Compatibility of *Schistosoma mansoni* and *Biomphalaria pfeifferi* in Northern Senegal

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(*Received 2 November 1998; revised 7 January 1999; accepted 7 January 1999*)

SUMMARY

The construction of the Diama dam on the Senegal River and the ensuing ecological changes have led to a massive outbreak of *Schistosoma mansoni* infection in Northern Senegal, associated with very high intensity of infections, due to extremely intense transmission. The vectorial capacity of *Biomphalaria pfeifferi* from Ndombo, near Richard-Toll was investigated in order to assess the role of the snail–parasite relationship in this particular epidemiological situation. The results revealed an unusually high compatibility between the Senegalese *S. mansoni* strain and its local snail intermediate host, *B. pfeifferi*. The snail infection rate after exposure to a single miracidium per snail was 87%. The cercarial production of infected snails was very high, with a mean total production of 50456 cercariae per snail. No significant difference was found in the total cercarial output between snails exposed to 1 miracidium and those exposed to 5 miracidia. The increase in the rate of cercarial output was significantly greater in snails exposed to 5 miracidia, but there was a higher mortality in this group. The chronobiological cercarial production pattern showed a peak around mid-day. The implications of these findings on the epidemiology of schistosomiasis in Northern Senegal are discussed.

Key words: *Schistosoma mansoni*, *Biomphalaria pfeifferi*, Senegal, chronobiology, snail–parasite compatibility.

INTRODUCTION

Schistosoma mansoni was introduced in Northern Senegal around 1988 as a result of man-made ecological changes in the Senegal River Basin (Talla *et al*. 1990; Talla, Kongs & Verlé, 1992; Stelma *et al*. 1993; Gryseels *et al*. 1994). This unexpected outbreak of *S. mansoni* was associated with very high intensities of infections: in one survey, the prevalence was 100% in people above the age of 5 years, 41% of infected people excreted over 1000 eggs per gram (epg) of faeces, the mean egg count was 646 epg, and individual egg counts were as high as 24160 epg (Stelma *et al*. 1993).

Several hypotheses have been put forward to explain this unusual epidemiological situation in which transmission has been established very rapidly and intensely.

Since the construction of the Diama dam there have been major ecological changes in the Senegal River, particularly in relation to the reduction in salinity, and change of pH from an acid to an alkaline environment (Southgate, 1997). These changes appear to have provided a new and suitable habitat for *B. pfeifferi*, and may also be beneficial to the transmission of the larval schistosome stages, i.e. miracidia to snail and cercariae to definitive host (Donnelly, Appleton & Schutte, 1984; Christensen, Frandsen & Nansen, 1979).

The development of agriculture has resulted in an influx of people to the area, some of whom were apparently infected with *S. mansoni*, leading to the establishment of local transmission. The population still relies largely on natural water bodies for everyday living: water contact is frequent and intense (Gryseels *et al*. 1994).

Although *S. haematobium* has been established in parts of Senegal River, the human population of Richard-Toll was largely free of any schistosome infections until recently and had thus not developed any acquired immune resistance.

Such extremely high prevalences and intensities of infection, established in such a short period indicate intense transmission. A highly compatible intermediate host–parasite relationship was suspected as a possible factor contributing to this situation. Malacological field studies revealed high infection

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https://doi.org/10.1017/S0031182099004345 Published online by Cambridge University Press

rates (overall mean of 44 %) in *B. pfeifferi* collected from different sites in the environs of Richard-Toll (Diaw *et al*. 1991). The explosive development and spread of *B. pfeifferi* populations, not yet exposed to selective pressure of *S. mansoni* infection, led to the intriguing hypothesis on a 'naive' host–parasite relationship. Therefore, experiments were conducted to assess the vectorial capacity of the Senegalese snail population and the local *S. mansoni*. The specific aim of the present study was to evaluate the levels of snail–parasite compatibility, including cercarial production, and to describe the cercarial emergence rhythms (chronobiology).

MATERIALS AND METHODS

Schistosome and snail host

S. mansoni was isolated from naturally infected *B. pfeifferi* collected in Nidangue, near Richard-Toll, Northern Senegal, in January 1996. A total of 116 *B. pfeifferi* were collected and transported to the laboratory of the Biomedical Parasitology Division, The Natural History Museum, London. Of the 90 survivors, 43 were positive for *S. mansoni*. An isolate of *S. mansoni* was established in the laboratory from these 43 infected snails, and maintained in mice and *B. pfeifferi*.

Snails were bred from the wild-caught *B. pfeifferi* in the laboratory for experimental infections.

Snail infection experiments

In order to study the vectorial capacity of the Senegalese *B. pfeifferi*, 2 groups of 100 laboratory-bred snails, with a shell diameter of 5–7 mm and aged approximately 4 weeks, were selected. These were designated as group MD1 and group MD5 (MD = miracidial dose). They were exposed individually to 1 miracidium or 5 miracidia of *S. mansoni*, respectively, in a well of Disposo-Tray™ containing 1 ml of snail conditioned water. They were left overnight before being transferred to polypropylene trays and fed *ad libitum* with lettuce. The water temperature was maintained at 26 °C for the duration of the experiment, and the snails were regularly examined for cercarial shedding from day 20 onwards.

