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Experimental *Trypanosoma evansi* Infection in Mice, Rats and Rabbits-Studies on Blood Glucose and Liver Glycogen

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ABSTRACT

Fifteen albino mice were inoculated intraperitonially each with 2.0 ml of blood obtained from the artificially infected buffalo calves with *Trypanosoma evansi* (*T. evansi*). Wet blood examination of the buffalo calves under microscope, however, did not reveal *T. evansi*. Fifteen rats and six rabbits were inoculated intraperitonially with 2.0 ml of blood containing 1.7×10^4 trypanosomes. Blood glucose level estimated in each of the laboratory animal prior to inoculation and after inoculation revealed significant decrease in blood glucose level. Sections of liver stained with Best carmine stain revealed progressive depletion of glycogen from the hepatic cells of mice, rats and rabbits.

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INTRODUCTION

Trypanosoma evansi (*T. evansi*) are known to utilise glucose causing hypoglycemia and death of animal (Jatkar and Singh, 1974; Pegran and Scott, 1976; and Mallick and Dwivedi, 1981). No reports are available on the effect of hypoglycemia on glycogen content of hepatic cells. The present work deals with such studies in laboratory animals infected with *T. evansi*.

MATERIALS AND METHODS

Fifteen albino mice were inoculated intraperitonially each with 2.0 ml of blood obtained from the artificially infected buffalo calves with *Trypanosoma evansi*. Wet blood film examination of the buffalo calves, however, did not reveal *T. evansi*. While 15 rats and six rabbits were inoculated intraperitonially with 2.0 ml of diluted blood of infected mice containing 1.7×10^4 trypanosomes. After inoculation wet blood films were prepared everyday from each animal and examined for trypanosomes. Parasitemia was determined by counting the number of trypanosomes per microscopic field of wet blood film and was graded according to Cabrera and Lui (1956).

Blood glucose levels were estimated from each animal prior to inoculation and periodically after inoculation (Tables 1, 2, 3) with EYE-TONE* Reflectance colorimeter. Tissues of liver from each animal were preserved in absolute alcohol, processed and paraffin sections were stained with Best carmine method (Culling, 1963). Liver tissues from healthy animals were used as control.

* Miles India Limited.

TABLE 1

Blood glucose level of mice before and after artificial infection with *T. evansi.*

Mice No.	*Before inoculation mg/ 100 ml*	*After inoculation mg/ 100 ml*	*Number of days after inoculation*	*Intensity of parasitemia*
1.	170	155	3	---
2.	170	160	3	---
3.	110	90	5	---
4.	155	145	5	---
8.	165	145	7	†
10.	155	140	7	††
14.	170	140	9	†††
15.	165	135	9	†††
6.	175	130	11	†††
7.	160	125	11	†††
11.	175	120	11	††††
5. @	125	—	12	Died
9.	135	95	12	††††
12.	120	65	13	†††††
13.	170	100	13	†††††
Mean	156.78	124.64		
S.E.	5.51	7.37		
't' value		6.40*		

*Significant ($P<0.05$)

@ As mice died, not included for statistically analysis.

† Occasional Trypanosomes.

†† An average of one to two Trypanosomes per high power field.

††† An average of three to four Trypanosomes per high power field.

†††† Many Trypanosomes per high power field.

††††† Teeming with Trypanosomes.

RESULTS AND DISCUSSION

Blood glucose levels in mice, rats and rabbits before and after inoculation are presented in Tables 1, 2 and 3. In all the three species blood sugar levels decreased progressively as intensity of parasitemia increased.

