Mem. Inst. Oswaldo Cruz, Rio de Janeiro, Vol. *84* (3): 281-288, jul./set. 1989

A POTENTIAL VECTOR OF *SCHISTOSOMA MANSONI* IN URUGUAY

W. LOBATO PARAENSE & LYGIA R. CORRÊA

Instituto Oswaldo Cruz, Departamento de Malacologia, Caixa Postal 926, 20001 Rio de Janeiro, RJ, Brasil

Susceptibility experiments were carried out with a Biomphalaria straminea*-like planorbid snail (*Biomphalaria *aff.* straminea, *species inquirenda) from Espinillar, near Salto (Uruguay), in the area of the Salto Grande reservoir, exposed individually to 5 miracidia of* Schistosoma mansoni *(SJ2 and BH2 strains).*

Of 130 snails exposed to the SJ2 strain, originally infective to Biomphalaria tenagophila, *30 became infected (23%). The prepatent (precercarial) period ranged from 35 to 65 days. The cercarial output was irregular, following no definite pattern, varying from 138 to 76,075 per snail (daily average 4.3 to 447.5) and ending up with death. Three specimens that died, without having shed cercariae, on days 69 (2) and 80 after exposure to miracidia, had developing secondary sporocysts in their tissues, justifying the prospect of a longer precercarial period in these cases. In a control group of 120* B. tenagophila, *exposed to the SJ2 strain, 40 became infected, showing an infection rate (33.3%) not significantly different from that of the Espinillar snail ($\chi^2 = 3.26$).*

No cercariae were produced by any of the Espinillar snails exposed to miracidia of the BH2 strain, originally infective to Biomphalaria glabrata. *Four specimens showed each a primary sporocyst in one tentacle, which disappeared between 15 and 25 days post-exposure, and two others died with immature, very slender sporocysts in their tissues on days 36 and 54. In a control group of 100* B. glabrata *exposed to BH2 miracidia, 94 shed cercariae (94%) and 6 remained negative.*

Calculation of Frandsen's (1979a, b) TCP/100 index shows that "Espinillar Biomphalaria*-SJ2* S. mansoni" *is a vector-parasite "compatible" combination. Seeing that* tenagophila*-borne schistosomiasis is prevalent in Rio de Janeiro and São Paulo states and has recently spread southwards to Santa Catarina state, and that the range of* B. tenagophila *overlaps that of the Espinillar* Biomphalaria, *the possibility of schistosomiasis establishing itself in Uruguay, although not imminent, is not to be disregarded.*

Key words: *Biomphalaria* aff. *straminea* – Planorbidae – *Schistosoma mansoni* – potential schistosome vector – Uruguay

In late November 1987, during a course on schistosomiasis organized by the Comisión Técnica Mixta de Salto Grande (Argentina-Uruguay) and the Universidad Nacional del Nordeste (Argentina), we took several samples of snails in the area of the reservoir. One of them consisted of a small planorbid, abundant in creeks, ditches and artificial ponds at Espinillar, a sugar mill and plantation complex near Salto (31°23S, 57°58'W), Uruguay, morphologically similar to *Biomphalaria straminea* (Dunker, 1848), and certainly corresponding to the snail identified as such by Olazarri (1984). Descendants of about 100 specimens of that population, taken from an artificial pond, are being kept in this laboratory. This paper reports the results of experiments to ascertain their susceptibility to infection with *Schistosoma mansoni.* As the specific identity of the planorbid involved is under investigation, since it shows some degree of morphologic and genetic divergence from typical *B. straminea*, its definitive identification depends on the result of that investigation.

In previous searches for freshwater snails from Paso de los Libres to Concordia and Salto (March 1973), the senior author found the planorbids *Biomphalaria peregrina, B. tenago-*

Received December 27, 1988.
Accepted May 22, 1989.

phila, Drepanotrema anatinum, D. kermatoides, D. lucidum and *Antillorbis nordestensis*, besides physids, lymnaeids, ancylids, ampullariids, hydrobiids and sphaeriids. The same taxa, plus *Drepanotrema heloicum*, had been found in March and October 1965 in the region from Montevideo to Maldonado (Paraense, 1965). It seems, therefore, that the present abundance of that *straminea*-like planorbid in the area of the Salto Grande reservoir may be ascribed to recent colonization.