Snail size was measured before exposure to the parasite, at 4 weeks post-exposure, at 12 weeks post-exposure and at snail death, using a calliper ruler. For each snail, the largest diameter, the smallest diameter and the width of the shell were measured.

Cercarial production

Twenty-eight days after exposure, 20 snails were randomly selected from each of the 2 groups of infected snails. These 40 snails were then separated and maintained individually in small plastic pots containing 25 ml of snail-conditioned water until they died. The water was changed daily, and the snails were fed daily with dry lettuce. The number of cercariae produced per individual snail was counted daily from day 30 post-infection (p.i.) to day 57 post-infection. After this period, counting of cercariae was performed on a weekly basis until the death of the snail. For each individual snail, the number of cercariae contained in three 1 ml aliquots (stained with Lugol's iodine solution) of the thoroughly mixed parasite suspension was counted, and the cercarial production was calculated. Water continued to be changed for each snail on a daily basis. For the total cercariae produced by each snail, the daily count (performed once per week) from day 64 onwards was multiplied by 7 to produce a weekly estimate of cercarial production.

Chronobiology of cercarial emergence

The rhythm of emergence of *S. mansoni* cercariae was studied according to the methods described by Théron (1982): water kept at a constant temperature (26 °C), balanced photoperiod (light/dark: 12 h/12 h) and photophase from 0600 h to 1800 h (2000 lux), with light intensity gradually increasing at the beginning and decreasing at the end of the photophase. Quantitative determination of cercarial emergence was ascertained by using a cercariometric apparatus, allowing the automatic hourly deposition of emitted cercariae into collecting vessels. The contents of the vessels were concentrated by filtration through a polyamide filter (25 μm pore size), and stained with Lugol's iodine solution. Cercariae trapped on the filter were counted under a stereoscopic microscope. The emergence rhythms were studied for 12 snails, on 2 consecutive days. The chronobiological data were transformed into circular variables (Chassé & Théron, 1988), after which the mean vector was calculated.

Ageing and cercarial viability

To test the relationship between ageing and the viability of *S. mansoni* cercariae, 6 mice were individually exposed to 200 cercariae each for 30 min by the paddling technique immediately after shedding from the snail, and 6 mice were individually exposed to 200 cercariae each 24 h after shedding. The cercarial suspension was kept undisturbed in a beaker in the snail room at 26 °C. All the mice were killed 6 weeks post-infection, and the worms were recovered by perfusion.

Statistical analysis

Cercarial production was analysed for individual snails using linear regression on log-transformed

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Table 1. Cercarial production and longevity of *Biomphalaria pfeifferi* from Ndiangue, Northern Senegal, infected with sympatric *Schistosoma mansoni*

No. of miracidia/snail	No. of snails/group	Mean total cercariae/ infected snail (Minimum–maximum)	Mean daily cercariae/ infected snail* (Maximum)	Mean longevity of infected snails† (Minimum–maximum)
1	20	51692 (18703–73446)	670 (2675)	155 (57–239)
5	20	49121 (18713–65079)	701 (3033)	115 (57–237)

* Means of shedding during the first month only (i.e. between days 30 and 57 post-infection).
† Number of days post-infection.

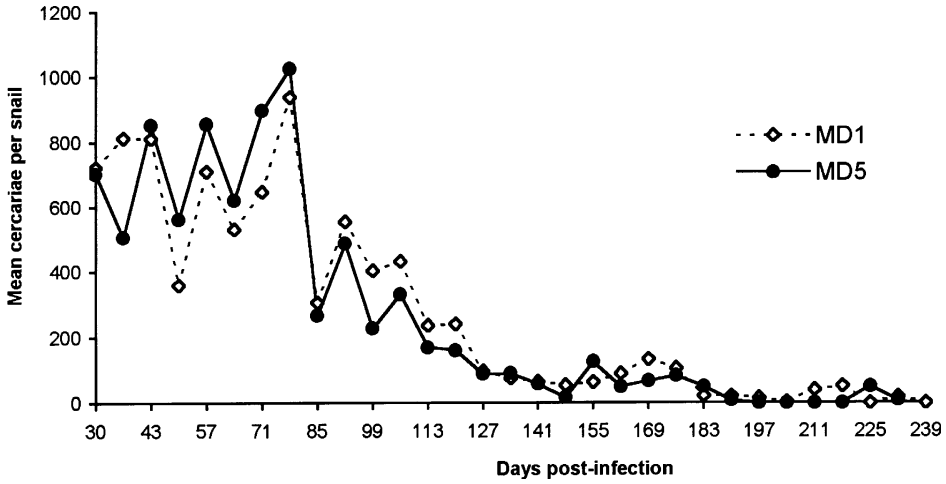

Fig. 1. Mean daily number of cercariae of *Schistosoma mansoni* from Ndiangue produced per individual *Biomphalaria pfeifferi* Richard-Toll exposed either to 1 miracidium (MD1) or 5 miracidia (MD5). The starting number of snails was 20 individuals per group.

