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Initial Plasma Protein Profile as Putative Index of Susceptibility in West Africa Dwarf and Red Sokoto Goats Experimentally Infected with *Trypanosoma brucei* and *T. congolense*.

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Abstract
Plasma protein in experimental trypanosome infection of West African Dwarf (WAD) and Red Sokoto goats (RSG) were compared considering mortalities incurred. The comparison aimed at establishing initial breed picture, discerning pattern of change under infection, in order to determine trend that serve as putative index of susceptibility which may be used as marker to assist early husbandry decisions within endemic area.
16 WAD, 16 RSG were subgrouped into 4 groups of 4 *T. brucei* (RSG_{tb}, WAD_{tb}), *T. congolense* (RSG_{tc}, WAD_{tc}), mixed *T.brucei/T.congolense* (RSG_{tbc}, WAD_{tbc}) or left as controls (RSG_c, WAD_c). RSG_{tb} and RSG_{tc} each had 25% mortality, RSG_{tbc} had 50% mortality but no infected WAD group had mortality. Serum samples analysed using HITACHI 902 automatic analyser showed Albumin ($RSG_c > WAD_c)_{IALB}$ had significantly higher ($P < 0.05$) than infected goats ($RSG_{tb} < WAD_{tb}$,$RSG_{tc} < WAD_{tc}$, $RSG_{tbc} < WAD_{tbc})_{LALB}$. Globulin – controls ($RSG_c < WAD_c$)IGLB had significantly lower ($P < 0.05$) than infected goats ($RSG_{tb} < WAD_{tb}$, $RSG_{tc} < WAD_{tc}$, $RSG_{tbc} < WAD_{tbc})_{LGLB}$.Total Protein – control ($RSG_c > WAD_c)_{ITP}$ had significantly lower ($P < 0.05$) than infected goats ($RSG_{tb} < WAD_{tb}$, $RSG_{tc} < WAD_{tc}$, $RSG_{tbc} < WAD_{tbc})_{LTP}$. Higher initial albumin and higher initial globulin of uninfected controls related to susceptibility and resistance in reversion where goat breed having comparatively higher initial albumin was more susceptible while goat breed having comparatively higher initial globulin was more resistant to African trypanosomosis. Higher initial Albumin levels could be used as putative index of susceptibility to the disease in goats.
Keywords: African Trypanosomosis, goats, plasma proteins, putative index of susceptibility.

1. Introduction

The dynamics of relative susceptibility of different breeds of goats in Nigeria to experimental trypanosomosis is poorly understood (Lasisi, 2009). Small ruminants (sheep and goats) are susceptible to influence of African trypanosomosis which induces changes in levels of their total proteins, globulin and albumin, with altered Albumin: globulin ratios (Taiwo et. al. 2003, Anosa and Isoun, 1976).
Generally, animals susceptible to African Animal Trypanosomosis (AAT) suffer syndromes that range from sub-clinical, mild or chronic to acute fatal disease (Mare, 1998). The severity of clinical trypanosomosis depends on factors such as specie and strain of trypanosome, dose of infecting trypanosomes, breed of animal, nutritional status (Adeiza et. al. 2008, Awobode, 2006, Mare, 1998). Differences exist in tolerance shown by animals to the infection which needs to be measured both between and within animal breeds (Verhulst and Pandey, 1998). However, practical reliable markers of resistance or susceptibility of animals to trypanosomosis which could assist decision making in animal husbandry within the endemic area are lacking (d'Ieteren et. al. 1998). Previous works had shown differences in susceptibility of goats to trypanosome infections. In the same Savannah goats experimentally infected with *T. brucei* and *T. vivax*, the *T. brucei* was reported to inflict more severe infection than *T. vivax* (Adeiza et. al. 2008). Also following an experimental infection of *T. congolense* to west African Dwarf and Red Sokoto goats, the Red Sokoto goats were reported to be more susceptible, incurring eventual mortalities unlike the West African dwarf goats (Adah et. al. 1993). However, even *T. congolense* which is known to be the commonest cause of infection in small ruminants (Jordan, 1986, Samdi et. al. 2010) exists in phenotypically similar but genotypically different types whose DNA may not hybridise with each other, like *T. congolense* – Savanna type, *T. congolense* - Kilifi type, *T. congolense* – West African forest/riverine type (Majiwa, 1992). The experimental infection of *T. congolense* to the same breed of Small East African goat was reported to produce pathology that showed regional differences marked by underlying heterogeneity (Mutayoba et. al. 1989). There is therefore the need to look beyond breed of goat or specie of Trypanosome for a putative easily measurable index which can indicate individual animal's susceptibility among the goat population in an endemic area. Between the different species of animals it is probable that what obtains for goats may not be the same for cattle. The mechanism of tolerance to trypanosomosis in goats which are unable to maintain PCV during infection (regarded as only showing resilience) differs from that in cattle which can show classical

