

Seroprevalence of Abortion Causing Agents in Egyptian Sheep and Goat Breeds and Their Effects on the Animal's Performance

Hamada Dahy Mahboub¹, Mohamed Atif Helal¹, Mabrouk Attia Abd Eldaim², Emad Mahmoud Abd El-Razek³ & Ahmed Mahmoud Elsify⁴

¹ Department of Husbandry and Animal Wealth Development, Faculty of Veterinary Medicine Sadat Branch, Menoufia University, Egypt

² Department of Biochemistry and Chemistry of Nutritions, Faculty of Veterinary Medicine Sadat Branch, Menoufia University, Egypt

³ Department of Theriogenology, Faculty of Veterinary Medicine Sadat Branch, Menoufia University, Egypt

⁴ Department of Animal Medicine and infectious diseases, Faculty of Veterinary Medicine Sadat Branch, Menoufia University, Egypt

Correspondence: Mabrouk Attia Abd Eldaim, Faculty of Veterinary Medicine, Menoufiya University, Egypt. Tel: 20-111-748-4718. E-mail: mabroukatia@yahoo.com

Received: May 20, 2013 Accepted: June 29, 2013 Online Published: August 15, 2013

doi:10.5539/jas.v5n9p92

URL: <http://dx.doi.org/10.5539/jas.v5n9p92>

Abstract

This study was carried out to illustrate seroprevalence of *Toxoplasma gondii* and *Brucella melitensis* in local sheep and goat flocks living in Nile Delta regions of Egypt as well as to investigate its effects on animals' health and performance. In trial 1: 492 serum samples were collected from domestic ewes and does flocks suffering from abortion and stillbirths were used in this survey. In trial (2): 100 serum samples collected from toxoplasma or brucella infected or non infected sheep and goats were used to study the effects of toxoplasmosis and brucellosis on animals' health. This study revealed that 142 (28.86%) sera contained antibodies against *Toxoplasma gondii* and 89 (18.09%) were positive for *Brucella melitensis*. The incidences of toxoplasmosis and brucellosis were 31.01% and 12.26% in sheep, respectively and 17.11% and 36.84% in goats, respectively. Higher seroprevalence of *Toxoplasma gondii* was recorded in sheep with high flock size. Farmers using set stocking management had lower seroprevalence toxoplasmosis and brucellosis. Toxoplasmosis and brucellosis lowered serum albumin level and liver enzymes activities while, increased serum levels of globulin, urea, triglyceride and cholesterol in ewes. Serum levels of total protein, albumin and urea and the activity of serum aspartate amino transferase were decreased, however serum level of C reactive protein was increased in infected does. These results indicated that toxoplasmosis is widely spread than brucellosis in small ruminants living in the provinces of Nile Delta. Both toxoplasmosis and brucellosis inversely affected animals' health. Management practices had a role in spread of these agents.

Keywords: toxoplasmosis, brucellosis, sheep, goats

1. Introduction

Small ruminant fetus could be exposed to viral (bluetongue virus, pest viruses and caprine arthritis encephalitis virus), bacterial (*Brucella spp.*, *Salmonella spp.*) and protozoa (*Toxoplasma gondii*) infection from early embryonic term to the end of the gestation period. These diseases cause abortion, fetal loss and congenital abnormalities in lambs and kids (Aydin, 1997; Burgu et al., 1992; Fieni et al., 2003). Toxoplasmosis and brucellosis are major zoonotic diseases in many countries and eradication of these diseases in animals is a required step to control the human diseases (Al-Sim'ani, 2000; Blood et al., 1986).

Toxoplasma gondii is a highly ubiquitous and prevalent parasite. Despite the cat being the only definitive host, it is found in almost of geographical areas and warm blooded animals. The prevalence of seropositivity for toxoplasma antibodies varies with geographic location, flock and country (Tütüncü et al., 2003). Acute acquired toxoplasmosis is most commonly asymptomatic, but it can range from mild symptomatic in the normal host to fulminant and fatal illness in the immunocompromised host. Toxoplasmosis is associated with the occurrence of embryonic death and absorption, fetal death and mummification, abortion, stillborn and neonatal mortality

(Dubey, 2009). Moreover, toxoplasmosis has harmful effects on the health and performance of ewes and does after parturition and sometimes leads to their death (Radostits et al., 2007). Therefore, toxoplasmosis leads to major economic losses in livestock production (Freyre et al., 1999; Maki et al., 1996).

Brucellosis in sheep and goats caused by *Brucella melitensis* frequently results in abortions and decreased the milk production (Shareef, 2006). Brucellosis is one of the most economically devastating diseases that cause great losses among the offspring and health problems in the rural and urban population. Brucella infection in human beings occurs through the contact with the infected materials or consumption of the contaminated dairy products due to traditional feeding habits such as usage of raw milk and milk products (Garin-Bastuji et al., 2006).

Toxoplasmosis and brucellosis have serious effects on animal health because they infect vital organs in the body such as liver, kidney, muscle and heart leading to their damage, impairment of their function and increase or decrease the release of their enzymes according the stage of infection (Radostits et al., 2007, Al-Majali, 2005, Moss & Henderson, 1999; Remington et al., 2000). In addition serum lipids are important mediators for host defense. Infection and inflammation decrease serum total cholesterol, but may increase triglycerides (Jahangiri al., 2009; McGullicuddy et al., 2009). Some studies reported that cholesterol maintains a specialized type of membrane domain thus it acts as platform through which pathogens enter to infect host cells (Maxfield, 2002). Thus toxoplasmosis and brucellosis lead to serious changes in the blood metabolites of infected animals. These changes in blood metabolites can be used as indicators for the infection with these parasites and consequently help in their diagnosis.