values, and tests for specific effects were obtained as *t*-tests or Mann–Whitney *U*-test. Results were considered statistically significant at $P < 0{\cdot}05$. The 28-day production (i.e. from days 30 to 57) was analysed to assess the difference between the two groups MD1 and MD5. The rate of change of cercarial production (i.e. the slope of a regression of log of cercarial production on day for the period 30–57 days) was analysed allowing for initial size and 28-day production. The mortality rate of snails (log transformed) was regressed allowing for 28-day production and initial size index to determine the differences between the two groups. The survival times of the two exposure groups were plotted as life tables.

RESULTS

Snail–parasite compatibility

The results of the snail infection experiments revealed very high infection rates: 87% of the *B. pfeifferi* exposed to a single miracidium were infected, and all the surviving snails (100%) exposed to 5 miracidia were positive. The minimum prepatent period was 22 days. At day 28 post-infection, only 1 (from the MD5 group) of the 200 exposed snails had died, hence this particular intermediate host–parasite relationship had a very high survival rate.

Cercarial production

The results of the cercarial production counts are summarized in Table 1. The total production of cercariae per infected snail during its life-span ranged from 18703 to 73446 cercariae, with a mean of 51692 cercariae for snails exposed to a single miracidium, and 49121 cercariae for those exposed to 5 miracidia ($U = 236$, $P > 0{\cdot}05$).

The pattern of daily cercarial production per snail is shown in Fig. 1. The mean daily cercarial production per snail was high between day 30 and day 80 post-infection, reaching a peak at day 78. Thereafter, the cercarial production decreased gradually until the death of the snail. As for the total production of cercariae per snail, there was no significant difference in the mean daily production of cercariae per snail between the groups exposed to 1

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Table 2. Frequency distribution of daily levels of *Schistosoma mansoni* cercarial production per individual *Biomphalaria pfeifferi* from Ndiangue, Northern Senegal

Levels of cercariae output	Group MD1*		Group MD5		MD1+MD5	
	N	Frequency (%)	*N*	Frequency (%)	*N*	Frequency (%)
< 100	114	13·6	44	6·1	158	10·1
100–199	82	9·8	52	7·2	134	8·6
200–299	74	8·8	64	8·8	138	8·8
300–399	76	9·0	68	9·4	144	9·2
400–499	80	9·5	69	9·5	149	9·3
500–999	302	40·0	294	40·6	596	38·1
1000–1499	92	11·0	109	15·0	201	12·9
1500–1999	12	1·4	18	2·5	30	1·9
2000–2499	7	0·8	5	0·7	12	0·8
⩾ 2500†	0	0	1	0·1	1	0
Total no. of counts made	839	100	724	100	1563	100
Median of cercariae output/snail	483		583		533	

* Group MD1 = group of snails exposed individually to 1 miracidium; Group MD5 = group of snails exposed individually to 5 miracidia.
† The highest daily production obtained was 3033 cercariae.
N = number of counts made.

Table 3. Mean sizes of *Biomphalaria pfeifferi* from Ndiangue, Northern Senegal, infected individually with 1 miracidium (group MD1) or 5 miracidia (group MD5) of sympatric *Schistosoma mansoni*, at different times pre- and post-infection

	Group MD1			Group MD5		
	LD*	SD	W	LD	SD	W
Before the infection	6·46 ± 0·50	5·34 ± 0·35	2·91 ± 0·12	6·08 ± 0·61	5·28 ± 0·52	2·72 ± 0·18
At 4 weeks post-infection	7·22 ± 0·64	5·92 ± 0·53	3·04 ± 0·19	7·34 ± 0·70	6·31 ± 0·74	3·19 ± 0·20
At 12 weeks post-infection	8·50 ± 0·60	7·18 ± 0·49	3·69 ± 0·34	8·26 ± 0·67	7·08 ± 0·61	3·39 ± 0·25
At snail death	8·85 ± 0·66	7·44 ± 0·65	3·75 ± 0·27	8·45 ± 0·74	7·26 ± 0·74	3·44 ± 0·24

* LD = large diameter; SD = small diameter; W = width of snail shell. All measurements are in mm.

miracidium and 5 miracidia. Nevertheless, the average number of cercariae produced between day 30 and day 57 was higher in group MD5 than in group MD1 ($t = 1·04$, D.F. = 37, $P > 0·05$). Interestingly, the increase in the rate of cercarial productivity was significantly higher in group MD5 than in group MD1 between day 30 and day 57 ($t = 3·08$, D.F. = 37, $P < 0·001$).

