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ORIGINAL ARTICLE

Modification of erythrogram, testosterone and prolactin profiles in jacks following experimental infection with *Trypanosoma congolense*

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Abstract

Equine trypanosomosis, a haemoparasitic disease leads to anaemia, low testosterone and prolactin levels. The aim of this study was to examine the effect of experimental *Trypanosoma congolense* (*T. congolense*) infection in jacks on erythrogram, testosterone and prolactin profiles. Four apparently healthy jacks aged 6–7 years, housed in fly-proof stables, were used for this study. To establish infection, they were, each, intravenously inoculated with 2 ml of blood from infected donor jack containing 2.0×10^6 trypanosomes. Daily rectal temperatures were measured, for 2 weeks pre-infection and 4 weeks post-infection. Parasitaemia was measured daily and scored. Blood of 3 ml was collected from jugular venipuncture, twice in a week at 09:00 h for 2 weeks pre-infection and 4 weeks post-infection and 1 ml is used for erythrogram analysis, while 2 ml was used for testosterone and prolactin assay using competitive enzyme-linked immunosorbent assay kits (ELISA). *T. congolense* infection, caused by variation of rectal temperatures, from day 4 post-infection, were significantly higher ($P < 0.05$) than those of pre-infection. Parasitaemia was detected on day 3 post-infection reaching peak on day 9 post-infection. There was a significant decrease in packed cell volume (PCV), haemoglobin concentration (Hgb) and red blood cell count (RBC) ($P < 0.05$) from pre-infection to post-infection. The average mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH) and mean corpuscular haemoglobin concentration (MCHC) decreased non-significantly ($P > 0.05$) from day 5 post-infection compared to pre-infection period. The mean average testosterone decreased significantly ($P < 0.05$) from day 5 post-infection, throughout the experimental period compared to pre-infection values; also the mean average prolactin increased significantly ($P < 0.05$) from day 5 post-infection compared to pre-infection values. It was concluded that experimental *T .congolense* infection in jacks altered testosterone, prolactin and erythrogram.

Keywords Erythrogram · Experimental infection · Jacks · Prolactin · *T. congolense* · Testosterone

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Introduction

Donkeys or asses (*Equus asinus asinus*) provide draught power (ploughing, hauling) and transport (riding, cart pulling), contributing substantially to people's livelihoods in terms of food, security, mobility and income generation (Blench et al. 2013; Dimpho and Ruishan 2020). It is a domesticated member of the horse family, Equidae (Grubb 2005; ICZN 2003). They are largely found in the arid and semi-arid areas, providing a reliable, ecologically friendly and renewable source of power to millions of poor communities (Blench et al. 2004; The Donkey Sanctuary 2017). Their population is estimated to be 43.5 million worldwide (FAO 2016), majority of which are working animals, supporting people living in the world's poorest communities, by transporting a wide

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range of goods and materials by pack or cart (Pritchard et al. 2009; Hassan et al. 2013). Approximately, 95% of the world's donkey population can be found in developing countries (FAO 2013) and they constitute 70% of the equine species in Africa.

African animal trypanosomes are intravascular, extracellular haemo-parasites, belonging to the genus *Trypanosoma*, affecting livestock, humans and wild animals (Jill et al. 2020). African animal trypanosomosis are caused by *Trypanosoma vivax* (*T. vivax*); *T. brucei* and *T. congolense* (Irungu et al. 2002; Taylor and Authié 2004). These trypanosomes are transmitted biologically or cyclically through the bite of tsetse fly (*Glossina* spp.) and/or mechanically by bites of other hematophagous Diptera of genera *Tabanus*, *Stomoxys*, *Hematopota* and *Lyperosia* (Itard 1989; Ahmed et al. 2016). The disease is widespread in Africa and occurs in 37 sub-Saharan countries, covering more than 9 million km^2, corresponding approximately to one-third of Africa's total land mass (Maudlin 2006). Trypanosomosis is recognised as one of the major factors limiting livestock production in Africa especially in the sub-Saharan region (Swallow 2000; Shaw et al. 2014; Raftery et al. 2019). It has caused economic losses in livestock production in Africa, the Middle East, Asia and Latin America (Maudlin 2006; Büscher et al. 2019). It is a potential impediment to food security in Nigeria (Samdi et al. 2010).

