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Susceptibility of Hedgehog, *Hemiechinus auritus* to *Schistosoma mansoni* under Experimental Infection

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ABSTRACT

This study aims to evaluate the susceptibility of the hedgehog (*Hemiechinus auritus*) to *Schistosoma mansoni* under the experimental condition. The susceptibility of this animal to *S. mansoni* infection was compared to the hamster (Permissive host) infection with the same parasite. In this study, 24 male of both hedgehogs and hamsters were used. Half of animals were infected with 250 *S. mansoni* cercariae. Eight weeks post infection; animals were sacrificed to determine the total worm burden, total eggs count, granuloma volume. The results revealed that a fecal examination of the infected hedgehogs showed no eggs were detected in the feces. The average of hamster's pre-patent period (the onset of patency) was 44.0±1.0 days. The average of *S. mansoni* worms recovered from hedgehogs was 31.0±10.0 worms, while the worm recovered from hamsters was 82.0±18.0. The granuloma volume measured from hedgehogs and the hamsters were 74.0±56.0 and 119.0±75.0, respectively. Marked reduction in the some biochemical parameters after the infection with *S. mansoni* was recorded in infected hedgehogs and hamsters. In conclusion, the hedgehogs showed non possibility for this animal to be a risk for schistosomiasis in the field.

Key words: Susceptibility, *Hemiechinus auritus, Schistosoma mansoni*, experimental, infection

INTRODUCTION

Schistosomiasis is an endemic disease in tropical and subtropical areas representing the second in importance to malaria from the socioeconomic point of view. It is endemic in 74 developing countries and effected between 391 to 597 million people worldwide (King, 2010). Recent studies showed that over 800 million, mostly children are at risk of infection (Gray *et al*., 2010; Siddiqui *et al*., 2011; Omonijo *et al*., 2013). In particular, the risk of infection in Egypt is still high due to the presence of S. *haematobium* and *S. mansoni* in the endemic areas of delta (El-Khoby *et al*., 2000), middle and Upper Egypt (Hammam *et al*., 2000). In Egypt, the epidemiology of schistosomiasis has been extensively investigated (Hotez, 2009), since the distribution and prevalence were documented for most affected areas (Gabr *et al*., 2000; Hammam *et al*., 2000). In recent study, over 89,000 persons in 251 rural communities found

to be infected. In 4 governorates, the prevalence of *S. mansoni* where it is endemic averaged 36.4% while *S. haematobium* where it is endemic average 7.8 % [http://www.pharmawriterpro.com/high-Prevalence-of schistosomiasis-in-Egypt.html].

So far, control of schistosomiasis depends mainly on praziquantel (PZQ) which becomes the drug of choice for the treatment worldwide. Due to the extensive uses of this drug as the only drug of choice, raise a question about the possibility of its resistance development, therefore, researchers have been trying to break down this resistance (Lar and Oyerinde, 2007). Furthermore, controversy studies showed that the new anti-schistosomal drug Mirazid may or may not be suitable to replace PZQ (Abdel-Aziz *et al*., 2006; El-Kott *et al*., 2011). Finding a potential vaccine for schistosomiasis is still under trials (Bashtar *et al*., 2006; Maghraby *et al*., 2007; Soliman, 2008).

It is well known that human is the most definitive host of *S. mansoni* and *S. haematobium* for maintenance of the life cycle, however, several studies showed that other mammalian species may contribute in maintaining the life cycle as well (Costa-Silva *et al*., 2002; El-Naggar *et al*., 2011). Several studies have been reported that most of the mammals with natural infection with *S. mansoni* were rodents, chimpanzee (Gentile *et al*., 2006; El-Naggar *et al*., 2011). In Egypt foci, however, it has found that the gerbil, the Nile rats and the field rats were naturally infected with both of *S. mansoni* and *S. haematobium* (Mansour, 1973). Recently, it has reported that under the experimental conditions, wild rodent species including Nile rats, house rats, Cairo spiny mice, black mice (Sisi) and field rats were found to be infected with *S. mansoni* and could be able to close the life cycle similar to human (El-Naggar *et al*., 2011). Similarly, in Brazil, it has found that the water-rats; *Nectomys squamipes* may present different levels of importance in the transmission of *S. mansoni* and could be considered important wild-reservoirs to this disease (D'Andrea *et al*., 2002; Gentile *et al*., 2006).