The maximum production of cercariae by 1 snail for 1 day was 3033 cercariae, observed in a snail from group MD5 at day 39 post-infection. A frequency distribution of daily production of cercariae was evaluated and the results are summarized in Table 2. From the total of 1563 individual daily counts made, 54 % exceeded 500 cercariae shed per snail, and 15 % exceeded 1000 cercariae. The median was 533 cercariae per snail each day.

Snail growth

The results of snail measurements are summarized in Table 3. Before the infection, the mean large diameter of snails per group was 6·46 mm and 6·08 mm for groups MD1 and MD5, respectively ($U = 118·5$, $P < 0·05$); it was 7·22 mm and 7·34 mm at 4 weeks post-infection ($U = 169·5$, $P > 0·05$), 8·50 mm and 8·26 mm at 12 weeks post-infection ($U = 159·5$, $P > 0·05$), and 8·85 mm and 8·45 mm at snail death ($U = 132$, $P > 0·05$), for groups MD1 and MD5, respectively.

Snail mortality

Figure 2 shows the mortality rates of infected *B. pfeifferi* in the 2 groups of snails selected and

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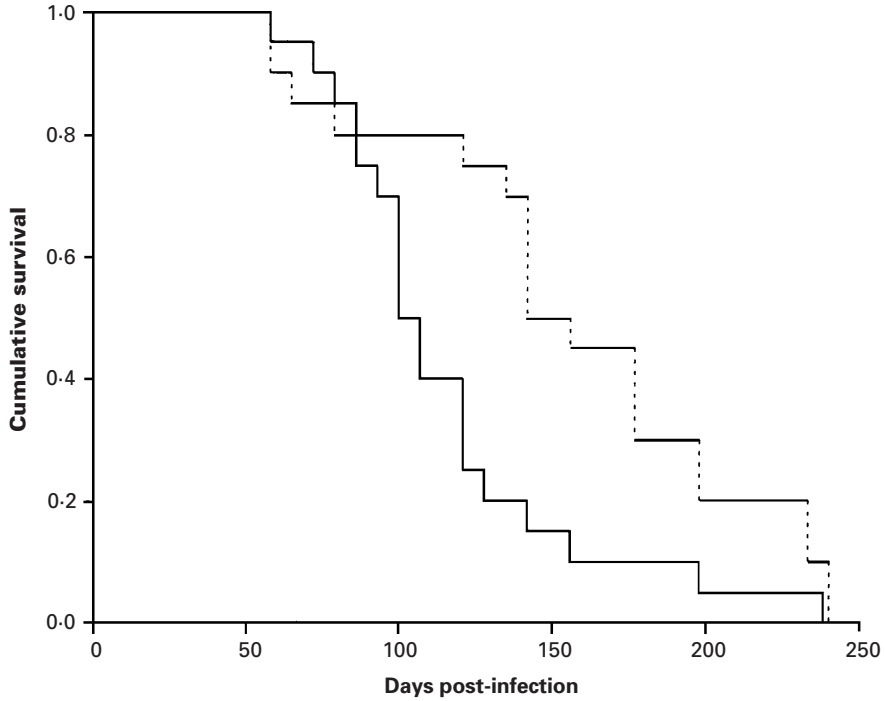

Fig. 2. Comparative survival curves of *Biomphalaria pfeifferi* from Ndiangue exposed either to 1 miracidium (- - -) or 5 miracidia) ——- of sympatric strain of *Schistosoma mansoni*. The starting number of snails was 20 individuals per group.

