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366 TRANSMISSION OF ENZOOTIC VESICULAR STOMATITIS VIRUS (NEW JERSEY) BY *SIMULIUM VITTATUM*. Cupp EW*, Mare CJ, Cupp MS, and Ramberg FB. Department of Veterinary Science, University of Arizona, Tucson, AZ.

We recently demonstrated that *Simulium vittatum* is a biological vector for an epizootic strain of vesicular stomatitis virus (New Jersey serotype - Camp Verde strain). Flies infected per os amplified and then secreted virus in their saliva 9-10 days postinfection. As part of our effort to develop a suitable model for the inter-epidemic maintenance of this group of viruses, we are currently evaluating the vector competency of *S. vittatum* for enzootic strains (as defined by nucleotide fingerprinting) of VS- NJ. We report here that this black fly can also serve as a laboratory vector of the Ossabaw Island strain which is maintained in nature by *Lutzomyia shannoni*. When infected per os (mean of 5,400 pfu/fly), 67% (10/15) of the flies amplified virus (mean of 8,900 pfu/fly by day 10); 60% (9/15) transmitted virus in their saliva at that time. An eclipse phase occurred approximately 24-48 hours after infection. These data suggest that black flies may be involved in circulation of enzootic VS-NJ strains that are ordinarily associated with phlebotomines. To further test the hypothesis that VS-NJ may operate in a two vector system, we are currently evaluating the vector competency of *S. vittatum* for two enzootic Mexican strains isolated from porcine and bovine hosts.

367 BIOLOGICAL TRANSMISSION OF LANGAT VIRUS BY THE SOFT TICK, *ORNITHODOROS SONRAI*. Durden LA and Turell MJ*. Institute of Arthropodology and Parasitology, Georgia Southern University, Statesboro, Georgia; and Disease Assessment Division, U. S. Army Medical Research Institute of Infectious Diseases, Fort Detrick, MD.

Studies were conducted to determine if the soft tick, *Ornithodoros sonrai* is a competent biological vector of Langat (LGT) virus (a member of the tick-borne encephalitis virus complex). Ticks were allowed to feed on suckling mice that were inoculated 4 days previously with LGT virus. Individual ticks, and brains of moribund or dead mice were tested for the presence of LGT virus by plaque assay on MK-2 cells. Virus was recovered from 67% (n=24) of the ticks sampled 4-17 days post infectious blood meal (PIB), and viral titers increased with increasing extrinsic incubation (mean titer of 8 infected ticks at 17 days PIB was $10^{3.0}$ plaque-forming units/tick, while at 50 days PIB, the mean titer of infected ticks was $>10^5$ plaque-forming units/tick). Transmission studies with individual ticks, conducted 35-70 days PIB, indicated that 39% (n=56) transmitted virus while feeding on mice. Virus was recovered from the brains of all dead or moribund suckling mice tested. These studies indicate that the soft tick, *O. sonrai*, is a competent laboratory vector of LGT virus. Soft ticks may play a role in the natural maintenance of tick-borne encephalitis viruses.

368 EXPERIMENTAL TRANSMISSION OF CRIMEAN-CONGO HEMORRHAGIC FEVER VIRUS FROM WEST-AFRICAN GROUND-FEEDING BIRDS TO *HYALOMMA MARGINATUM RUFIPES* TICKS. Zeller HG*, Cornet JP, and Camicas JL. Laboratoire des Arbovirus, Institut Pasteur, Dakar, Senegal; and Laboratoire ORSTOM de Zoologie Medicale, Dakar, Senegal.

In western Senegal, antibodies to Crimean-Congo Hemorrhagic Fever (CCHF) virus were recorded in some ground-feeding birds: Bucerotidae *Tockus (Lophoceros) erythrorhynchus* (Red-beaked Hornbill) and Sturnidae *Lamprotornis (Lamprocloius)* sp. (Glossy Starlings). These bird species are commonly infested by larvae and nymphs of *Hyalomma truncatum* or *Rhipicephalus guihoni* ticks. These ticks are potential vectors for CCHF virus in West-Africa. In the present study, an experimental model for understanding the role of wild birds in the CCHF virus cycle was developed. *Tockus erythrorhynchus* birds were infested by *Hyalomma marginatum rufipes* larvae and then inoculated with 500 LD_{50} CCHF virus by intraperitoneal route. Virus transmission to larvae/nymphs was obtained without detectable viremia in birds suggesting a non viremic transmission of the virus. Seroconversions were recorded. CCHF virus was detected in 90% of the tested nymphs unregardless the level of blood meal, as well as in adults. Titers were $10^{4.4}$ LD_{50} in nymphs and $10^{4.9}$ LD_{50} in

adults. From these results, the role of wild ground-feeding birds in CCHF virus ecology in West-Africa is discussed and revalued.