Although the serious economic losses caused by toxoplasmosis and brucellosis in small ruminants, there is no report concerning the distribution of *Toxoplasma gondii* and *Brucella melitensis* among the local sheep and goat flocks in the Nile Delta region of Egypt. Therefore, this study was carried out to:

- i)-estimate the seroprevalence of toxoplasmosis and brucellosis in local sheep and goats concerning localities, breeds, grazing system and flock size.
- ii)-investigate the changes in blood biochemistry of the infected animals which reflect the effects of these diseases on animals' health and performances.

2. Materials and Methods

2.1 Trial 1: Survey Study of Agents Causing Abortion

492 blood samples were randomly collected from female's sheep and goats from five provinces in Nile Delta region. According to management practices, these flocks were classified into two flock sizes (≤ 15 and > 15) and two grazing systems (grazed and non grazed flocks). These animals were not vaccinated against *Toxoplasma* and *Brucella*. Obtained sera were stored at -20°C till analysis.

The sera were screened for *Toxoplasma gondii* specific IgM antibodies with an indirect haemagglutination test (Toxo-HAI, Fumouze, France), according to the manufacturer's instructions. The negative and positive controls provided in the kit were included in all plates. The sera were also screened for *Brucella melitensis* (stained antigen suspensions) with agglutination test (Plasmatec Laboratory products, UK) (Farrell & Robertson, 1975).

2.2 Trial 2: Effect of Toxoplasmosis and Brucellosis Infections on Animals' Health and Performance:

100 sheep and goats (50 animals in each species) were divided into three groups according to serological examination. One group was negative for toxoplasmosis and brucellosis ($n=20$), the second group was positive for toxoplasmosis ($n=15$) and negative for brucellosis and the third group was positive for brucellosis ($n=15$) and negative for toxoplasmosis. Females were weighted before breeding. Blood samples were collected for biochemical analysis. Litter size and incidence of abortion or still births were recorded.

The following metabolites were measured, serum activities of liver enzymes, aspartate amino transferase (AST) and alanine amino transferase (ALT) because they give an indication about liver damage as they produced intracellularly and their elevated serum levels indicate hepatocytes damage. Also serum levels of total protein, albumin, cholesterol, triglyceride and urea because elevated serum level of urea indicates impairment of kidney damage by a spectrophotometer (Spekol 11, Carl Zeiss Jena, Germany) according to the instructions of manufacture (Diagnostic diamond, Egypt) (Young, 2001). Globulin concentration was calculated as the difference between serum total protein and albumin concentrations. C-reactive protein, inflammation marker, was measured by agglutination test (Spinreact, Spain). All the tests in trials 1 and 2 were carried out at the Laboratory of Husbandry and Animal Wealth Development Department, Faculty of Veterinary Medicine, Sadat City.

3. Statistical Analysis

Data were collected, organized and analyzed by using SPSS program. Data were expressed as mean $\pm$ SEM except mortality rates are expressed as a percentage. The data were analyzed statistically by ANOVA method and Duncan's test was used to detect differences among means using SPSS® Statistical Software (SPSS® 11.01 for Windows, 14). The differences between species, breed, flock sizes, grazing systems and provinces were tested using a chi-square test.

4. Result

4.1 Trial 1

4.1.1 Survey Study of Agents Causing Abortion

4.1.2 Clinical Findings

Clinically examined sheep and goats had no specific clinical signs of diseases. But their flocks were suffered from abortion in the previous pregnancy periods. The incidence of abortion recorded during this survey was ranged from 1-25%.

4.1.3 Serological Findings

Out of 492 serum samples 142 (28.86%) and 89 (18.09%) were seropositive for toxoplasmosis and brucellosis respectively (Table 1). Differences among provinces were significant for toxoplasmosis ($P < 0.0004$) and brucellosis ($P < 0.0097$). Seroprevalence of toxoplasmosis presented the highest value in Kafr El-Sheikh province 91 (36.25%). However, Menoufia province had a higher brucellosis (28.57%) compared to other Delta provinces (Table 1). A statistically significant association was detected between *T. gondii* ($P=0.027$) and *B. melitensis* ($P=0.0001$) seroprevalence between species (Table 2). *T. gondii* seroprevalence was higher in sheep than goats. In contrast, goats had a higher *B. melitensis* seroprevalence than sheep. Serological screening of the breeds revealed that the prevalence rate of *Toxoplasma gondii* among sheep was higher in Baladi breeds 35.42% (119 out of 336, $P=0.001$) than other sheep breeds (Table 2). There was no significant difference among sheep breeds could be detected for brucellosis. All samples collected from Barki sheep had no detectable toxoplasma infection. Seroprevalence for 76 goat breeds showed that Zarabi had higher toxoplasma infection than other breeds ($P=0.033$). However, Baladi goats had the highest percent of Brucella infections (59.09%, $P=0.024$, Table 2).

Table 2 revealed that large flocks size and grazed animals had higher toxoplasma infection than small size and non grazed flocks ($P=0.0001$, $P=0.001$) respectively. In contrast, small size and non grazed flocks were shown more seropositive for brucella infection than large and grazed flocks ($P < 0.003$, $P < 0.001$, respectively).

Table 1. *Toxoplasma gondii* and *Brucella melitensis* seroprevalence in sheep and goat flocks from different provinces

Provinces	No. tested	Toxoplasma gondii ¹		Brucella melitensis ²	
		No. seronegative	No. seropositive	No. seronegative	No. seropositive
Menoufia	126	92 (73.02%)	34 (26.98%)	90 (71.43%)	36 (28.57%)**
Kafr El-Sheikh	251	160 (63.75%***)	91 (36.25%)	215 (85.66%)	36 (14.34%)
Behera	84	73 (86.90%)	11 (13.10%)	70 (83.33%)	14 (16.67%)
Giza	12	8 (66.67%)	4 (33.33%)	11 (91.67%)	1 (8.33%)
Gharbia	19	17 (89.47%)	2 (10.53%)	17 (89.47%)	2 (10.53%)
Total No.	492	350 (71.14%)	142 (28.86%)	403 (81.91%)	89 (18.09%)

¹ Chi square value $\chi^2 = 20.29$ *** ($P < 0.0004$).

