

Seroepidemiological survey on Rift Valley fever among small ruminants and their close human contacts in Makkah, Saudi Arabia, in 2011

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Summary

This study describes a seroepidemiological survey on Rift Valley fever (RVF) among small ruminants and their close human contacts in Makkah, Saudi Arabia. A total of 500 small ruminants (126 local, 374 imported) were randomly selected from the sacrifice livestock yards of Al-Kaakiah slaughterhouse, in the holy city of Makkah, during the

pilgrimage season 1432 H (4–9 November 2011). In addition, blood samples were collected from 100 local workers in close contact with the animals at the slaughterhouse. An RVF competition multi-species enzyme-linked immunosorbent assay (ELISA) detecting anti-RVF virus immunoglobulin G (IgG)/immunoglobulin M (IgM) antibodies and an RVF IgM-specific ELISA were used for serological investigations. In total, 84 (16.8%) of the 500 sacrificial sheep and goats tested seropositive in the competition ELISA but no IgM antibodies were detected in the IgM-specific assay. All seropositive samples, comprising 17.91% of the imported animals and 13.49% of the local ones, were therefore designated positive for anti-RVF virus IgG antibody. Among the local personnel working in close contact with the animals, 9% tested seropositive in the RVF competition ELISA. The study indicates that two factors may increase the likelihood of an RVF outbreak among sacrificial animals and pilgrims: *i*) the large-scale importation of small ruminants into Saudi Arabia from the Horn of Africa shortly before the pilgrimage season, and *ii*) the movement of animals within Saudi Arabia, from the RVF-endemic south-western area (Jizan region) to the Makkah region, particularly in the few weeks before the pilgrimage season. From these findings, it is recommended that *i*) all regulations concerning the import of animals into Saudi Arabia from Africa should be rigorously applied, particularly the RVF vaccination of all ruminants destined for export at least two weeks before exportation, and *ii*) the movement of animals from the RVF-endemic south-western area (Jizan region) of Saudi Arabia to the Makkah region should be strictly prohibited.

Keywords

Epidemiology – Makkah – Pilgrimage – Rift Valley fever – Saudi Arabia – Serological survey – Small ruminant.

Introduction

Rift Valley fever (RVF) is a severe mosquito-borne viral disease affecting humans and domestic ruminants and caused by a *Phlebovirus* (*Bunyaviridae*) (1). The fever was first described in 1930 in the Rift Valley of Kenya (2) and is assumed to have spread from

there to other regions of Africa (3). Among animals it mainly affects ruminants, causing abortions in gravid females and mortality among their young. In humans the infection is usually asymptomatic or is characterised by a moderate fever; however, in 1% to 3% of cases more severe forms of the disease (hepatitis, encephalitis, retinitis, haemorrhagic fever) can lead to major sequelae or death (4).

The first confirmed RVF outbreak outside Africa was reported in 2000 in Yemen and Saudi Arabia in the Arabian Peninsula (5, 6). In September 2000, the Centers for Disease Control and Prevention confirmed a diagnosis of RVF in all of four serum samples submitted from Saudi Arabia. The diagnostic tests included polymerase chain reaction, virus isolation, immunohistochemistry and an enzyme-linked immunosorbent assay (ELISA) for the detection of antigen and immunoglobulin M (IgM) antibody (7). Patients presented with a febrile haemorrhagic syndrome accompanied by liver and renal dysfunction. By the end of the outbreak, April 2001 statistics from the Saudi Ministry of Health documented a total of 882 confirmed cases, with 124 deaths. However, both the severity of the disease and the relatively high death rate of 14% might be a consequence of underreporting the less severe cases (8).

Before the outbreak in Saudi Arabia, the potential for RVF to spread out of its usually recognised endemic area had already been exemplified by an epidemic in Egypt in 1977; Table I shows reported epizootics of RVF virus (RVFV) in numerous countries in Africa and the Middle East (2, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39). Outbreaks of RVF are associated with persistent heavy rainfall with sustained flooding and the appearance of large numbers of mosquitoes, which are the main vector. Localised heavy rainfall is seldom sufficient to create conditions for an outbreak; the simultaneous emergence of large numbers of first-generation transovarially infected mosquitoes or the introduction of a viraemic animal is also required. After amplification of the virus in vertebrates, mosquitoes act as secondary vectors to sustain the epidemic (40). The annual emergence of RVFV has been reported in Zambia during or

after the seasonal rains, and an increase in the number of seroconverted animals has been associated with vegetational changes (41). The detection of mosquitoes of the genus *Aedes*, which are competent vectors of the virus, during inter-epizootic periods in Kenya, suggests that a cryptic vector-to-vertebrate cycle of the virus may exist during these periods (42).