trypanotolerance or trypanoresistance (Goossens et. al. 2001).
Since trypanosomes are haemotropic parasites (Akinboade, 1991) there is need to explore for intrinsic and easily measurable factors in the vascular system which could have predictive value for the susceptibility of animals.

Objective

The objective of this investigation was to compare plasma protein profiles of uninfected or control goats (initial profiles) with those of infected goats showing differences in susceptibility (later profiles) to see if a consistent trend could be discerned. Such a trend found across gradient of susceptibility would be considered as candidate for a putative index to predict susceptibility of goats to trypanosome infection.

2. Plasma Proteins in Trypanosomosis

In general, animals affected by trypanosomosis have poor immune response, are intolerant to stress and exhibit poor productivity (Abebe, 1991). Plasma proteins (Total protein, albumin, globulin) could assume critical importance in such a scenario. Directing attention on physiological parameter like plasma proteins of control and infected host animal can further enable better understanding and management of the condition (Abubakar et. al. 1999). The plasma protein normally participate in maintenance of body immune status and haemodynamic balance directing fluid movement across vascular and interstitial compartments. Derangement in fluid movement could result in oedema fluid accumulating in interstitial spaces or body cavities. Such accumulation produce hydroperitoneum or ascites in peritoneal cavity, hydrothorax in pleural cavity and hydropericardium in pericardial sac. The fluid may be non-inflammatory (transudate) usually low in proteins or colloids, or inflammatory (exudate) caused by escape of plasma proteins (especially albumin) from leakages allowed by increased vascular permeability (Vegad, 1995).
The lesion caused by African animal trypanosomiasis include subcutaneous oedema which is particularly prominent and usually accompanied by hydrothorax, hydropericardium and ascites (Mare 1998, Urquhart et. al., 1998). The heart is usually damaged with marked cellular infiltrates found in perivascular and interstitial locations so that perivascular and interstitial oedema is common especially in terminal cases of *T. brucei brucei*, *T. congolense* and *T. vivax* (Taylor and Authie, 2004). Other inflammatory reactions (Urquhart, et. al. 1998) and parasite factors such as proteases contribute to pathology, like congopain that is a cysteine protease from *T. congolense* occurring as a circulating antigen (Taylor and Authie, 2004).

3. Materials and Methods

Thirty two goats comprising 16 each of WAD and RSG breed were purchased from markets and farms in Makurdi in Benue State of Nigeria. These were housed in insect proof pen, fed a mixture of fleshly cut legumes and grasses. Supplemental concentrate formulated as dried brewers grain 20%, Maize offal 64.5%, soya beans 12.0%, Bone ash 2.5% and salt 1%. Water was provided ad Libitum. Prophylactic medications given included Terramycin (LA) (Farvet Bladel Holland) sulphonamide injection (kepro B.V. Deventer Holland) Aldendazole dewormer (Eagle Chemical Co. Ltd. Chungchongnam, Korea) and Peste des petits Ruminants vaccine (NVRI, Vom Nigeria) according to manufacturer's instructions. They were also dusted with pyrethrin preparation (piff paff, Gongoni, Kano) during the one month quarantine period prior to infection.
Subgrouping of 4 goats of each breed were either infected with *T. brucei* (Federe strain), *T. congolense* (Karu strain), a mixture of *T. brucei / T. congolense* or left as uninfected controls.
Serum samples collected once weekly for 7 weeks post infection were subjected to autoanalysis using HITACHI 902 autoanalyser.

