



RESEARCH ARTICLE

Evolution of Bovine Brucellosis over 11-Years Period in the Sudan

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ABSTRACT

The objective of this work was to project the evolution of bovine brucellosis in an 11-years period in Kuku Dairy Scheme, Khartoum North, Sudan. The prevalence was estimated in the baseline year and the required data on other parameters were obtained from primary and secondary sources. Two scenarios were projected; in the first one, the animal population was considered to grow at the rate obtained from the Ministry of Animal Resources and Fisheries while in the second, the population was held constant over the study period. The deterministic transmission model of Zinsstag *et al.* (2005) was modified and used as an analytical framework. The study revealed in the first scenario that the number of susceptible animals will increase from 8,798 in the baseline year, 2004, to 14,384 head in the final year, 2014, with a growth rate of 63.5%. The number of sero-positive animals will also increase from 1,508 to 3,064 head with a growth rate of 103.2%. The incidence of the disease is 154 heads in the initial year, this number changes over the 11 years to 563 with a growth rate of 265.6%. In the second scenario, the incidence is 166; it changes over the 11 years to 1,008 with a growth rate of 507.2%. The number of sero-positive animals will increase from 1,508 to 5,400 head with a growth rate of 258.1%. The number of the susceptible animals will decrease over time as result of new infections. It will decrease from 8,798 to 4,906 head with a growth rate of -44.2%. It can be concluded that brucellosis will evolve in the scheme until all animals become infected if no control strategies adopted. More investigations on the impact of the disease on fertility and its control are recommended.

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INTRODUCTION

Brucellosis is a highly contagious and zoonotic disease with a cosmopolitan distribution (Roth *et al.*, 2003). It is endemic in many countries and across various animal production settings, and is responsible for considerable economic losses and public health burden (Hou *et al.*, 2013; Raclouz *et al.*, 2013). The causative is a gram negative bacterium called *Brucella*, it mainly affects cattle, sheep, goats, camels and pigs, as well as humans disease (Roth *et al.*, 2003; Raclouz *et al.*, 2013). However, bovine brucellosis caused by *B. abortus* is the most important among animal brucellosis (Corbel, 1997). It has been declared in 70% of the world's countries (Nilson and Duncan, 1990) being endemic in several areas as the Mediterranean region, Arabian Peninsula, India, Mexico, Central and South America (Hurtado, 2001; Hou *et al.*,

2013). The prevalence of bovine brucellosis is variable in cattle although it is generally higher in dairy cattle than range cattle due to the intensive farm management (Langoni *et al.*, 2000). The eradication of bovine brucellosis has been achieved in some countries as Australia, Canada, and many others by applying test and slaughter policy (OIE, 2012; Hou *et al.*, 2013). Contrary, in the Sudan, it is still widely spread among cattle and is reported in different parts of the country as the most prevalent when compared to brucellosis in other animal species (Omer *et al.*, 2007).

The main clinical signs of animal brucellosis are a high incidence of abortions as well as reduced fertility and milk yield along with increased mortalities of the newborns. The high incidence of abortions depends on the timing of the infection whether it is a recent or chronic (Raclouz *et al.*, 2013; Hou *et al.*, 2013). However,

the disease can be present for several years without showing any clinical signs (Racloz *et al.*, 2013). The spread of the disease between herds usually occurs by the introduction of chronically asymptomatic infected animals (Robinson, 2003). Infection is transmitted to susceptible cattle through mucous membranes of the alimentary or respiratory tract or through the conjunctiva from aborted feti, placental membranes and infected vaginal discharges and fluids (Thim, 1982). Transmission and spread of brucellosis among animals is affected by a variety of factors including: farming system and practices, farm sanitation, livestock movement, mixing and trading of animals, and sharing of grazing grounds (Musa, 2004; Omer *et al.*, 2007). Good knowledge of these factors is essential and central to the success of a control policy (Reviriego *et al.*, 2000; Bikas *et al.*, 2003; Minas *et al.*, 2004). The control of the disease depends on the system of animal management (Musa, 2004). The approach for controlling, preventing, or eradicating of brucellosis in a country or region depends on the level of the infection in the herds or flocks, type of husbandry, economic resources, public health impacts, and potential international trade implications (Bikas *et al.*, 2003). However, decisions for managing brucellosis are likely to be intuitive unless accurate and current epidemiological information are available (Robinson, 2003).