In animals, RVF may be inapparent in non-pregnant adults but outbreaks are characterised by the onset of abortions and high neonatal mortality. Jaundice, hepatitis and death are seen in older animals. In humans, RVF may present as a haemorrhagic disease with liver involvement; there may also be ocular and neurological lesions (40). The haemorrhagic form was the most frequent presentation of RVF during the 2008 outbreak in Madagascar and was responsible for a high level of mortality in humans (30).

Direct contact with animals, particularly contact with the body fluids of sheep, has been identified as the most important modifiable risk factor for infection with RVFV; thus, public education during epizootics may reduce human illness and death in future outbreaks (10). The use of multivariable logistic models in a post-epidemic analysis of RVFV transmission in north-eastern Kenya indicated that age, residence in a village (as opposed to a town) and drinking raw milk were significantly associated with RVFV seropositivity. Visual impairment was much more likely in the RVFV-seropositive group (43).

Laboratory diagnosis of RVF is based on histopathology or the demonstration of viral antigen or antibody (44). Clinical diagnosis of the fever must be confirmed by serological tests, detection of RNA and/or isolation of the virus (3).

In a serological survey (45) of RVFV among 580 sacrificial animals in the holy city of Makkah (120 local and 460 imported animals randomly selected from sacrificial herds at Al-Kaakiah slaughterhouses), during the pilgrimage season of 1430 H (25–30 November 2009), an overall recent exposure rate of 2.59% (0.83% among local animals, 3.04% among imported ones) was detected,

demonstrated by IgM-positive sera. Among immunised animals, the overall herd rate based on IgG seropositivity was 47.06% (55% among local animals, 45% among imported ones). Animals that test seropositive for IgM may increase the potential risk of a further outbreak among sacrificial animals during the pilgrimage season, with all the subsequent socio-economic and public health consequences. The importance of an effective and controlled vaccination programme for local animals and verification of the immune status of imported herds was emphasised (45).

During the past three decades, and particularly in recent years, RVFV has become an important subject of interest as public health agencies have become alerted to the possible emergence of this arbovirus in temperate countries. Aspects of the epidemiology of the virus are still not fully understood, and safe effective vaccines are still not freely available for protecting humans and livestock against the dramatic consequences of this infection (46). In Saudi Arabia, some millions of small ruminants, mostly imported from the Horn of Africa, where RVF is endemic, are sacrificed annually during the pilgrimage season. During traditional sacrificial slaughtering, aerosols of infected blood may be generated, increasing the risk of infection among abattoir workers and butchers. Moreover, the increase in populations of mosquitoes, the main RVFV vector, among the overcrowded pilgrims is another potential risk factor for human infection (47).

In the present study, a seroepidemiological survey on RVFV was carried out among small ruminants and workers in close contact with the animals during the pilgrimage season of 1432 H (4–9 November 2011) in order to detect any potential risk of the eruption of new RVF outbreaks among the animals and pilgrims.

Materials and methods

Sample population

A total of 500 sacrificial animals were randomly selected from the livestock yards of Al-Kaakiah slaughterhouse, the main slaughterhouse in Makkah, during pilgrimage season 1432 H (4–9

November 2011). The investigated animals included 126 local and 374 imported animals. The local animals comprised 51 sheep (Nejdi) and 27 goats (Heijazi) from Makkah and 23 sheep (Nejdi) and 25 goats (Heijazi) from Jizan; the imported animals comprised 132 sheep from Sudan (Red Moosai, Tanganyika), 136 Somali sheep (Berbera black head) and 106 Somali goats (Deg hier, Deg yer) (Table II). Blood samples were collected from the jugular veins of the animals and sera were separated the same day. In addition, blood samples were collected from 100 local workers at the livestock yards of the slaughterhouse (south of Makkah). All serum samples were stored at -80°C until serological testing.