² Chi square value $\chi^2 = 13.34$ ** ($P < 0.0097$).

The significant differences are within column.

Table 2. Effect of species, breed, flock size and grazing systems on prevalence of toxoplasmosis and brucellosis

Items		No. tested	Toxoplasmosis		Brucellosis	
			No. seropositive	P- value	No. seropositive	P- value
Species:	Sheep	416	129 (31.01%)	0.027	51 (12.26%)	0.0001
	Goats	76	13 (17.11%)		28 (36.84%)	
Breeds:						
Sheep	Rahmani	28	6 (21.43%)	0.001	1 (3.57%)	0.298
	Barki	22	0		3 (13.64%)	
	Ossemi	30	4 (13.33%)		6 (20.00%)	
	Baladi	336	119 (35.42%)		41 (12.20%)	
Goats	Damascus	20	2 (10.00%)	0.033	4 (20.00%)	0.024
	Zarabi	34	10 (29.41%)		11 (32.35%)	
	Baladi	22	1 (4.55%)		13 (59.09%)	
Flock size:						
	≤ 15	152	20 (13.16%)	0.0001	36 (23.68%)	0.003
	> 15	340	122 (35.88%)		43 (12.65%)	
Grazing system:						
	Grazed flocks	350	125 (35.71%)	0.001	43 (14.10%)	0.001
	Non-grazed flocks	142	16 (11.27%)		46 (32.39%)	

4.2 Trial 2

4.2.1 Effect of Toxoplasmosis and Brucellosis on Performance and Blood Chemistry of Ewes and Does

In this study we investigated the effects of *Toxoplasma gondii* and *Brucella melitensis* infection on sheep and goats' health and performances through examining the changes in the blood metabolites of the infected animals. These parameters give an indication about the effects of these causative agents on animals' health and performance.

These results revealed that *Toxoplasma gondii* and *Brucella melitensis* infection had no significant effect on serum total protein and C reactive protein levels in ewes (Table 3). However, they decreased albumin/globulin ratio (A/G ratio) and AST enzyme activity. *Toxoplasma gondii* infection significantly decreased serum albumin level in ewes however; *Brucella melitensis* infection had no significant effect on it. In addition, the activity of serum ALT enzyme was significantly increased in *Toxoplasma gondii* and *Brucella melitensis* infected ewes. Also, serum levels of globulin, urea, triglyceride and cholesterol were significantly increased in infected ewes (Table 3).

Table 3. Effect of toxoplasmosis and brucellosis on serum biochemical parameters in ewes

Biochemical parameters	Healthy sheep	Sheep infected with <i>Toxoplasma gondii</i>	Sheep infected with <i>Brucella melitensis</i>
Total protein (g/dl)	4.20 ± 0.15	4.10 ± 0.37	4.3 ± 0.45
Albumin (g/dl)	1.81 ± 0.20 ^a	1.24 ± 0.13 ^b	1.66 ± 0.11 ^{ab}
Globulin (g/dl)	2.39 ± 0.07 ^a	3.11 ± 0.28 ^b	3.64 ± 0.49 ^b
A/G ratio	0.85 ± 0.08 ^a	0.41 ± 0.087 ^b	0.56 ± 0.1 ^b
AST (unit/ml)	58.87 ± 1.92 ^a	33 ± 2.69 ^b	28.56 ± 3.53 ^b
ALT (unit/ml)	15.11 ± .43 ^a	22.86 ± 1.99 ^b	21.90 ± 3.24 ^b
BUN (mg/dl)	24.35 ± 2.07 ^a	32.26 ± 2.26 ^b	31.28 ± 2.42 ^b
Triglyceride (mg/dl)	17.82 ± 1.29 ^a	51.49 ± 6.65 ^b	51.22 ± 3.78 ^b
Cholesterol (mg/dl)	41.61 ± 4.33 ^a	57.84 ± 5.17 ^b	66.22 ± 4.63 ^b
C-reactive protein (mg/dl)	8.40 ± 1.70	10.71 ± 2.48	6.60 ± 2.94

BUN: Blood urea nitrogen.

Values carrying the different letters are significantly different at $P < 0.05$.

The effects of Toxoplasmosis and Brucellosis on goat blood metabolites were somewhat different from those in sheep. Toxoplasmosis and Brucellosis significantly decreased serum levels of total protein, albumin, urea, A/G ratio and the activity of serum AST in does. In addition, *Toxoplasma gondii* caused significant decrease in cholesterol level while *Brucella melitensis* caused significant decrease in globulin concentration in serum of does ($P=0.05$, Table 4). In contrast, brucellosis significantly increased serum triglyceride and C reactive protein levels in goat however; toxoplasmosis has no significant effect on them. Both toxoplasmosis and brucellosis have no significant effect on serum activity of ALT (Table 4).