Statistical Analysis

Data obtained was subjected to Analysis of Variance (ANOVA) $P < 0.05$.

4. Result

Mean Total Protein Table 1 was significantly ($P < 0.05$) higher in infected WAD and RSG than control WAD and RSG. Among the controls, it was non - significantly ($p > 0.05$) higher in RSG control than WAD control. However, it was significantly ($P < 0.05$) higher in infected WAD than infected RSG of each infected inoculum i.e. *T. brucei, T. congolense*, mixed *T. brucei / T. congolense*.
Mean albumin Table 2 was significantly ($p < 0.05$) higher in control RSG and WAD than their infected counterparts except *T. brucei* infected WAD which had higher mean albumin than WAD control. Among the controls, it was higher in RSG than WAD but among all infected groups (*T. brucei, T. congolense*, mixed *T. brucei/T. congolense*) it was higher in WAD than RSG.
Mean globulin was significantly higher in infected WAD and infected RSG than their uninfected counterparts. In each infected group (*T. brucei, T. congolense*, mixed *T. brucei / T. congolense*) as well as the uninfected controls, globulin values were higher in the WAD than their RSG counterparts.
Albumin : globulin ratios were significantly higher ($P < 0.05$) in WAD and RSG controls than infected groups(*T.*

brucei, T. congolense, mixed *T. brucei/T. congolense*). The Albumin : Globulin ratios of RSG control was higher than WAD control and that of mixed infected RSG higher than mixed infected WAD. Although the ratio in WAD control was higher than all infected WAD groups and that in RSG control higher than all infected RSG groups, the comparative ratio in single infected WAD (*T. brucei*, or *T. congolense*) was higher than that of equivalent single infected RSG (*T. brucei,* or *T. congolense*) due to the greater relative decline in value of Albumin in the single infected RSG groups (*T. brucei*, or *T. congolense*).

5. Discussion

Higher Albumin in control RSG (initial) than control WAD (initial) became reversed to lower Albumin in all infected RSG groups (later) than equivalent infected WAD groups (later) i.e.

$$\frac{RSG_{(IAB)} > WAD_{(IAB)}}{RSG_{(LAB)} < WAD_{(LAB)}} = SPIN\ 1 \qquad (1)$$

On the other hand, higher Globulin in control WAD(initial) than control RSG(initial) was maintained as higher Globulin in all infected WAD groups (later) than equivalent infected RSG groups (later) i.e.

$$\frac{WAD_{(IGB)} > RSG_{(IGB)}}{WAD_{(LGB)} > RSG_{(LGB)}} = SPIN\ 2 \qquad (2)$$

Where IAB = initial Albumin, LAB = Later Albumin
IGB = initial Globulin, LGB = Later Globulin.
ITP = Initial Total Protein, LTP = Later Total Protein
SPIN 1 and 2 = Susceptibility Putative Index 1 and 2

This finding is considered against the backdrop of mortalities that occurred in all infected RSG groups while no mortality occurred in infected WAD groups during the 8 week period of investigation.

Lasisi (2009) made similar report showing occurrence of higher mean serum Albumin in non-Parasitised RSG (initial) than equally non-parasitised WAD (initial) goats, but those having haemoparasitic infection showed reversed trend with higher Albumin in WAD (Later) than RSG (Later). This fits into susceptibility putative index I (SPIN 1) described above.

Biryomumaisho et. al. 2003 reported reduced total protein in *T. congolense* infection but increased total protein in *T. brucei* during primary infection of East African goats. They reported increased total protein in both T. congolense and *T. brucei* secondary infection of the goats whose reduced albumin was thought to be due to either trypanosome uptake of albumin bound fatty acids and lipoproteins or increased catabolism by host. Other plausible explanations given for reduced albumin in trypanosomosis are plasma expansion, proteinuria, hepatocellular damage (Abubakarr et. al. 1999, Anosa and Isoun 1983, Saror, 1980), increased loss through ascites (Lording and Friend, 1991).