Many epidemiological studies have been carried out, in different parts of the world including the Sudan, on the prevalence and risk factors of bovine brucellosis, yet the evolution of the disease over certain years has not been paid enough consideration, with exception to the studies of Gonzalez-Guzman and Naulin (1994) and Zinsstag *et al.* (2005) who developed and analyzed the spreading and transmission of bovine brucellosis and brucellosis over time among animals and humans in Mongolia, respectively. Therefore, the objective of this work was to predict the evolution of bovine brucellosis in Kuku Dairy Scheme in the absence of control measures.

MATERIALS AND METHODS

The study area

The study was conducted in Kuku Dairy Scheme, Khartoum State, the Sudan. The scheme was established in November 1963 on the nucleus of small Milk Producers Co-operative dated from 1953 with the aim of settling the nomadic tribe (Bataheen) and to supply Khartoum with milk. The scheme covers an area of about 2600 acre of flat levelled land stretching out from the old riverain cultivation area on the Blue Nile bank, east of Khartoum North. The scheme was based on the famous Bombay Dairy where local traditional producers were banded together to produce milk under modern hygienic conditions (El Hadari and Simpson, 1967). The co-operative operation renders services in reduced charges per feddan for irrigation. Animal medication is valued at market rate and the farmers shoulder all the expenses related to the agricultural operations (Angara, 1998). The efforts of upgrading the local breed were successful but the efforts of controlling animal diseases in particular brucellosis lagged far behind. The scheme was proved to be endemic with brucellosis. No formal control strategy was adopted.

Sources of data

The required data were obtained from both primary and secondary sources. The primary source was the sero-prevalence survey conducted during the period from January to June/2004 using Rose Bengal Plate Test (RBPT) and Competitive Enzyme Linked Immuno-Sorbent Assay (cELISA) as described by Angara *et al.* (2009). On the other hand, secondary sources of data like animal mortalities (0.06% and 0.05%) for susceptible and sero-positive animals and extraction rate (0.184%), included: Text books, journals, relevant studies, Food and Agriculture Organization (FAO) beside General Administration of Planning and Livestock Economics (GAPLE), Ministry of Animal Resources and Fisheries (MARF), Khartoum, the Sudan.

Evolution of bovine brucellosis

The evolution of bovine brucellosis in a 11-years period (2004-2014), was predicted by the deterministic transmission model as described by Zinsstag *et al.* (2005). Nevertheless, to suit the situation in Kuku Dairy Scheme, the following modifications were introduced to the model: (a) only one animal species (cattle) was included in the model and (b) the model omitted immunity due to vaccination compartment ($Z=zero$) because, although strain 19 is produced in the Sudan, herders and owners did not use it to protect their animals against brucellosis. Accordingly, the disease situation in the scheme can be described as follows: The total number of cattle in the Scheme included: the susceptible cattle (X) or compartment 1 and the infected cattle (Y) or compartment 2. Moreover, the normal cattle growth rate was considered α_c , as brucellosis affects mainly fertility and milk production; the sero-prevalence was considered a dependent effect (η) on cattle birth rates α_c (Bernues *et al.*, 1997). Therefore, the effective birth rate $\alpha_{c(effective)}$ is calculated as follows:

$$\alpha_{c(effective)} = \alpha_{c(baseline)} (1 - (\eta)) (Y / X+Y) \dots \dots \dots (1)$$

Where:

η = the prevalence dependent reduction of birth rates α_c , including abortions among the sero-positive which equals the number of sero-positive multiplied by the abortion rate (0.12%) (Angara *et al.*, 2009).

The incidence or newly infected cattle is calculated by multiplying the proportion of infected cattle (γ_c) by the contact rate (β_c) by the number of susceptible (X) and the number of sero-positive (Y).

$$\text{Incidence}_{(cattle)} = (\gamma_c \beta_c XY) \dots \dots \dots (2)$$

Where:

γ_c = the proportion of infected animals that the number of sero-positive animals divided by the total number of animals in the population.

β_c = the cattle contact rate that equals $k / (n-1) /$ number of sero-positive animals (Carpenter *et al.*, 1978). However, k equals the effective contact (Annual abortion and delivery of sero-positive animals); n equals the total number of animals in the population.