In equines (mules, donkeys and horses), infection leads to equine trypanosomosis also known as durin, nagana and surra, characterised by overlapping clinical features including anaemia, generalised lymphadenitis, intermittent anorexia, episodic pyrexia, lethargy and progressive loss of body condition (Blood and Radostits 2007; Samuel et al. 2016; Coetzer and Tustin 2005). Equine trypanosomosis has various means of transmission; dourine is caused by *T. equiperdum* and is transmitted sexually, nagana is caused by *T. vivax*, *T. congolense* and/or *T. brucei* subspecies and is transmitted by tsetse flies and surra is produced by *T. evansi* and transmitted mechanically by biting flies (Blood and Radostits 2007; Coetzer and Tustin 2005).

The sire, an important contributor to herd genetic potential subfertility in male, has a greater impact on the overall herd fertility (Healy et al. 1993). Testosterone produced by the interstitial cells is needed for spermatogenesis, sperm maturation, development of male secondary characteristics and libido (Khisk 2008; Clark et al. 2018). Prolactin is secreted by lactotroph, in the anterior pituitary gland, and plays a role in the synthesis of testosterone through upregulation and increased sensitivity of LH receptors on Leydig cells (Bole-Feysot et al. 1998; Dombrowicz et al. 1992; Bernard et al. 2019). Studies have revealed the involvement of central nervous system in trypanosomosis and impairment of the hypothalamic-pituitary-gonadal axis (Batista et al. 2007; Abebe et al. 1993). African animal trypanosomosis induced local inflammation of the tuberal region of the hypothalamus, pituitary gland and gonads; this is linked with rise in serum level of cytokines and generalised tissue damage which may reduce the amount of GnRH released from the hypothalamus (Adenowo et al. 2005; Reincke et al. 1998). This effect will alter the serum levels of testosterone and prolactin in the affected animals which consequently will reduce their reproductive efficiency (Noakes et al. 2009). The alteration of testosterone, prolactin and erythrogram profiles following *T. congolense* infection has been reported in other domestic animal but scarcely in jacks. This study examined changes that occur in the testosterone, prolactin and erythrogram profiles of jacks following experimental infection with *T. congolense*. This could be used to suggest the effect of *T. congolense* on the fertility of jacks.

Materials and methods

Ethical statement

This work was carried out in line with the ethical guidelines approved by the Ahmadu Bello University Committee on Animal Welfare and Use.

Experimental animals

Four apparently healthy adult jacks, aged 6–7 years, were purchased from Sheme market, in Katsina State, Nigeria, which was known to be tsetse-free and were used for this study. The jacks were housed in fly-proof stables, fed with woolly finger grass (*Digitaria smutsii*) and signal grass (*Brachiaria decumbens*) and supplemented with maize and sorghum bran. They were given access to water ad libitum during the experimental period. The donkeys were treated against ecto- and endoparasites using ivermectin and albendazole at the dose rate of 200 and 10 mg/kg (Imam et al. 2010). The jacks were acclimatised for a week and were ear tagged for proper identification.

The trypanosome stock

The *T. congolense* stock was obtained from the Nigerian Veterinary Research Institute (NVRI), Vom in Plateau State, Nigeria. The parasite was inoculated into two mice, intramuscularly and the other intraperitoneally, and transported to the Faculty of Veterinary Medicine, where the experiment was carried out.

Inoculation of donor donkey

On arrival, the infected mice were kept in a laboratory and fed adequately. Blood samples were collected daily to monitor

parasitaemia using the method described by Woo (1970) and as estimated using the modified method of Paris et al. (1982) as described by Mutayoba et al. (1994). Parasitaemia was detected in the mice 7 days post-infection. When parasitaemia was 1.0×10^6 trypanosomes/ml on day 9 post-infection, one of the mice was euthanized by severing its jugular vein, following chloroform anaesthesia, to obtain blood that was used to intravenously infect a donor donkey.

Animal allocation and infection

Consequent to the infection of the donor jack with 1 ml of blood containing 1×10^6 *T. congolense* parasite, 0.5 ml of blood was collected from the donor jack and tested daily for the appearance of the parasites. *T. congolense* parasites were first observed in the blood of the donor jack on day 7 post-inoculation. Peak parasitaemia was observed on day 9 post-inoculation. The experimental jacks were, each, administered with 2 ml of blood from the donor jack containing 2.0×10^6 trypanosomes (Sekoni et al. 2004) as estimated using the modified method of Paris et al. (1982) as described by Mutayoba et al. (1994) and was tagged day 0 of infection.