Increased in globulin has been commonly attributed to rise in immunoglobulin during trypanosome infection. This may or may not be protective, depending on isotype of immunoglobulin involved (Taylor et. al. 1996). In Trypanosomosis, increase in concentration of serum IgM fraction occurs but it has no affinity for the parasite and is rather non specific (Chatterjee, 2009). Animals resisting trypanosomosis show isotype switch from IgM to IgG that is more protective (Taylor et. al. 1996).

6. Conclusion

African Animal trypanosomosis is a serious endemic haemotropic disease confronting Livestock including goats in Sub-saharan Africa.

There is an absence of markers of susceptibility to the disease among the goat population. Plasma proteins altered in the course of the infection affect pathophysiology of the disease.

Result of this investigation indicate that using combined factor index, goat with higher initial albumin and lower initial globulin in their total proteins as shown by Red Sokoto goats were more susceptible when infected with either *T. brucei, T. congolense* or combined inoculums of both parasites. Consequently, higher initial albumin and lower initial globulin in uninfected goats are proposed as putative indexes of susceptibility to trypanosomosis in goats.

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Table 1 Total Protein (g/L) levels in *T. brucei* and *T. congolense* infected West African Dwarf and Red Sokoto goats

Treatments (Mean±SE)

Week	RSG_*T. congolense*	WAD_*T. congolense*	RSG_*T. brucei*	WAD_*T. brucei*	RSG_mixed infection	WAD_Mixed infection	RSG_control	WAD_control
0	71.99±4.85^{a}	81.32±3.24^{a}	76.31±4.31^{a}	76.02±2.21^{a}	73.57±3.48^{a}	71.24±1.1.97^{a}	78.45±5.75^{a}	74.25±4.34^{a}
1	74.44±1.90^{a}	70.89±5.28^{a}	64.87±5.58^{a}	72.21±3.62^{a}	70.65±4.06^{a}	66.34±7.86^{a}	70.18±3.92^{a}	65.15±4.43^{a}
2	89.95±7.08^{abc}	104.81±2.87^{a}	88.08±7.25^{abc}	93.74±4.65^{ab}	92.88±6.51^{ab}	101.93±6.02^{a}	76.16±1.46^{c}	79.45±3.52^{bc}
3	88.27±0.18^{abc}	99.21±3.59^{a}	81.91±5.65^{bcd}	89.68±2.98^{abc}	79.84±4.54^{bcd}	93.58±6.45^{ab}	70.70±1.78^{d}	75.94±6.28^{cb}
4	83.97±1.06^{b}	101.85±3.70^{a}	95.87±6.98^{ab}	89.77±3.98^{ab}	82.20±4.03^{b}	94.40±4.53^{ab}	64.23±5.22^{c}	69.52±2.95^{c}
5	89.21±4.54^{ab}	101.96±6.88^{a}	100.44±3.70^{a}	97.65±7.26^{a}	86.88±0.02^{ab}	92.50±2.96^{a}	73.71±1.17^{bc}	68.44±2.34^{c}
6	65.40±0.01^{d}	99±4.76^{a}	90.25±3.60^{ab}	88.74±4.46^{abc}	79.11±3.99^{bcd}	85.46±5.50^{abc}	70.62±2.58^{cd}	77.65±9.03^{bcd}
7	90.03±2.79a	100.24±6.70^{a}	94.69±4.11^{a}	92.57±6.64^{a}	84.80±1.66^{a}	91.25±8.97^{a}	89.34±12.65^{a}	78.86±4.40^{a}
Total	81.97±2.04^{bc}	95.76±2.45^{a}	85.20±2.74^{bc}	87.55±2.09^{b}	80.13±1.85^{cd}	86.91±2.84^{bc}	74.13±2.01^{d}	73.52±1.85^{d}

Note: Mean±SE across a row with different superscripts are significantly different with a>b>c>d. Mean separation done with Duncan Multiple Rangetest

Table 2 Albumin (g/L) levels in *T. brucei* and *T. congolense* infected West African Dwarf and Red Sokoto goats

Treatments (Mean±SE)