The number of susceptible cattle grows every year by cattle birth which equals $\alpha_c (X+Y) (1- \eta) (Y/(X+Y))$, where: α_c (birth rate of cattle), multiplied with the sum of compartment 1 and 2. This term is multiplied by a sero-prevalence $(1- \eta) (Y/(X+Y))$ dependent decrease of cattle birth rate.

Sero-prevalence reduction in births equals the number of sero-positive animals multiplied by the reduction in fertility (0.15%). On the other hand, this number decreases as a result of mortality of susceptible cattle and the increase in prevalence due to new infections. So the change in the number of susceptible cattle (dx/dt) is calculated as follows:

$$dx/dt = \alpha_c (X+Y)(1-(\eta (Y/(X+Y)))) - \mu_c X - Ex - \gamma_c \beta_c XY \dots (3)$$

Where:

dx/dt = the annual change in the number of susceptible cattle.

The number of infected cattle will increase as long as there were new infections and will decrease as a result of the mortality of the infected cattle. So the change in the number of infected cattle (dY/dt) is computed as follows:

$$dY/dt = \gamma_c \beta_c XY - \mu_c X \dots (4)$$

Where:

dY/dt = the annual change in the number of sero-positive cattle.

Analysis of data

Modeling of bovine brucellosis in Kuku Dairy Scheme, Khartoum North, the Sudan was conducted using a software Microsoft® Excel for Windows® 2007 data base.

RESULTS

As presented in Table 1, the herd of the Scheme consisted of 27.5% (n = 2838) calves less than one year, 12.3% (n = 1269) heifers, 58.8% (n = 6056) adult cows and 1.4% (n = 143) bulls and the overall sero-prevalence of brucellosis was found to be 24.9% by c-ELISA. The number of sero-positive aborters was found to be 12.0% (17/143) (Angara *et al.*, 2009). The baseline year estimates of brucellosis are summarized in Table 2.

Evolution of bovine brucellosis: scenario one

In the first scenario the total number of animals in the population was left to grow according to the normal growth rate. This scenario considered the nature of the traditional producers in keeping large herd sizes. The modeling revealed an increase in the cattle population from 10.306 in 2004 to 17.448 heads in 2014 with a growth rate of about 69.3% (Table 4 and 5; Figure 2 and 3). The disease in cattle will evolve if no control strategy is adopted and put in place over the 11 years as follows: The number of susceptible animals will increase from 8.798 in the baseline year to 14.384 head in the final year with a growth rate of 63.5%. The number of sero-positive animals will increase from 1.508 to 3.064 head in the final year with a growth rate of 103.2%. The prevalence of the disease was 141 heads in the initial year and the incidence

Table 1: The composition of the dairy herd in kuku dairy scheme, Khartoum North, the Sudan in the Baseline Year 2004

	No. of all animals	No. of animals in the selected herds	Herd composition (%)
Calves	2838	396	27.5
Heifers	1269	177	12.3
Adult Cows	6056	845	58.8
Bulls	0143	020	01.4
Total	10306	1438	100.0

Table 2: Baseline Estimates for Brucellosis Deterministic Transmission Model among Cattle in Kuku Dairy Scheme, Khartoum North, the Sudan

Parameter	Description	Estimate
X	number of susceptible cattle	8798 head
Y	number of sero-positive cattle	1508 head
X+Y	total number of cattle	10306 head
% ca-infectious (γ_c)	proportion of infected cattle	14.63

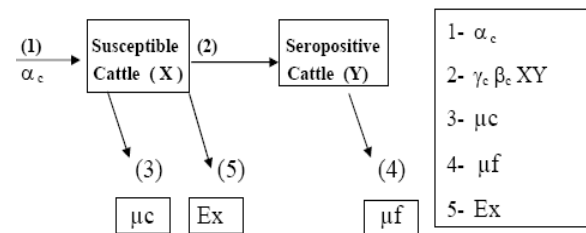


Fig. 1: Cattle Deterministic-Transmission Model.