369 A MODEL SYSTEM FOR STUDYING THE ABILITY OF A TICK TO INGEST AN ARBOVIRUS WHILE FEEDING ON VIREMIC GUINEA PIGS. Linthicum KJ*, and Logan TM. Disease Assessment Division, U.S. Army Medical Research Institute of Infectious Diseases, Fort Detrick, MD.

Previous studies indicate that ticks, fed on a vertebrate host that has had multiple exposures to ticks, ingest significantly less blood than ticks fed on naive hosts. This may affect the amount of virus ingested from viremic hosts. A model system was developed for studying the ability of *Hyalomma truncatum* to ingest an arbovirus while feeding on guinea pigs previously exposed to ticks. Guinea pigs previously infested with either >2000 larval or ca. 200 nymphal *H. truncatum* ticks were inoculated with Venezuelan equine encephalitis (VEE) virus and allowed to develop high viremias [$>10^{7.0}$ plaque-forming units (PFU/ml of blood]. Concurrent with developing host viremias, larval, nymphal, and adult *H. truncatum* were infested on guinea pigs and were tested after drop off for the presence of VEE virus. In each life stage, there was no relationship between VEE virus titer ingested and either weight gain or hemoglobin uptake. Pools of larval ticks with undetectable or very low hemoglobin levels contained greater than $10^{3.0}$ PFU/tick of VEE virus at drop off. Individual nymphs and adults that ingested less than 0.30 mg of hemoglobin contained $10^{2.3}$ PFU of VEE virus/tick. Although the previous exposure of a host to ticks can have a significant effect on weight gain and hemoglobin uptake during subsequent tick infestations, in our model system, it had no effect on the ability of ticks to ingest virus.

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370 IDENTIFICATION OF *LEISHMANIA* PARASITES FROM DESERT STORM PARTICIPANTS AND A GENETIC COMPARISON WITH OTHER OLD WORLD ISOLATES. Kreutzer RD*, Magill A, Neva F, Fryauff DJ, Aleman-Munoz MM, and Grogl M. Biology Department, Youngstown State University, Youngstown, OH; Division of Experimental Therapeutic, Walter Reed Army Institute of Research, Washington, DC; Laboratory of Parasitic Diseases, NIAID, National Institutes of Health, Bethesda, MD; US Naval Medical Research Unit No. 3, Cairo, Egypt; and Medical School, University of Panama, Panama, Republic of Panama.

Leishmania parasites were isolated from U.S. military participants in Desert Storm. A partial list of the isolates we have cultured, identified by enzyme electrophoresis and cryopreserved includes 6 *L. tropica* and 4 *L. major*. A complete enzyme analysis was mad of these 10 as well as 16 *L. tropica* and 31 *L. major* isolates from Africa and the middle east. Most human hosts of the latter group had simple cutaneous leishmaniasis (SCL), but a majority of the Desert Storm patients presented with viscerotropic disease. Data will be included reporting clinical forms in all human hosts as well as enzyme data noting the *L. tropica* isolates are on average twice as polymorphic at the 10 and 20% levels than are the *L. major* isolates. There are only minor enzyme differences (2 enzymes) among the 4 *L. major* Desert Storm isolates (WR1070A, 1075A, 1077, 2036). Among the 6 *L. tropica* isolates there are 2 or 3 distinct genetic (enzyme) types (WR1063C and 1091 and 1092A - 1095 - 2029A and 2044). Thus far *L. tropica* and *L. major* are the two *Leishmania* parasites which have been identified from U.S. Desert Storm participants. As is the case with other *L. tropica* from the region a large amount of enzyme polymorphism has been observed among the *L. tropica* Desert Storm parasites. The *L. major* isolates are much less polymorphic.

371 URBAN OUTBREAK OF VISCERAL LEISHMANIASIS IN NORTHEASTERN BRAZIL: ASSESSMENT OF T CELL RESPONSES OF PATIENTS. Jeronimo SM*, Higgs E, Mackay S, Luz K, Morais R, Tomas E, Fernandes MK, Evans T, Petri WA, and Pearson RD. Universidade