Serological surveillance of Rift Valley fever virus among sacrificial animals and their human contacts

Serum samples from the sacrificial animals and their human contacts were evaluated for total anti-RVFPV IgM/IgG antibodies using ID screen® RVF competition multi-species ELISA kits (ID-Vet Innovative Diagnostics, Montpellier, France). In a second step, seropositive samples were tested for specific anti-RVFPV IgM antibodies using ID screen® RVF IgM ELISA kits (ID-Vet Innovative Diagnostics, as above). All samples that tested positive for total RVFPV antibodies but negative for IgM were assumed to be positive for anti-RVFPV IgG antibodies. Serological assays were carried out at the diagnostic veterinary laboratory at Jizan port; the manufacturer's instructions were followed.

Results

Seroprevalence of Rift Valley fever virus among contact workers

Among the serum samples of 100 local personnel working in close contact with the sacrificial animals, nine (9%) were positive in the RVFPV competition ELISA.

Seroprevalence of Rift Valley fever virus among sacrificial animals

In total, 84 (16.8%) sera among 500 samples from sacrificial animals tested positive in the RVFV competition ELISA. No IgM antibodies were detected and therefore all the positives were considered as anti-RVFV IgG antibody positive (Table III). The 84 seropositive animals comprised 17 (13.49%) of 126 Saudi Arabian animals and 67 (17.91%) of 374 animals imported from Africa.

The 17 seropositive cases among the Saudi Arabian animals comprised 7 (8.97%) of 78 animals from Makkah and 10 (20.83%) of 48 animals from Jizan (Table IV). The 67 imported seropositive cases comprised 28 (21.21%) of 132 animals from Sudan and 39 (16.11%) of 242 from Somalia (Table V).

Discussion

Rift Valley fever is an acute viral disease that affects humans and ruminant animals (48) and is considered one of the most important viral zoonoses in Africa (49). The epidemic of RVF in south-western Saudi Arabia in 2000 coincided with simultaneous occurrences of the disease in Yemen. These epidemics were the first documented evidence of RVFV transmission outside Africa (5, 6, 39), and since that date RVFV has been considered an important animal and human pathogen in Saudi Arabia and Yemen.

In the present study, RVFV seropositivity was detected among small ruminants and personnel in close contact with the animals in the western region (Makkah Al-Mukarmah) of Saudi Arabia during the pilgrimage season of 1432 H (4–9 November 2011). It is speculated that the virus was introduced into the Arabian Peninsula in 1997–1998 via infected imported livestock or windborne infected mosquitoes during the RVF epidemic in East Africa, and that climatic conditions have supported sufficient vector populations to enable transmission of the virus in Saudi Arabia and Yemen since then.

Two lines of evidence support this speculation. First, the epidemic in 2000 began spontaneously in geographically diverse areas in Saudi Arabia and Yemen, suggesting that dissemination of the virus probably occurred before the epidemic period (6). Second, the genetic sequence of the virus isolated in Saudi Arabia and Yemen is closely related to that of the virus isolated in the 1997–1998 outbreaks in East Africa (50). This situation is similar to the introduction of RVFV from Sudan into Egypt, where an extensive epizootic of RVF was reported for the first time in 1977 and 1978 and caused high morbidity and mortality in domestic ruminants (21, 22, 23). Before this first outbreak in Egypt, RVF had been reported in 20 countries in sub-Saharan Africa (51). Sudan was the most likely origin of the Egyptian outbreak and the most probable hypotheses were introduction of the virus by infected animals, especially camels, or by insects carried on the wind, or a combination of both (52). Another hypothesis suggested an introduction via sheep transported along Lake Nasser (53). The reintroduction of RVF or other diseases from Sudan into Egypt or from Yemen into Saudi Arabia could be facilitated by roads linking Sudan and Egypt, and Yemen and Saudi Arabia, and by the absence of sufficiently strict quarantine measures at international borders.

The annual importation of some millions of ruminant animals into Saudi Arabia from the Horn of Africa, where RVF is endemic, may further facilitate the recurrence of outbreaks, not only in the southern region (Jizan) but also in other regions of the country. However, epidemics of RVF in Saudi Arabia have occurred principally in the Tehama zones (54), which are the floor of the Rift Valley in the west of both Yemen and Saudi Arabia, and are close to the Red Sea. The greatest RVFV activity was associated with alluvial fans of soil brought down from highland zones by river systems close to the eastern wall of the Rift Valley. The onset of cases in Yemen and Saudi Arabia was simultaneous, indicating that the emergence of RVFV-infected mosquitoes was multi-focal, driven by common climatic changes. Retrospective studies in both countries have confirmed that the virus had been present in these areas before the clinical outbreaks occurred.