Measurement of rectal temperature

The temperature of the jacks were measured daily at the same hour for 2 weeks pre-infection and throughout post-infection from day 0 to day 28 using rectal digital thermometer. The rectal temperatures were measured in degree Celsius.

Measurement of parasitaemia in the experimental jacks

After the infection of the experimental jacks, ½ ml of blood was collected daily from the jugular vein (Ahmed et al. 2020; Abdelmoneim et al. 2020) of each jack for the detection of *T. congolense* parasite and to establish infection. Parasitaemia (1×10^6 trypanosomes/ml) was detected in the blood of one of the infected jacks 4 days post-infection and at day 5 post-infection, all the infected jacks were parasitaemic. Parasitaemia was measured daily and scored from day 0 throughout the experimental period that lasted for 28 days (4 weeks) by the method described by Woo (1970) and scored using the modified method of Paris et al. (1982) as described by Mutayoba et al. (1994).

Determination of the erythrogram parameters

One millilitre of blood was collected through jugular vein (Mohamed et al. 2018; Essam et al. 2020), twice in a week, for 2 weeks pre-infection and 4 weeks post-infection, from each of the experimental jacks. The blood was dispensed into screw-capped test tube containing ethylenediaminetetraacetic acid (Prasanna et al. 2017; Amany et al. 2019; Amany et al. 2020) and used for erythrogram analysis according to methods described by Woo (1970) and Coles (1974), respectively.

Determinations of testosterone and prolactin concentrations

Two millilitres of blood samples were collected twice in a week at 09:00 h from each jack for 2 weeks pre-infection period and 4 weeks post-infection period. Each blood sample was drawn aseptically by jugular venipuncture using 18-gauge needles and 10-ml syringes and emptied into a plain tube. Serum was separated by centrifugation at 2000 × g for 10 min, after which it was harvested and kept frozen at − 20 °C until analysis (Ramnath et al. 2008).

Serum testosterone was determined using competitive ELISA kits (AccuBind^B ELISA, Monobind Inc. 100 North Pointe Drive Lake Forest, CA 92630, USA), intended for a quantitative determination of testosterone concentration in serum or plasma using ELISA microplate reader (EL × 800). The sensitivity of the assay was 0.0576 ng/ml. Within assay precision, coefficients of variation for low, normal and high

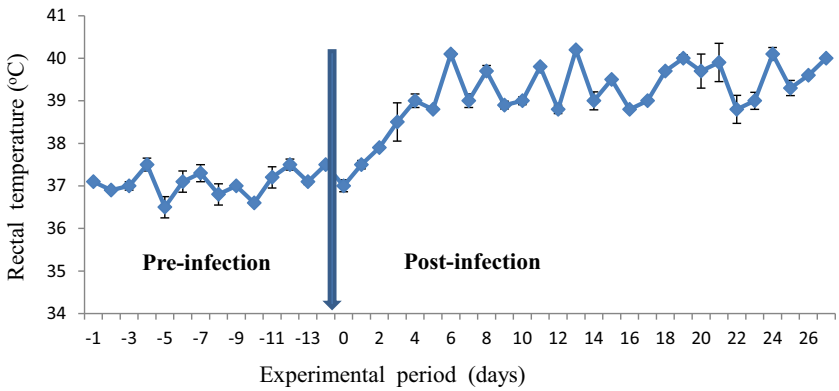

Fig. 1 Rectal temperature variation at pre- and post-infection period following experimental *T. congolense* infection in jacks

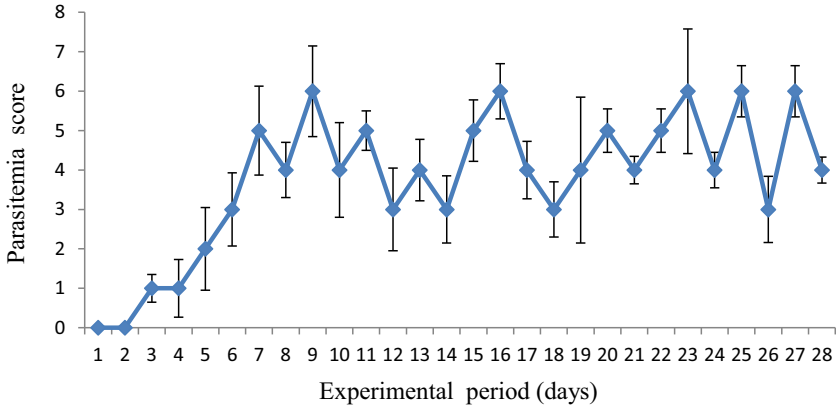

Fig. 2 Fluctuating mean (± SEM) parasitaemia following experimental *T. congolense* infection in jacks

pooled controlled serum samples were 9.8%, 4.8% and 5.6%, respectively.