MATERIAL AND METHODS

The experimental snails were 230 juveniles of the Espinillar strain, 3-4 mm in shell diameter (about 3 weeks old), exposed each to 5 *S. mansoni* miracidia of the SJ2 strain (130 snails) and of the BH2 strain (100 snails). The SJ2 *S. mansoni* strain was isolated on March 31, 1982 from four naturally infected *Biomphalaria tenagophila* among 50 collected from the same biotope at São José dos Campos, São Paulo state, from which a discontinued strain mentioned as SJ in previous papers originated. The BH2 *S. mansoni* strain was isolated on May 13, 1985 from 11 naturally infected *Biomphalaria glabrata* among 50 collected from an irrigation ditch at Ressaca, a district of Belo Horizonte city, Minas Gerais state.

Both strains have been kept by passages through syntopic (from the same breeding place) SJ2 *B. tenagophila* and BH2 *B. glabrata*, and female Swiss albino mice.

The miracidia were obtained from eggs concentrated, by Lutz's (1919: 116) sedimentation technique, from feces of mice infected transcutaneously (SJ2 strain, 5th passage; BH2 strain, 3rd passage), 8-10 weeks earlier, by exposure to cercariae. Mice were induced to defecate by putting them in a layer of water, about 1 cm thick, at the bottom of a jar high enough to prevent jumping over the rim. Fecal pellets were collected, comminuted, suspended in dechlorinated tap water, and poured through previously water-soaked surgical gauze (four thicknesses) into a conic sedimentation glass. The sediment was washed by decantation, three or more times, at intervals of 30 min, until the water got clear. Manipulation and sedimentation of fecal material was carried out at about 10 °C to prevent early hatching of miracidia. The final concentrate of eggs was transferred to a petri dish and exposed to the light of electric lamps (28-30 °C). As the miracidia hatched, they were pipetted under a stereomicroscope and dropped into tissue culture wells, in each of which a snail had already been put. Then just enough dechlorinated tap water was added to cover the snail, the well plate was covered, and the snails were left there overnight. The snails were then removed to aquaria (kept covered with an acrylic plate to prevent cooling of water below room temperature by evaporation), and observed at least two times daily. If any specimen happened to die, it was dissected and examined for developing stages of the schistosome. On the 25th day after exposure to miracidia, and then every 5th day, the snails were isolated in vials with water and exposed to the light of electric lamps (28-30°C) to induce shedding of cercariae. Moribund specimens, as well as those that survived for 90 days without shedding cercariae, were dissected and examined.

The emission of cercariae was followed until death in all the positive specimens. Each infected snail, kept separately in a small aquarium, was exposed to light, as described above, on Mondays, Wednesdays and Fridays, from 9 AM to 5 PM, in a vial with 2 ml dechlorinated tap water, after which they were returned to the aquaria. To each vial 0.5 of 2% aqueous ninhydrin was added, the mixture was gently swirled and emptied into a 10 cm diam pyrex petri dish lined with a somewhat wider (11 cm) filter paper molded into it so as to form an internal dish with raised border. The paper was steamed until dry by placing the petri dish on a hot plate. The dry paper was stored for subsequent count of the cercariae, which appear violet against a white background. The total count for each snail was divided by the number of examined samples to find the average number of cercariae per sample. The product of this individual average by the duration in days of cercarial output gave an estimate of the total number of cercariae shed by that snail if daily counts had been made.

The aquaria were kept at a room temperature of 24-26°C throughout the experiment.

For comparison of infection rates, 120 *B. tenagophila* (SJ2 strain) and 100 *B. glabrata* (BH2 strain), 3-5 mm in shell diameter, were exposed each to 5 syntopic miracidia, and followed up as described above, except for counts of cercariae.