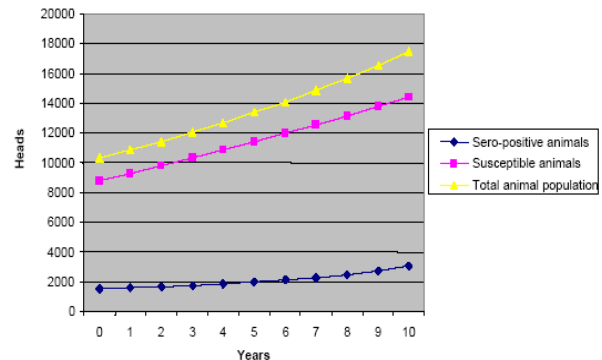


Fig. 2: Evolution of bovine brucellosis in Kuku scheme over 11 years with normal growth rates.

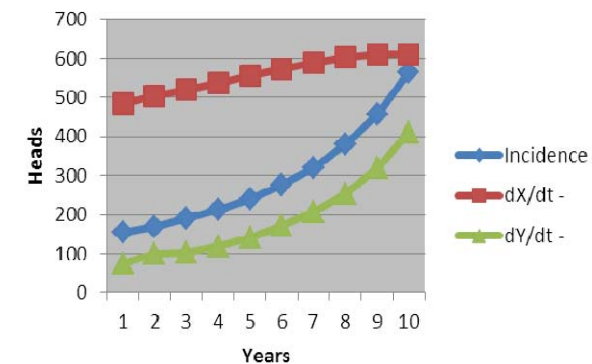
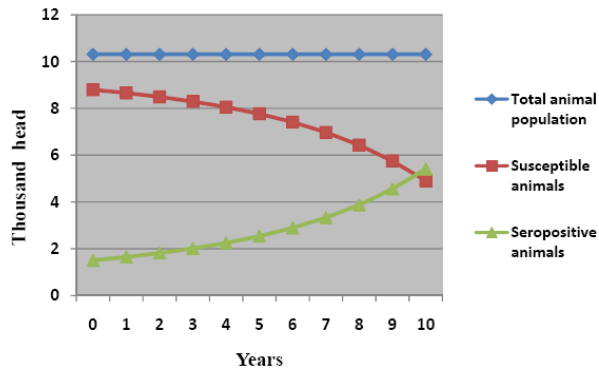


Fig. 3: Incidence of bovine brucellosis and the change in number of susceptible and Sero-positive animals over 11 years.

Table 3: Estimated Parameters used in projecting the Evolution of Brucellosis in Kuku Dairy Scheme, Khartoum North, the Sudan

Cattle Parameter	Description	Estimate	Source
α_c	Cattle birth rate	27.5%	Calculated from field data (2004)
β_c	Cattle contact rate	7.2780×10^{-05}	Calculated based on Carpenter (1986)
γ_c	Proportion of infectious sero-positive cattle	14.63%	Computed from field data (2004)
μ_c	Cattle mortality rate	6%	Adapted from GAPLE (2004)

**Fig. 4:** Evolution of Bovine Brucellosis in Kuku Dairy Scheme over 11 years with constant animal population

was 154. The incidence evolves over the eleven years to 563 with a growth rate of 265.6%.

The change in the number of susceptible animals (dX/dt) will increase from 484 heads in the first year to 612 heads in the final year with a growth rate of 26.4%, while the number of sero-positive animals (dY/dt) will increase from 66 to 320 heads with a growth rate of 384.8% (Table 4 and 5; Figure 2 and 3).

Evolution of bovine brucellosis: scenario two

In the second scenario total number of animals in the population was kept constant considering the availability of resource endowment with regard to fencing and cultivated areas. The total number will remain 10,306 heads over the whole period with zero growth rate. The disease in cattle will evolve if no control strategy is adopted and put in place over the eleven years as shown in Table 6 and 7.

The sero-prevalence of the disease in the baseline year is 141 heads. The incidence in the first year is 166 new cases. This number evolves over the eleven years to 1,008 with growth rate of 507.2%. The number of susceptible animals decrease along the time from 8,798 head to 4,906 due to the new animals infected with growth rate of -44.2%, on the other hand, the number of sero-positive animals will increase from 1,508 to 5,400 head in the final year with a rate of growth of 258.1% (Table 6 and 7; Figure 4). The change in the number of susceptible (dX/dt) animals evolves from -166 head in the baseline year to -1008 head in the final year with a growth rate of -507.2%, while the change in the number of seropositive animals (dY/dt) will increase from 166 baseline year to 1008 with a growth rate of 507.29%.