Serum prolactin (PRL) was determined by using direct ELISA kits (AccuBind[B] ELISA Monobind Inc. 100 North Pointe Drive Lake Forest, CA 92630, USA), intended for a quantitative determination of PRL concentration in serum or plasma using ELISA microplate reader. The sensitivity of the assay was 0.150 ng/ml PRL concentration. Within assay precision, coefficients of variation for low, normal and high pooled controlled serum samples were 4.3%, 3.6% and 6.8%, respectively, and between assay precision, coefficients of variation for low, normal and high pooled controlled serum samples were 9.8%, 8.8% and 6.8%, respectively.

Data analysis

Rectal temperatures, parasitaemia, testosterone and prolactin levels were analysed using Student's *t* test to compare between pre-infection and post-infection values. Data were analysed using GraphPad Prism, version 7.04 for Windows (GraphPad Software, San Diego, CA, USA, www.graphpad.com.). Values of $P < 0.05$ were considered significant (Snedecor and Cochran 1994).

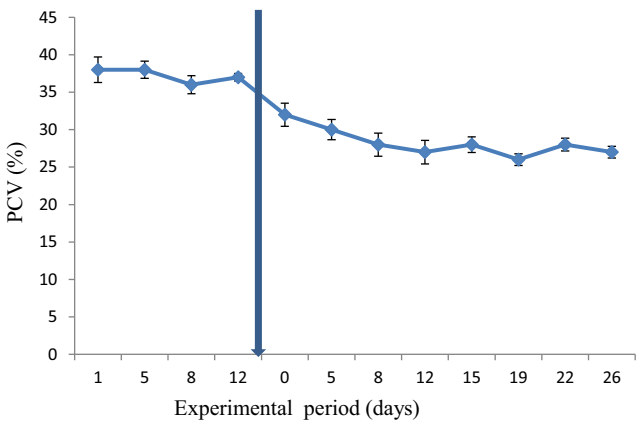

Fig. 3 Changes in mean (± SEM) packed cell volume following experimental *T. congolense* infection in jacks

Results

Clinical observations

Beginning from the day of infection, all the experimental jacks were closely observed for change in demeanour/behaviour. Following parasitaemia, the jacks were observed to be dull, weak, intermittent appetite, fluctuating pyrexia (39–41 °C) and rough hair coat. As the infection progressed, mucous membranes became pale and superficial lymph nodes were enlarged.

Rectal temperature

The mean rectal temperatures of the jacks vary ranging from 36.5 ± 0.22 °C to 37.9 ± 0.32 °C at pre-infection period ranging from 37 ± 0.12 °C to 40.1 ± 0.22 °C at post-infection period. Pyrexia was first observed on day 4 post-infection and thereafter the temperatures throughout the experimental period were significantly higher ($P < 0.05$) than those of pre-infection values (normal range, 37.2–38.2 °C), (Fig. 1). The pyrexia was fluctuating/intermittent throughout the experimental period (Fig. 1).

Parasitaemia

The variation in parasitaemia following experimental *T. congolense* infection in jacks was shown (Fig.2). The parasitaemia gradually increased from day 3 post-infection, reaching the peak parasitaemia on day 9 post-infection and thereafter, and continued to fluctuate throughout the experimental period (Fig. 2).

Erythrogram parameters

Packed cell volume

The changes in PCV at pre- and post-infection following experimental *T. congolense* infection in jacks were shown (Fig.