Table 4. Effect of toxoplasmosis and brucellosis on serum biochemical parameters in does

Biochemical parameters	Healthy goats	Goats infected with <i>Toxoplasma gondii</i>	Goats infected with <i>Brucella melitensis</i>
Total protein (g/dl)	5.44 ± 0.27 ^a	4.03 ± 0.80 ^b	2.81 ± 0.16 ^b
Albumin (g/dl)	2.15 ± 0.23 ^a	0.84 ± 0.3 ^b	1.34 ± 0.14 ^b
Globulin (g/dl)	3.47 ± 0.27 ^a	3.19 ± 0.63 ^a	1.47 ± 0.29 ^b
A/G ratio	0.72 ± 0.07 ^a	0.34 ± 0.23 ^b	0.75 ± 0.46 ^{ab}
AST (unit/ml)	67.23 ± 5.09 ^a	47.33 ± 3.56 ^b	32 ± 2.78 ^b
ALT (unit/ml)	15.071 ± 0.81	14.67 ± 2.04	14.61 ± 0.51
BUN (mg/dl)	42.50 ± 2.74 ^a	25.34 ± 5.64 ^b	30.62 ± 2.41 ^b
Triglyceride (mg/dl)	25.28 ± 2.53 ^a	20.38 ± 5.95 ^a	67.11 ± 2.22 ^b
Cholesterol (mg/dl)	84.15 ± 5.85 ^a	51.79 ± 17.80 ^b	97.96 ± 3.73 ^a
C-reactive protein (mg/dl)	8.12 ± 2.37 ^a	12.00 ± 8.49 ^{ab}	17.14 ± 2.76 ^b

BUN: Blood urea nitrogen.

Values carrying the different letters are significantly different at $P < 0.05$.

Effects of toxoplasmosis and brucellosis on animal performance were revealed that litter size was significantly reduced in goats infected with *Brucella melitensis* compared to healthy and *Toxoplasma gondii* infected animals ($P < 0.05$) as shown in Table 5. However, mortality rates were higher in kids born from does infected with *Toxoplasma gondii* than those born from healthy or *Brucella melitensis* infected does ($P < 0.05$). There were no significant differences between sheep groups either healthy, infected with *Toxoplasma gondii* or *Brucella melitensis* in litter size and mortality rates of lambs. In addition, toxoplasmosis or brucellosis had no effect on maternal body weight of both ewes and does in comparison to healthy ones.

Table 5. Effect of toxoplasmosis and brucellosis on litter size, mortality rate (%) and maternal body weight (kg) in sheep and goats

		Animal condition		
		Healthy	Infected with <i>Toxoplasma gondii</i>	Infected with <i>Brucella melitensis</i>
Sheep:	Litter size	1.56 ± 0.14	1.33 ± 0.32	2.00 ± 0.45
	Mortality rate	6.15 ± 4.75	16.67 ± 10.07	10.00 ± 14.25
	Ewe body weight	45.30 ± 1.20	42.33 ± 2.56	50.00 ± 3.61
Goats:	Litter size	2.00 ± 0.19 ^a	2.33 ± 0.45 ^a	0.67 ± 0.45 ^b
	Mortality rate	10.71 ± 7.25 ^b	27.67 ± 11.07 ^a	3.33 ± 11.25 ^b
	Doe body weight	41.69 ± 1.81	38.67 ± 3.78	31.33 ± 3.78

Values carrying the different letters are significantly different at $P < 0.05$.

5. Discussion

Toxoplasma gondii causes more important reproductive disease in both human and animals as a zoonotic protozoon. *Toxoplasma gondii* causes heavy economic losses to sheep and goats industry worldwide and is considered as one of the main causes of infectious ovine and caprine abortion (Buxton et al., 2007). The spread of ovine toxoplasmosis in Kafr El-Sheikh province as compared with other Nile Delta provinces may be attributed to its climatic condition that was favorable for oocysts survival because toxoplasma infection occurs in temperate sheep-rearing countries worldwide (Aitken, 2007). Significant increase of seroprevalence of *T. gondii* in Kafr El-Sheikh province (>50%) might reflect an increased contamination of the environment (mainly food production or storage places, pastures) with oocysts. The rates of *Toxoplasma gondii* infection in sheep and goats revealed in this study were in agreement with the results of Hassan et al. (2000) who reported that the incidence of *Toxoplasma gondii* infection in Egyptian sheep and goat flocks suffering from abortion were 9.4% to 6.9%, respectively. The toxoplasma prevalence rates found in this study were higher in sheep than goats (31.01% vs. 17.11%). This finding was disagreed with the results reported by Ahmed et al. (2008) and Ramzan et al. (2009) who concluded that goats are more susceptible to toxoplasmosis than sheep due to their higher activity and movement. These activities increase the probability of contact with contaminated sources. These disagreements may attributed to grazing pattern where many sheep flocks were grazed daily while the goats' flocks that examined in this survey were housed. Therefore, the possibility of contact with contaminated pastures and food during grazing was high in sheep flocks. Flock size may consider a significant risk factor because it increases the chance of infection by increasing contact between animals and contaminated sources of infection such as cats' faces. In addition, management of large-sized flocks usually requires more work and effort than small-sized flocks. This finding was consistent with a previous study by Caballero-Ortega et al. (2008) who reported that large flocks had an approximately 2.23-fold higher risk of having positive cases of *Toxoplasma gondii* among aborted animals compared to smaller flocks. Transmission of *Toxoplasma gondii* transplacentally was recorded in one lamb in this study. This agreed with many studies (Morley et al., 2008; Buxton et al., 2007), although toxoplasmosis causes abortion only once in infected animals. Dubey and Kirkbride (1989) reported that 80 ewes produced 144 lambs, 30 of which were born dead and toxoplasma was detected in 11 of the dead lambs.

Brucellosis is a zoonotic disease transmitted by direct or indirect contact with infected animals, soiled pasture and corrals, ingesting infected milk and with semen from male to female animals (Renukaradhya et al., 2002; Al-Majali, 2005). The overall estimation of seroprevalence of *T. gondii* in Nile Delta regions was generally higher than *B. melitensis*. This may be attributed to periodical examination of small ruminants by the Veterinary Service Agency to brucella infection and slaughtering the infected animals. These screening procedures reduce the spread of brucellosis among small ruminant compared to toxoplasmosis. Brucellosis is endemic at high levels among the large ruminant population of the studied village in the Nile Delta region of Egypt, where the main risk factor for cattle and buffalo seropositive status is the presence of sheep or goats in the same household (Hannah et al., 2011).

