and explanted to determine if virus was present. Similar pools and explant cultures were made from liver, spleen, kidney, and salivary glands. Organs were placed in plastic petri dishes and minced into pieces less than 0.5 mm^3 using a scissors. After three washings with phosphate buffered saline, the tissue pieces were aspirated into a pasteur pipette and transferred to 25 cm^2 tissue culture flasks. Three ml of M199

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media were added to each of the flasks which were then incubated at 37 C in 5% CO_2. Explant media was changed twice per week and assayed for virus. After 10 wk postexplanation, cells were lysed by three cycles of freeze-thawing. The media was centrifuged and the supernatant assayed for virus. Titer of virus in explant samples was determined by inoculating explant growth media intracranially into suckling mice.

After determining that virus localized in the lungs, a follow-up experiment was conducted to determine when the virus reached the lungs, in which cell type it replicated, and if it caused any pathologic changes. Fifty mice were inoculated with the test dilution of stock virus. Another 50 mice were inoculated with diluent to act as controls. Every 7 days for 70 days five inoculated and five control mice were killed by pentobarbital overdose. Lungs were removed from three mice in each group and frozen in preparation for the indirect fluorescent antibody (IFA) test as described below to determine the presence of virus and cell type in which it localized. In the remaining two mice in each group, Bouin's solution was infused under pressure into the lungs to inflate the alveoli. The entire lung was fixed for 24 hr in Bouin's solution prior to sectioning and staining with hematoxylin and eosin for histopathologic examination.

Virus transmission. Viral persistence in lung tissue of infected mice suggested transmission could occur by aerosol or through salivary secretions during mutual grooming. To test for transmission by these routes, five mice that had been infected with a test dose of virus 42 days previously were placed in each of two 32.5 × 20 × 12.5 cm rat boxes with eight uninoculated mice for an additional 42 days. After this transmission period, serum samples were collected from all the mice and assayed by the micro-complement-fixation (CF) test (Casey, 1965) for antibody determinations. The mice were killed by pentobarbital overdose and their lungs examined for the presence of virus by the IFA technique described below.

Viral and antibody assays

Whole blood, tissue culture media, urine, fecal, and oral swab samples were assayed for the presence of virus by IC suckling mouse inoculations. Titration endpoints were calculated by the method of Reed and Muench (1938). Suckling mice were observed for 14 days PI or until death. Antibody assays were performed by the micro-CF test using infectious antigen prepared from suckling mouse brains.

Virus was detected in situ in lung tissues using an indirect fluorescent antibody (IFA) test. Mice were killed by overdosing with pentobarbital and their lungs were removed immediately. One-cm^2 pieces of tissue were snap-frozen by immersing for 30 sec in a beaker of 2-methylbutane (iso-pentane, Scientific Products, McGaw Park, Illinois 60085, USA) sitting in liquid nitrogen. Tissue pieces were transferred to cryotubes and stored at −70 C until sectioning with a cryotome. After sectioning and mounting on microscope slides, specimens were fixed in cold acetone for 10 min and antiserum and fluorescein conjugate were added as previously described (Lyerla and Forrester, 1979). MOD antiserum was produced as mouse ascitic fluid in white mice (Field and Kalter, 1972). Prior to use, the antiserum was adsorbed for 1 hour with deer mouse liver powder to remove nonspecific antimouse antibodies. Antimouse immunoglobulin G conjugated to fluorescein dye was purchased commercially (Cappel Laboratories, Malvern, Pennsylvania 19355, USA). Tissue sections were counterstained with 1% Evans blue for 30 sec prior to mounting the coverslip. Slides were then examined with an ultraviolet microscope. Known positive and negative controls were tested with each group of samples processed.

RESULTS

Viremia and antibody patterns

Mean viremia and antibody titers are shown in Figures 1 and 2. Viremias lasted for 2 days for all mice, for 3 days for 40% of the mice and for 4 days for 20% of the animals. Maximum mean viremia titer reached 1.9 $\log_{10}$ $SMICLD_{50}$/ml on day 2 PI. No viremia was detected on day 46 PI. Measurable CF antibody titers were present in all mice by day 8 PI, peaked at a mean of 1:160 (range: 1:60–1:500) on day 13–20 PI, and gradually declined to a mean titer of less than 1:60 when the study was terminated at day 63 PI. Three samples used to determine antibody response patterns and three samples taken during the transmission experiment, a total of only 6% (6/104) of the samples tested, were anticomplementary and were excluded from the data analysis.