DISCUSSION

This study projected the evolution of bovine brucellosis in a 11-years period in Kuku Dairy Scheme, Khartoum North, the Sudan. Contrary to Zinsstag *et al.* (2005) Gonzalez-Guzman and Naulin (1994) the model

Table 4: Evolution of Bovine Brucellosis in Kuku Scheme over 11 years (2004-2014) with growing Animal Population, Khartoum North, the Sudan

Year	Incidence	dX/dt	dY/dt
0	141*	-	-
1	154	484	066
2	169	504	075
3	190	520	099
4	213	538	103
5	241	555	120
6	276	573	142
7	321	590	170
8	379	602	207
9	457	610	245
10	563	612	320

*Values of the baseline year are based on prevalence; Source: (computed from Table 2 and 3 using equation 2, 3 and 4).

Table 5: Growth Rates of Susceptible, Sero-positive and Total Animal Population in 11 Years in Kuku Dairy Scheme, Khartoum North, the Sudan

Description	0	10	Growth rate (%)
Total animal (X+Y)	10306	17448	069.3
Susceptible Animals (X)	08798	14384	063.5
Sero-positive animals (Y)	01508	03064	103.2
incidence	00154	00563	265.6
dX/dt	00484	00612	026.4
dY/dt	00066	00320	384.8

Source: (computed from Table 2 using equation 2, 3 and 4).

Table 6: Evolution of Bovine Brucellosis in Kuku Scheme over 11 year with constant Animal Population, Khartoum North, the Sudan

Year	Incidence	dX/dt	dY/dt
0	0141*	-	-
1	0166	-0166	0166
2	0189	-0198	0198
3	0237	-0237	0237
4	0288	-0288	0288
5	0353	-0353	0353
6	0438	-0438	0438
7	0549	-0549	0549
8	0682	-0682	0682
9	0843	-0843	0843
10	1008	-1008	1008

* Values of the baseline year are based on prevalence; Source: (computed from Table 2 using equation 2, 3 and 4).

Table 7: Growth Rates of Susceptible, Seropositive and Total Animal Population in 11 Years in Kuku Dairy Scheme, Khartoum North, the Sudan.

Description	Baseline year	Final Year	Growth rate
Total animal (X+Y)	10306	10306	000.0
Susceptible Animals X	08798	04906	- 044.2
Seropositive animals Y	01508	05400	258.1
Incidence	00166	01008	507.2
dX/dt	-00166	-01008	- 507.2
dY/dt	00166	01008	507.2

Source: (computed from Table 2 and equations 2, 3 and 4).

used in the present study consisted only of two compartments: (X) susceptible animals or compartment 1 and (Y) sero-positive animals or compartment 2. The Model of Zinsstag *et al.* (2005) consisted of three compartments, (X) susceptible, (Y) sero-positive and (Z) immune animals, while the one of Gonzalez-Guzman and Naulin (1994) consisted of four compartments: (S) susceptible, (I₁) aborting infectious, (I₂) infectious carriers and (Ø) immune by vaccination. Modifications introduced to the Model used in the present study were because Zinsstag *et al.* (2005) tracked the issue in a comprehensive approach and at a macro-level whereas Gonzalez-Guzman and Naulin (1994) studied the spread of brucellosis at a micro-level. Furthermore, Zinsstag *et al.* (2005) and Gonzalez-Guzman and Naulin (1994) dealt with a single fattening farm. In the present study the spread of bovine brucellosis was projected in a collection of dairy cattle farms (Kuku Dairy Scheme) and at a meso-level. However, only few studies focused on the evolution of bovine brucellosis such as Carpenter *et al.* (1987) and Gonzalez-Guzman and Naulin (1994); they developed and analyzed the spread of bovine brucellosis and investigated the epidemiology and economics of *Brucella ovis* control, respectively. In addition, Zinsstag *et al.* (2005) developed a model combining both epidemiological and economic analysis of animal and human zoonosis.

In this study the flow into the compartment of susceptible animals were the sero-positive dependant births (head/year), contrasting Zinsstag *et al.* (2005) who included loss of immunity due to both vaccination and natural infection, besides the sero-positive dependant births (head/year). Gonzalez-Guzman and Naulin (1994) included loss of vaccination immunity and healthy cows' births. Moreover, the present study and Gonzalez-Guzman and Naulin (1994) did not include loss of immunity of sero-positive because infected cows remained immune. Zinsstag *et al.* (2005) set this rate to zero as the Model was developed to be applied to all zoonotic diseases.