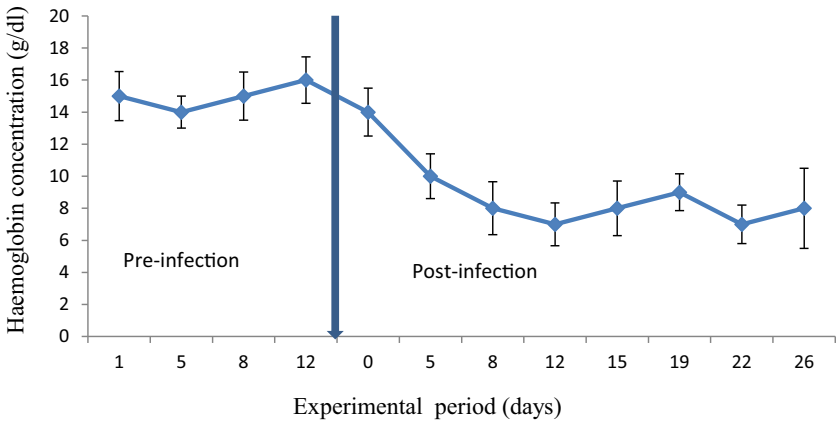

Fig. 4 Changes in mean (± SEM) haemoglobin concentration following experimental *T. congolense* infection in jacks

3). There was a significant decrease in PCV ($P < 0.05$) from pre-infection ranging from 36 ± 0.12 to 38 ± 0.32% to post-infection period ranging from 26 ± 0.21 to 36 ± 0.11%. Anaemia developed in the infected jacks on day 5 post-infection (Fig. 3).

Haemoglobin concentration (g/dl)

The changes in the mean average haemoglobin concentration in pre- and post-infection period are shown (Fig. 4). There was a marked significant decrease ($P < 0.05$) in Hgb concentration from day 5 and throughout post-infection period ranging from 7 ± 0.48 to 10 ± 0.37 g/dl compared to the pre-infection value ranging from 14 ± 0.5 to 16 ± 0.52 g/dl. This indicated decreasing Hgb concentration due to *T. congolense* infection (Fig. 4).

Red blood cell count

The average mean RBC varies at pre- and post-infection period (Fig. 5). A significant decrease ($P < 0.05$) was observed in RBC from day 5 and throughout post-infection period ranging from 3 ± 1.28 × 10^6µl to 6 ± 1.01 × 10^6µl compared to the pre-infection value ranging from 8 ± 1.15 ×10^6µl to 9 ± 1.2 × 10^6µl in experimentally infected jacks.

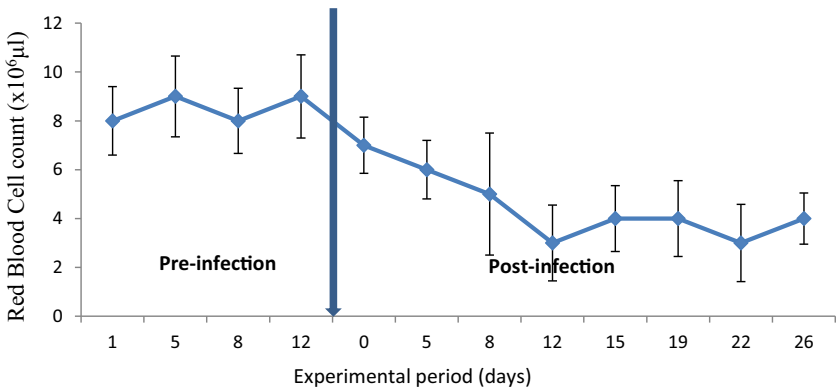

Fig. 5 Alteration in mean (± SEM) red blood count following experimental *T. congolense* infection in jacks

Erythrocytic indices

Erythrocytic indices were also observed to vary from pre-infection to post-infection period of the experiment (Fig. 5). The average mean corpuscular volume (MCV) (fl), mean corpuscular haemoglobin (MCH) (pg) and mean corpuscular haemoglobin concentration (MCHC) (g/dl) were found to decrease non-significantly ($P > 0.05$) from day 5 of infection and throughout the post-infection experimental period compared to pre-infection period (Fig. 6, 7, and 8).

Testosterone

The mean (± S.E.M) testosterone profile variation at pre- and post-infection period was shown, (Fig. 9). The mean average testosterone decreased continually and significantly ($P < 0.05$) from day 5 post-infection and throughout the experimental period ranging from 0.5 ± 0.4 to 2.5 ± 0.86 ng/ml compared to the pre-infection values ranging from 2.8 ± 0.8 to 4.5 ± 0.87 ng/ml. Testosterone levels were higher at pre-infection period than the post-infection and elaborated pulsatile pattern (Fig. 9).