Virus shedding

There was no evidence of viral shedding in urine, feces, or oral swabs. All of the

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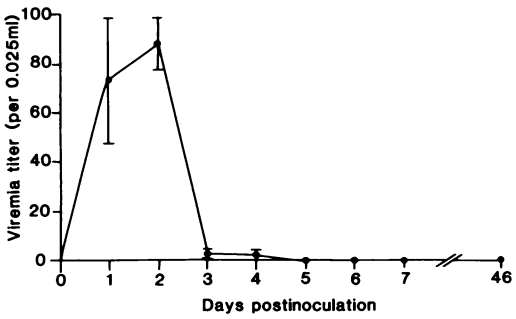

FIGURE 1. Mean (log_{10}) viremia titers per 0.025 ml of deer mice (*Peromyscus maniculatus*) inoculated intraperitoneally with Modoc virus (0.5 ml of $10^{7.6}$ $SMICLD_{50}$/ml). Vertical bars = ±1 standard error of the mean.

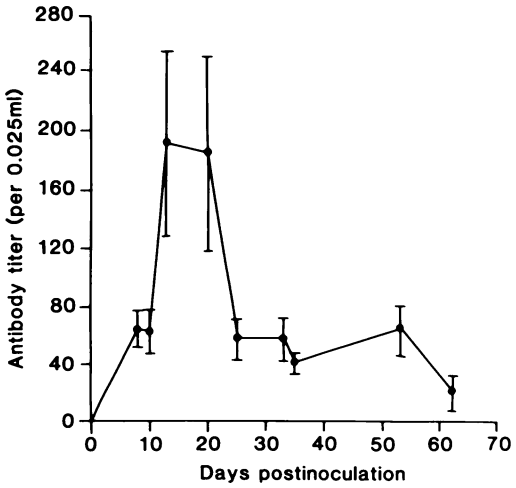

FIGURE 2. Mean antibody titers per 0.025 ml of deer mice (*Peromyscus maniculatus*) inoculated intraperitoneally with Modoc virus (0.5 ml of $10^{7.6}$ $SMICLD_{50}$/ml). Vertical bars = ±1 standard error of the mean.

animals had CF antibody titers of ≥1:80 when bled on day 50 PI, indicating that infection had occurred.

Tissue tropism

Virus was detected in all lung tissues explanted 65 days PI. No virus was found in liver, spleen, kidney, or salivary gland tissue explanted at the same time. Although the virus did not cause any cytopathic effect (e.g., cell death or malformation) in the mouse lung cell cultures, it did replicate, reaching maximum titers (3 log_{10} per 0.025 ml) 13 days postexplantation and maintaining approximately the same titer until 34 days postexplantation, when the experiment was terminated.

Virus was seen in the lung parenchyma by the IFA test in all mice killed from 42 to 70 days PI but was not seen in any of the mice killed prior to that time. It was present in the cytoplasm of connective tissue and was never seen in bronchiolar or alveolar cells. Virus was present in the cytoplasm of only a few cells widely scattered throughout the sections. Nonspecific background fluorescence was present in all sections, but was greatly reduced by adsorption of the specific antiserum with deer mouse liver powder and by counterstaining with Evans blue. Lung sections from control mice not inoculated with MOD virus and sections from MOD-positive mice stained without the addition of specific antiserum also exhibited nonspecific fluorescence but did not exhibit intracytoplasmic bright spots characteristic of virus infection. No pathologic changes were seen in any lung sections examined.

The IFA test was useful for determining which cells harbored virus but, due to the nonspecific background fluorescence, the test was difficult to read. The background staining probably could have been reduced further by using a direct FA test, instead of the indirect test. However, because of the widely scattered nature of the virus in the lung parenchyma, false negatives could still result due to random selection of tissue sections. Therefore, tissue explantation probably is a more sensitive test for routine diagnosis of viral persistence.

Virus transmission

All inoculated (known positive) mice had virus in their lungs as evidenced by the IFA test and a positive CF antibody titer (mean: 1:50, range: 1:20–1:80; two samples anticomplementary) when examined 84 days PI. Of the 16 uninoculated mice ex-

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amined 42 days after being housed with the inoculated mice, only one had virus in its lungs detectable by the IFA test. That mouse and seven others (50% of the test animals) developed CF antibody titers of 1:20 (six mice), 1:40 (one mouse) or 1:160 (one mouse). Serum from one additional mouse was anticomplementary.

DISCUSSION

This study has shown that a field strain of MOD virus establishes a persistent infection in deer mouse lungs and may be transmitted horizontally through close, prolonged contact of infected and susceptible individuals.

Viremia and antibody patterns induced by this strain of virus were similar to those caused by the prototype virus (Davis et al., 1974). Viremias occurred during days 1–4 PI and lasted 3 days in 40% of the mice and 4 days in 20% of the animals. CF antibody titers peaked about 1 wk earlier in this experiment (day 13–20 PI) than they did in experiments with the prototype virus (day 27 PI) (Davis et al., 1974). The subsequent decline in CF antibody titer observed here is consistent with previous observations of the prototype virus as well as many other antigenic agents (Wilfert, 1980). The presence of measurable antibody titers does not preclude the persistence of virus. This has been shown for other persistent viruses (Fraenkel-Conrat and Kimball, 1982) and has been demonstrated for MOD virus infection in hamsters (Yamada and Hardy, 1982). In the MOD virus system, two subclasses of IgG have been identified in the hamster; virus can persist in the presence of IgG_2 whereas IgG_1 appears to be neutralizing (Yamada and Hardy, 1982). The CF test does not distinguish between these two immunoglobulin subclasses.

