
Sialyltransferase Activity from Thyroid Gland of Sheep Experimentally Infected With *Trypanosoma Congolense*

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ABSTRACT

African animal trypanosomosis (AAT) is a disease complex caused by trypanosomes parasites and transmitted via tsetse fly vector. Transplacental and mechanical transmission can also occur. Trypanosomosis can affect both domestic and wild animals in natural and experimental infections. AAT is one of the most devastating disease in African continent particularly Nigeria. The disease has direct impacts on livestock productivity, hence affecting farmer's production opportunity and income, leading to poverty and hunger. Sialidase (SD) has been implicated in the de-sialylation of erythrocyte surface sialic acid (SA), hence the pathogenesis of anaemia in AAT. Trypanosusceptible animals were found to survive AAT induced anaemia for a long period of time or even recover. This could be possible because of the host response to anaemia by the release of sialyltransferase (ST). ST mediates attachment of SA to the cell surface glycoprotein including erythrocytes. Thus, re-sialylation phenomenon is believed to contribute to the stabilization of red cells mass in anaemia induced by AAT. In this study, ST activities were expressed from thyroid glands of T. congolense-infected and non-infected sheep. The median ST activities from both groups were determined on DEAE-cellulose and Sephadex G-100 columns. The observed increase in ST activities particularly in the T. congolense-infected group was the major finding in this research. Therefore, the results lean support on ST involvement in the recovery phase of anaemia due to AAT. The P<0.05 was considered statistically significant.

Keywords: African animal trypanosomosis, Anaemia, Sialidase, Sialic acid, Sialyltransferase

INTRODUCTION

African animal trypanosomosis (AAT) is a very complex and debilitating protozoan disease affecting animals in sub-Saharan Africa (Esievo *et al.*, 1990;

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Umar *et al.*, 2007; Mbaya *et al.*, 2009). The disease is caused by trypanosomes species and transmitted mostly via tsetse flies (*Glossina spp*). In Africa, AAT has been described as a major challenge to livestock production, food security and public health (Samdi *et al.*, 2010). Clinical disease have been produced in cattle, sheep, goats and other species, morbidity and mortality vary with the breed of animal but can reach 50% (Raheem, 2014). In Nigeria, the most important trypanosomes species are the *Trypanosoma brucei*, *T. congolense*, *T. vivax* and *T. evansi* in livestock, while *T. brucei gambiense* infect human (Samdi *et al.*, 2010). Apart from intermittent fever, lethargy, loss of body condition and decreased fertility, anaemia is the most important clinical feature of AAT (Anosa *et al.*, 1997). Several mechanisms of anaemia in AAT were defined by many researchers (Mbaya *et al.*, 2012).

Esievo *et al.* (1979) suggested that trypanosomal sialidase (SD) is involved in the pathogenesis of anaemia in AAT; this is because of its ability to remove sialic acid (SA) on the galactose residues of an erythrocyte. In consequence, erythrocytes are rapidly cleared from the blood circulation by erythrophagocytosis, which result in anaemia. Beside trypanotolerant breeds, however it is most intriguing to found that some trypanosusceptible animals recover from anaemia over several months of trypanosomes infection, while others live chronically infected with persistently low erythrocyte counts (Anosa *et al.*, 1997). This could be possible because of the host response to AAT induced anaemia through the action of sialyltransferase (ST). ST is believed to mediate the attachment of SA to the erythrocyte surface, therefore causing some degree of stabilization of the red cells mass, hence playing an important role in the recovery phase from anaemia due to AAT (Esievo, Saror, Kolo and Eduvie., 1986). ST activities have been found in various tissues and on cell surfaces, with more activities produced in the thyroid glands than in salivary glands, liver and serum of *T. congolense*-infected sheep (Baraya *et al.*, 2013). Considering the huge implication on health and economic burden of AAT in Africa, further studies on host response to anaemia due to AAT need to be advanced to shade more light on re-sialylation phenomenon in trypanosomes infection. Therefore, in this study we compared the ST activity from thyroid glands of *T. congolense*-infected and non-infected sheep.