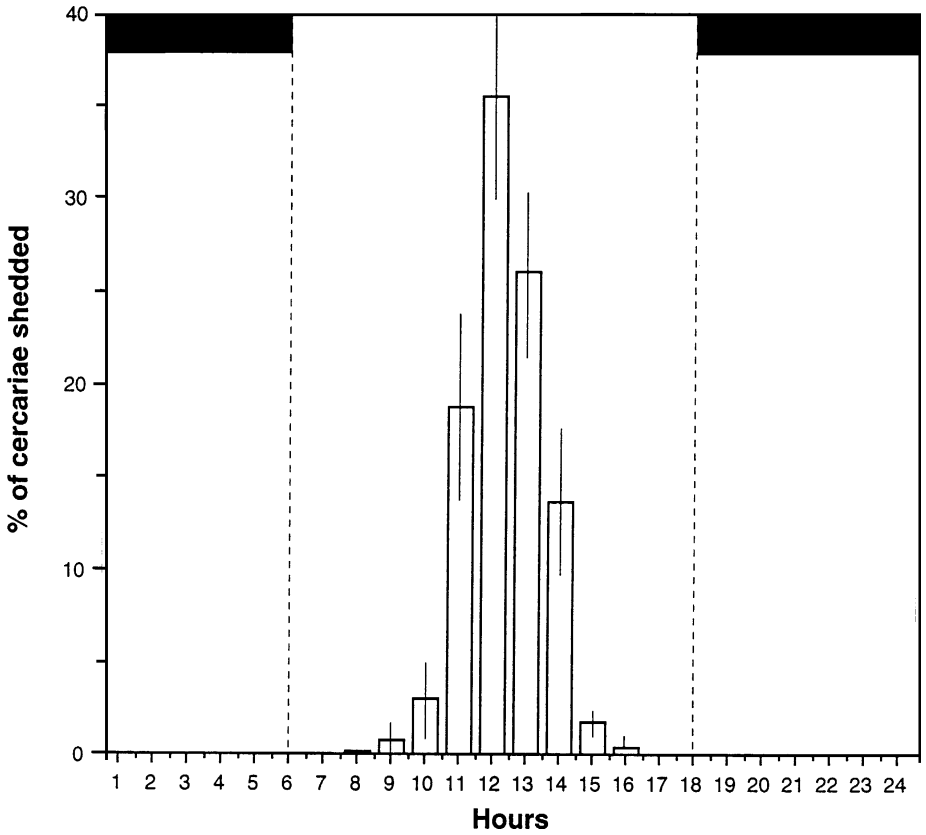

Fig. 3. Histogram of the daily emergence pattern of cercariae of *Schistosoma mansoni* from Ndiangue, Northern Senegal. Vertical bars represent S.D.

followed up. No mortality was observed up to day 57 p.i., after which mortality evolved gradually. The maximum life-span of an individual infected snail was 239 days p.i. for group MD1, and 237 days p.i. for group MD5. The mean longevity of snails was significantly higher in group MD1 than in group

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MD5 ($t = 2{\cdot}05$, D.F. = 37, $P < 0{\cdot}05$) (Table 1). Those snails whose cercarial output increased fastest died sooner.

Chronobiology of cercarial emergence

The results of the chronobiological cercarial emergence pattern are represented in Fig. 3, showing a circadian type. There was only 1 shedding peak at 12 h 21 min, with an angular deviation of 1 h 07 min, representing 36% of the daily production of cercariae.

Ageing and cercarial infectivity

The overall worm return from mice (6 mice per group) individually exposed to 200 cercariae of *S. mansoni* immediately and 24 h after shedding from laboratory-bred *B. pfeifferi* was 261 (21·8%) and 90 (7·5%), respectively ($U = 0{\cdot}0$, $P < 0{\cdot}001$). Thus, approximately one third of the cercarial population remain viable for 24 h, but interestingly the sex ratio changes from 1·58:1 (160M/101F) to 0·38:1 (25M/65F) after 24 h. Female cercariae significantly retain viability for a longer period than male cercariae ($U = 2{\cdot}5$, $P < 0{\cdot}01$).

DISCUSSION

The results of this study demonstrated a highly compatible intermediate host–parasite relationship between *S. mansoni* and *B. pfeifferi* from Ndiangue, near Richard-Toll. The snails were very susceptible to the parasite strain: the high infection rate (i.e. 87%) obtained with a single miracidial dose per snail is exceptional. Infection rates reported elsewhere for various snail populations exposed to 1 miracidium per snail are generally lower than 50% (Frandsen, 1977; Imbert-Establet & Combes, 1986; Liberatos, 1987; Lwambo *et al*. 1987; Ward *et al*. 1988; Tchuem Tchuenté & Jourdane, 1993; Moukrim, Zekhnini & Rondelaud, 1996). There are some cases of infection levels slightly greater than 50%, for example, Frandsen (1977) and Tchuem Tchuenté & Jourdane (1993) recorded infection rates of 54·3% and 53·2% between *S. intercalatum* and *Bulinus forskalii*, respectively; and Imbert-Establet & Combes (1986) reported 52% between *S. mansoni* and *B. pfeifferi*. The snail infection rate varies widely according to schistosome species and strains, and is, to some extent, related to the snail host–parasite compatibility.

Also, the cercarial production by the Senegalese *B. pfeifferi* was very high compared to existing data with different human schistosome species and strains. The mean total production of cercariae (i.e. 50406) of the Senegalese *S. mansoni* strain per individual *B. pfeifferi* in this study was approximately 2·5 times the maximum number of cercariae (20251) obtained by Frandsen (1979) in a *S. mansoni*/*B. glabrata* combination. Average values for total number of cercariae produced in the life-span of infected snails have been summarized by Loker (1983); the values range from 232 cercariae for *S. japonicum* to 20251 cercariae for *S. mansoni*. However, it should be noted that cercarial production may vary considerably among the human schistosomes. Cases of mean cercarial output levels as high as 2000–4000 cercariae per day per *B. glabrata* have been reported (Ward *et al*. 1988); and, exceptionally, some individual daily counts may exceed 16000 cercariae (Sturrock & Sturrock, 1970; Hairston, 1973).

