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REPRODUCTION AND *TRYPANOSOMA CONGOLENSE* IN NIGERIAN WEST AFRICAN DWARF EWES: II. GENITAL AND ENDOCRINE LESIONS

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Abstract

Aim: The study was designed to determine the effect of *Trypanosoma congolense* on the genital and endocrine organs of West African Dwarf (WAD) ewes.

Methods: Ten WAD ewes obtained for the study were divided into two groups comprising 5 ewes each. Group A was infected with *Trypanosoma congolense*, while group B was uninfected and allowed to run for eight weeks. At the end of the study period, three infected and two control ewes were sacrificed. The weights of the ovaries were determined and sections taken for histopathological examination. Sample sections from other parts of the reproductive tract, brain, pituitary gland, and hypothalamus were also taken.

Results: The mean ovarian weight, number of follicles and number of corpera lutea of the infected ewes decreased compared to the control ewes but were not statistically significant ($p > 0.05$). No parasites were seen following impression smears conducted on the ovaries. However, there were slight areas of necrosis and mild degeneration of the ovarian stroma. These were absent in the control ewes. No detectable gross lesions were seen in the adenohypophysis of both the infected and control ewe, although slight areas of focal necrosis were seen at histology. There were no detectable gross and histological lesions in the uterus, cervix, vagina, brain and hypothalamus of both infected and control ewes.

Conclusion: The findings from this study are of great importance for the economic exploitation of WAD sheep in tsetse infected area.

Keywords: Adenohypophysis, Ovaries, *Trypanosoma congolense*, WAD Ewe.

INTRODUCTION

Trypanosoma congolense is a haemoparasite affecting cattle, pigs, goats, sheep, horses, and dogs (OIE, 2013). It is a pathogenic parasite with two known strains, one from West Africa and the other from East Africa. However, the West African strain is more pathogenic (Osaer *et al*., 1994). *T. congolense* is transmitted biologically (Mbaya et al, 2012), although mechanical and congenital transmissions have been reported (Griffin, 1983; Desquesnes & Dia, 2003). Pregnant animals infected with *T. congolense* may abort or give birth to weak neonates (Faye et al., 2004). There is anorexia, anaemia, lacrimation, weight loss, weakness and death of the dam in some instances (Llewelyn et al, 1987). In non-pregnant females, there is anestrus (Llewelyn et al., 1988) and genital lesions (Ogwu and Njoku, 1991). Infected males show pathological changes characterized by testicular degeneration, penile protrusion, haemorrhage, prepucial inflammation, decrease testosterone levels, increase cortisol concentration and depressed pituitary and adrenocortical functions (Adeyemo et al., 1990; Sekoni et al. 1990; Raheem et al., 2009; Victor et al. 2012; Okubanjo et al. 2014; Okubanjo et al. 2015). The pathogenesis of trypanosomosis-induced reproductive losses has been the subject of numerous researches (Ogwu *et al*., 1986; Edeghere et al., 1992; Faye et al., 2004; Leigh and Fayemi, 2013; Silver *et al*., 2013; Allam *et al*., 2014;

Adeyeye *et al*., 2016a), with a few reviews available on the disease (Ikede *et al*., 1988; Sekoni, 1994; Raheem, 2014). The mechanism responsible for these losses is not widely known. However, Bawa (2000) suggested fetal hypoxia and stress, pyrexia, anemia and direct invasion of body tissues as possible mechanisms. The attack on the body tissues is characterized by pathological changes. *T. congolense* mainly resides in plasma (Seifert, 1996) and is believed to cause injury by anaemia without major histological changes on the tissue (Ikede and Loses, 1972). However, Ogwu and Njoku (1991) reported histopathological changes in *T. congolense* infected heifers. To the best of our knowledge, no study has been designed to ascertain this position in West African Dwarf ewe which is a trypanotolerant breed (Geerts *et al*., 2009). This study was carried out to determine the gross and histopathological changes associated with *T. congolense* infection in the genital and endocrine organs of WAD ewes.