Hemiechinus auritus (the hedgehog) is a common insectivore in Egypt and it is widely distributed all over the Nile Delta. It's susceptibility to *S. mansoni* infection was not investigated yet. The present study was conducted to primarily evaluate its sensitivity to *S. mansoni* infection and to what extend the hepato-pathological changes may affects the life span of the affected animals.

MATERIALS AND METHODS

Hedgehogs (*Hemiechinus auritus)* and hamsters: Hedgehogs, *Hemiechinus auritus,* belong to order Insectivora, family Erinaceidae (Strocker, 2005). Twenty four male hedgehogs of body weight of 202.0±29.0 g were collected from the field and used in this study. Animals were treated with a single dose of praziquantel (PZQ), 2 weeks before the experimentation; therefore these animals were free from helminthic infections as judged by fecal examinations. They were acclimatized to the laboratory conditions for 2 weeks before experimentation and they maintained on laboratory standard balanced diet. Twelve males of the laboratory permissive host Syrian golden hamsters, *Mesocresitus auratus* were used as a control group. Hamsters of body weight of 92.0±13.0 g were obtained from the Experimental Animal Research Unit of the Schistosome biological supply program at Theodor Bilharz Research Institute, Giza, Egypt. Hedgehogs and hamsters were infected and housed under the laboratory condition at the animal facility of the Department of Zoology, Faculty of Science, Tanta University, Egypt.

Parasite and animal's infection: *Schistasoma mansoni* cercariae maintained in *Biomphalaria alexandrina* were used in this study. The parasite life cycle was kept in the

laboratory according to the method described by Christensen *et al.* (1984). Twelve hedgehogs and twelve hamsters were individually exposed to 250 *S. mansoni* cercariae for 2 h, by partial immersion of their body into the cercarial suspension (Christensen *et al.*, 1984). Twelve animals of each species were saved as normal controls.

In a different experiment, hamsters and hedgehogs were used to determine the survival rate upon 9 weeks post-infection with 250 *S. mansoni* cercariae.

Determination of the pre-patent period and the fecundity of female worms: Coprologic study was conducted to determine the pre-patent period of *S. mansoni* in the infected animals (the time from initial infection to the first appearance of eggs in the animal feces), in addition to the fecundity of female worms (the mean number of eggs produced per female worm per day). The pre-patent period was determined by daily inspection of the feces, according to the technique of Marshall *et al.* (1989), started from the 35th day post-infection. The fecundity of *S. mansoni* female was obtained by determination of the number of eggs per gram of feces every 3 days over a period of 2 weeks started from the 38th day post-infection. The fecundity was determined by dividing the average daily egg output in the feces of the animals by the number of mature female worms recovered.

Enumeration of males, females and the total worm burden: Eight weeks post infection, the animals were sacrificed. Briefly, to sacrifice hedgehogs, individual animals were anesthetized with suitable anesthesia to relax the abdominal muscle. Each animal was sacrificed and livers were perfused according to the technique of Christensen *et al.* (1984). Using normal saline containing anti-coagulant, liver of each animal was perfused to eliminate single and coupled worms from the liver. All worms were picked and transferred into clean petri dishes to be counted under dissecting microscopes. The mesenteric veins were then examined for picking any remaining adult worms trapped in.

Enumeration of eggs lodged in different organs: Tissue samples, each of 0.1 g, were excised from the lung, spleen, liver and intestine of the infected animals and then digested in 5% KOH solution. The number of eggs g^{-1} of tissue was estimated.