In addition to their serious effects on sheep and goat performance, Toxoplasmosis and Brucellosis have harmful effects on animals' health as they disturb vital organs' function, which appear in the form of changes in blood biochemical parameters.

Toxoplasmosis and Brucellosis decreased serum total protein level in goat and serum albumin levels in sheep and goat while, the activity of serum ALT was elevated in sheep. These findings were in line with that indicated by (Suzuki, 1971, Portugal et al., 2004, Al-Kaysi et al., 2010; Al-Hussary & Al-Zuhairy, 2010) who, found that serum levels of total protein and albumin were reduced in animals infected with *Toxoplasma* and *Brucella* while, the activities of AST and ALT were increased. However, El-Shazly et al. (2001) and Ustun et al. (2004) found no significant differences in the liver function between infected and non-infected rats. These changes may due to liver damage caused by *Toxoplasma* and *Brucella* which increased the release of liver enzymes into the serum while, decreased albumin synthesis by reticuloendothelium cells in the liver. Blais and Chamberland (1993) and Calderaro et al. (2009) recorded that toxoplasmosis leads to extensive damage in the liver cells and changes their enzymes activities. This liver damage leads to metabolic changes causing decrease in hepatic protein synthesis (Boothroyd et al., 1997; Montoya & Liesenfeld, 2004). Also *Toxoplasma* and *Brucella* may infect and damage kidney, which increase protein excretion in the urine and lead to hypoalbuminemia (Stockham & Scott, 2002).

Toxoplasmosis and Brucellosis increased serum globulin level in sheep. This finding was agreed with that of Abenga and Anosa (2005) and Torda (2001) who indicated that parasite infection increased serum gamma globulins in infected animals that may due to host parasite interaction. However, brucellosis decreased serum globulin level in goat that may due to the harmful effect of brucella on the immune system in the body or due to the difference in the body reaction against the infective agent (Mordue et al., 2001).

Toxoplasmosis and Brucellosis decreased the serum AST activity in sheep and goat this effect was in contrast to the result of previous studies (Suzuki, 1971; Portugal et al., 2004; Al-Kaysi et al., 2010; Al-Hussary & Al-Zuhairy, 2010). This difference may due to the activities of liver enzymes differed according to intensity of the inflammatory condition caused by infective agent (Khan et al., 1997). The other possible cause is that AST is not produced by liver only but also by muscle and heart cells. Thus, any defect in these cells leads to changes in serum AST activity. It was indicated that toxoplasma and brucella infect the heart and muscles and harm their function (Radostits et al., 2007; Al-majali, 2005; Moss & Henderson, 1999).

In addition to their adverse effect on the liver function brucellosis and toxoplasmosis also increased the blood urea nitrogen in sheep that may due to their deleterious effects on the kidney which decrease the excretion of urea from the body and subsequently increased its serum level (Dubey, 1997). Toxoplasma cysts were found in the kidneys of the infected mice and led to many pathological changes in their tissues (Fayed et al., 2004; Hammouda et al., 2006). In contrast, brucellosis and toxoplasmosis decreased blood urea nitrogen in goat this result may due to restricted food intake in infected does or increased food intake in non-infected does. It was reported that intake of protein rich diet increased blood urea nitrogen (Torell et al., 1974)

This study revealed the toxoplasmosis increased serum triglyceride and cholesterol levels in infected ewes. Our finding was in agreement with that of (Stockham & Scott, 2002). Also Djoumessi (1989) and Rimland et al. (2006) found that parasitic and HIV infection elevates lipoproteins like high density lipoprotein (HDL), low density lipoprotein (LDL) and total cholesterol levels. However toxoplasmosis decreased serum cholesterol levels in infected does and this finding was in consistence with that of Azhar et al. (2012) who indicated that *T. gondii* decreases serum cholesterol and triglyceride and LDL levels, while increases serum HDL level. *T. gondii* alters host cell metabolism because it uses host metabolic products for its own metabolic pathways (Al-Kennany et al., 2007) as Toxoplasma cannot synthesize cholesterol and depends upon acquisition of LDL derived from the host cells (Coppens et al., 2000; Portugal et al., 2008). These differences in effect of *toxoplasma gondii* on the triglycerides and total cholesterol levels in sheep and goats may attributed to the difference in the stage of the disease as acute parasitic infection results in decreased levels of HDL and LDL and moderately increased triglycerides (Nilson et al., 1990; Khovidhunkit et al., 2004).

Our study showed that brucellosis increased serum levels of triglyceride and cholesterol in infected ewes and serum triglyceride in infected does and this result agreed with that of Amal (2009) who indicated that Brucellosis increase in plasma total cholesterol, LDL-cholesterol and high level of LDL/HDL ratio in infected patients. Little information is available on the effect of brucella on the plasma lipid profile in infected patients and animals.

Generally, toxoplasmosis and brucellosis did not affect body weight of infected animals. However, litter size was markedly reduced in goats infected with *Brucella melitensis*. Because *Brucella* spp. is known to produce abortion at all stages of gestation and prevalence of brucellosis in this study was higher in goats than sheep. Therefore, this may explain why infected goats with *Brucella melitensis* had few litter size. In current study, mortality rates in both infected goats and sheep with *T. gondii* were high significantly in goats and numerically in sheep in comparison to non infected goats and sheep. The effect of maternal infection with *T. gondii* on the surviving neonates was studied in mice by Maria et al. (2009) who found that mortality increased at first week of age due to maternal infection with *T. gondii*.

