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WESSELSBRON VIRUS INFECTION IN WEST AFRICAN DWARF GOATS (*FOUTA DJALLON*): VIROLOGICAL AND IMMUNOLOGICAL STUDIES

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Summary. — West African dwarf goats were experimentally infected with Nigerian strain of Wesselsbron virus. Viraemia was detected in infected goats 2 days after infection and lasted for one day. A 100 % mortality was observed among the infected animals; the virus was reisolated in mice from almost every tissue obtained from the bodies of infected goats. In addition, the infected goats developed complement-fixing and haemagglutination inhibiting antibodies to Wesselsbron virus.

Key words: *Wesselsbron virus*; *flaviviridae*; *West African dwarf goats* (*Fouta Djallon*)

Introduction

Wesselsbron virus belongs to the family *Flaviviridae*. It has been responsible for severe disease outbreaks in sheep in South Africa (Weiss et *al.*, 1956). The virus has been isolated from man (Weiss *et al.*, 1956; Smithburn *et al.*, 1957; Heyman *et al.*, 1958; Justines and Shope, 1969; Tomori *et al.*, 1981), camel (Kemp *et al.*, 1973) and mosquitoes (Kokernot *et al.*, 1960). Clinical infection in sheep and goats is characterized by fever and severe leukopaenia, abortion in pregnant ewes and high mortality in lambs and kids (Weiss *et al.*, 1956; Weiss, 1957; Coetzer *et al.*, 1978; Coetzer *et al.*, 1979; Theodoridis and Coetzer, 1980; Fagbami, 1980).

Wesselsbron virus was first isolated from a camel during a routine virus surveillance in Northern Nigeria in 1968 (Kemp *et al.*, 1973). Although immunity surveys have shown that antibodies to Wesselsbron virus and other flaviviruses are prevalent in domestic animal sera (Fagbami, A. H. unpublished data), no clinical episode of Wesselsbron disease has been described in Nigeria. In the present study, West African dwarf goats were infected with Wesselsbron virus in order to determine the virological and immunological response of this breed of goats to Wesselsbron virus and to determine the pattern of serological cross reactions with other flaviviruses.

Material and Methods

Virus. The Nigerian strain Wesselsbron virus (Ib-AN 31956) isolated from the blood of camel was used after 5 suckling mouse brain passages. Ten per cent infected suckling mouse brain suspension was prepared in bovine albumin phosphate buffered solution (BAPS) containing 2000 I.U./ml of penicillin and 600 mg/ml of streptomycin; after centrifugation at 10,000 rev/min it served as stock virus. Titre of stock virus was 5 $\log_{10}$ LD_{50}/0.02 ml. Other flaviviruses used in serological tests are shown in Table 1.

Experimental procedure. Four West African dwarf goats (Nos. 1—4) (4—5 months old) were infected subcutaneously (s.c.) with 2.5×10^{-5} suckling mouse intracerebral (i.c.) LD_{50}. The control animal (No. 5) received 0.05 ml of 10% normal suckling mouse brain in minimum essential medium (MEM). Blood samples were obtained daily for 7 days for viraemia studies. Tissue' from the bodies of Wesselsbron virus infected animals following death were assayed for virus presence.

Sera were obtained on days 0, 8, 15 and 22 post inoculation (p.i.) and tested by complement-fixation (CF) and haemagglutination inhibition (HI) for antibodies against Wesselsbron and other flavivirus antigens.

Viraemia as well as presence of virus in tissues were determined by (i.c.) inoculation of whole blood and 10 % tissue suspension in MEM into mice, samples which yielded virus were further titrated for endpoints, calculated by the method of Reed and Muench (1938) and expressed in $\log_{10}$ LD_{50}.

Immunological studies. A microtitre complement fixation tests was used (Weinbren, 1958). HI tests were performed on kaolin treated sera according to the methods of Clarke and Casals (1958) adapted to microtitre plates. Wesselsbron (WSL), Yellow fever (YF), Potiskum (POT) and Uganda S. (UGS) virus antigens were prepared by sucrose acetone extraction on infected suckling mouse brains as described by Clarke and Casals (1958).

Results

All four infected goats developed clinical disease characterized by fever, anorexia, diarrhoea, dehydration, weakness, recumbency and death. Virus was detected in the blood of Wesselsbron infected 48 hr p.i. and lasted for 24 hr (Table 2).

Tissues from the bodies of all infected animals which died from Wesselsbron virus infection were assayed for presence of the virus. Virus was present in liver, spleen, adrenal glands, brain, lungs, kidney and in messenteric lymph

Table 1. Viruses used in complement fixation and haemagglutination inhibition tests

Virus	Source	Passage level	Complement-fixing titre	Haemagglutination inhibition titre
Wesselsbron Ib-AN 31956	Camel	5	16	128
Yellow fever VIR 114978	Man	5	16	64
Potiskum Ib-AN 10069	African giant rat (*Gricetomys gambianus*)	15	8	64
Uganda S. AN 8829	Sentinel mouse	5	32	512

Table 2. Viraemia pattern in West African dwarf goats infected with Wesselsbron virus

Animal No.	Days p.i. 1	2	3	4
1	—	3.7*	3.2	—
2	—	2.7	3.2	—
3	—	2.0	3.0	—
4	—	1.5	2.0	—

— = No viraemia detected
* in $\log_{10}$ LD_{50}/0.02 ml

nodes. The virus was detected in almost every tissue assayed from goats that died on days 22 and 24 p.i. (Table 3).