Davis et al. (1974) demonstrated viral shedding in urine and from the oral cavity of deer mice within 10 days of inoculation with the prototype virus. In the present experiment, urine samples were assayed as early as 2 days PI (continuing at selected intervals until 50 days PI) but virus shedding never was observed. The MOD virus strain used by Davis et al. (1974) had undergone two passages in hamsters prior to use in their experiments. Previous studies (Davis and Hardy, 1974) have shown that MOD virus persists in hamster kidneys and is shed in their urine. It may be that passage of the virus through hamsters prior to inoculation in deer mice, altered its tissue tropism sufficiently to allow urine shedding. The present experiment used field strain MOD virus that had been passaged three times in brains of suckling white mice. Additionally, the positive throat swab results obtained by Davis et al. (1974) may have been due to their having inoculated the mice intranasally. In the present experiment, mice were inoculated intraperitoneally and there was no evidence of virus in oral swabs collected 37 days PI.

Davis et al. (1974) determined that MOD virus persisted for long periods of time in the lungs of inoculated animals. It is possible that virus persisted there because it was introduced in large amounts during the intranasal inoculation process. Also, they used young (4–8-wk-old) mice that may not have been fully immunocompetent and so were unable to successfully rid themselves of the virus. The present experiment used adult mice (>3 mo of age) and the virus was administered intraperitoneally. Virus persistence in the lungs still occurred. Virus was found both by explantation of lung tissue and by IFA examination.

The failure of Davis et al. (1974) to demonstrate direct horizontal transmission of the virus may have been due to too short of a time period allowed for transmission. Animals were put together only 28 days PI and left together for an additional 28 days prior to determining if transmission had occurred. The present experiments showed that virus does not reach the lungs until 42 days PI. Therefore, normal mice

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were not put with inoculated animals until 42 days PI when virus was known to be in the lungs of inoculated mice. To allow sufficient time for virus to reach the lungs of the normal, contact animals, mice were kept together for an additional 42 days prior to determining if transmission had occurred. Additionally, in this experiment the mice were crowded in an attempt to more closely mimic their natural huddling behavior (Johnson, 1970). All inoculated animals had MOD virus antibody titers and virus in their lungs when assayed 84 days PI, indicating that virus was present as assumed. Under this regimen, 50% of the contact animals developed antibody. However, only one of the contact mice had virus in its lung tissue. This might be an underestimate since the virus is difficult to detect due to background staining and low concentration. Additionally, the virus is so widely scattered in the lung tissue that the sections examined may have been incorrectly interpreted as negative. Therefore, it appears that virus can be transmitted horizontally but that this is not a very efficient system for maintenance of the virus within a population of mice. The deer mouse may not be the only host for this virus. MOD virus antibodies have been found also in the least chipmunk (*Tamias minimus*) and the red squirrel (*Tamiasciurus hudsonicus*) (Zarnke and Yuill, 1985). These species may be more efficient transmitters of the virus. Alternatively, the laboratory conditions under which these experiments were conducted may not have been conducive to virus transmission, whereas stressful conditions encountered in the field (such as cold temperatures or food shortage) may increase virus replication and shedding, hence promoting transmission.

In summary, field strain MOD virus produced a transient viremia in deer mice with production of measurable antibody titers and persistence of virus in the lungs. Unlike hamsters, deer mice did not maintain virus in their kidneys or shed it in their urine. Instead, the virus persisted in their lungs beginning 42 days PI and may have been transmitted by direct contact.

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BOOK REVIEW . . .

Parásitos y Enfermedades del Bagre, *Ictalurus* spp. (Parasites and Diseases of Catfish Species), F. Jimenez Guzman, L. Galaviz Silva, F. Segovia Salinas, H. Garza Fernandez, and P. Wesche Ebeling. Laboratorio de Parasitologia, Universidad Autónoma de Nuevo León, Aptd. 22, San Nicolás de la Garza, Nuevo León, Mexico. 1986. 320 pp.

This book, written in Spanish, consists of 18 chapters covering protozoa, trematodes, cestodes, nematodes, acanthocephalans, leeches, crustacea, molluscs (glochidia), parasitological techniques, a list of parasites of catfish, bacteria, viruses, nutritional diseases, culture of pathogens, and miscellaneous methods. There are clear illustrations designed to assist in the identification of parasites and the diagnosis of other catfish diseases.

With its 1985 companion volume, "Parásitos de la Lobima, *Micropterus* spp.," this book becomes one of a very few fish disease books written in Spanish for the growing profession of fish culture in Nuevo León and adjacent areas. The authors are from a parasitology laboratory which serves in the training of leaders in this developing resource.

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