Prolactin

The mean (± SEM) average profile changes at pre- and post-infection period were shown (Fig. 10). The mean average

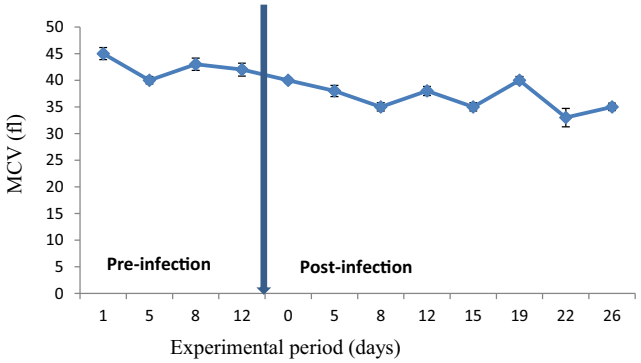

Fig. 6 Variation in mean (± SEM) MCV following experimental *T. congolense* infection in jacks

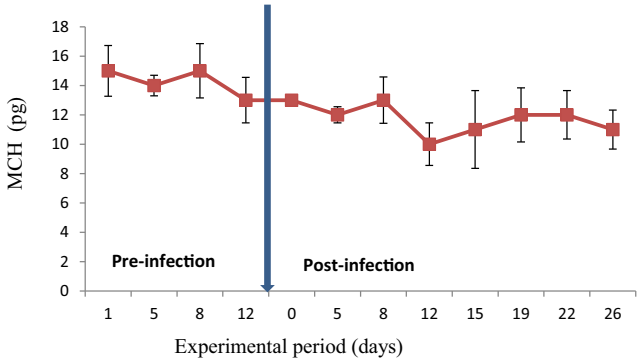

Fig. 8 Variation in mean (± SEM) MCH following experimental *T. congolense* infection in jacks

prolactin decreased significantly ($P < 0.05$) from day 5 post-infection and throughout the experimental period ranging from 2 ± 1.1 to 5 ± 0.55 ng/ml compared to the pre-infection values ranging from 2.1 ± 0.7 to 2.4 ± 0.3 ng/ml. Prolactin levels were higher at pre-infection period than the post-infection (Fig. 10).

Discussion

This study revealed that experimental infection of jacks with *T. congolense* produced changes in behaviour, erythrogram parameters, testosterone profile and prolactin profile. The clinical changes which included dullness, weakness, lymph node enlargement, intermittent pyrexia and anorexia observed in this present study agreed with the report of Samuel et al. (2016), who showed same clinical changes in donkeys infected experimentally with *T. congolense*. Déthié et al. (2001) and Vourchakbé et al. (2020) reported similar findings in donkeys naturally infected with trypanosomes. These clinical features observed in this study could be due to extravascular inflammation on the skin and the localization of the parasite in small blood vessels by attaching with their flagella to the endothelial cells of small blood vessels of the jacks and dissemination in cerebrospinal fluid, leading to parasitaemia and inflammations which consequently resulted in the production of pyrogens, thereby altering the thermoregulatory centre (Emery and Moloo 1981; Masake et al. 1984; Murray 1989; Abebe et al. 1993).

The rectal temperatures were observed to be higher in the post-infection period than pre-infection period and fluctuated at higher range beyond the normal reference values for donkeys. The higher than normal temperature indicated that pyrexia was intermittent and consistent with trypanosomosis in animals; this agreed with the findings of Whicher and Westacott (1992) in domestic animals. Similar findings were reported by Vourchakbé et al. (2020) in natural infection of donkeys with *T. congolense*. Whicher and Westacott (1992) reported that undulating pyrexia following trypanosome infection could be due to the activation and subsequent interaction of several endogenous pyrogenic cytokines in the blood and the parasites' localization in microvasculature; this could be the reason for the intermittent pyrexia observed in this present work.

The variation in the blood parasite level (parasitaemia) observed in this present study could be attributed to the parasite multiplication in the blood stream and subsequent localization in the microvasculature. This is consistent with the findings of Mamoudou et al. (2015) in cattle trypanosomosis; Samuel et al. (2018) in packed donkeys experimentally infected with *T. congolense*; Vourchakbé et al. (2020) and Déthié et al. (2001) in *T. congolense* natural infection in donkeys; and