Granuloma measurements: For histo-pathological examination, selected liver specimens were fixed in 10% neutral formalin paraffin, sections of 6 μm thickness were prepared and then stained with hematoxylin and eosin methods. Lesions containing single eggs in their centers were selected for measurement and the diameter of each granuloma was obtained by measuring two diameters of the lesion at right angles to each other using an ocular micrometer. The mean diameter of at least 70 lesions from the hedgehog of each species was determined and the volume of each lesion was calculated, assuming a spherical shape (Cheever *et al.*, 1993), from its mean diameter using the following equation:

$$\text{Volume} = r^3 \times \frac{22}{7} \times \frac{4}{3}$$

$$r = \text{radius of granuloma } (\mu m)$$

Determination of the biochemical assays: Determination of the liver cholinesterase activity was estimated according to Ellman *et al.* (1961), arginase (Brown and Cohen, 1959). Alanine

aminotransferase (ALT) and aspartate aminotransferase (AST) were assessed according to Reitman and Frankel (1957). In addition, total lipids (Frings *et al.,* 1972), total protein (Lowry *et al.*, 1951) in the liver homogenate and serum albumin (Doumas *et al.*, 1971) were determined.

Statistical analysis of the data: Student's t- test was used to compare the mean volumes of the granulomas and the biochemical parameters measured. Values of $p<0.05$ were considered statistically significant.

RESULTS

Survival rate of infected animals: Exposure of hedgehogs and hamsters individually to *S. mansoni* infection at a dose of 250 cercariae per animal resulted into marked mortalities. Hedgehog was much more affected than hamster where 10 hedgehogs out of 24 exposed to the infection survived by the end of the observation period (9 weeks) whereas the number of hamster survived were 12 out of the 15 exposed to the infection (Fig. 1).

The onset of patency of *S. mansoni* infection and the fecundity of female worms: Fecal examinations of hedgehogs exposed to 250 *S. mansoni* cercariae per animal showed that no eggs were detected in the feces of the hedgehogs whereas the average pre-patent period (the onset of patency) was 44±1 days for the hamster. No significant difference was observed among the 5 fecal examinations carried out for the hamster ($H = 0.7$, $p = 0.9$) (Table 1). Also, the number of eggs per

Table 1: No. of eggs per gram of feces (epg) of the hamsters and hedgehogs in five coprologic examinations

	Post infection (days)							
Animals	38th	41st	44th	47th	50th	Epg Mean±SD	SmF No.	Fecundity (Epg SmF day^{-1})
Hamsters								
Mean±SD	1048±401	1089±325	1217±220	1147±194	1208±337	1142±65	43	29±8
Hedgehogs								
Mean±SD	0	0	0	0	0	0	16	0

SmF: *S. mansoni* female, SD: Standard deviation

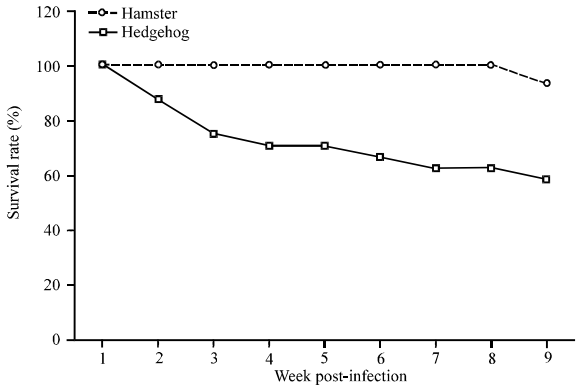

Fig. 1: Survival curve of *S. mansoni* infected hamsters and hedgehogs. Hamsters and hedgehogs were infected with 250 cercaria/animal and were housed for 9 weeks under experimental conditions

gram of feces did not correlate significantly with the number of female worms recovered ($r_s = 0.02$, $p>0.05$). Miracidial hatching tests showed that the eggs recovered from tissue of the hedgehog were viable and infective. The same observation was recorded for the hamster.