Our study revealed no significant difference in the total cercarial output between the group of snails exposed to 1 miracidium and that exposed to 5 miracidia. This result is similar to that obtained by Moukrim *et al*. (1996) with *Planorbarius metidjensis* exposed to different miracidial doses of *S. haematobium*, and by Touassem & Théron (1989) with *B. glabrata* exposed to 1 or 10 miracidia of *S. rodhaini*. However, it differs from the results obtained by Théron, Pagès & Rognon (1997) with *B. glabrata*/*S. mansoni* and by Mouahid & Combes (1987) with *Bulinus truncatus*/*S. bovis*. Surprisingly, the results vary considerably, and cercarial production may be either higher for plurimiracidial infections compared to monomiracidial (Théron, 1985; Théron *et al*. 1997) or inversely smaller for plurimiracidial infections (Mouahid & Combes, 1987).

It should be highlighted that though the difference between the two groups MD1 and MD5 in the daily cercarial production was not significant, the mean daily number of cercariae shed per snail was greater in *B. pfeifferi* exposed to 5 miracidia than in those exposed to 1 miracidium. Careful analysis of the data demonstrated that the increase in the rate of cercarial output was significantly greater in snails of group MD5 than in those of group MD1. Interestingly, there was a correlation between the level of cercarial production and the mortality of snails; those snails whose cercarial output increased fastest died sooner. Hence, the balance between the 2 groups of snails in terms of total cercarial production was due to the difference in snail mortality, the mean longevity for snails of group MD1 being greater than that for snails of group MD5.

The analysis of the frequency distribution of cercarial counts confirms the heavy shedding of cercariae by the Senegalese *B. pfeifferi*, with more than 81% of the daily counts over 200 cercariae per snail, and more than 15% over 1000 cercariae. On the contrary, studies by Frandsen (1979) on various *S. mansoni*/*Biomphalaria* spp. showed that only 20–30% of the daily counts were greater than 200 cercariae, and only 1% of counts over 1000 cercariae per day per snail.

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All these data of the snail susceptibility and cercarial production demonstrated an extremely high compatibility between the Northern Senegal *S. mansoni* strain and its local snail host, *B. pfeifferi*. These observations confirm the fact that the number of cercariae shed, which is shown to vary considerably with snail species and snail populations, is an indication of the compatibility between the snail and the schistosome (Frandsen, 1979; Ward *et al*. 1988; Théron *et al*. 1997). Studies by Ward *et al*. (1988) showed dramatic differences in cercarial output per snail between 2 populations of *B. glabrata* differing slightly in their susceptibility to *S. mansoni*. When exposed to 5 or more miracidia, the more susceptible snail group (with 90–100 % infection rate) produced nearly twice the number of cercariae as those from the lower susceptible snail group (with 70–80 % infection rate). Théron *et al*. (1997) confirmed that levels of cercarial production are parasite strain dependent, are determined during the early development of primary sporocysts, and are correlated with the number of developed primary sporocysts.

The data from the chronobiology experiments demonstrated clearly that there is only 1 peak of cercarial emission, around mid-day. This circadian rhythm is a typical 'human' pattern. Cercarial emergence rhythms are considered as adaptive behaviours of the parasite (Combes *et al*. 1994), which are genetically controlled (Théron, 1989; Théron & Combes, 1988), shaped under the selective pressures exerted by the behaviour of the definitive hosts. This indicates that the parasite was introduced in Northern Senegal by humans, and not from migrant or resident rodents. The cercarial emergence pattern obtained indicates that in Senegal the concentration of cercariae will be at a maximum in natural water bodies in the afternoon. The question arose as to whether the local community would be equally at risk of infection with water contact activities early in the morning compared with those in the afternoon. In an attempt to answer this question mice were exposed to cercariae 0 h and 24 h, at 25 °C, after shedding from *B. pfeifferi*. In the 24 h batch there was a loss of infectivity by about two thirds. Contrary to the usual situation, with newly shed cercariae, the sex ratio of worms resulting from 24-h-old cercariae was highly biased in favour of female worms (i.e. 2·6 females : 1 male).

This result shows that in these transmission foci in Northern Senegal cercariae are present in the natural water bodies throughout day and night, and human infection may thus occur whenever water contact arises. The risk may certainly be greater in the (late) afternoon, when highest cercarial densities are obtained. Sex ratios of adult schistosomes are usually biased towards males; reports on several species have shown that in natural and experimental infections adult males usually outnumber females by 1·5 : 1 to 5 : 1 (Liberatos, 1987). Liberatos (1987) reported that the adult male bias sex ratio of *S. mansoni* is caused by greater male infectivity, and survival, of miracidia for snails and cercariae for mammals. Our result obtained with ageing cercariae suggests a greater longevity of female cercariae compared to male cercariae. The resulting adult female biased sex ratio when cercariae become older is of interest in the context of parasite dynamics as this would, to some extent, mitigate the surplus of male worms in definitive hosts; and would contribute to mating dynamics (Tchuem Tchuenté *et al*. 1995, 1996).