TABLE I

Results of exposure of 130 *Biomphalaria* aff. *straminea* (species inquirenda) from Espinillar (Salto, Uruguay), 3-5 mm in shell diameter, to the SJ2 strain of *Schistosoma mansoni* (5 miracidia per snail)

Days after exposure	Exposed snails	Results[a]
22-34	4	Dead, negative
34	1	Dead, SP in DG, OT
35	7	Shed cercariae (nos. 1, 2, 3, 4, 9, 10, 17)
39	1	Dead, negative
39	1	Dead, SP in RR, DG, OT
40	8	Shed cercariae (nos. 5, 6, 7, 8, 11, 16, 18, 19)
40	1	Dead, negative
47	1	Dead, SP in RR, DG, OT
50	4	Shed cercariae (nos. 12, 13, 14, 15)
50	1	Dead, SP in HE, FO, MC, RR, PW, DG, OT
50	1	Dead, negative
63	1	Dead, negative
65	1	Shed cercariae (no. 20)
67	1	Dead, negative
69	1	Dead, SP in MC, PN, RR, PW, DG, OT
69	1	Dead, SP in FO, MC, PN, RR, PW, DG, OT
78	1	Dead, negative
80	1	Dead, SP in MC, RR, PW, DG, OT
84	1	Dead, negative
90	1	Dissected, few SP with cercariae in FO, PN, RR, PW, DG, OT
90	2	Dissected, few encapsulated slender SP without cercariae in FO, DG, OT
90	89	Dissected, negative

[a] Infection rate 23%. DG, digestive gland; FO, foot; HE, head; MC, mantle collar; OT, ovotestis; PN, pneumostome; PW, pulmonary wall; RR, rectal ridge; SP, sporocyst. Snail nos. refer to Table III.

RESULTS

Of the 130 Espinillar snails exposed to SJ2 miracidia (Table I), 30 became infected (23%). Of the latter, 20 shed cercariae, 7 died between 34 and 80 days post-exposure with unripe sprocysts in the body tissues, and 3 also proved infected with sporocysts when dissected at the end of the experiment (90 days post-exposure).

Five of the infected specimens showed secondary sporocysts restricted to the digestive gland and ovotestis (Tables I and II). In the remaining 25 the sporocysts were spread more or less extensively through other organs in addition to the mentioned ones. Developing sporocysts were present in one or more external organs (head, foot, tentacles, mantle collar, pneumostome and pseudobranch) in 14 specimens which died between 48 and 80 days post-exposure, and 3 which were killed and dissected on the 90th day.

As shown in Table I, the earliest shedding of cercariae was observed on the 35th day post-exposure (7 specimens), and in the majority of the snails the onset of shedding occurred later: 40 days in 8 specimens, 50 days in 4, 65 days in 1. Thus the prepatent (precercarial) period varied from 35 to 65 days.

In the infected snails that died without shedding cercariae (Table I), including one killed on day 90, cercariae were present in variable numbers within secondary sporocysts, indicating that cercarial output would have occurred should the snails live longer, in which case a much longer prepatent period would be recorded. Two other surviving snails dissected on day 90 showed slender secondary sporocysts, still immature (without cercariae), in the foot, digestive gland and ovotestis.

The emission of cercariae was irregular, following no definite pattern. Representative examples are shown in Fig. 1. It can be seen that quite different curves of cercarial emission are described in infections of longer duration (snails nos. 9 and 16), as well as in those of shorter and middle duration. In the days that preceded death cercarial output gradually decreased in about half of the snails and dropped to zero in the other half.

TABLE II

Cercarial output, survival after exposure to miracidia (5 per snail) and postmortem findings in *Biomphalaria* aff. *straminea* (species inquirenda) from Espinillar (Salto, Uruguay), infected with the SJ2 strain of *Schistosoma mansoni*