MATERIALS AND METHOD

Six (n = 6) apparently healthy adult *yankassa* breed of sheep were used in each group, namely group A (*T. congolense*-infected group) and group B (non-infected group). These animals were acclimatized for the period of two months before



the commencement of the study, housed separately in a fly-proof pen; feed and water were provided *ad libitum*. During the time of acclimatization, packed cell volume (PCV) was determined from each group to set baseline data, animals were screened and dewormed for internal and external parasites. At the end of acclimatization period, an approximately 1×10^2 trypanosomes/ml (Savannah strain) was inoculated into group A via the jugular vein using sterile procedure and monitored for parasitaemia. PCV for each group was determined over a period of 30 days (data not shown in this study). Then, both groups were humanely sacrificed and the thyroid glands harvested and stored at -20°C for further analysis. Sialyltransferase from the thyroid glands of both groups was extracted separately using Triton X-100 as described by Sticher *et al.* (1991). The crude enzyme extracts from each group were separately passed through ammonium sulphate (70%) saturation, then ion exchange chromatography (diethylaminoethyl (DEAE)-cellulose) and sephadex G-100 columns (1.6 x 30 cm). In each separate columns, 5 ml eluates were collected into 25 (n = 25) samples bottles for sialyltransferase assay. Sialyltransferase activity was determined from both groups as described by Køen and Thiem (1997). The absorbance was taken at 549nm.

RESULTS AND DISCUSSION

The median sialyltransferase activities for *T. congolense*-infected and non-infected groups on DEAE-cellulose columns were 0.5 U/ml (0.37) and 0.4 U/ml (0.30), respectively (Table 1). On sephadex-G 100 columns, the median sialyltransferase activities for *T. congolense*-infected and non-infected groups were 2.7 U/ml (1.80) and 1.8 U/ml (1.77), respectively (Table 2).

Table 1: Comparison of the median sialyltransferase activities of *T. congolense*-infected and non-infected groups on DEAE-cellulose columns

Variable	Median (IQR)		Z statistic	P value*
	<i>T. congolense</i> -infected (n = 25)	Non-infected (n = 25)		
Sialyltransferase activity (U/ml)	0.50 (0.37)	0.40 (0.30)	-1.971	0.049

*Mann Whitney test.

Table 2: Comparison of the median sialyltransferase activities of *T. congolense*-infected and non-infected groups on sephadex G-100 columns

Variable	Median (IQR)		Z statistic	P value*
	<i>T. congolense</i> -infected (n = 25)	Non-infected (n = 25)		
Sialyltransferase activity (U/ml)	2.73 (1.80)	1.80 (1.77)	-1.582	0.114

*Mann Whitney test.



Even though there were marked increase in ST activities from both groups on sephadex G-100 columns, the result was not significant. Nevertheless these findings are still biochemically relevant since ST activity can be affected by the changes in pH and temperature which are common to AAT. Previously we have shown that, *T. congolense* infection in sheep can alter the physical and biochemical properties of ST. The increase in ST activities observed in both DEAE-cellulose and sephadex G-100 columns can suggest the enzyme involvement in re-sialylation of erythrocyte because of anaemia caused by AAT. ST activities mediates the attachment of the new or cleaved SA produced by the action of SD in AAT to their normal site on an erythrocyte to bring about stabilization of red cells mass. This process is assumed to play an important role in the survival of some trypanosusceptible animals.

CONCLUSION

The observed changes in ST activities can be used as a model to understand the re-sialylation mechanism in AAT. Therefore, in the future this phenomenon can be developed to modulate the host response to anaemia by generating more glycoconjugates such as SA on an erythrocyte; thereby preserving the physiological life span of an erythrocyte. Hence, the host protection against AAT induced anaemia.

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