The hatching meracidia were used to infect clean snails (*Bimophalaria alexandrina*) and interestingly, snails infected with meracidia emerged from *S. mansoni* eggs harvested from either hedgehogs or hamster produced infective cercariae which were able to infect albino mice again.

The total worm burden recovery after the infection with *S. mansoni* cercariae: To address the capability and the susceptibility of hedgehogs to *S. mansoni* infection, after 8 weeks post-infection, all exposed animals to *S. mansoni* were sacrificed and the numbers of male, female and total worm number were assessed through liver perfusion and picking of any remaining worms from the intestinal mesenteries. The results showed that significant differences in the number of adult worms recovered between the two species were found. The hedgehogs were found to be loaded with 31.0±10.0 worms per animal, while the hamster harbored 82.0±18.0 per animal. Interestingly, the number of male worms was less than thenumber of female ones on both of infected hamsters and infected hedgehog (Table 2).

No significant difference was observed in the distribution of worms among the mesenteric, portal and intrahepatic veins of the hedgehog ($H = 5.7$, $p = 0.05$) and the hamster ($H = 3.5$, $p = 0.1$) (data not shown). In both species, the mesenteric veins contained the greatest average number of schistosomes, followed by the portal and intrahepatic veins. However, no significant difference was recorded between the mean number of male and female worms in the three regions.

The distribution of schistosome eggs in animal tissues: Tissue digestion showed that the greater number of eggs was contained in the intestine, followed by the liver. These results were common to the hedgehog and the hamster. The difference observed was not significant ($t = -1.2$, $p = 0.3$) in hedgehog but significant ($t = -7.9$, $p = 0.0$) in the hamster. In the hedgehog, few eggs were found in the lung and no eggs were recorded in the spleen, while in the hamster few eggs were found in both organs. With respect to the total tissue egg count, a greater number of eggs were observed for the hamster than for the hedgehog ($t = -7.9$, $p = 0.0$) (Table 3).

Table 2: No. of worms recovered from the hedgehog and the hamster eight weeks post exposure to *S. mansoni* cercariae

	Worm burden and granumola volume (after 8 weeks post-infection)			
Animal groups	SmM	SmF	Total	Granuloma volume
Hamsters (Cont.)	39.0±8.50	43.0±10.0	82.0±18.0	119.0±75.0
Hedgehogs	15.0±4.50*	16.0±5.5*	31.0±10.0*	74.0±56.0

*SmM: *S. mansoni* male, SmF: *S. mansoni* female. (μm^3) *Significant at $p<0.05$

Table 3: Distribution of *S. mansoni* eggs in the tissues of the hamsters and the hedgehogs

Animals	Intestine	Liver	Lung	Spleen
Hamsters				
Mean±SD	71116±1447	17741±3712	166±45	166±235
Hedgehogs				
Mean±SD	11891±6339*	8008±2260*	102±35	0

*Significant at $p<0.05$

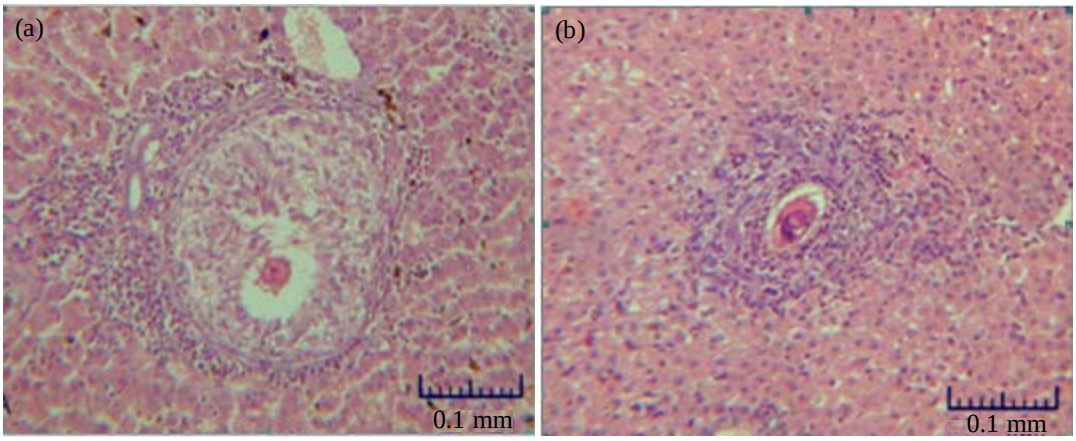

Fig. 2(a-b): Typical granulomatous reaction of the infected (a) Hamster and (b) Hedgehogs