In this study loss of vaccination immunity was omitted because there were no vaccination strategies in place. On the other hand, flows out of the compartment were mortality (head/year), extraction (head/year) and infected cattle (head/year). Zinsstag *et al.* (2005) added vaccinated newborns to the out flow. Gonzalez-Guzman and Naulin (1994) included abortions, slaughtered and infected animals as an out-flow. Furthermore, resembling Zinsstag *et al.* (2005), flows into compartment (Y) were infected cattle. But for Gonzalez-Guzman and Naulin (1994) this compartment was for aborting infectious (I₁). Additionally and in agreement with Gonzalez-Guzman and Naulin (1994), flow out of the compartment was mortality of sero-positive females. However, no loss of immunity was added as done by Zinsstag *et al.* (2005). No third compartment was found to contain immune animal similar to the case of ecozoo for the reason mentioned before. In this study there was no fourth compartment, as did in the study of Zinsstag *et al.* (2005), because the two studies considered the sero-positive as one compartment instead of two; infectious abortive and infectious non-abortive compartments. Flows into the third compartment were vaccinated newborns and adults as described by Zinsstag *et al.* (2005), and the infectious carrier as described by Gonzalez-Guzman and Naulin (1994) for

whom flows into the fourth compartment were the immunized cattle.

The Model accounts 0.15% reduction in fertility (Failure of conception and abortion), abortion account to 0.12% (field data) and used slightly conservative figure of 0.03% for failure of conception and abortion because it is difficult to estimate the effect of brucellosis alone on fertility as there are many other causes and factors for infertility.

In the first scenario the total number of animals is expected to increase by 0.667% in the 11 years, the number of the sero-positive animals (Y) is expected to increase by 1.574 at the expense of the susceptible animals (X) which will increase by 0.549%. The increase in the number of susceptible animals (X) is less than the growth of the whole population; this indicates that two effects act to give the final number of the susceptible animals. These are the normal growth of the total number of animals in the population that increase the animal numbers and the incidence of the disease, which reduce them. Under the assumption of no disease control the effect of the incidence of the disease out weighted the growth in herd population resulting in positive increase of the number of susceptible animals but at a lower rate (0.549%) compared with the growth rate of the population (0.667%).

In the second scenario, the size of animal population was held constant, the number of susceptible animals will decrease by time (-0.2%) and the number of sero-positive animal will grow faster than in scenario one (2.27%). Under the assumption of a constant population size where the growth in animal population was set to be equal to mortality and extraction, the only remaining effect on the number of susceptible animals is the incidence of the disease which has negative impact on the number of susceptible animals. To keep a constant herd size the producers tend to reduce the number of non-productive animals and keep the productive ones. By doing so the proportion of mature productive animals increases the matter that enhances the evolution of the disease by intensifying the number of infectious sero-positive animals and hence acts to more than doubling the rate of the disease evolution over the 11 years period. The incidence of the disease, followed by the number of sero-positive animals, will increase at an increasing rate. In contrast to the number of susceptible animals which will decrease at the same rate, until the last year when the number of sero-positive will exceed the number of susceptible. The situation will continue after year ten until all animal population become sero-positive (i.e the number of susceptible animals will go to zero) if no control strategy is adopted and put in place.

Conclusions and Recommendations

The study concluded that brucellosis in Kuku Dairy Scheme will evolve until all animal population become infected if no control strategy is adopted and put in place. This will continue to exert negative impact on the environment and the public health beside the economic losses to both farmers and the economy of the scheme. The study recommended more investigation on the impact of the disease on fertility and the control of the disease in animals for human benefits and the scheme's economy.

There is an urgent need to raise the awareness of at-risk-groups (producers, dairies, sellers and consumers) toward the characterization of the disease, the related public health risk and how to avoid infection.