Snail no.[a]	Survival after exposure (days)	Cercarial output					Postmortem findings[d] (organs with SP)
		Duration (days)	Examined samples[b]	Total count	Total estimate[c]	Daily average	
1	94	53	26	2,751	5,607	105,8	FO, MC, PN, RR, PW, DG, OT
2	64	23	13	563	996	43.3	MC, PN, RR, DG, OT
3	43	7	4	209	365	52.2	MC, PW, DG, OT
4	82	47	21	3,142	7,031	149.6	PN, PW, DG, OT
5	71	24	13	2,297	4,240	176.7	DG, OT
6	48	7	4	175	306	43.7	MC, PB, RR, PW, DG, OT
7	74	32	15	314	669	20.9	DG, OT
8	74	32	15	65	138	4.3	DG, OT
9	128	92	40	437	1,002	10.9	FO, MC, PN, RR, PW, DG, OT
10	75	38	17	670	1,497	39.4	AG, DG, OT
11	73	31	14	1,045	2,312	74.6	DG, OT
12	62	10	5	338	676	67.6	RR, PW, DG, OT
13	74	22	10	112	246	11.2	RR, PW, AG, DG, OT
14	125	74	32	3,612	8,355	112.9	HE, MC, PN, RR, PW, AG, DG, OT
15	81	29	13	482	1,076	37.1	MC, PN, DG, OT
16	210	170	73	32,669	76,075	447.5	PW, DG, OT
17	41	5	3	515	858	171.7	MC, PN, RR, PW, DG, OT
18	88	46	21	1,155	2,530	55.0	MC, PW, DG, OT
19	120	79	35	4,669	10,539	133.4	RR, PW, DG, OT
20	99	34	15	2,670	6,052	178.0	FO, MC, PW, DG, OT
					130,570		
Range	41-210	5-170	3-73	65-32,669	138-76,075	4.3-447.5	

[a] Snail nos. refer to Table I.
[b] Triweekly samples.
[c] Duration of cercarial output in days x daily average.
[d] AG, albumen gland; DG, digestive gland; FO, foot; HE, head; MC, mantle collar; OT, ovotestis; PB, pseudobranch; PN, pneumostome; PW, pulmonary wall; RR, rectal ridge; SP, sporocyst.

With the exception of three specimens which were killed on day 90, the duration of infection after exposure to miracidia varied from 34 to 128 days in 26 snails and reached 210 days in one. Survival after onset of cercarial output varied from 5 to 10 days in 4 specimens and from 22 to 92 days in 15 (average 36 days). Snail no. 16 shed cercariae for 170 days and showed the highest production, both in daily average and total output. If its cercarial production is added to that of the preceding 19, the average duration of cercarial shedding for the 20 snails will up to 42.7 days.

Table II shows some data on survival after exposure, duration and quantification of cercarial output, and location of developing stages of the parasite, concerning the 20 snails that shed cercariae.

Of the 120 SJ2 *B. tenagophila* exposed to syntopic miracidia, 40 became infected (33.3%). One died negative on day 31 after exposure, two died with developing sporocysts on days 14 and 34, 38 began shedding cercariae from days 25 to 75, and 79 proved negative on dissection on day 90.

No cercariae were produced by any of the 100 Espinillar snails exposed to BH2 miracidia. Four specimens showed each a primary sporocyst in one tentacle, which disappeared between 15 and 25 days post-exposure; one of them died negative on day 47. Two other specimens died with immature, very slender sporocysts on days 36 (4 in the digestive gland) and 54 (2 in the digestive gland and 1 in the ovotestis). Seven died negative between 25 and 72 days post-exposure, and the remaining 90