Granuloma volume measurements: To measure the granuloma volume of the 8 week-infected hedgehogs and hamsters, the mean diameter of at least 70 lesions from each species were determined and then the volume of each lesion was calculated to determine the granuloma volume. Figure 2 (a and b), the histo-pathological examination of the liver sections of the infected hamsters and the infected hedgehogs showed typical granulomatous reactions formed mainly of histocytes lymphocytes and eosinophils. Microscopic examination showed that there are no any calcified dead eggs in the liver tissues of the infected hedgehogs and most of eggs contain live meracidia and this observation could explain that these animals were recently infected with *S. mansoni* and they did not naturally infected. The results showed also that the granulomas volume of the infected hedgehogs measured 74.0±56.0 μm^3 which was significantly smaller than those of the infected hamsters; 119.0±75.0 μm^3.

Effect of *S. mansoni* infection on the biochemical parameters in liver tissues: The current study showed also that *S. mansoni* infection caused marked hepatic dysfunction in the hedgehogs as well as in the hamsters.

The liver enzymatic activities of cholinesterase, arginase, alanine aminotransferase (ALT) and aspartate aminotransferase (AST) were markedly reduced after 8 weeks post *S. mansoni* infection as compared to their counterparts. Furthermore, infection of hedgehogs and hamsters with *S. mansoni* led to decrease in the liver total protein contents and serum albumin, with an increase in the total lipids when compared with their normal counterparts (Table 4).

DISCUSSION

Although, *Schistosoma mansoni* shows a low specificity in relation to its vertebrate host choice, only few species are capable of developing the infection and allowing the parasite to complete its biological cycle in natural environment (Rey, 1993). The high susceptibility of different mammalian species to *S. mansoni* infection was reported (Barbosa, 1972; Costa-Silva *et al*., 2002; El-Naggar *et al*., 2011). So far, there are few data concerning about the potential role of the hedgehog, *Hemiechinus auritus* to act as a reservoir host for *S. mansoni* infection. In this study,

Table 4: Biochemical disorders in the liver of animals infected with *S. mansoni* infection

	Hamster			Hedgehog		
	Normal	Infected		Normal	Infected	
Biochemical parameters	Mean±SD	Mean±SD	Diff. (%)	Mean±SD	Mean±SD	Diff. (%)
Arginase[1]	61.5±26	30.6±8.5*	-50.2	95±16.8	18.4±9.5*	-80.6
Cholinesterase[2]	17±7.9	3±0.6*	-82.3	6.5±0	2.9±0.5*	-55.4
ALT[3]	19±3.7	2±3.8*	-36.8	25.3±3.6	5.4±0.9*	-78.6
AST[4]	22.4±1.9	11±0.9*	-50.8	13±1.4	5.3±1.8*	-58.5
Total lipids[5]	42±14.2	121±27.4*	+188	65.5±1.0	104.8±22*	+60
Total protein[6]	100±11.4	98±7.2	-86	120±12.5	80±2.0*	-33.3
Albumin[7]	6±0.0	4.1±0.7*	-31.6	5.5±0	3±0.6*	-45.4

*Significant at $p<0.05$, activities and contents were expressed in: (1) µmol urea min^{-1} wet liver tissue (2) µmol SH min^{-1} wet liver tissue (3 and 4) µmol pyruvate min^{-1} wet liver tissue (5 and 6) mg g^{-1} wet liver tissue (7) mg (%)

some aspects of *S. mansoni* biology in the hedgehogs were studied. Susceptible hosts to *S. mansoni* have been defined as those in which development of worms is complete, excretion of viable eggs in feces is not interrupted and parasite life cycle is maintained (Cioli *et al.,* 1977).