6. Conclusion

These results indicated that toxoplasmosis is widely spread than brucellosis in small ruminants living in the provinces of Delta. Both toxoplasmosis and brucellosis had harmful effects on animals' health. Large flock size and grazing system should be considered in the eradication and control of the spread of these agents.

Acknowledgements

This project was supported financially by the Science and Technology Development Fund (STDF), Egypt, Grant No. 736.

References

- Abenga, J. N., & Anosa, V. O. (2005). Serum total protein & creatine levels in experimental Gambian trypanosomosis of Vervet monkeys. *African Journal of Biotechnology*, 4, 187-190.
- Ahmed, Y. F., Sokkar, S. M., Desouky H. M., & Soror A. H. (2008). Abortion due to toxoplasmosis in small ruminants. *Global Veterinaria*, 6, 337-342.
- Aitken, I. D. (2007). *Diseases of Sheep* (4th ed., pp. 137-142). Oxford: Blackwell. <http://dx.doi.org/10.1002/9780470753316>

- Al-Hussary, N. A. J., & Al-Zuhairy, A. S. M. (2010). Effect of toxoplasmosis and brucellosis on some biochemical parameters in ewes. *Iraq Journal of Vet. Sci.*, 24, 73-80.
- Al-Kaysi, A. M., Eid, R. A. A., Fahmy, B. G. A. (2010). Biochemical studies on the effect of *Toxoplasma* infection on liver and kidney functions in mice. *Egypt. J. Comp. Path. & Clinic. Path.*, 23, 174-185. <http://dx.doi.org/10.1016/j.smallrumres.2004.07.013>
- Al-Kennany, E. R. (2007). Pathological study on the capability of *Toxoplasma gondii* to induce oxidative stress and initiation a primary lesion of atherosclerosis experimentally in broiler chickens. *J. Anim. Veter. Adv.*, 6, 938-42.
- Al-Majali, A. M. (2005). Seroepidemiology of caprine Brucellosis in Jordan. *Small Rumin. Res.*, 58, 13-18.
- Al-Sim'ani, R. G. G. (2000). *A seriological study to diagnose Toxoplasmosis in sheep and human in Ninevah governorate*. M.Sc. Thesis, College of Veterinary Medicine, University of Mosul.
- Amal H. A. (2009). The Effect of Brucellosis on Lipid Profile and Oxidant-Antioxidants Status. *Iraqi J Pharm Sci.*, 18, 26-31.
- Aydin, N. (1997). Brucella infections. In M. ARDA (Ed.), *Veterinary microbiology*, Medisan Ltd. Sti., Ankara, 110.
- Azhar, H. A., Huda, H. H., & Shatha, M. J. A. (2013). Lipid Profile Changes in Toxoplasmosis Aborted Women. *J. Baghdad for Sci.*, 10, 168-175.
- Blais, J., & Chamberland. (1993). Inhibition of *Toxoplasma gondii* protein synthesis by azithromycin. *Antimicrob. Chemo.*, 37, 1701-1703. <http://dx.doi.org/10.1098/rstb.1997.0119>
- Blood, D. C., Radostits, O. M., & Henderson, J. A. (1986). *Veterinary Medicine* (6th ed.). Philadelphia: Bailliere Tindall. <http://dx.doi.org/10.1016/j.vetpar.2007.07.003>
- Boothroyd, J., Black, M., Bonnefoy, S., Hehl, A., Manger, I., & Tomavo, S. (1997). Genetic and biochemical analysis of development in *Toxoplasma gondii*. *Phil. Trans. R. Scotland*, 352, 1347-1354.
- Burgu, I., Urman, H. K., Akca, Y., Yonguc, A., Mellor, P. S., & Hamblin, C. (1992). Serological survey and vector surveillance for bluetongue in southern Turkey. In E. T. WALTON & B. I. OSBURN (Eds), *Proceedings of The Second International Symposium on Bluetongue, African Horse Sickness & Related Orbiviruses* (pp. 168-174). Boca Raton, Florida: CRC Press.
- Buxton, D., Maley, S. W., Wright, S. E., Rodger, S., Bartley, P., & Innes, E. A. (2007). *Toxoplasma gondii* and ovine toxoplasmosis: new aspects of an old story, *Veterinary Parasitology*, 149, 25-28.
- Caballero-Ortega, H., Palma, J. M., García-Márquez, L. J., Gildo-Cárdenas, A., & Correa, D. (2008). Frequency and risk factors for toxoplasmosis in ovines of various regions of the State of Colima, Mexico. *Parasitology*, 135, 1385-1389. <http://dx.doi.org/10.1017/S0031182008004873>
- Calderaro, S., Peruzzi, S., Piccolo, G., Montecchioni, S., Rossi, S., Chezz, C., & Eeltori, G. (2009). Laboratory diagnosis of *Toxoplasma gondii* infection. *Int. J. Med. Sci.*, 6, 135-136. <http://dx.doi.org/10.7150/ijms.6.135>
- Coppens, I., Sinai, A. P., & Joiner, K. A. (2000). *Toxoplasma gondii* exploits host low-density lipoprotein receptor-mediated endocytosis for cholesterol acquisition. *J. Cell Biol.*, 149, 167-180. <http://dx.doi.org/10.1083/jcb.149.1.167>
- Djoumessi, S. (1989). Serum lipids and lipoproteins during malaria infection. *Pathol. Biol.*, 37, 909-11.
- Dubey, J. P. (1997). Tissue cyst tropism in *Toxoplasma gondii*: a comparison of tissue cyst formation in organs of cats, and rodents fed oocysts. *Parasitology*, 115, 15-20. <http://dx.doi.org/10.1017/S0031182097008949>
- Dubey, J. P. (2009). Toxoplasmosis in sheep the last 20 years. *Veterinary Parasitology*, 163, 1-14.
- Dubey, J. P., & Kirkbride, C. A. (1989). Economic and public health considerations of congenital toxoplasmosis in lambs. *J. Am. Vet. Med. Assoc.*, 195, 1715-6.
- El-Shazly, A. M., Soliman, M., El-Kalla, M. R., Rezk, H., El-Nemr, H., Hgoussa, A. E., ... Morsy, T. A. (2001). Evaluation of soluble adhesion molecules in the diagnosis of amoebiasis, giardiasis and toxoplasmosis. *J. Egypt Soc Parasitol*, 31, 691-700.
- Farrell, I. D., & Robertson, E. (1975). Serum antibody response in acute brucellosis. *J. Hyg.*, 74, 23. <http://dx.doi.org/10.1017/S0022172400046672>

