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ECOLOGY OF THE LARVAL PARASITIC STAGES OF *SCHISTOSOMA MANSONI*

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SUMMARY

The behavior of the larval parasitic stages of *Schistosoma mansoni* in regard to their hosts is discussed in this paper.

Relationships between the larval stages of *S. mansoni* and the tissues of their intermediate hosts show a great range of variation. In highly susceptible snails the larval stages develop without any tissue reaction. On the other hand it has been shown that in partially resistant snails most of the sporocysts are destroyed by phagocytic action of the amoebocytes.

Susceptibility of the snails to the infection with *S. mansoni* varies a great deal. Differentiation of the human schistosomes into geographic strains is well known and in the last few years evidence has been produced to show that differences in susceptibility of the snail vectors of *S. mansoni* is regulated genetically. Such differences in susceptibility may occur within one same species. Small genetic differences between two Brazilian strains of *Australorbis glabratus* showing different degrees of susceptibility to the same strain of *S. mansoni* have been demonstrated.

The vector efficiency in transmitting *S. mansoni* depends on the balance between the parasite and their intermediate hosts. Several aspects of the snail-parasite relationships are analised particularly the ability of the vectors to harbour anl transmit Schistosomiasis mansoni.

The penetrating tropism observed in the miracidia of *S. mansoni* is by no means specific. Miracidia penetrate fresh-water snails belonging to different families as *Physidae* and *Ampullariidae*. Moreover miracidia of *S. mansoni* eagerly attack and penetrate the skin of tadpoles.

INTRODUCTION

Schistosomiasis mansoni is transmitted in Brazil by three planorbid species: *Australorbis glabratus* (Say), *Australorbis tenagophilus* (D'Orb.) and *Tropicorbis centimetralis* (Lutz). During the last few years the relationships between the parasitic larval stages of *Schistosoma mansoni* and their hosts have been intensively investigated in this country. In the present paper the Author gives a brief review of the literature on that subject.

VECTOR CAPABILITIES

According to BARBOSA & COELHO [13] the ability of the vectors to harbor and transmit *Schistosoma mansoni* depends on three factors: 1) susceptibility of the snails to in-

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fection; 2) density and distribution of the snail populations; and 3) host-parasite balance. Only items 1 and 3 will be discussed here.

SUSCEPTIBILITY TO THE INFECTION

It has been shown that different geographical strains of *S. mansoni* exist in regard to their ability to infect geographical strains of the vectors. Studies by FILES & CRAM [30] and FILES [29] revealed that differences in the results of cross-infection experiments may be due to variations both in the snails and in the trematode.

Studies conducted in Northeaster Brazil (BARBOSA & COELHO [13], BARBOSA, COELHO & DOBBIN [17], COELHO & BARBOSA [24]) revealed that the two known vectors of Schistosomiasis mansoni in that region, *Australorbis glabratus* and *Tropicorbis centimetralis* behave differently in regard to their capabilities to harbor and transmit the infection.

Under experimental conditions *A. glabratus* is a much more efficient vector of *S. mansoni* than *T. centimetralis*. While *A. glabratus* can be easily infected in the laboratory, *T. centimetralis* showed to be partially resistant. Although miracidia penetrate successfully in both snails reactions opposed by the hosts were substancially different. In *A. glabratus* the great majority of the sporocysts develop successfully while in *T. centimetralis* the larvae excite precocious reactions from the host. After 24 hours most of the sporocysts which had penetrated *T. centimetralis* were surrounded by slight cellular reaction. This reaction becomes stronger in the following days and 5 or 6 days after infection the degenerated sporocysts are involved in an extensive cellular reaction and the larvae are destroyed by phagocytosis.

These reactions had already being described by *Brooks* [19] in *Tropicorbis havanensis* (= *peregrinus*) and by NEWTON [34] in a Brazilian strain of *A. glabratus* which is resistant to the strain of *S. mansoni* from Puerto Rico.

