RIFT VALLEY ANTIBODIES IN SERA OF EGYPTIANS

THESIS

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BY

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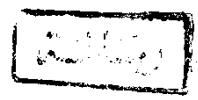

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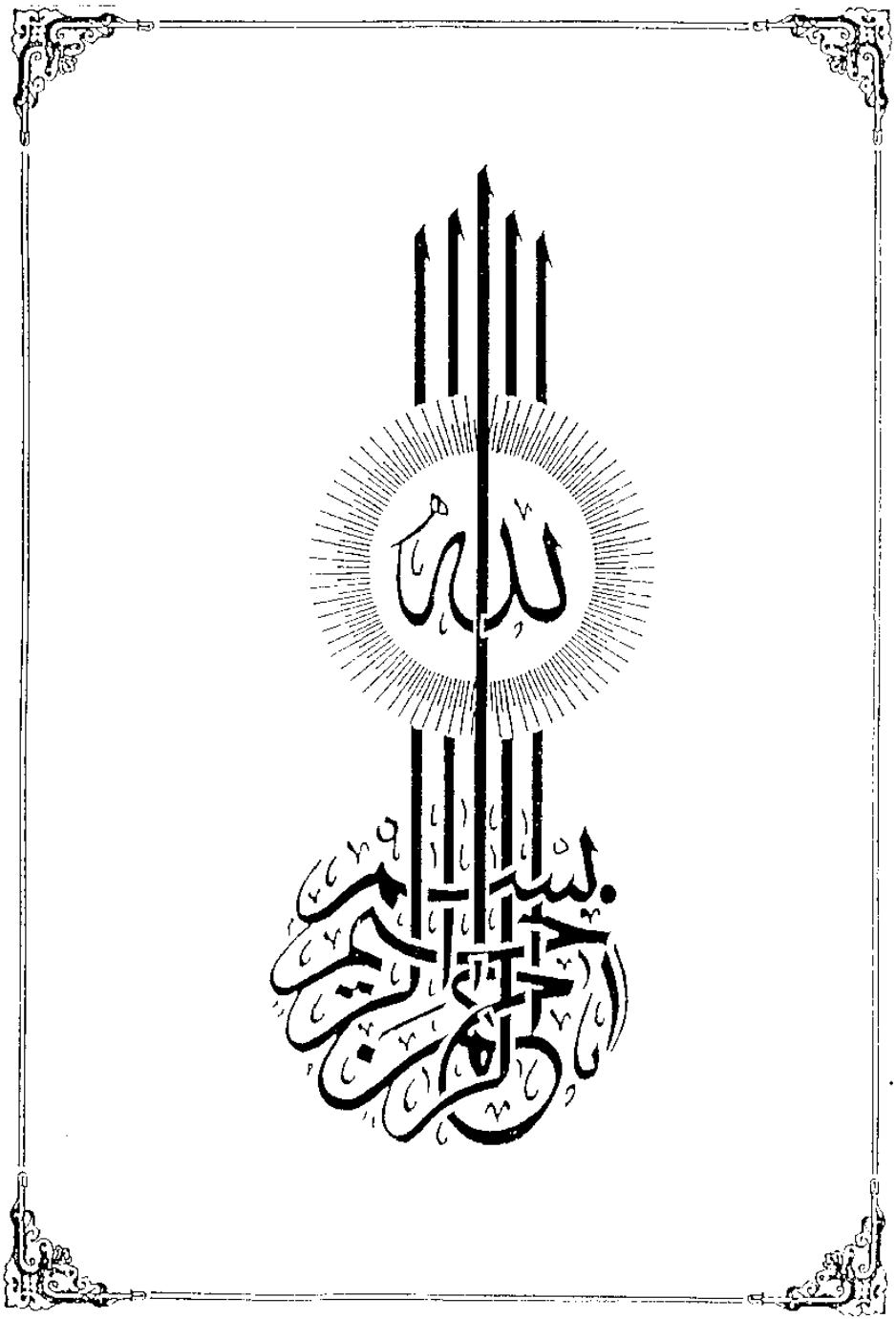