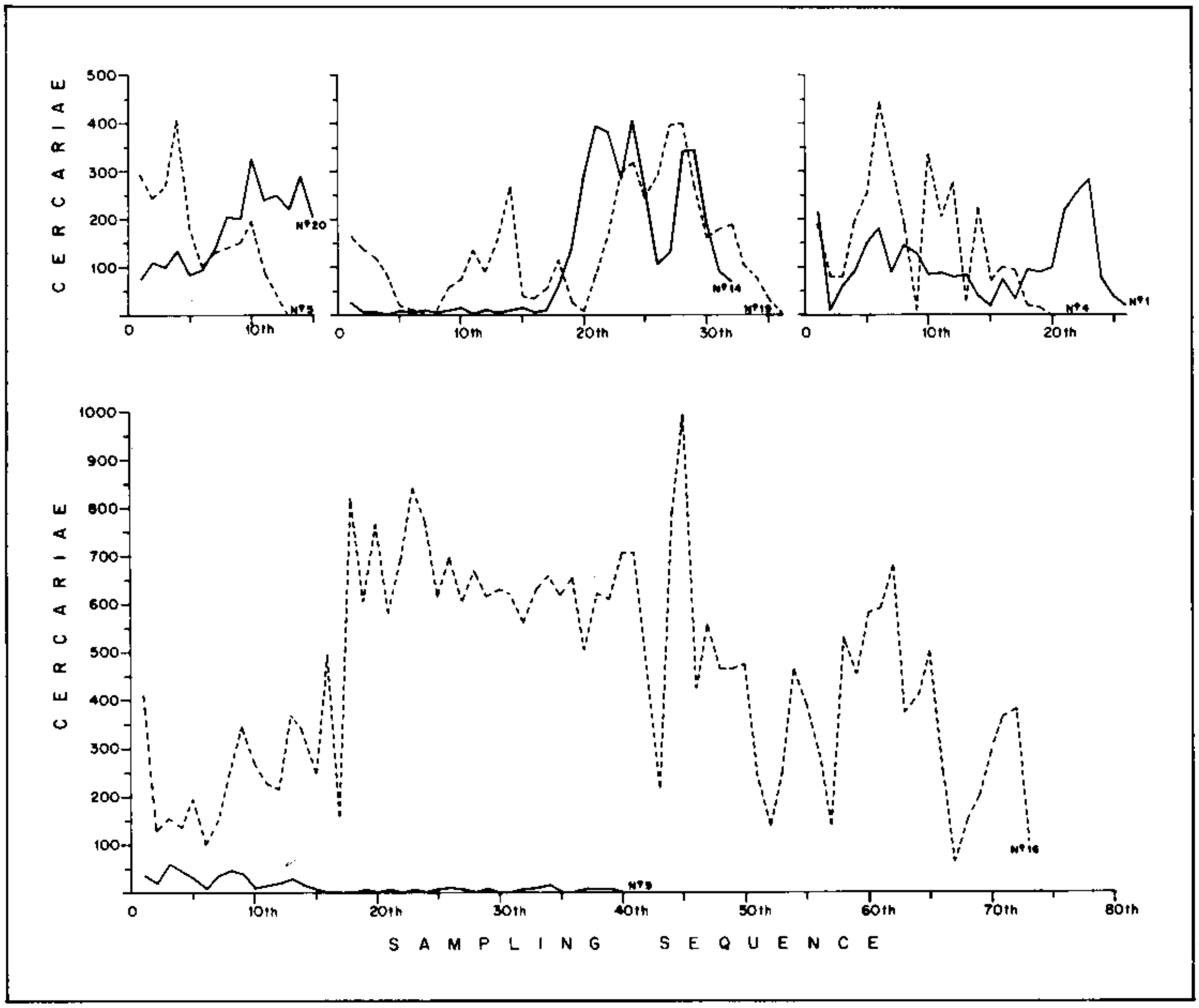

Fig. 1: cercarial output from *Biomphalaria* aff. *straminea* (species inquirenda) from Espinillar (Salto, Uruguay), infected with the SJ2 strain of *Schistosoma mansoni.* Triweekly samples, taken on Mondays, Wednesdays and Fridays.

(including the three survivors that had shown primary sporocysts in the tentacles) were negative when dissected on day 90. On the other hand, of the 100 BH2 *B. glabrata* (control group) exposed to BH2 miracidia, 82 shed cercariae on the 25th day post-exposure and 12 on the 30th (infection rate 94%), the remaining 6 proving negative on dissection 90 days post-exposure.

DISCUSSION

The above results point to a significant level of susceptibility of the *Biomphalaria* from Espinillar to infection with the SJ2 strain of *S. mansoni.* Several characteristics of the infection, however, suggest some degree of incompatibility between the parasite and the snail, as discussed now.