All results obtained from this study are highly relevant to the Northern Senegal situation, and may provide an explanation of the epidemiological peculiarities observed in human populations. The very high prevalences and intensities of *S. mansoni* infections in the community probably result from a combination of several factors: (1) presence of heavily infected *B. pfeifferi* snails in water bodies throughout much of the year, with very high levels of natural infections due to a highly compatible snail–parasite relationship; (2) extremely high cercarial production by these *B. pfeifferi* populations; (3) circadian rhythm of cercarial emergence with a concentration period of high shedding around 12.00 h; (4) viability of cercariae over 24 h, leading to a risk of infection at any time of day; and (5) intense and frequent water contacts.

Apart from the levels of infection, these observations on massive and continuous exposure may also be of relevance to the development of acquired (immune) resistance and of morbidity in this population (Gryseels, 1994; Stelma *et al*. 1994).

The present study raises the question whether this highly compatible intermediate host–parasite relationship is due to the snail, to the parasite or to both. Further studies are in progress attempting to answer this question, using *S. mansoni* and *B. pfeifferi* from Senegal and other foci to compare the vectorial capacity of *B. pfeifferi*/*S. mansoni* in various sympatric and allopatric combinations.

The authors are most grateful to the European Commission INCO-DC (IC18.CT960112) for their financial support; to M. Anderson and V. Tuffney for their technical assistance in London; to Dr D. De Clerq, Dr M. Diop, Dr M. Sène for help in the field; to C. B. Moncrieff for help with the statistical analysis of the data; and to Dr Dirk Engels for his comments on the manuscript.

REFERENCES

CHASSÉ, J. L. & THÉRON, A. (1988). An example of circular statistics in chronobiological studies: Analysis of polymorphism in the emergence rhythms of *Schistosoma mansoni* cercariae. *Chronobiology International* **5**, 433–439.

CHRISTENSEN, N. O., FRANDSEN, F. & NANSEN, P. (1979). The effects of some environmental conditions and final-host and parasite-related factors on the penetration of *Schistosoma mansoni* cercariae into mice. *Zeitschrift für Parasitenkunde* **59**, 267–275.

COMBES, C., FOURNIER, A., MONÉ, H. & THÉRON, A. (1994). Behaviours in trematode cercariae that enhance parasite transmission: patterns and processes. *Parasitology* **109**, S3–S13.

DIAW, O. T., VASSILIADES, G., SEYE, M. & SARR, Y. (1991). Epidémiologie de la bilharziose intestinale à *Schistosoma mansoni* à Richard-Toll (Delta du Fleuve Sénégal): étude malacologique. *Bulletin de la Société de Pathologie Exotique* **84**, 174–183.

DONNELLY, F. A., APPLETON, C. G. & SCHUTTE, C. H. J. (1984). The influence of salinity on the ova and miracidia of three species of *Schistosoma*. *International Journal for Parasitology* **13**, 539–545.

FRANDSEN, F. (1977). Investigations of the unimiracidial infection of *Schistosoma intercalatum* in snails and the infection of the final host using cercariae of one sex. *Journal of Helminthology* **51**, 5–10.

FRANDSEN, F. (1979). Studies of the relationship between *Schistosoma* and their intermediate hosts. III. The genus *Biomphalaria* and *Schistosoma mansoni* from Egypt, Kenya, Sudan, Uganda, West Indies (St Lucia) and Zaire (two different strains: Katanga and Kinshasa). *Journal of Helminthology* **53**, 321–348.

GRYSEELS, B. (1994). Human resistance to *Schistosoma* infections: age or experience. *Parasitology Today* **10**, 380–384.

GRYSEELS, B., STELMA, F. F., TALLA, I., VAN DAM, G. J., POLMAN, K., SOW, S., DIAW, M., STURROCK, R. F., DOERING-SCHWERDTFEGER, E., KARDORFF, R., DECAM, C., NIANG, M. & DEELDER, A. M. (1994). Epidemiology, immunology and chemotherapy of *Schistosoma mansoni* infections in a recently exposed community in Senegal. *Tropical and Geographical Medicine* **46**, 209–219.

HAIRSTON, N. G. (1973). The dynamics of transmission. In *The Epidemiology and Control of Schistosomiasis* (*Bilharziasis*) (ed. Ansari N.), pp. 250–353. S. Karger, Basel.

IMBERT-ESTABLET, D. & COMBES, C. (1986). *Schistosoma mansoni*: comparison of a Caribbean and African strain and experimental crossing based on compatibility with intermediate hosts and *Rattus rattus*. *Experimental Parasitology* **61**, 210–218.

LIBERATOS, J. D. (1987). *Schistosoma mansoni*: male-biased sex ratios in snails and mice. *Experimental Parasitology* **64**, 165–177.

LOKER, E. S. (1983). A comparative study of the life-histories of mammalian schistosomes. *Parasitology* **87**, 343–369.