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REFERENCES

- AngaraT-EE, 1998. Impact of currency devaluation on dairy production in Khartoum State *MSc Thesis*. University of Khartoum. Khartoum, Sudan
- AngaraT-EE, AA Ismail, H Agab and NM Saeed, 2009. Seroprevalence of Bovine Brucellosis in Kuku Dairy Scheme. *Sudan J Vet Sci Anim Husb*, 48: 27-35.
- Carpenter TE, SL Berry and JS Glenn, 1987. Economics of *Brucellaovis* Control Simulation Model. *J Am Vet Assoc*, 190: 977-982.
- Corbel MJ, 1997. Brucellosis: An Overview: International Conference on Emerging Zoonoses Emer Infec Dis, 3: 213-221.
- Bikas C, E Jelastopulu, M Leotsinidis, X Kondakis, 2003. Epidemiology of Human Brucellosis in a rural area of north-western Peloponnese in Greece. *Eur J Epidemiol*, 18: 267-274.
- Bernues A, E Manrique and MT Maza, 1997. Economic evaluation of bovine brucellosis and tuberculosis eradication programmes in a mountain area of Spain. *Prev Vet Med*, 30: 137-149.
- El Hadari A and MC Simpson, 1967. The production and Marketing of Milk in Khartoum province: an economic study of traditional and modern methods. Research bulletin No.7.
- General Administration of Planning and Livestock Economics (GAPLE). 2004. Ministry of Animal Resources and Fisheries, Khartoum, the Sudan.
- Gonzalez-Guzman J and R Naulin, 1994. Analysis of a Model of Bovine Brucellosis using Singular Perturbations. *J Math Biol*, 33: 211-223.
- Hou Q, X Sun, J Zhang, Y Liu, Y Wang and Z Jin, 2013. Modeling the transmission dynamics of sheep brucellosis in Inner Mongolia Autonomous Region, China. *Math Biosci*, 242: 51-58.
- Hurtado R, 2001. Brucellosis, new and old issues regarding diagnosis and management. Accessed on January the 31st 2001, ([http://www.mgh.harvard.edu/id/images/brucellosis](http://www.mgh.harvard.edu/id/images/brucellosis.pdf)). pdf, Harvard education online.
- Langoni H, M Silvio, V Aristeu, B Renata, B Flávia, J Lia and A José, 2000. Isolation of *Brucella* species from milk of brucellosis positive cows in São Paulo and Minas Gerais states. *Braz. J Vet Res Anim Sci*, 37: 1413-9596.
- Minas A, M Minas, A Stournara and S Tselepidis, 2004. The effects of Rev-1 vaccination of sheep and goats on human brucellosis in Greece. *Prev Vet Med*, 64: 41-47.
- Musa MT, 2004. Epidemiology of brucellosis in animals and man. The national training workshop in: Surveillance, diagnosis and control of brucellosis. Khartoum, the Sudan.
- Nilson JR and JR Duncan, 1990. Animal Brucellosis. Boca Raton. Florida, USA. Press, Vet Bull, 454.
- Omer MM, AA Abdelaziz, MAS Abusalab and MA Ahmed, 2007. Survey of brucellosis among sheep, Goats, Camels and Cattle in Kassala Area, Eastern The Sudan. *J Anim Vet Adv*, 6: 635-637.
- Racloz V, E Schelling, N Chitnis, F Roth and J Zinsstag, 2013. Persistence of brucellosis in pastoral systems. *Rev Sci Tech Off Int Epiz*, 32: 61-70.
- Reviriego FJ, M Moreno and L Domingues, 2000. Risk factors for brucellosis sero-prevalence of sheep and goat flocks in Spain. *Prev Vet Med*, 44: 167-173.
- Robinson A, 2003. Guidelines for coordinated human and animal brucellosis surveillance, FAO Animal production and health Paper 156. Room
- Roth F, J Zinsstag, D Orkhon, G Chimed-Ochir, G Hutton, O Cosivi, G Carrin and J Otte, 2003. Human health benefits from livestock vaccination for brucellosis: case study. *Bull WHO*, 81: 867-876.
- Thim MBM, 1982. Brucellosis, Distribution in Man, Domestic Animals and Wild Animals publishers Springer -Verlag, Berlin. (Hiedelberg, New York. BV association, 1968).
- Zinsstag JR, F Orkhon, D Chimed-Ochir, G Nansalma, JM Kolar and P Vounatsou, 2005. A model of animal-human brucellosis transmission in Mongolia. *Prev Vet Med*, 69: 77-95.