TABLE 2

Blood glucose level of rats before and after artificial infection with *T. evansi.*

Rat No.	*Before inoculation mg/ 100 ml.*	*After inoculation mg/ 100 ml.*	*Number of day after inoculation*	*Intensity of parasitemia*
15	90	90	3	---
14	70	65	5	---
13	75	45	7	---
5	70	65	9	---
1	70	60	11	†
6	80	60	13	†
10	65	55	15	††
7	65	30	17	†††
8	85	50	19	††††
11	75	40	21	††††
2	70	50	23	†††††
12	90	60	23	††††
4	65	35	24	†††††
9	85	30	24	†††††
3 @	85	—	25	Died
Mean	75.35	52.50		
S.E.	2.43	4.34		
't' value	5.69*			

*Significant ($P<0.05$)

@ As rat died, not included for statistical analysis.

From the Tables 1, 2 and 3 it was observed that as the disease progressed, the blood sugar level dropped gradually in all the three species of laboratory animals and on 15th, 25th and 82nd day of infection in mice, rats and rabbits, respectively, it was found extremely low. The increased intensity of parasitemia during the course of infection was responsible for causing hypoglycemia by utilising the host's blood sugar. However, in case of rabbits, even when the trypanosomes were scarce in the circulating blood a lowered blood sugar level was observed which co-incided with the findings of Sen *et al.* (1959b).

TABLE 3

Blood glucose level (mg%) of rabbits before and after artificial infection with *T. evansi*.

Rabbit	*Before inoculation*	*Days after inoculation*										
		7	*15*	*21*	*30*	*37*	*45*	*52*	*60*	*67*	*75*	*£82*
1	105	100	80	—	—	—	—	—	—	—	—	—
2	125	120	110	105	95	—	—	—	—	—	—	—
5	140	135	120	115	105	100	100	—	—	—	—	—
3	155	150	150	140	130	125	115	80	75	—	—	—
4	130	125	120	120	115	105	95	80	80	75	70	—
6	145	145	140	135	135	120	100	90	85	75	75	70
Mean	133.33	129.16	120.00	123.00	116.00	112.50	102.50	83.33	80.00	75.00	72.50	70.00
S.E. ±	7.17	7.49	10.03	6.46	7.50	5.95	4.33	3.33	2.89	00.00	2.04	00.00
't' value		4.98*	3.99*	5.47*	4.95*	8.49*	19.60*	7.85*	7.18*	12.97*	27.00*	—

* Significant ($P < 0.05$)

£ As number of observations were very less on 82nd day P.I.
Statistical analysis was not done for the same.

A significant decrease in the blood sugar particularly during the terminal parasitemia with trypanosomes infection was reported by Jatkar and Singh (1974); Pegran and Scott (1976) and Mallick and Dwivedi (1981). Contrary to these findings, Gill (1977) reported normal or high blood sugar in cases of surra prior to death.

Disturbed carbohydrate metabolism in trypanosomes infection was reported by Balis (1964) and Kligler *et al.* (1929). Break down in glycolytic activity of liver resulted in glycopyruvic intoxication and death of animal (Gill, 1977). Parasitemia in rabbits was not found high even with the strain which was highly fatal to the mice. This evidence is against the hypothesis that sugar consumption by trypanosomes causing hypoglycemia, as observed in mice and rats, is a major pathogenic mechanism (Von Brand, 1966).

The sections of liver stained with Best carmine stain revealed progressive depletion of glycogen from the hepatic cells of mice, rats and rabbits. Glycogen depletion started after 5th day post inoculation (P.I.) in mice and rats, and 15th day P.I. in rabbits. Hepatic cells around the central vein were first to reveal glycogen depletion. The depletion slowly progressed towards the peripheral cells of hepatic lobules. By the end of 11th, 19th and 60th day P.I. all the hepatic cells were found to be devoid of glycogen in mice, rats and rabbits, respectively. Control animals did not reveal depletion of glycogen from hepatic cells.

Progressive total depletion of glycogen from hepatic cells noticed in all the three species of laboratory animals is possibly due to utilisation of considerable amount of glucose from the blood stream of host. Hypoglycemia with its consequences might be the primary cause of death (Sen *et al.*, 1959a).

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