The antibody responses of goats infected with Wesselsbron virus are shown in Tables 4 and 5. All inoculated animals developed CF and HI antibodies to WSL and other flavivirus antigens tested. The highest CF antibody titre of 32 was found in sera obtained on days 15 and 22 p.i. Two of the infected animals developed HI antibody to Wesselsbron virus and other flavivirus antigens used in the tests. HI antibody to Wesselsbron virus was at least four fold higher than to other flavivirus antigens. The highest level of HI antibody (160) was detected in sera obtained on day 8 p.i.

Table 3. Wesselsbron virus concentration in tissues in $\log_{10}$ (SMIC) LD_{50}/0.02 ml

Death (days p.i.)	Goat 1	Goat 2	Goat 3	Goat 4
	5	22	8	24
Blood				
Liver	1.0	1.5	1.0	1.5
Spleen	—	1.0	—	1.0
Adrenal gland	1.0	1.5	1.0	1.5
Lungs	—	1.0	—	1.0
Brain	—	1.5	1.0	1.0
Kidney	—	1.0	—	—
Messenteric lymph nodes	—	1.5	1.5	1.5

— = No viraemia detected
SMIC = suckling mouse intracerebral

Table 4. Complement-fixing antibody titres of sera collected from WSL virus infected goats

Animal No.	Day of sampling	Antigen			
		WSL virus	POT virus	UGS virus	YF virus
1	0	—	—	—	—
	0	—	—	—	—
2	8 p.i.	—	—	—	—
	15 p.i.	16	8	16	16
3	0	—	—	—	—
	0	4	4	4	4
	8 p.i.	8	8	8	8
4	15 p.i.	32	32	16	16
	22 p.i.	32	32	16	32

— = No complement-fixing antibody detected

Discussion

The present experimental transmission of Wesselsbron virus to dwarf goats showed that although clinical disease has not been previously described in Nigeria, the virus is pathogenic for West African dwarf goat. The short period of viraemia which ceased several days before fever subsided was in agreement with the findings of Theodoridis and Coetzer (1980); Fagbami (1980). Fagbami (1980) attributed the low level of viraemia to previous passages of the virus in suckling mouse brain. The maximum virus titre in the blood of infected goat was 3.7 $\log_{10}$ LD_{50}/0.02 ml with a mean value of

Table 5. Haemagglutination-inhibition test on sera collected from WSL virus-infected goats

Animal No.	Day of sampling	Antigen			
		WSL virus	POT virus	UGS virus	YF virus
1	0	—	—	—	—
	0	—	—	—	—
2	8 p.i.	160	10	10	10
	15 p.i.	80	10	20	20
3	0	—	—	—	—
	0	—	—	—	—
	8 p.i.	160	10	20	20
4	15 p.i.	80	10	20	20
	22 p.i.	80	10	20	20

— = No HI antibody detected

2.8 $\log_{10}$/0.02 ml. This was found to be pathogenic for the age group of infected goats used in the experiment. The detection of the virus in various organs of the bodies of infected goats was also consistent with the findings of Theodoridis and Coetzer (1980) and demonstration of a relatively higher virus titres in the liver, adrenal glands and brain as compared to other infected organs were also in agreement with the findings of Theodoridis and Coetzer (1980).

Detection of CF and HI antibodies characteristic of natural and experimental infections in sheep (Weiss *et al.*, 1956; Smithburn *et al.*, 1957; Fagbami, 1980) and in goats (Theodoridis and Coetzer, 1980) were also observed. The higher titres of CF and HI antibodies in the animals that died later in the experiment probably contributed to their survival for a longer period when compared to those that died in the early phase of infection. HI antibody to Wesselsbron virus antigen was at least four-fold higher than to other flaviviruses used in the experiment. This was similar to that reported by Fagbami (1980). Antibodies were detected between day 8 and day 22 p.i. with the highest titre 32 on day 22 p.i. for CFT and 160 on day 8 p.i. for HI test.

Clinical cases of Wesselsbron virus infections in domestic animals have not been documented in Nigeria. However, there is serological evidence that such infections are not uncommon in the country. The lack of further isolation of the virus and the absence of severe natural disease in domestic animals may be due to the low level of virus surveillance in sheep, goats and other domestic animals. It is possible that the high prevalence of flavivirus antibodies in the West African dwarf goats might limit the activity of Wesselsbron virus in Nigeria.

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