These laboratory experiments on the susceptibility of the two vectors of *S. mansoni* in Northeastern Brazil were confirmed in the field. Natural infection rates have been always substantially higher in *A. glabratus* than in *T. centimetralis* (BARBOSA & COELHO [16]).

Although *A. glabratus* has been considered an efficient vector of *S. mansoni* and undoubtedly the main vector of Schistosomiasis in the West Indies and South America, FILES & CRAM [30] and FILES [29] had already demonstrated that *A. glabratus* from various American origins showed differences in susceptibility when exposed to geographical strains of *S. mansoni*. The above mentioned workers further suggested that such differences in susceptibility may be due to variations both in the vector and in the parasite.

One of the most striking differences in susceptibility of the snail vectors demonstrated by the above mentioned workers was that related to the resistance of the Brazilian strains of *A. glabratus* to most of the foreign strains of the parasite. FILES & CRAM [30] employed for their experiments two lots of *A. glabratus* from Recife, and FILES [29] used the same snail species for similar experiments using two lots from Recife and two lots from Salvador.

According to FILES & CRAM [30], *A. glabratus* from Recife was refractory to most strains of the parasite but was susceptible to a *S. mansoni* hybrid strain (Recife × Puerto Rico). These workers did not used any pure Brazilian strain of *S. mansoni*.

Similar results were obtained by FILES [29]. *A. glabratus* from Recife was refractory to one pure strain of *S. mansoni* from Salvador. *A. glabratus* from Salvador was refractory to all the *S. mansoni* strains used by FILES even to two Brazilian cross strains. However, the Salvador snail not tested against any parasite pure strain for Brazilian origin. The *S. mansoni* cross strain mentioned PARAENSE & CORRÊA [40] as Salvador × Recife and quoted from FILES [29] apparently is his Salvador cross 2 which is a cross Salvador × Recife cross. In turn, Recife cross resulted from the cross Recife × Puerto Rico.

Until that time there were no published data concerning the susceptibility of the Bahian strain of *A. glabratus* to the infection with *S. mansoni* from Brazilian origin *.

Following that BARBOSA & BARRETO [11] demonstrated that *A. glabratus* from Bahia showed very low susceptibility to the infection with *S. mansoni* when compared to a high susceptible strain from Pernambuco. Using three lots of snails from Bahia these workers obtained 1.7% of infection, while Pernambuco snails showed 83.9% of infection rate.

BARBOSA & BARRETO [11] call the attention to the problem of the physiological differences between populations of one same species in relation to their ability to transmit a disease. In the particular case of *A. glabratus* they have demonstrated that two populations of this snail, living not very far from each other, behave differently in regard to their capacity to harbor and transmit the Brazilian strain of *S. mansoni*.

BARRETO [18] confirmed the low susceptibility of the Bahian snails in respect to the infection with a local strain of *S. mansoni*. The infection rate obtained in his experiments was 0.92%. Examining 2,217,638 snails from Salvador, Bahia, during the period of 1952 to 1959, BARRETO [18] verified that natural infection rates were always very low, ranging monthly from 0.45 to 1.39%.

Confirming and extending these studies on the variation of susceptibility of strains of *A. glabratus* to the infection with the Brazilian strain of *S. mansoni* PARAENSE & CORRÊA [40] presented the results of infection experiments made on 23 strains of *A. glabratus* originated from Brazil, Venezuela and West Indies. The great majority of the studied snail populations were highly susceptible to the strain of *S. mansoni* from Belo Horizonte, Brazil. Negative rates were observed in three populations from Salvador, Bahia, which were exposed to 10 miracidia per snail. However 19.8% infection rate was observed in one of these Bahian snail populations when exposed to 1,000 miracidia per snail.

NEWTON [35] demonstrated that susceptibility to infection with *S. mansoni* in *A. glabratus* is regulated genetically.