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INTRODUCTION

Rift Valley fever (R.V.F.) is a disease pathogenic for sheep and other domestic animals with an occasional extension to man. Its occurence in the Nile Valley and Delta in the late 1970s is only the most recent episode of the R.V.F. Saga. Recognition of the disease as an entity with high epizootic potential can be traced back to the early 1930s in Kenya, then to the South Africa in the early 1950s, followed by successive outbreaks of zoonotic disease in the 1960s and 1970s generated from persistent endemic foci, all limited to Sub-Saharan Africa (Swartz et al., 1981). Uptill now, there is no appearence of the disease outside Africa. It may mean that there are still unappreciated factors in establishment of the transmission cycles which give protective areas outside Africa (Morens, 1981).

Kenya has been very much involved in the history of Rift Valley fever. The classical first investigation was made by Daubney, et al.in 1931 near Naivasha area where the disease was occuring, which caused a high mortality in lambs. Those workers demonstrated that the disease was caused by a filterable virus which was

transmitted by a mosquito vector. A measure of control was achieved by moving Susceptible animal up to the top of the Rift valley wherever this wa possible. This meant an increase in altitude from 5500 to 8000 feet where it was found that transmission of the disease didn't occur. (Davis, 1981).

Rift valley fever was discovered in Egypt in early october,1977. The epidemic was first recognised in Sharkia governorate "Basatin El Esmailia".In retorspect, cases of the same Signs and Symptoms were found to occur as early as September,1977. The outbreak was explosive, this may be due to that the virus invaded a virgen area (El Akkad,1981). The epidemic at 1977 affected man and animals and affected all age groups, but males were more commonly affected than females. It was responsible for deaths, both by direct infection and spontaneous abortion , morbidity Suffering from such chronic disability as blindness, dementia and transmition or amplification of R.V.F. Another human out-break was recorded in 1978 and Sporadic cases were detected in 1979 and 1980, which meant that the virus was circulating in the country until 1980. (Darwish and Hoogstral,1981).

Daubney and his colleagues in 1931 continued their Studies on the fever in the field and laboratories. They arrived at certain couclusions based on the investigation of 1930 epizootic. first, the death in sheep was associated with extensive hepatic necrosis and the agent was an infectious agent. Since similar disease Could be transmitted from sheep to sheep, sheep to man and man to sheep by serum inoculation, and to sheep by inoculation of minced liver tissue extract from an ill sheep. Secondly, the agent was highly infectious in the laboratory since all four European (and presumably susceptible) persons working with it, developed the illness. This agent was a virus since specimens produced no growth on artificial media, but disease occured in sheep inoculated with serum from other ill sheep passed through chamberland filter up to and possibly beyond L II grade. (Davis, 1981).

Experimental studies were conducted to determine capability of mechanical transmission of Rift Valley fever Virus by Hematophagous Diptera. All species were tested (Glossina morsitans, Aedes Egypti, Aedes taeniorhynchus, culex pipiens, stomoxys calcitrans, Lutzomyia longipalpis and culicoides variipennis). The findings demonstrated that mechanical transmission of R.V.F. virus by hematophagous flies may

contribute to the natural transmission and dessimination of this virus. (Lynn Hoch, et al., 1985). Rift Valley Fever virus that yields potent antigens and antisera, so the serological diagnosis had and still have a Significant role in diagnosis of R.V.F. (Swanepoel, et al., 1983). Complement fixation test (C.F.T.) seems to be the most widely functional test in Egypt, other tests should be used for confirmation as haemagglutination test, heamagglutination inhibition test and immuno flourescent antibody technique (Casals, 1967)

It is important to state that R.V.F. virus is not related by C.F.T. to any of the bunya viruses or bunya virus like agent. So, it is nearly a unique virus among bunya virus in its lack of relationship to other viruses (Shope et al, 1978).

There are other serological tests as neutralization test and Enzyme linked immuno sorbent assay (ELISA). ELISA was used to measure the quantity of R.V.F. viral antigen in infected culex pipiens. It may be useful in detecting R.V.F. infected arthropods in the field it provides rapid, sensitive and specific test (Niklasson and Thomas 1985).