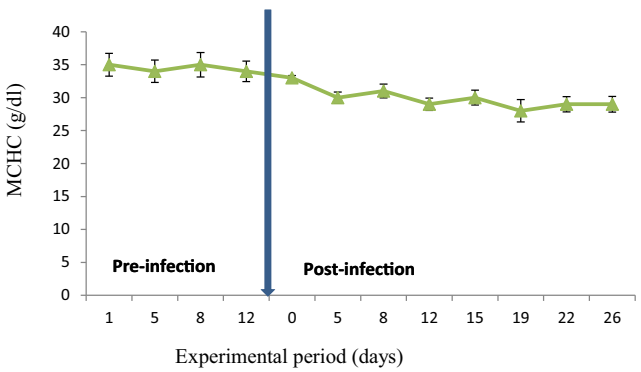

Fig. 7 Variation in mean (± SEM) MCHC following experimental *T. congolense* infection in jacks

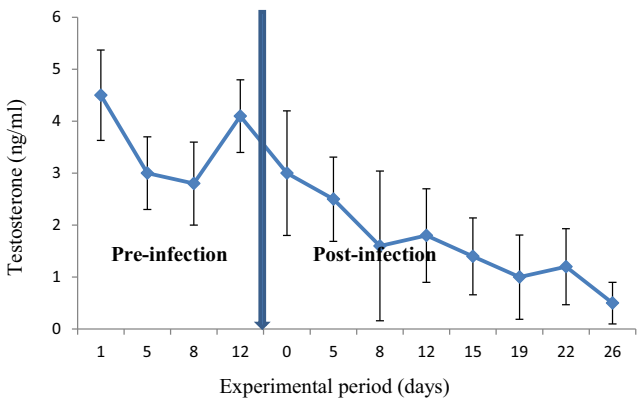

Fig. 9 Changes in mean (± SEM) testosterone profile following experimental *T. congolense* infection in jacks

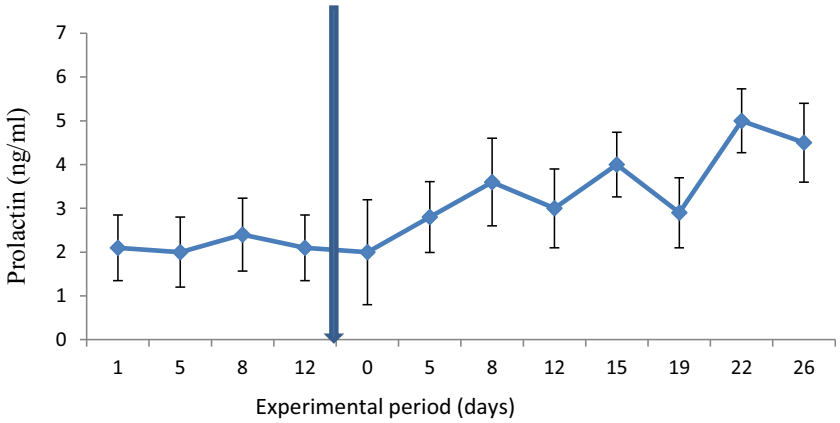

Fig. 10 Changes in mean (± SEM) prolactin profile following experimental *T. congolense* infection in jacks

Adamu et al. (2009) in pigs. Persistent parasitaemia could be responsible for the activation of endogenous pyrogen which precipitated pyrexia and undulating parasitaemia led to fluctuating pyrexia in *T. congolense* infection as reported by Croos (2003). The persistent parasitaemia could also be due to the expression of variant surface glycoproteins responsible for antigenic variation and evasion of the host defence mechanism and this variation could be responsible for the recurrence of parasitaemia in the course of *T. congolense* infection in this present study (Paiva et al. 2000; Horn 2014).

The packed cell volume (PCV), haemoglobin concentration and red blood cell count of the experimentally infected jacks were observed to be lower at post-infection compared to pre-infection. Anaemia (erythrocyte below physiological range) is a consistent sign of trypanosomosis in domestic animal. This agreed with the report of Adamu et al. (2008) in sheep experimentally infected with trypanosomes, Adamu et al. (2009) in experimental infection in pigs, Vourchakbé et al. (2020) and Déthié et al. (2001) in *T. congolense* natural infection in donkeys and Samuel et al. (2018) in experimentally infected jacks. The anaemia observed in this study could be due to increased red cell destruction by the parasite (Mamo and Holmes 1975), erythrocytes desialylation (Guegan et al. 2013), dyshaematopoiesis (Preston et al. 1975), haemodilution (Holmes 1976) and expanded mononuclear erythrophagocytosis (Katunguka-Rwakishaya et al. 1992). Anaemia could also be due to acute haemolysis, oxidative damage to red blood cell membrane due to glutathione depletion in red cell (Akanji et al. 2009), release of proteolytic lysosomal enzymes (proteases) from pockets on their flagella and from damaged or dead trypanosomes (Igbokwe 1994) and possible reduction of erythrocyte plasticity and longevity due to pyrexia (Woodruff et al. 1973)