Studying the comparative susceptibility to *S. mansoni* of two Brazilian strains of *A. glabratus*, BARBOSA & BARRETO [11] admitted that genetic diversity presented by these two strains may have led to differences in susceptibility. All the 18 crosses made between members of the two strains of *A. glabratus* revelead a reduced fertility. Only 54.5% of the eggs laid were viable. Furthermore, out of the 18 pairings, five albino snails failed to show a complete substitution of their autofertilization process when in presence of sperm from his conspecific partner.

BARRETO [18] however, was not able to confirm the above results. In his crossing experiments between Bahia and Pernambuco snails (*A. glabratus*) 91.4% of the eggs laid by the albino snails were viable.

PARAENSE & CORRÊA [40] observed no relationship between the degree of susceptibility of *A. glabratus* to *S. mansoni* and the distance separating the snail populations. They admit that differences in susceptibility observed were related to the genotypes of the snail populations.

Australorbis tenagophilus from Southern Brazil has been considered as a poor vector of Schistosomiasis *mansoni*. Cellular reactions which follow laboratory infection of this species with *S. mansoni* were studied by COELHO [22, 23]. Review of the literature recently presented by PARAENSE & CORRÊA [41] showed that natural infection rates of *A. tenagophilus* recorded by several workers, were usually low. However, in few instances this snail showed high natural infection rates.

COELHO [23], studying the susceptibility of several strains *A. tenagophilus* from Brazilian Southern States, concluded for the great resistance of this snail to *S. mansoni* infection. Discussing his results, COELHO [23] pre-

* However, in a document presented to the first meeting of the W.H.O. Expert Committee on Bilharziasis which was held in San Juan, Puerto Rico, Wright presented data showing that *A. glabratus* from Bahia was completely refractory to the infection with a strain of *S. mansoni* from Pernambuco, Brazil. W. H. Wright — The snail factor in the epidemiology of Bilharziasis. W.H.O. Working Document presented to the Expert Committee on Bilharziasis, 11 Aug. 1952 (quoted by permission of the Author). CRAM [26] refers to this document without much detail.

sents the interesting suggestion of a possible adaptation between the snail (*A. tenagophilus*) and the local strain of the parasite to explain some of the high natural infection rates which were, found by other workers in certain areas of the States of São Paulo and Rio de Janeiro.

Suggestions presented by COELHO [23] were confirmed by PARAENSE & CORRÊA [41]. These workers showed that at least in one particular case, physiological adjustment between snail and parasite exist. This was demonstrated by theses workers in laboratory infection experiments with *A. tenagophilus* from São José dos Campos, São Paulo. This snail strain which was insusceptible to infection with *S. mansoni* from Belo Horizonte (Minas Gerais) was easily infected with a sympatric strain of *S. mansoni*. On the other hand, this latter parasite strain was uninfective to *A. glabratus* from Belo Horizonte which in turn is highly susceptible to the local strain of *S. mansoni*.

THE HOST-PARASITE BALANCE

The host-parasite balance depends on the activity of the parasite and the response of the host to the invader.

Parasite-host specificity has received much attention for a long time. Specificity should be looked upon as a relative and an evolving concept. Relationships between parasite and vector is part of a complex unit involving a biological chain which can be written as parasite ⟶ vector ⟶ definitive host. In the case of the relationships between *S. mansoni* and their snail intermediate hosts, the problem can be discussed as follows.

The trematode larvae are not harmless to their snail hosts. Damage produced by *S. mansoni* in *A. glabratus* was studied by FAUST [28] and MARCUZZI [33].

Snails infected with *S. mansoni* are usually killed as a result of the infection. The life-span of these snails infected in the laboratory is short. Infected *A. glabratus* has an average life-span of 39 days. About 50 per cent of these snails died after 45 days from the beginning of the shedding of the cercariae and the last infected snail lived for 150 days (BARBOSA, COELHO & DOBBIN [17]). The life-span of infected *T. centimetralis* in the laboratory was determined by COELHO & BARBOSA [24] as 5.2 days. One snail lived for 91 days but the great majority died within the first 10 days.