AIM OF THE WORK

The aim of the work is to study the serum antibodies against Rift valley fever virus in Egyptians by complement fixation test and Haemagglutination inhibtion test in an attempt to elucidate the degree of exposure to the virus.

REVIEW OF LITERATURE

Rift Valley Fever Virus Classification

Rift valley fever is caused by a virus which is classified as a member of the phlebotomus fever serogroup of the family Baunya viridae (Bischope and shope, 1980). They described the characters of the family Baunya viridae and the characters of R.V.F. virus as follows :

a) It is spherical RNA virus, 90-100 nm in diameter.
   The diameter of R.V.F virus was estimated to be 60 - 75 nm by Levitte et al. (1963).

b) It is an enveloped virus, two virus specific glyco proteins designated G1 and G2 located on the outer surface of the virus particle. Rice et al. (1980) and Each et al. (1981) revealed the presence of three major proteins in R.V.F virus, anonglycosylated and two glyco proteins with molecular weight 25, 26 and 65×10^3 respectively.

c) Virus replication occurs in the cytoplasm of infected cells and virus particles are formed by buding into the Golgi cisternae. Mc Gavran and Easterday (1963) studying the R.V.F virus

in mouse livers found that new viruses are formed in the cytoplasm within a membrane limited system resembling Golgi apparatus.

d) Virions are liberated from infected cells by fusion of the intra cellular vacules with cellular plasma membrane or by cell membrane disruption and discharge of cell contents (Easterday and Murphy, 1963).

R.V.F Virus Dissemination and Transmission

R.V.F virus may be disseminated from infected to susceptible vertebrate by arthropod vector or by inhalation or contamination (Hoogstraal et al., 1979). There is no way to determine from previous report on R.V.F. the relative epidemiological importance of each of these R.V.F virus transmission. Dissemination routes in an enzootic or an epizootic situation has never been precisely assesed. The role of each disseminating route has propably differed in various R.V.F outbreaks in Africa and possibly even in Nile valley and Delta areas where domestic animal population and hematophagous arthropod densities differ.
However Culex Pipiens is a likely and experimentally efficient vector of the virus in Egypt.

1- Arthropod vector.

In the study by Khalil and NAMRU-3 team in 1981, they found that the culex pipiens population densities were extremely high in the southeastern belt of the Delta. During the fall of 1977 when R.V.F was first recognised in Egypt, the major drainage canal traversing this area and the abundant pools, puddles, ditches and canals characteristic of the rice fields, gardens and groves were swarmed with larva. The culex pipiens species constituted over 90% of the collections of adult mosquitoes mostly aspirated from the walls of buildings in and near which humans and animals infected by R.V.F virus lived. The collections were chiefly from Sharkia and Qalyubiya, also from Giza (Meegan et al., 1980).

The decline at the onset of cooler winter weather was probably hastened by the governmental emergency aerial insecticiding of R.V.F foci. This program was definitely effective in reducing mosquito numbers in villages and farms. Before and after spraying during the autumn season of intense breeding octivity the fresh water and sewage canals running from Cairo area to the east near the southern margin of the Nile Delta

and the irrigated fields near these canals, are well known mosquito breeding habitats. All persons professionally concerned with the R.V.F epidemic, have agreed that during the fall season of 1977 mosquito (Culex pipiens) population densities in the southeastern Nile Delta were even higher than usual. There have been no significant long term studies of culicine population dynamics in this area on the basis of observations on mosquito population densities and data on ecological and geographical distribution of mosquitoes (khalil, 1981).

In relation to R.V.F morbidity in humans and domestic animals, and seasonal dynamics, C. pipiens is the chief candidate for consideration as the R.V.F virus in Egypt (Hoogstraal et al., 1979).No other mosquito species is consistently present or numerous in the chief R.V.F foci that were investigated.In the spring of 1980, C. pipiens larvae were present in 26 of 40 (65%) water Samples taken in 35 localities in cultivated areas of Aswan and Qena Governorates (khalil , 1981). Results of NAMRU-3 studies of the blood meals of numerous C.pipiens samples from R.V.F foci in the Nile Delta and upper Egypt showed human and domestic