LWAMBO, N. J. S., UPATHAM, E. S., KRUATRACHUE, M. & VIYANANT, V. (1987). The host-parasite relationship between the Saudi Arabian *Schistosoma mansoni* and its intermediate and definitive hosts. 1. *S. mansoni* and its local snail host *Biomphalaria arabica*. *Southeast Asian Journal of Tropical Medicine and Public Health* **18**, 156–165.

MOUAHID, A. & COMBES, C. (1987). Genetic variability of *Schistosoma bovis* cercarial production according to miracidial dose. *Journal of Helminthology* **61**, 89–94.

MOUKRIM, A., ZEKHNINI, A. & RONDELAUD, D. (1996). *Schistosoma haematobium*: influence of the number of miracidia on several characteristics of infection in newborn *Planorbarius metidjensis*. *Parasitology Research* **82**, 267–269.

SOUTHGATE, V. R. (1997). Schistosomiasis in the Senegal River Basin: before and after the construction of the dams at Diama, Senegal and Manantali, Mali and future prospects. *Journal of Helminthology* **71**, 125–132.

STELMA, F. F., TALLA, I., POLMAN, K., NIANG, M., STURROCK, R. F., DEELDER, A. M. & GRYSEELS, B. (1993). epidemiology of *Schistosoma mansoni* infections in a recently exposed community in northern Senegal. *American Journal of Tropical Medicine and Hygiene* **49**, 701–706.

STELMA, F. F., TALLA, I., VERLÉ, P., NIANG, M. & GRYSEELS, B. (1994). Morbidity due to heavy *Schistosoma mansoni* infections in a recently established focus in northern Senegal. *American Journal of Tropical Medicine and Hygiene* **50**, 575–579.

STURROCK, B. M. & STURROCK, R. F. (1970). Laboratory studies of the host–parasite relationship of *Schistosoma mansoni* and *Biomphalaria glabrata* from St Lucia, West Indies. *Annals of Tropical Medicine and Parasitology* **64**, 357–363.

TALLA, I., KONGS, A. & VERLÉ, P. (1992). Preliminary study of the prevalence of human schistosomiasis in Richard-Toll (the Senegal river basin). *Transactions of the Royal Society of Tropical Medicine and Hygiene* **86**, 182.

TALLA, I., KONGS, A., VERLÉ, P., BELOT, J., SARR, S. & COLL, A. M. (1990). Outbreak of intestinal schistosomiasis in the Senegal River Basin. *Annales de la Société Belge de Médecine Tropical* **70**, 173–180.

TCHUEM TCHUENTÉ, L. A. & JOURDANE, J. (1993). Further data on the compatibility between *Schistosoma intercalatum* (lower Guinea strain) and *Bulinus forskalii*: epidemiological consequences. *Tropical Medicine and Parasitology* **44**, 221–222.

TCHUEM TCHUENTÉ, L. A., SOUTHGATE, V. R., IMBERT-ESTABLET, D. & JOURDANE, J. (1995). Change of mate and mating competition between males of *Schistosoma intercalatum* and *S. mansoni*. *Parasitology* **110**, 45–52.

TCHUEM TCHUENTÉ, L. A., SOUTHGATE, V. R., COMBES, C. & JOURDANE, J. (1996). Mating behaviour in schistosomes: are paired worms always faithful? *Parasitology Today* **12**, 231–236.

THÉRON, A. (1982). Le compartiment cercaire dans le cycle de *Schistosoma mansoni* Sambon, 1907. Ecologie de la transmission bilharzienne en Guadeloupe. Doctoral Thesis. University of Perpignan, France.

THÉRON, A. (1985). Dynamics of the cercarial production of *Schistosoma mansoni* in relation with the miracidial dose exposure to the snail host *Biomphalaria glabrata*. *Annales de Parasitologie Humaine et Comparée* **60**, 665–674.

THÉRON, A. (1989). Hybrids between *Schistosoma mansoni* and *S. rodhaini*: characterization by cercarial emergence rhythms. *Parasitology* **99**, 225–228.

THÉRON, A. & COMBES, C. (1988). Genetic analysis of cercarial emergence rhythms of *Schistosoma mansoni*. *Behavior Genetics* **18**, 201–209.

THÉRON, A., PAGÈS, J. R. & ROGNON, A. (1997). *Schistosoma*

mansoni: distribution patterns of miracidia among *Biomphalaria* snail as related to host susceptibility and sporocyst regulatory processes. *Experimental Parasitology* **85**, 1–9.

TOUASSEM, R. & THÉRON, A. (1989). *Schistosoma rodhaini*: dynamics and cercarial production for mono- and pluri-miracidial infections of *Biomphalaria glabrata*. *Journal of Helminthology* **63**, 79–83.

WARD, R. D., LEWIS, F. A., YOSHINO, T. P. & DUNN, T. S. (1988). *Schistosoma mansoni*: relationship between cercarial production levels and snail host susceptibility. *Experimental Parasitology* **66**, 78–85.

https://doi.org/10.1017/S0031182099004345 Published online by Cambridge University Press