- Fayed, H. M., Allam, K. A., & Ali, N. E. (2004). Merogony of *Toxoplasma gondii* (Apicomplexa: Coccidia) and its effect on the mortality and histopathology in the house mouse *Mus musculus*. *J. Egypt Soc. Parasitol.*, 34, 45-64.
- Fieni, F., Rowe J., Van-Hoosear, K., Burucoa, C., Oppenheim, S., Anderson, G., ... Bondurant, R. (2003). Presence of caprine arthritis-encephalitis virus (CAEV) proviral DNA in genital tract tissues of superovulated dairy goat does. *Theriogenology*, 59, 1515-1523.
- Freyre, A., Benino, J., Falcon, J., Caslells, D., Correa, O., & Casaretto, A. (1999). The incidence and Economic Significance of ovine toxoplasmosis in Uruguay. *Vet. Parasitol.*, 81, 85-88.
- Garin-Bastuji, B., Blasco, J. M., Marin C., & Albert, D. (2006). The diagnosis of brucellosis in sheep and goats, old and new tools. *Small Ruminant Research*, 62, 63-70. <http://dx.doi.org/10.1016/j.smallrumres.2005.08.004>
- Hammouda, N. A., Amin, S. M., Khalifa, A. M., Abou-El, N. I., Gaafar, M. R., & Nasr, M. A. (2006). The use of ELISA and immunohistochemistry techniques for detection of *Toxoplasma gondii* antigen in tissues of experimentally infected mice. *J. Egypt Soc. Parasitol.*, 36, 925-935.
- Hannah, R. H., Mahmoud, M. E., Yamen, M. H., Wael, F. E., Ahmed, A. T., & Javier, G. (2011). *Brucella* spp. infection in large ruminants in an endemic area of Egypt: cross-sectional study investigating seroprevalence, risk factors and livestock owner's knowledge, attitudes and practices (KAPs). *BMC Public Health*, 11, 341. <http://dx.doi.org/10.1186/1471-2458-11-341>
- Hassan, H. M., Abd El-Aal, A., Ghazy, A. A., & Malaka, F. L. (2000). Seroprevalance of *Neospom caninum* and *Toxoplasma gondii* antibodies in sheep and goat in Egypt. *J. Egyptian Veterinary Medical Association*, 60, 19-24.
- Jahangiri, A., de Beer, M. C., Noffsinger, V., Tannock, L. R., Ramaiah, C., Webb, N. R., ... de Beer, F. C. (2009). HDL remodeling during the acute phase response. *Arterioscler Throm.Vasc.Biol.*, 29, 261-267. <http://dx.doi.org/10.1161/ATVBAHA.108.178681>
- Khan, I. A., Schwartzman, J. D., Matsuura, T., & Kasper, L. H. (1997). A dichotomous role for nitric oxide during acute *Toxoplasma gondii* infection in mice. *Proc. Natl. Acad. Sci. USA*, 94, 13955-13960. <http://dx.doi.org/10.1073/pnas.94.25.13955>
- Khovidhunkit, W., Kim, M. S., Mmon, R. A., Shigenaga, J. K., Moser, A. H., Feingold, K. R., & Grunfeld, C. (2004). Effects of infection and inflammation on lipid and lipoprotein metabolism: mechanisms and consequences to the host. *J. Lipid Res.*, 45, 1169-1196. <http://dx.doi.org/10.1194/jlr.R300019-JLR200>
- Maki, Y., Seng, S., Kato, M., Hoshi, Y., Igarashi, I., Nagasaw, H., ... Suzuki, N. (1996). Sequence analysis of three major antigens (P30.P23.P22) of Virulent strains of *Toxoplasma gondii*. *J. Protozoo. Res.*, 6, 83-93.
- Maria, A. C., Elba, R., Laura, M., Maria, L. D., Luis, J., & Ethel, G. (2009) *Toxoplasma gondii*: Effect of maternal infection in the development of lymphoid organs of BALB/c neonates. *Experimental Parasitology*, 121, 279-287. <http://dx.doi.org/10.1016/j.exppara.2008.11.015>
- Maxfiel, F. R. (2002). Plasma membrane microdomain. *Curr. Opin. Cell Biol.*, 14, 483-87. [http://dx.doi.org/10.1016/S0955-0674\(02\)00351-4](http://dx.doi.org/10.1016/S0955-0674(02)00351-4)
- McGullicuddy, F. C., de la Llera Moy, M., Hinkle, C. C., Joshi, M. R., Chiquoine, E. H., Billheimer, J. T., ... Reilly, M. P. (2009). Inflammation impairs reverse cholesterol transport in vivo. *Circulation*, 119, 1135-1145. <http://dx.doi.org/10.1161/CIRCULATIONAHA.108.810721>
- Montoya, J., & Liesenfeld, O. (2004). Toxoplasmosis. *Lancet*, 363, 1965-1976. [http://dx.doi.org/10.1016/S0140-6736\(04\)16412-X](http://dx.doi.org/10.1016/S0140-6736(04)16412-X)
- Mordue, D. G., Monroy, F., La Regina, M., Dinarello, C. A., & Sibley, L. D. (2001). Acute toxoplasmosis leads to lethal overproduction of The cytokines. *J. Immunol.*, 167, 4574-4584.
- Morley, E. K., Williams, R. H., Hughes, J. M., Thomasson, D., Terry R. S., Duncanson, P., ... Hide, G. (2008). Evidence that primary infection of Charollais sheep with *Toxoplasma gondii* may not prevent foetal infection and abortion in subsequent lambings, *Parasitology*, 135, 167-173. <http://dx.doi.org/10.1017/S0031182007003721>
- Moss, D. W., & Henderson, A. R. (1999). Clinical enzymology. In C. A. Burtis & E. R. Ashwood (Eds.), *Tietz-Textbook of Clinical Chemistry* (pp. 617-721). USA: W.B.Sounders Co.