Following 15 *A. glabratus* found naturally infected in a small pool, BARBOSA [4] concluded that although the life-span of these snails could not be determined, the infected snails rapidly died under natural conditions probably as a result of the infection.

Infected snails may lose their infections when kept at routine laboratory breeding conditions. Spontaneous healing of infected snails was first noticed by GORDON & co-workers [31] and further observed by LAGRANGE & co-workers [32], by STIREWALT [44] and by BARBOSA, COELHO & DOBBIN [17].

The effect of the infection on the reproduction of the snails has been studied by several Authors. The presence of sporocysts of *S. mansoni* in the hermaphrodite gland of *A. glabratus* is rather common. BRUMPT [20] observed a reduction in the number of eggs laid by infected *A. glabratus*. COELHO [21] observed that infection *A. glabratus* with *S. mansoni* was responsible for a significant reduction in the number of eggs and egg-masses laid by the infected group in comparison with the uninfected group. Restoration of the reproductive activity was observed when the snails lost their infection. True castration did not occur and cross-sections made through the ovotestis of highly infected snails revealed always some sexual cells to be functionally active. Viability of the eggs from infected parents was very low.

Mortality of the "new-born" snails was also investigated by COELHO [21]. In the group of the infected snails mortality of the young (up to 10 days) was greater than in the uninfected group.

Very little is known on the physiology of the snail hosts of the Schistosomes and even less is known on the pathology of the infection.

The single layer cell skin of the snails does seem to offer any special resistance to the penetration of the miracidia. However as soon as the miracidia get in contact with the tissues of the snails they are immobilized and lose their ciliary cover.

Larval stages of *S. mansoni* usually develop without any cellular response by the host when the infection occurs in highly susceptible snail vectors, as in the majority of *A. glabratus strains*. However when partially or totally resistant snails are penetrated by the miracidia of *S. mansoni* within 24 hours a slight cellular reaction is seen around the larvae. In partially resistant snails some sporocysts are able to complete its development while in totally resistant strains the larvae degenerate and are surrounded by extensive cellular reaction.

Recent studies made on laboratory cultures of tissues of *A. glabratus* infected with *S. mansoni* open new possibilities for the study of the host-parasite relationships.

Although BARBOSA & COELHO [16] were not able to observe any immobilizing effect of the blood of infected *A. glabratus* against the miracidia of *S. mansoni*, new immunological tecniques may prove to be useful in detecting specific antibodies in the tissues of the infected snails. Fluorescent antibody reactions have been applied with success to all the stages of the life-cycle of *S. mansoni* (SADUN & co-workers [43]).

Other experiments by BARBOSA & COELHO [13] have shown that *A. glabratus* which had lost their infection can be easily reinfected with *S. mansoni* miracidia. Snails which harbor one sex miracidia can be reinfected with the opposite sex miracidia. In the re-infected snail groups the tissue reactions were more marked than in the group infected for the first time.

As mentioned before the relationships between the schistosomes and their vectors are regulated genetically. Incompatibilities between *S. mansoni* and their vectors of different endemic areas, are due to physiologically different strains of both parasite and snail as demonstrated by several Authors. A good example of small genetic differences presented by two different populations of the same snail species, already referred to, was reported by BARBOSA & BARRETO [11]. These workers demonstrated that two Brazilian strains of *A. glabratus*, one from Bahia and another from Pernambuco, which showed striking differences in susceptibility to the same strain of *S. mansoni*, presented an incipient reproductive isolation.

It has been demonstrated that at least two climatic factors directly influence the course of infection in the snails: temperature and estivation.

The fresh-water habitats in tropical regions are likely to have less seasonal variation in temperature than those in temperate zones. Snails transmitting Schistosomiasis show a high degree of tolerance to variation of temperature. For *A. glabratus* from Pernambuco the optimum temperature lies between 25 and 28°C. They are rapidly destroyed by freezing and by temperature of 42°C during 2 hours. They have however a very broad range of favourable temperature (18 to 32°C).