Historically, explosive outbreaks of the disease have occurred simultaneously over a wide area of Africa at intervals of between five and 15 years, generally after periods of heavy rainfall in otherwise dry areas, the long interval between outbreaks in animals allowing for the development of susceptible populations. For many years the reservoir during the inter-epidemic periods was unknown, then the virus was detected in dormant eggs of the mosquito *Aedes lineatopinnis* in the soil of grassland depressions known as dambos. When these depressions fill with rainwater, the eggs hatch and infected mosquitoes develop. The mosquitoes infect an amplifying host (ruminant), which serves as a source of infection for many other genera of mosquito that can rapidly spread the disease (41). In Kenya, the virus was isolated from mosquitoes collected at or near naturally or artificially flooded grassland depressions serving as developmental sites for the immature stages of many mosquito species. Transovarial transmission of the virus was also suggested (55).

Before the present study, RVF was considered endemic only in the Tehama zones (south-western Jizan and Asir regions) of Saudi Arabia. In Jizan, the mass vaccination of ruminant animals, primarily sheep and goats, with the live attenuated RVFV Smithburn vaccine strain immediately after the outbreak in 2000 resulted in remarkable control of the disease in this region, manifested by the absence of serological evidence of recent RVFV infection in 2003 (56). However, a later serological survey in 2006 reported the persistence of RVFV in Jizan (57).

During the present study, no clinical signs of the disease were noticed in animals or in human beings. In Madagascar in 2008 it was suggested that there had been country-wide circulation of RVFV, including parts of the country where no clinical illness in animals or in humans had been reported. The virus may spread when ecological conditions favour its amplification (29) and most outbreaks occur in remote locations after floods. To determine the environmental risk factors and long-term sequelae of human RVF, the rates of previous RVFV exposure by age and location were examined during an inter-epidemic period in north-eastern Kenya in 2006. Over all, the RVFV

IgG ELISA seropositivity rate was 13% and evidence of inter-epidemic RVFV transmission was detected. Increased seropositivity was found among older people, those who were male, those who lived in a rural village as opposed to a town, and those who had disposed of animal abortus. Greater exposure to mosquitoes and animals were reported in rural villages. Seropositive people were more likely to have visual impairment and retinal lesions; however, other physical findings did not differ (58).

An indirect ELISA was developed by Paweska *et al.* for detection of antibody against RVFV in domestic and wild ruminant sera (59). In the present study, serum samples from investigated sacrificial small ruminants and their human contacts were evaluated for total RVF antibodies (IgM/IgG) using RVF competition multi-species ELISA kits. Samples that tested positive were then retested for specific anti-RVF IgM antibody. Paweska *et al.* (60) have also described the development and validation of an inhibition ELISA based on gamma-irradiated antigen derived from tissue culture for the detection of antibody against RVFV in humans and domestic and wild ruminants (60). It was demonstrated that this ELISA can be used as a safe, robust and highly accurate diagnostic tool in disease surveillance and control programmes, in import/export veterinary certification and for monitoring immune responses in vaccinees. In a more recent study, an indirect ELISA based on recombinant nucleocapsid protein of RVFV was validated by Paweska *et al.* (61) for the detection of specific IgG antibodies in sera of African buffalo; the diagnostic sensitivity of the assay was 98.7% and diagnostic specificity 99.36% (61).

In the present study, 84 (16.8%) of 500 sacrificial animals tested seropositive for anti-RVFV antibodies and, in the absence of IgM antibodies, all these samples were presumed to be positive for IgG antibody. In the Senegal River Basin, however, antibody prevalence in some areas was as high as 85%, with approximately 80% of the sera testing positive for both anti-RVFV IgG and viral-specific IgM antibodies among animals from areas with active transmission of the virus (62). It is known that IgM persists for about two to three months after infection (46), thus the absence of anti-RVFV IgM antibodies in

all the serum samples in the present study indicated that there was no recent infection in the seropositive animals or in the workers in close contact with them and no active transmission of the virus at the study site.