Week	RSG_*T. congolense*	WAD_*T. congolense*	RSG_*T. brucei*	WAD_*T. brucei*	RSG_mixed infection	WAD_Mixed infection	RSG_control	WAD_control
0	26.78±3.07^{b}	32.63±2.21^{ab}	32.10±2.59^{ab}	34.48±0.80^{a}	33.98±2.29^{a}	33.13±2.06^{ab}	27.60±1.63^{ab}	31.48±1.32^{ab}
1	27.53±1.10^{a}	31.95±1.16^{a}	26.65±4.48^{a}	30.40±1.31^{a}	30.23±0.99^{a}	27.95±2.26^{a}	25.40±3.18^{a}	28.28±4.39^{a}
2	26.45±0.95^{ab}	27.38±3.57^{ab}	21.97±1.95^{b}	30.88±2.09^{ab}	26.33±3.97^{ab}	26.55±1.74^{ab}	33.53±1.53^{a}	28.50±1.40^{ab}
3	27.07±1.12^{ab}	27.70±3.53^{ab}	22.67±2.37^{b}	28.50±2.04^{ab}	25.65±3.92^{ab}	25.85±0.91^{ab}	31.58±1.37^{a}	29.95±2.05^{ab}
4	26.23±1.97^{a}	25.50±1.90^{a}	27.45±2.05^{a}	30.95±4.48^{a}	23.88±3.74^{a}	28.32±1.68^{a}	30.63±5.10^{a}	31.03±3.82^{a}
5	31.73±8.75^{a}	28.33±3.02^{a}	25.20±1.97^{a}	31.00±2.47^{a}	21.25±5.15^{a}	35.38±5.15^{a}	35.97±2.82^{a}	30.98±1.07^{a}
6	24.07±3.72^{b}	33.70±8.46^{ab}	19.70±1.47^{b}	35.75±15.63^{ab}	23.13±3.17^{b}	26.90±1.26^{ab}	44.93±6.54^{a}	37.63±6.65^{ab}
7	25.23±3.73^{bc}	31.95±0.75^{abc}	26.87±1.77^{bc}	31.53±2.10^{abc}	23.35±6.65^{c}	30.60±1.17^{abc}	37.35±2.95^{a}	33.43±1.71^{ab}
Total	26.92±1.22^{bc}	29.82±1.38^{ab}	25.56±1.15^{c}	31.68±1.18^{a}	26.50±1.32^{bc}	29.29±1.11^{abc}	32.66±1.48^{a}	31.52±1.19^{a}

Note: Mean±SE across a row with different superscripts are significantly different with a>b>c>d. Mean separation done with Duncan Multiple Range test

Table 3 Globulin (g/L) values of control and infected goats.

Week	RSG_*T. congolense*	WAD_*T. congolense*	RSG_*T. brucei*	WAD_*T. brucei*	RSG_mix infection	WAD_Mix infection	RSG_control	WAD_control
0	45.21±4.14^{a}	43.39±3.65^{a}	44.21±2.65^{a}	46.84±3.82^{a}	39.60±5.46^{a}	38.12±3.25^{a}	50.85±5.23^{a}	42.78±3.24^{a}
1	46.91±2.40^{a}	40.26±2.80^{a}	38.22±2.52^{a}	41.46±4.17^{a}	40.43±4.98^{a}	38.39±5.83^{a}	44.78±4.08^{a}	36.88±3.13^{a}
2	57.37±0.00^{bcd}	66.36±4.78^{abc}	66.11±5.80^{abc}	73.93±3.13^{ab}	66.38±7.98^{abc}	75.38±5.87^{a}	42.64±2.78^{d}	51.41±4.87^{cd}
3	61.20±1.14^{abc}	60.53±3.42^{abc}	56.95±6.36^{abc}	70.71±4.11^{a}	54.19±6.86^{bcd}	67.73±5.76^{ab}	39.13±0.90^{d}	45.99±5.10^{cd}
4	57.73±0.92^{b}	64.27±4.71^{ab}	73.14±10.96^{a}	70.90±3.78^{ab}	58.33±3.42^{b}	66.08±3.30^{ab}	33.60±2.71^{c}	38.06±6.04^{c}
5	57.48±5.81^{a}	69.32±5.22^{a}	75.24±2.84^{a}	70.96±9.08^{a}	65.63±5.13^{a}	64.23±2.59^{a}	37.74±2.30^{b}	37.46±1.93^{b}
6	41.80±6.40^{bc}	55.04±11.24^{ab}	70.55±2.95^{a}	63.87±11.60^{ab}	55.98±5.91^{ab}	58.56±6.22^{ab}	25.68±7.41^{c}	40.03±2.71^{bc}
7	64.79±5.13^{a}	66.68±1.00^{a}	67.82±4.73^{a}	68.72±8.59^{a}	61.45±5.00^{a}	55.74±10.42^{a}	54.82±18.41^{a}	45.43±5.50^{a}
Total	53.61±2.05^{b}	57.57±2.71^{ab}	59.44±3.07^{ab}	64.13±2.94^{a}	53.61±2.60^{b}	57.90±2.98^{ab}	41.12±2.22^{c}	42.09±1.53^{c}