MATERIALS AND METHODS

Experimental animals

Ten matured non-pregnant but cycling West African Dwarf (WAD) ewes obtained from the Small Ruminant Research Program, National Animal Production Research Institute, Shika-Zaria, Nigeria. They were selected from the sheep stock of the institute, and were initially used to study the effect of *Trypanosoma congolense* on the oestrous cycle of WAD, their management has therefore been described in Abubakar *et al*. (2015).

Study design

They were divided into two groups comprising 5 ewes each. Group A was infected with *Trypanosoma congolense* while group B was uninfected, and the study ran for eight weeks. The parasite used was obtained from the Department of Veterinary Parasitology and Entomology, Ahmadu Bello University Zaria, Nigeria.

Pathological examination

At the end of the study period, three infected and two control ewes were humanly euthanized and necropsied. The necropsy was carried out to examine for gross lesions on the reproductive tract and the endocrine glands. The weights of the ovaries were determined and sections taken for histopathological examination. Sample sections from other parts of the reproductive tract, brain, pituitary gland, and hypothalamus were also taken. All these were fixed in Bouin`s solution and used for histopathological evaluation.

RESULTS

The infected ewes had a pre-patent period of 10.2 ± 1.2 days. Other clinical signs were undulating parasitemia, intermittent pyrexia, anemia and emaciation. The mean ovarian weight, number of follicles and number of corpera lutea of the infected ewes is presented in Table 1. These parameters decreased compared to the control ewes but were not statistically significant ($p > 0.05$). Petechial haemorrhage was observed on the left ovary of one of the infected ewe. No parasites were seen following impression smears conducted on the ovaries. However, they had slight areas of necrosis and mild degeneration of the ovarian stroma. These were absent in the infected ewes. No detectable gross lesions were seen in the adenohypophysis of both infected and control ewe (Figure 1), although some slight areas of focal necrosis were seen histologically in the infected ewes (Figure 2). There were no detectable gross and histological lesions in the uterus, cervix, vagina, brain and hypothalamus of both infected and control ewes.

DISCUSSION

The ovarian weights of the infected ewes decreased but were not substantially different from the uninfected ewes. Similarly, the number of follicles and corpera lutea of the infected ewes did not differ from ewes in the control. Our observation contradicts the report of Isoun and Anosa (1974) as well as Adenowo *et al*. (2005) in *T. vivax* infected ewes, where substantial decreases were observed. Likewise, ovarian atrophy, reduction in number of follicles and corpera lutea has also been reported in *T. congolense* infected goats (Mutayoba *et al*., 1988) and heifers (Ogwu and Njoku, 1991), as well as in

Table 1: Mean±SEM ovarian weight, number of follicles and number of corpora lutea in *Trypanosoma congolense* infected WAD ewes

	Mean ovarian weight	Mean number of follicles	Mean number of corpora lutea
Infected ewes (n=3)	1.33 ± 0.06	2.00 ± 0.29	1.50 ± 0.09
Control ewes (n=2)	1.45 ± 0.05	3.25 ± 0.43	1.75 ± 0.31

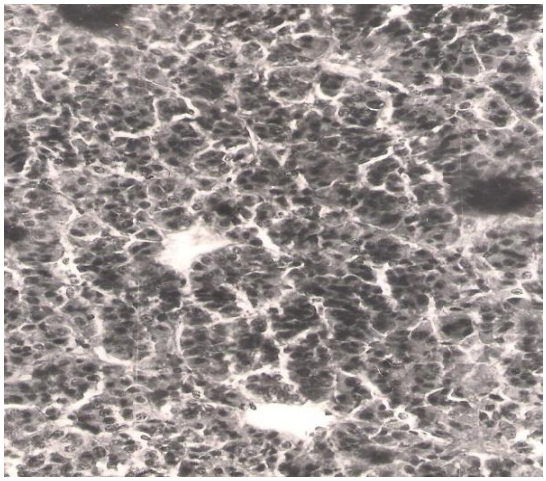

Fig 1: Adenohypophysis of a the uninfected WAD ewe showing normal parenchymal cells (H&E x 400).