Anti-RVfV IgG antibody was detected in 13.49% of local animals and 17.91% of imported animals. Among the seropositive animals from the local breeds, 9% were from Makkah and 20.83% from Jizan. However, seropositivity in animals from Jizan might not necessarily indicate previous infection and could be due to animal vaccination, as a programme against RVF has been implemented there since the outbreak in 2000. However, if the currently detected IgG antibodies were due to vaccination, this would suggest that the vaccination process is still only partial. Among the imported animals, only 17.91% tested IgG positive, thus a large portion are not properly vaccinated and are at high risk for RVFV viraemia. Seropositivity in animals from Makkah usually indicates previous exposure to the virus. This is supported by findings in an earlier serological survey of RVF during the pilgrimage season of 1430 H (25–30 November 2009), in which a recent exposure rate of 2.59%, manifested by IgM-positive sera, was detected among the investigated sacrificial animals (45).

The appearance of RVF outside the African continent might be related to the export of infected animals from Africa (8). Among animals imported into Saudi Arabia from Sudan, 21.21% tested positive for anti-RVfV IgG antibodies; among those from Somalia, 6.11% were seropositive. The vaccination histories of these animals were unavailable but all imported ruminants should be vaccinated in the exporting countries before shipping, in accordance with the regulations of the Saudi Ministry of Agriculture. The findings of the present study indicate that the rules covering importation and quarantine of live animals are not being followed effectively.

Commercial ELISA kits that can distinguish naturally infected animals from those that have been vaccinated are urgently required. A recently developed simple ELISA has been described as distinguishing naturally infected animals from those vaccinated with

an attenuated virus. The assay has potential for use in both human and animal populations (63).

In the present study, 9% of serum samples from local personnel working in close contact with sacrificial animals at Al-Kaakiah slaughterhouse yards tested seropositive in an RVFV competition ELISA, indicating previous exposure of these people to the virus either within or outside Makkah. However, none of the seropositive human cases had suffered from clinical illness and it is speculated that they contracted the infection through the butchering of viraemic animals. This hypothesis is in line with the findings of a national cross-sectional serosurvey among slaughterhouse workers in Madagascar during the 2008–2009 epidemic, where it was demonstrated that 21.7% of them had IgG or IgM antibodies against RVFV (64).

Conclusions

Rift Valley fever continues to present a threat to public health during pilgrimage seasons in Saudi Arabia. Two factors may increase the possibility of RVF outbreaks among sacrificial animals and pilgrims: the first is the intensive importation of small ruminants from the Horn of Africa shortly before the pilgrimage season; the second factor, from within Saudi Arabia, is the movement of animals from the RVF-endemic south-western area (Jizan region) to the Makkah region (Fig. 1), particularly in the few weeks before the pilgrimage season. The findings of the study demonstrate that better understanding of the epidemiology of the disease is needed, in order to have an efficient and effective strategy for control and prevention. It is further suggested that, to facilitate the launch of such a strategy, better collaboration between the veterinary and public health authorities in Saudi Arabia is required.

Recommendations

The following ten recommendations may contribute to the prevention and control of RVF in Saudi Arabia:

- when ecological conditions are favourable for circulation of the virus, the movement of animals from the RVF-endemic south-western area (Jizan region) to any other region of the country should be prohibited at all times of the year, particularly with regard to the Makkah region shortly before the pilgrimage season
- the relative risk of RVFV activity should be monitored and predicted continuously through the application of simulation models
- quarantine regulations for live animals imported from abroad should be improved, especially at Jiddah Islamic port, to enable safety assurance at the entrance point; collaboration could be achieved by establishing checkpoints and laboratory testing for RVFV in the exporting countries, with technical support from Saudi Arabia
- a good network of information on RVF epidemiology should be established with the countries that export millions of live ruminant animals to Saudi Arabia every year
- the surveillance system for tracking RVFV activity in the south-western (Jizan) region of Saudi Arabia should be improved to prevent the expansion of the disease into new non-infected regions
- more consideration should be given to the risk of RVFV spread during the pilgrimage season, when millions of ruminant animals are slaughtered annually
- public awareness about the risk factors for RVF should be improved
- collaboration between veterinary and human health authorities, entomologists, environmental specialists and biologists should be improved as the best strategy for the prevention and control of RVF

- early warning information derived from satellite data should be coordinated
- RVF emergency preparedness protocols should be created.