The erythrocytic indices, i.e. MCV, MCH and MCHC, measured in this present study were used to determine the type of anaemia in this study. MCV, MCH and MCHC were found to vary non-significantly between the pre- and post-infection. This resulted in normocytic normochromic anaemia which is a non-regenerative anaemia; this agreed with the report of Mbaya et al. (2010) and Anosa and Isoun (1980), who demonstrated normocytic normochromic anaemia in acute trypanosomosis with a tendency to become microcytic. This type of anaemia is non-regenerative as red bone marrows are not stimulated to increase the process of haematopoiesis.

Testosterone profile was significantly lower from day 5 post-infection compared to pre-infection period. The decline in the testosterone profile in this present study agreed with the report of Waindi et al. (1986) in goats, Mutayoba et al. (1994) in rams and Boly et al. (1994) in bulls infected with trypanosomosis. This could be due to impaired hypothalamic-pituitary-gonadal axis function leading to reduced luteinizing hormone production, reduced sensitivity of Leydig cells to an already low circulating LH, reduced steroidogenesis and reduced testosterone production (Mutayoba et al. 1994; Noakes et al. 2009). It has also been reported that tissue hypoxia and formation of reactive oxygen species consequent to anaemia may also be responsible for low level of testosterone in trypanosomosis infection (Logan-Henfrey et al. 1992). Testosterone contributes to the processes of spermatogenesis and sex drive or libido in domestic animals (Noakes et al. 2009). The decline in the testosterone levels in this present study could impair the fertility of the infected jacks.

Prolactin levels in this present study were significantly higher compared to pre-infection levels. This could be due to induction of local inflammation of the tuberal region of the hypothalamus and pituitary gland by *T. congolense*, resulting in increased prolactin production (Adenowo et al. 2005). The increase in prolactin levels at post-infection could also be due to increased level of stress causing oxidative damage due to the *T. congolense* infection leading to the production of higher serum levels of prolactin as stress marker; this agreed with the report of Logan-Henfrey et al. (1992). It has been reported by Koike et al. (1991) that the production of TNF-α and IL-6 due *T. congolense* infection, though not measured in this current work, stimulates prolactin release from the anterior pituitary cells inhibiting the production of gonadotropins. Trypanosomosis induced activation of the

hypothalamo-pituitary-adrenal (HPA) axis causing an increase in prolactin production and might be responsible for subsequent infertility observed in male rat (Rivier et al. 1986), bulls (Johnson et al. 1982), boars (Litrap and Raeside 1975) and rams (Naylor et al. 1990). Levels above or below the physiologic level of prolactin altered male reproductive function by inhibiting gonadotropin production and reducing the sensitivity of the Leydig cells to luteinizing hormones and hence reduced testosterone production in male domestic animals; however, physiologic levels enhanced luteinizing hormone receptors in Leydig cells, resulting in testosterone production, which enhanced spermatogenesis (Grattan et al. 2007). Prolactin (PRL) is involved in the pituitary control of male reproduction due to the presence of specific binding sites, influence on pituitary gonadotropin release, trophic effects on the testis and male accessory organs, stimulation of steroidogenesis and steroid metabolising enzymes (Hoehn and Marieb 2007).

Conclusion

Experimental *T. congolense* infection in jacks resulted in decreased serum testosterone and increased prolactin profiles and also reduced erythrogram parameters leading to normocytic normochromic anaemia. The decreased serum testosterone could lead to low libido, reduced spermatogenesis and infertility in the jacks. The high prolactin levels could indicate stress generation, reduced responsiveness of Leydig cells due to receptor downregulation and reduced testosterone production. The reduced erythrogram parameters, PCV, Hgb and RBC, resulted in anaemia which could lead to lower reproductive function.

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Authors' contributions Samuel F.U, N. Kolo, M.Shinkut, Echeku, O. W and S.Y.Idris were responsible for the research design, paper draft and most of laboratory work followed by obtained data analysis. Kolawole B. J, N.P. Chiezey and P.I. Rekwot were involved in the supervision of the work, data analysis and the manuscript edition.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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