Note: Mean±SE across a row with different superscripts are significantly different with a>b>c>d. Mean separation done with Duncan Multiple Range test

Table 4 Albumin: Globulin Ratio of *T. brucei* and *T. congolence*

Week	RSG_*T. congolense*	WAD_*T. congolense*	RSG_*T. brucei*	WAD_*T. brucei*	RSG_mix infection	WAD_Mix infection	RSG_control	WAD_control
0	0.61±0.10^{ab}	0.78±0.11^{ab}	0.73±0.06^{ab}	0.75±0.07^{ab}	0.93±0.17^{a}	0.90±0.13^{a}	0.56±0.07^{b}	0.74±0.04^{ab}
1	0.59±0.05^{a}	0.80±0.05^{a}	0.70±0.13^{a}	0.72±0.05^{a}	0.79±0.13^{a}	0.75±0.06^{a}	0.59±0.20^{a}	0.79±0.15^{a}
2	0.45±0.00^{b}	0.42±0.08^{b}	0.33±0.03^{b}	0.42±0.04^{b}	0.43±0.14^{b}	0.36±0.04^{b}	0.80±0.09^{a}	0.57±0.07^{b}
3	0.44±0.03^{bc}	0.46±0.07^{bc}	0.40±0.05^{c}	0.71±0.05^{c}	0.53±0.15^{bc}	0.39±0.02^{c}	0.81±0.04^{a}	0.67±0.07^{b}
4	0.46±0.04^{b}	0.41±0.05^{b}	0.38±0.08^{b}	0.45±0.08^{b}	0.42±0.07^{b}	0.43±0.02^{b}	0.93±0.20^{a}	0.89±0.24^{a}
5	0.59±0.20^{bc}	0.41±0.04^{c}	0.34±0.03^{c}	0.47±0.09^{c}	0.33±0.10^{c}	0.44±0.01^{c}	0.97±0.14^{a}	0.83±0.05^{ab}
6	0.60±0.25^{b}	0.94±0.55^{b}	0.28±0.01^{b}	0.74±0.34^{b}	0.45±0.11^{b}	0.48±0.08^{b}	2.47±1.28^{a}	0.93±0.11^{b}
7	0.40±0.09^{a}	0.48±0.02^{a}	0.40±0.05^{a}	0.49±0.09^{a}	0.39±0.14^{a}	0.60±0.13^{a}	0.75±0.20^{a}	0.77±0.10^{a}
Total	0.53±0.04^{c}	0.60±0.08^{bc}	0.47±0.04^{c}	0.55±0.05^{bc}	0.56±0.06^{bc}	0.55±0.04^{bc}	0.95±0.16^{a}	0.77±0.04^{ab}

Note: Mean±SE across a row with different superscripts are significantly different with a>b>c>d. Mean separation done with Duncan Multiple Range test

Table 5 Post Infection Case Fatality Rate

		Time (Weeks)								
Breed	Inoculum	1	2	3	4	5	6	7	Total Mortality	Percentage (%)
WAD	*T. congolense*	-	-	-	-	-	-	-	0	0
	T. brucei	-	-	-	-	-	-	-	0	0
	Mixed Infection	-	-	-	-	-	-	-	0	0
RSG	*T. congolense*	-	-	-	1	-	-	-	1	25
	T. brucei	-	-	-	-	-	-	1	1	25
	Mixed Infection	-	-	-	-	-	2	-	2	50