The correlation between temperature and the prepatent period in snails infected with *S. mansoni* is well known (GORDON & co-workers [31], STIREWALT [44]). Low temperature prolongs the prepatent period while high temperature speeds the evolution of the larval parasitic stages.

Moreover, low temperature stimulates the healing and reduce the production of cercariae (STIREWALT [44]). Snails (*A. glabratus*) infected with *S. mansoni* and kept continuously at temperatures of 10°C or of 35°C die off more rapidly in comparison with a group of uninfected snails submitted to the same conditions. At a temperature of 10°C, 37.6% of the infected snails lost their infection (BARBOSA [2]).

As has been demonstrated by several Authors, the daily cycle in elimination of the cercariae of infected snails is influenced by light and temperature. Shedding of cercariae is however almost completely inhibited when infected *A. glabratus* is maintained at 35°C to be restored within few days after temperature dropped to 33°C (BARBOSA [2]).

Rainfall cycles may influence profoundly the fresh-water habitats. In the coastal area of Northeastern Brazil, as in many other tropical zones, rainfal cycles are the main factor in disturbing the life-cycle of the snail vectors of schistosomiasis.

In Eastern Pernambuco there is a marked rainy season which occurs from March or April to July or August. During the dry season many bodies of water dry up completely.

Field studies by OLIVIER & BARBOSA [37, 38] on the natural history of *A. glabratus* and *T. centimetralis* revealed that these snails can survive the dry season in sufficient number to repopulate the area when the water returns. A resumé of the ecology of these snails was recently presented by BARBOSA [3].

During estivation the snails are obviously submitted to adverse conditions and during this period the mortality of the snails is very high.OLIVIER, BARBOSA & COELHO [39] demonstrated that infected *A. glabratus* die in much greater number than uninfected ones when they are kept out of water. However when infected *A. glabratus* are kept out of water over a two weeks period the survining snails lose their infection (BARBOSA & COELHO [12]). Later on, evidence was presented (BARBOSA & COELHO [14]) to show that this phenomenon occurs only when the infection in the snail is mature. However, when snails harboring immature *S. mansoni* infection are taken out of water the sporocysts stop their development while the snails remain in estivation. The activity of the sporocysts is resumed when the snails return to the water. Five-day-old sporocysts are able to remain dormant for at least 150 days (BARBOSA [2]). Moreover, collections of snails regularly made during three consecutive summers on the dry soil of one endemic area of the State of Pernambuco revealed 21 snails harboring immature infections after variable periods of desiccation (BARBOSA & BARBOSA [5]).

When infected snails are kept alternately in and out of water (5 and 30 days respectively) for three periods, the sporocysts remain in dormancy while the snails are in desiccation to resume their activity when the snails are replaced in the water (BARBOSA [2]).

Apparently, the conditions created in the host tissues during the estivation period make the parasite stop its development. The phenomenon is seasonal and characterized by the temporary lack of growth and reproduction of the parasite during the period in which the snail is subjected to desiccation.

The phenomenon of diapause of the sporocysts in the estivating snails has been regarded as an adaptation of the parasite to its host in areas submitted to regular periods of estivation (BARBOSA & BARBOSA [5]).

Working in Belo Horizonte, Minas Gerais, COELHO & CHAIA [25] confirmed results obtained in Northeastern Brazil on the behavior of *S. mansoni* sporocysts in estivating *A. glabratus*. Infected snails harboring mature infections survive free of infection. Diapause was observed in snails with 6 days old infection which were kept for 25 days out of water in the laboratory. High mortality observed in estivating snails from Belo Horizonte is explained by differences in ability of the snails to resist desiccation. There is enough evidence to show (OLIVIER [36], BARBOSA [1], BARBOSA & BARBOSA [7]) that snails living in areas subject to annual drought become better adapted to resist desiccation. Although in Belo Horizonte the snail habitats are subjected to different conditions and only a very small number of snails resisted desiccation, yet the sporocysts of a local strain of *S. mansoni* were able to enter in diapause in the few snails which had escaped desiccation under laboratory conditions. This is explained by COELHO & CHAIA [25] as a pre-adaptation phenomenon which occur with *S. mansoni* when infecting snails living in environments not regularly liable to drought.