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Table I
The major recorded Rift Valley fever epizootics in African and Middle Eastern countries

Country	Date	Reference
Africa		
Kenya (Rift Valley)	1931	Daubney <i>et al.</i> (2)
Kenya	1989	Davies <i>et al.</i> (9)
Kenya (north-eastern)	1997–1998	Woods <i>et al.</i> (10)
Kenya	2006–2007	Munyua <i>et al.</i> (11)
Somalia	2006–2007	Nderitu <i>et al.</i> (12)
Tanzania	2007	WHO (13)
Sudan	1973	Davies (14)
Sudan	1979–1981	Davies <i>et al.</i> (9)
Sudan	1984	Eisa (15)
Sudan	2007	Hassan <i>et al.</i> (16)
Zambia	1973–1974	Hussein <i>et al.</i> (17)
Zambia	1977–1978	Hussein <i>et al.</i> (17)
Zambia	1985	Hussein <i>et al.</i> (18)
Zambia	1992	Davies <i>et al.</i> (19)
Zambia	1997	Samui <i>et al.</i> (20)
Egypt	1977–1978	Darwish <i>et al.</i> (21)
		Imam & Darwish (22)
		Meegan <i>et al.</i> (23)
Egypt (Upper Egypt)	1993	Gabery <i>et al.</i> (24)
Egypt (Upper Egypt)	1997	Abd El-Rahim <i>et al.</i> (25)
Mauritania and Senegal	1987	Walsh (26)
Madagascar (East Coast)	1990	Morvan <i>et al.</i> (27)
Madagascar (Central Highlands)	1991	Morvan <i>et al.</i> (28)
Madagascar	2008	Jeanmaire <i>et al.</i> (29)
		Rakotoarivelo <i>et al.</i> (30)
Mauritania (southern Mauritania)	1987	Saluzzo <i>et al.</i> (31)

Mauritania	1988	Lancelot <i>et al.</i>	(32)
Mauritania (southern Mauritania)	1993	Zeller <i>et al.</i>	(33)
Mauritania (northern Mauritania)	2010	El Mamy <i>et al.</i>	(34)
Mozambique	1963–1983	Nunes Perisca & Ferreira	(35)
Congo	1994	Olloy	(36)
Senegal	1997	Zeller <i>et al.</i>	(37)
South Africa	2008–2010	WHO	(38)
Middle East			
Saudi Arabia (south-western Jizan region)	2000	Al-Hazmi <i>et al.</i>	(5)
		CDC	(7)
		Madani <i>et al.</i>	(6)
Yemen (El Zuhrah district)	2000	CDC	(39)

CDC: Centers for Disease Control and Prevention

WHO: World Health Organization

Table II
Distribution of investigated sacrificial animals from the livestock yards of Al-Kaakiah slaughterhouse, Makkah, during the pilgrimage season of 1432 H (4–9 November 2011)

Animal species	Imported animals		Local animals		Total
	Sudan	Somalia	Makkah	Jizan	
Goats	0	106	27	25	158
Sheep	132	136	51	23	342
Sub-totals	132	242	78	48	
Total	374		126		500

Table III
Seropositivity to Rift Valley fever virus: detection of IgM and IgG antibodies among local and imported animals

Animal source	Investigated numbers	IgG/IgM positive		IgM positive		IgG positive	
		No.	%	No.	%	No.	%
Local	126	17	13.49	0	0	17	13.49
Imported	374	67	17.91	0	0	67	17.91
Total	500	84	16.80	0	0	84	16.80

IgG: immunoglobulin G

IgM: immunoglobulin M

Table IV
Seropositivity to Rift Valley fever virus: detection of IgG antibodies among local animals

Source of animals	Animal species	No. of animals tested	IgG positive	
			No.	%
Makkah	Sheep	51	7	13.72
	Goats	27	0	–
Sub-total		78	7	8.97
Jizan	Sheep	23	4	17.39
	Goats	25	6	24.00
Sub-total		48	10	20.83
Total		126	17	13.49

IgG: immunoglobulin G

Table V
Seropositivity (IgG) to Rift Valley fever virus among imported sacrificial animals and their source

Source of animals	Animal species	No. of animals tested	IgG positive	
			No.	%
Sudan	Sheep	132	28	21.21
	Goats	0	0	–
Sub-total		132	28	21.21
Somalia	Sheep	136	30	22.05
	Goats	106	9	8.49
Sub-total		242	39	16.11
Total		374	67	17.91