- Nilson, E. I., & Nilson, E. P. (1990). Changes in plasma lipoproteins in acute malaria. *J. Intern. Med.*, 227, 151-155. <http://dx.doi.org/10.1111/j.1365-2796.1990.tb00136.x>
- Portugal, L. R., Femanries, L. R., Cesar, Ge, Santiagjç, Oliveira, D. R., Silva, N. M., Silva, A. A., Gazzinelli, R. T., & Alvarez-Leite, J. I. (2004). Infection with *Toxoplasma gondii* increases atherosclerotic lesion in Apo E-deficient mice. *Infect. Immun.*, 76, 6571-5676.
- Portugal, L. R., Fernandes, L. R., Pietra Pedroso, V. S., Santiago, H. C., Gazzinelli, R. T., & Alvarez-Leite, J. I. (2008). Influence of low-density lipoprotein (LDL) receptor on lipid composition, inflammation and parasitism during *Toxoplasma gondii* infection. *Microbes Infect.*, 10, 276-284. <http://dx.doi.org/10.1016/j.micinf.2007.12.001>
- Radostits, O. M., Gay, C. C., Hinchcliff, K. W., & Constable, P. D. (2007). Veterinary Medicine: A textbook of the diseases of cattle, horse, sheep, pigs & goat. *Saunders*, 26, 1518-1522.
- Ramzan, M., Akhtar, M., Muhammad, F., Hussain, I., Hiszczyńska-Sawicka, E., Haq, A.U., Mahmood, M. S., & Hafeez, M. A. (2009). Seroprevalence of *Toxoplasma gondii* in sheep and goats in Rahim Yar Khan (Punjab), Pakistan, *Tropical Animal Health and Production*, 41, 1225-1229. <http://dx.doi.org/10.1007/s11250-009-9304-0>
- Remington, J. S., Mcleod, R., Thulliez, P., Desmants, G., & Toxoplasmosis. (2000). In J. S. Remington, & J. O. Klein (Eds.), *Infectious Diseases of the fetus and Newborn Infant* (5th ed., pp. 206-346). W.B. Saunders. Co. Philadelphia.
- Renukaradhya, G. J., Isloor, S., & Rajasekhar, M. (2002). Epidemiology, zoonotic aspects, vaccination and control/eradication of brucellosis in India. *Vet. Microbiol.*, 90, 183-195. [http://dx.doi.org/10.1016/S0378-1135\(02\)00253-5](http://dx.doi.org/10.1016/S0378-1135(02)00253-5)
- Rimland, D., Guest, J. L., Herná'ndez-Ramos, I., Del Rio, C., Le, N. A., & Brown, W. V. (2006). Antiretroviral therapy in HIV-positive women is associated with increased apolipoproteins and total cholesterol. *J Acquir. Immune. Defic. Syndr.*, 42, 307-313. <http://dx.doi.org/10.1097/01.qai.0000220164.72113.12>
- Shareef, J. M. (2006). A Review of Serological Investigations of Brucellosis among Farm Animals and Humans in Northern Provinces of Iraq (1974-2004). *J. Vet.Med. B.*, 53, 38-40. <http://dx.doi.org/10.1111/j.1439-0450.2006.01021.x>
- Stockham, S. L., & Scott, M. A. (2002). *Fundamentals of veterinary clinical pathology* (pp. 251-277). USA: Iowa State Press.
- Suzuki, N. (1971). Biochemical observation on rats infected with *Toxoplasma gondii*. *Res. Bull. Obihiro. Univ.*, 7, 15-25.
- Torda, A. (2001). Toxoplasmosis. Are cats really the source? *Aust. Fam. Physician*, 30, 743-747.
- Torell, D. T., Hume, I. D., & Weir, W. C. (1974). Factors affecting blood urea nitrogen and its use as an index of the nutritional status of sheep. *J. Anim. Sci.*, 39, 435-440.
- Tütüncü, M., Ayaz, E., Yaman, M., & Akkan, H. A. (2003). The seroprevalance of *toxoplasma gondii* in sheep, goats and cattle detected by indirect hemagglutination (IHA) test in the region of Van, Turkey. *Indian Vet. J.*, 80, 401-403.
- Ustun, S., Aksoy, U., & Daqci, F. (2004). Incidence of toxoplasmosis in patients with cirrhosis. *World J. Gastroenterol.*, 10, 452-454.
- Young, D. S. (2001). *Effects of disease and clinical lab testes* (4th ed AACC).

Copyrights

Copyright for this article is retained by the author(s), with first publication rights granted to the journal.

This is an open-access article distributed under the terms and conditions of the Creative Commons Attribution license (<http://creativecommons.org/licenses/by/3.0/>).