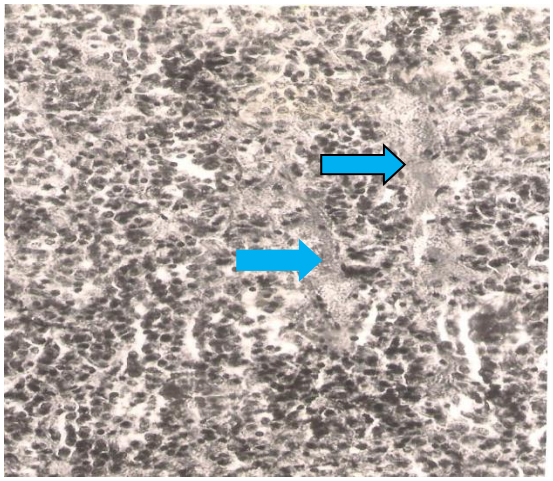

Fig 2: Adenophysis of a *T. congolense* infected WAD ewe showing focal necrosis (blue arrows) of the parenchymal cells (H&E x 400).

T. vivax infected goats (Rodrigues *et al*., 2013) and cattle (Ige and Amodu, 1975). The contradictions may be attributed to the trypanotolerant nature of WAD ewes. There was petechial haemorrhage on the left ovary of one of the infected ewe. Since this was not generalized nor found in other infected ewes, it maynot be attributed to *T. congolense* infection in this study. Slight areas of necrosis and mild degeneration of the ovarian stroma were observed histologically on the ovaries of the infected ewes. In other trypanosomosis susceptible animals, lesions ranging from fibrosis and degeneration of ovarian stroma and follicular cyst with atretic follicles have been reported (Mutayoba *et al*., 1988; Ogwu and Njoku, 1991; Adenowo *et al*., 2005), probably due to the breed variation suggested earlier. In the adenohypophysis, no gross lesions were seen among the infected ewes, although slight areas of focal necrosis were observed at histology. In *T. vivax* infected Yankasa ewes, Adenowo *et al*. (2005) reported mononuclear cell infiltration in the capsules and the parenchyma along with necrosis of the parenchyma cells of the adenohypophysis. These changes were observed in *T. evansi* infected ewes (Adeyeye, 2016b) as well as in goats infected with *T. congolense* (Mutayoba *et al*., 1988) and *T. brucei* (Leigh *et al*., 2015). Lesions on the adenohypophysis will lead to impairment in the release of gonadotropin-releasing hormone (GnRH) which is responsible for stimulating the production of follicular stimulating hormone (FSH) and luteinizing hormone (LH). This leads to impaired estrus cycle which was observed in our earlier report (Abubakar *et al*., 2015). The uterus, cervix, vagina, brain and hypothalamus had no detectable gross or histological changes. This is similar to the reports of (Adeyeye *et al*, 2016b) in *T. evansi* infected ewes, except for the hypothalamus which they reported had neuronal degeneration and infiltrated by microglial cells. Our observations disagree with Adenowo *et al*. (2005), who reported lesions in the hypothalamus of *T. vivax* infected ewes. Leigh *et al*. (2015) also reported pathological lesions in the uterus and hypothalamus of WAD does infected with *T. brucei*. The variation in sheep breeds maybe responsible for this difference with Adenowo *et al*. (2005). Although WAD goats are also trypanotolerant, Sheep have been observed to be more trypanotolerant than goats (Boid et al., 1981). This probably explains our variation with Leigh *et al*. (2015). In conclusion, infection of WAD ewes with *T. congolense* showed no marked genital and endocrine lesions, in contrast with observations made in previous studies using other sheep breeds and *trypanosoma* species. It is therefore suggested that WAD ewes are capable of controlling the pathological effect of *T. congolense*. The phenomenon of trypanotolerance in WAD sheep might have contributed to the relatively mild pathological lesion on the genital and endocrine organs. This finding is of great importance for the economic exploitation of this breed of sheep in tsetse infected area.

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