In a former experiment (Paraense & Corrêa, 1981), using one of the most compatible combinations so far recognized in this country — BH strain of *B. glabrata* and BH strain of *S. mansoni* —, we observed, in 69 snails exposed individually to 1 miracidium, a prepatent period of 30-35 days (mean 34.86 ± 4.62 SD) and no persistence of developing parasites after that period. On the other hand, in the present experiment the prepatent period was of 35-65 days (mean 41.50 ± 7.80 SD), with a highly significant difference between the means ($P < 0.001$, t test), and in 5 specimens that died after the 65th day or were killed on day 90 the infection had not yet reached the cercarial stage. Moreover, in those 5 specimens and in 11 of the 20 that shed cercariae (Table II) immature sporocysts were still present at the sites of penetration of miracidia in exposed soft

TABLE III

Natural and experimental infection of *Biomphalaria straminea* from Pernambuco state, Brazil, with *Schistosoma mansoni* from that state, and prevalence of human infection at the same localities[a]

Locality[b]	Natural infection[c]		Experimental infection[e]		Human infection[f]	
	Examined snails[d]	Positive %	Examined snails	Positive %	Examined subjects	Positive %
Bezerros	13,210	0.01	96	0.0	541	67.42
Carpina	8,060	0.07	98	0.0	890	14.28
Catende	5,300	0.0	45	0.0	648	51.90
Escada	15,985	0.13	49	0.0	453	82.11
Gravatá	21,000	0.00	62	0.0	571	67.96
Moreno	13,500	0.00	71	0.0	1,067	66.54
Paudalho	10,760	0.04	51	0.0	452	57.96
Ribeirão	7,495	0.03	41	2.4	401	74.06
Timbaúba	13,540	0.02	21	4.8	739	58.45
Vicência	15,715	0.01	48	4.2	222	85.58

[a] Correspondence between the above data (though not simultaneously collected) is valid, since no measures against snails or human infection were applied during the period: "Until 1975 there were no systematic attempts to control the disease. Isolated local efforts with limited resources have been reported. The only available drug, hycanthone, was not suitable for mass use because of its contraindications and side effects" (Machado, 1982).

[b] Localities where the only known vector is *B. straminea.*

[c] Data from Nov. 1954 to Oct. 1955 (Barbosa & Coelho, 1956).

[d] Named *Tropicorbis centimetralis* (a junior synonym of *B. straminea*).

[e] 10 miracidia per snail. Data from Sept. 1964 to Aug. 1967 (Barbosa & Figueiredo, 1970).

[f] Data from Aug. to Nov. 1948 (Pellon & Teixeira, 1950).

parts such as head, foot, mantle collar, pseudobranch and pneumostome. The lengthening of the prepatent period and the retention of developing primary sporocysts at the site of penetration can be seen as manifestations of host resistance to the parasite, leading to a slowing of development of the latter and indicating some degree of incompatibility between the host-parasite systems. That such level of incompatibility will not preclude the snail from acting as an efficient vector may be inferred from observations with *B. straminea.* As widely known, this species is a major vector of schistosomiasis in northeastern Brazil. It opposes a cellular defense mechanism to the penetrating miracidia, usually sheds much less than 500 cercariae on a daily average, and exceptionally survives the first month of the patent period (Coelho & Barbosa, 1956, as *Tropicorbis centimetralis,* a junior synonym). As a result of such unbalanced host-parasite relationship, the natural infection rate of *B. straminea* seldom reaches 1%, and in the great majority of instances is under 0.1%. Very low experimental rates are also the rule. If records of natural and experimental infection rates of *B. straminea* from a number of localities where this snail is the only known vector in the state of Pernambuco are compared with the prevalence of human schistosomiasis in the same localities (Table III), it can be concluded that *B. straminea,* in spite of behaving as a poor host of *S. mansoni,* must be considered in fact a good vector of the parasite, since it is able to maintain high endemicity levels in those localities.

A total cercarial output of 130,570 was estimated for the Espinillar *Biomphalaria*-SJ2 *S. mansoni* combination in the present study (Table II). Calculation of the TPC/100 index (total cercarial production $\times \frac{100}{130}$ exposed snails = 100,438) proposed by Frandsen (1979a) indicates that the present combination falls into Class III (compatible), i. e. midway between Class 0 (refractory) and Class VI (extremely compatible) (Frandsen, 1979b).