The present study showed that no eggs were passed in the feces of the hedgehog, therefore, the hedgehog considers as a dead-end host for schistosomiasis. This indicates that some factors may influence the excretion of eggs with feces. These may include intrinsic or extrinsic factors to the parasites. Furthermore, the physiological state of the intestinal mucus can also cause the differences in egg excretion. Eggs deposited in mesenteric veins must migrate through the intestinal wall before being excreted with feces (Sene *et al.*, 1996).

In this study, it has found that the enumeration of worm burden of the hedgehogs have low susceptibility to *S. mansoni* compared to the hamster. The worms were detected in the liver as well as in the mesenteric veins. These results were in agreement with Dias *et al.* (1978).

They reported that adult *S. mansoni* worms were found to be concentrated in portal and intra-hepatic veins in naturally infected *Cavia aprea* and *Holochilus leucogaster*. Similar findings were obtained from *Nectomys squamipes* naturally infected with *S. mansoni* (Silva *et al.,* 1992).

In the hedge hog and the hamster, male and female worms were found to be evenly distributed in the mesenteric, portal and intrahepatic veins. These data disagreed with those found in *N. squamipes,* where a greater proportion of male worms were observed in the liver as well as the mesentery (Silva *et al.,* 1992). Tissue digestion showed that in the hedgehogs and the hamsters, the largest number of eggs was contained in the intestine, followed by the liver. The total tissue eggs count was comparable in both animals. Quantitative oogram showed that a greater number of eggs were contained in the proximal portion of the intestine and that was in accordance with the results previously reported for *N. rattus* and *N. squamipes* (Silva *et al.,* 1992).

Infection of the hedgehogs with *S. mansoni* caused marked pathological changes at histo-pathological and biochemical levels. The infection with *S. mansoni* caused the development of the characteristic granulomatous inflammatory reaction reported in permissive hosts; hamsters as well as the hedgehogs. Although of smaller volume of the granulomas were found in the liver hedgehogs, the cellular characteristic features of the inflammations are the same as those developed in the permissive host (Fig. 2). The reduction of granuloma diameter could be due to the reduction of type III procollagen which is responsible for the granulomas formation (Badawy *et al.*, 1991).

As a result of *S. mansoni* infection, the liver is manifested by dramatic changes of the enzymatic activities. The current results showed that the values of the liver enzymatic activities of cholinesterase, arginase, ALT and AST, were markedly inhibited as a result of *S. mansoni* infection. These results were in agreement with the previous studies which showed that cholinesterase and arginase activities of the liver tissue homogenate showed a significant reduction in murine *S. mansoni* (Al-Sharkawi, 1996). With regard to transaminases, ALT is more specific for liver damage than AST (Wilkinson, 1976). The results showed also that the activities of ALT and AST in the liver tissue homogenate were reduced and this finding was in agreement with those found by Al-Sharkawi *et al.* (1989).

S. mansoni infection caused marked increase of the hepatic content of total lipids, which reflects the liver incapability for their mobilization. The liver contents of protein, serum albumin were also decreased while; the liver content of total lipids was elevated after the *S. mansoni* infection of hamsters and hedgehogs. Such increased water intake may explain the decrease in the total content of the liver protein reported in this study. However, the reduction of serum albumin is consistent and extends previous observations in experimentally infected monkeys (Bruce *et al.,* 1963) and mice (Goodgame *et al.,* 1978; Zakaria *et al.,* 1981). In conclusion, the hedgehog showed low susceptibility to *S. mansoni* infection as compared to the hamster and such infection results in marked severe hepatic dysfunction. Furthermore, this host is not appropriate for schistosomiasis transmission in the field.

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