IgG: immunoglobulin G

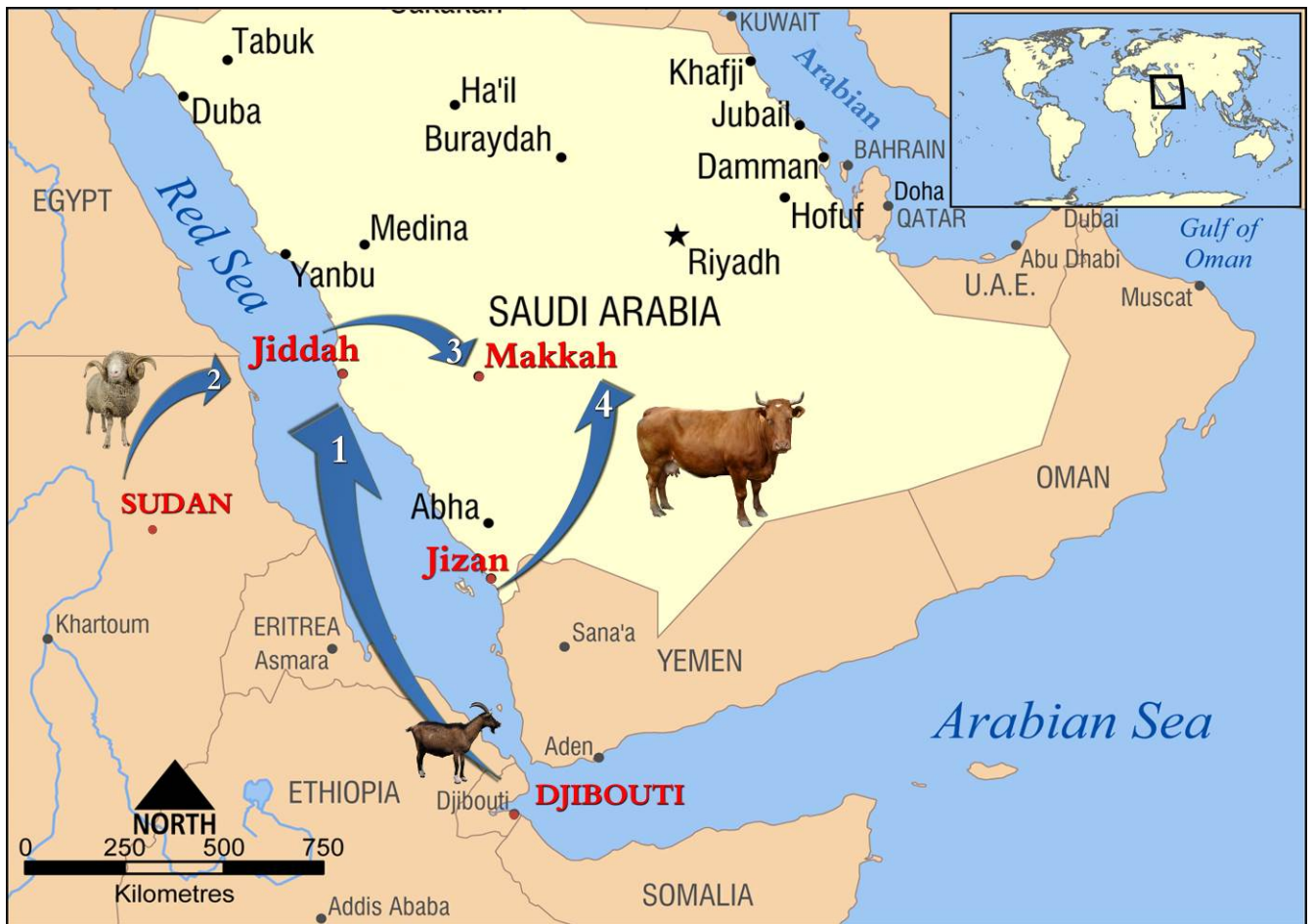


Fig. 1

Importation of live ruminant animals into Saudi Arabia

- 1) From the Horn of Africa through Djibouti
- 2) From Sudan into Saudi Arabia
- 3) From Jiddah to Makkah
- 4) Through the illegal movement of live animals from the Jizan region, where Rift Valley fever is endemic, to the Makkah region, where seropositive animals and human cases have been detected