The conclusion seems obvious that, from the standpoint of its degree of compatibility with the SJ2 strain of *S. mansoni,* the *Biomphalaria* from Espinillar can be considered a potential vector of schistosomiasis in its area of distribu-

tion. By the way, the difference between its infection rate (23%) and that of the SJ2 *B. tenagophila* (33.3%) is not significant ($\chi^2 = 3.26$), and the last-mentioned species is a major vector in the states of Rio de Janeiro and São Paulo.

As previously shown, the SJ strain of *S. mansoni* is infective not only to the syntopic (SJ) *B. tenagophila* (Paraense & Corrêa, 1963) but also to other populations of this planorbid species throughout its range (Paraense & Corrêa, 1978). In recent years *B. tenagophila*-borne schistosomiasis has spread southwards from Rio de Janeiro and São Paulo to Santa Catarina state (Bernardini & Machado, 1981). The possibility of further southward extension to Rio Grande do Sul state was recently demonstrated (Paraense & Corrêa, 1987).

Numerous observations along the last 30 years have witnessed a steady expansion of schistosomiasis through the geographic domain of *B. tenagophila*, which overlaps that of the planorbid species now under investigation. Since the latter and *B. tenagophila* are both susceptible to the SJ2 strain of *S. mansoni*, the possibility of the establishment of schistosomiasis in Uruguay must be contemplated. As to the BH2 strain, which is representative of *B. glabrata*-borne *S. mansoni* and, according to our experience, hardly infects *B. tenagophila*, its failure to infect the *Biomphalaria* from Espinillar indicates that it does not constitute so imminent a threat to the area of distribution of this snail as the *B. tenagophila*- borne schistosome.

RESUMO

Um vetor potencial do *Schistosoma mansoni* no Uruguai – Foram feitas provas de suscetibilidade com um molusco planorbídeo semelhante à *Biomphalaria straminea* (species inquirenda) de Espinillar, localidade próxima a Salto (Uruguai), na área da represa de Salto Grande, cada exemplar sendo exposto individualmente a 5 miracídios de *Schistosoma mansoni* (cepas SJ2 e BH2).

De 130 exemplares expostos à cepa SJ2, originalmente infectante para *B. tenagophila*, 30 se infectaram (23%). O período pré-patente (pré-cercariano) variou de 35 a 65 dias. A emissão de cercárias foi irregular, não seguindo padrão definido, variando de 138 a 76.075 por exemplar (média diária de 4,3 a 447,5) e terminando com a morte. Três exemplares que morreram, sem ter eliminado cercárias, no 69º (2) e no 80º dia após exposição aos miracídios, tinham esporocistos secundários em desenvolvimento nos tecidos, justificando a expectativa de um período pré-patente mais longo nestes casos. Em um grupo-controle de 120 *B. tenagophila*, exposto à cepa SJ2, 40 se infectaram, não diferindo significativamente seu índice de infecção (33.3%) daquele do planorbídeo de Espinillar ($\chi^2 = 3,26$).

De 100 exemplares de Espinillar expostos a miracídios da cepa BH2, originalmente infectante para *B. glabrata*, nenhum produziu cercárias. Um esporocisto primário formou-se em um tentáculo em 4 exemplares, desaparecendo entre 15 e 25 dias após a exposição. Dois outros exemplares morreram com esporocistos imaturos e muito delgados nos tecidos (4 em um caso e 3 no outro), no 36º e 54º dias. Em um grupo-controle de 100 *B. glabrata* exposto à cepa BH2, 94 emitiram cercárias (94%) e 6 permaneceram negativos.

De acordo com o índice TCP/100 de Frandsen (1979a, b), a combinação *Biomphalaria* de Espinillar-*S. mansoni* SJ2 constitui uma relação vetor-parasito "compatível". Tendo em vista que a xistosomose transmitida pela *B. tenagophila* é prevalente nos estados do Rio de Janeiro e São Paulo e recentemente propagou-se para o sul até o estado de Santa Catarina, e que a distribuição geográfica da *B. tenagophila* sobrepõe-se à da *Biomphalaria* de Espinillar, a possibilidade do estabelecimento da xistosomose no Uruguai, ainda que não iminente, não deve ser desconsiderada.

Palavras-chave: *Biomphalaria* aff. *straminea* – Planorbidae – *Schistosoma mansoni* – Vetor potencial de *Schistosoma* – Uruguai

ACKNOWLEDGEMENT

We are grateful to Arq. Winston Mañosa, head of the Department of Ecology, Comisión Técnica Mixta de Salto Grande, for facilities for field work at El Espinillar.

REFERENCES

BARBOSA, F. S. & COELHO, M. V., 1956. Alguns aspectos epidemiológicos relacionados com a transmissão da esquistossomose em Pernambuco, Brasil. *Publ. Av. Centro Pesq. Aggeu Magalhães, 5*: 31-47.

BARBOSA, F. S. & FIGUEIREDO, T., 1970. Susceptibility of the snail intermediate hosts of schistoso-

miasis from northeastern Brazil to the infection with *Schistosoma mansoni*. *Rev. Inst. Med. Trop. S. Paulo, 12:* 198-206.

BERNARDINI, O. J. & MACHADO, M. M., 1981. Esquistossomose mansoni em Santa Catarina: isolamento do *Schistosoma mansoni* do primeiro foco de transmissão ativa em São Francisco do Sul. *Arq. Catarin. Med., 10:* 213.

COELHO, M. V. & BARBOSA, F. S., 1956. Qualidades de vetor dos hospedeiros de *Schistosoma mansoni* no nordeste do Brasil. III. Duração da infestação e eliminação de cercárias em *Tropicorbis centimetralis*. *Publ. Av. Centro Pesq. Aggeu Magalhães, 5:* 21-30.

FRANDSEN, F., 1979a. Studies of the relationships between *Schistosoma* and their intermediate hosts. 1. The genus *Bulinus* and *Schistosoma haematobium* from Egypt. *J. Helminthol., 53:* 15-29.

FRANDSEN, F., 1979b. Discussion of the relationships between *Schistosoma* and their intermediate hosts, assessment of the degree of host-parasite compatibility and evaluation of schistosome taxonomy. *Z. Parasitenk., 58:* 275-296.

LUTZ, A., 1919. *Schistosomum mansoni* and schistosomatosis observed in Brazil. *Mem. Inst. Oswaldo Cruz, 11:* 109-140.

MACHADO, P. A., 1982. The Brazilian program for schistosomiasis control, 1975-1979. *Am. J. Trop. Med. Hyg., 31:* 76-86.

OLAZARRI, J., 1984. *Biomphalaria straminea* (Dunker, 1848) (Mollusca: Gastropoda) en la cuenca del río Uruguay. *Hist. Nat.*, Corrientes, *4:* 285-288.

PARAENSE, W. L., 1965. The Brazilian species of *Drepanotrema*. VIII. *D. heloicum* (Orbigny, 1835). *Rev. Brasil. Biol., 25:* 25-34.

PARAENSE, W. L. & CORRÊA, L. R., 1963. Susceptibility of *Australorbis tenagophilus* to infection with *Schistosoma mansoni*. *Rev. Inst. Med. Trop. S. Paulo, 5:* 23-29.

PARAENSE, W. L. & CORRÊA, L. R., 1978. Differential susceptibility of *Biomphalaria tenagophila* populations to infection with a strain of *Schistosoma mansoni*. *J. Parasitol., 64:* 822-826.

PARAENSE, W. L. & CORRÊA, L. R., 1981. Observations on two biological races of *Schistosoma mansoni*. *Mem. Inst. Oswaldo Cruz, 76:* 287-291.

PARAENSE, W. L. & CORRÊA, L. R., 1987. Probable extension of schistosomiasis mansoni to southernmost Brazil. *Mem. Inst. Oswaldo Cruz, 82:* 577.

PELLON, B. & TEIXEIRA, I., 1950. Distribuição da esquistossomose mansônica no Brasil. (Presented before the 8th Brazilian Congress of Hygiene, Recife). Divisão de Organização Sanitária, Rio de Janeiro. 117 p. mimeogr.