POTENTIAL INTERMEDIATE HOSTS

It is well know that the larval stages of the schistosomes have a very strict specificity regarding their intermediate hosts. Admitting, as generally agreed, that the snail vectors of *S. mansoni* belong to a single genus, the larval parasitic stages of this trematode take place in species included in one nominal genus. In the American continent the number of species known as actual vectors is very limited. In the extensive territories occupied by Schistosomiasis *mansoni* in Brazil, no more than three snail species are known to act as intermediate hosts. The same concept cannot be applied to Africa before clarification of snail taxonomy is carried out in that continent.

It seems likely that attention must be turned to the problems related to other human and animal schistosomes. Special attention should be given to aspects of transmission of *S. haematobium* in Portugal and

India where other molluks besides those usually known as vectors have been incriminated.

There is one record showing that *S. mansoni* is able to complete its larval development out of the *Biomphalaria*-group of snails. Evidence presented by BARBOSA, BARBOSA & MORAIS REGO [9] revealed that *Planorbarius metidjensis* from French Morocco can be infected with a Brazilian strain of *S. mansoni*. Examination of the sections made from the anterior part of the snails revealed that only few miracidia had been able to develop without any reaction by the host. Out of twelve snails exposed to the miracidia only one shed cercariae.

In the American continent since 1945 CRAM, JONES & WRIGHT [27] have reported that the snail *Tropicorbis havanensis* (= *obstructus*) from Lousiana is a potential host of *S. mansoni*.

It is now realized that other potential intermediate hosts exist in South America's non-endemic areas. Three species of the *Biomphalaria*-group inhabiting Chile and Ecuador may be considered potentially as intermediate hosts of *S. mansoni*.

BARBOSA & BARBOSA [6] succeeded in infecting *Tropicorbis chilensis* from Santiago, Chile, with the strain of *S. mansoni* from Recife, Brazil. Out of 44 surviving snails 3 (6.8%) became infected. The three infected snails were observed in the laboratory where they shed cercariae during 3, 28 and 28 days respectively. The maximum daily output of cercariae was 1,200 as given by one infected snail. The average daily number of cercariae shed per snail was 273.

Following that BARBOSA, BARBOSA & RODRIGUES [10] demonstrated that another species, *T. peregrinus* from Guayaquil, Ecuador, was susceptible to a strain of *S. mansoni* from Belo Horizonte, Brazil. Out of seventeen snails exposed to the miracidia, 14 were alive. From these, one shed cercariae.

Recently BARBOSA, BARBOSA & CARNEIRO [8] reported that another species from Ecuador, *Australorbis sericeus*, is susceptible to the Brazilian strain of *S. mansoni* from Recife. Out of 40 surviving snails, 14 (17%) resulted infected. Twelve of these snails were followed in the laboratory. The length of time that the infection lasted in the snails was 23.4 days (average) and the average daily number of cercariae shed per snails was 76.6. The maximum daily output of cercariae was 700.

In Puerto Rico *Tropicorbis riisei* (probably *peregrinus*) was successfuly infected with a local strain of *S. mansoni* by RICHARDS [42].

It should be finally mentioned that miracidia of *S. mansoni* are able to penetrate different species of unrelated families of fresh-water snails as well as in tadpoles (BARBOSA [2]).

RESUMO

Ecologia das fases larvais intramolusco de Schistosoma mansoni

No presente trabalho, o Autor discute o comportamento das fases larvais de *Schistosoma mansoni* vivendo em parasitismo em seus caramujos hospedeiros.

Os tecidos dos moluscos demonstram comportamento muito variável em relação à infecção por *S. mansoni*. Em caramujos muito suscetíveis os tecidos dos moluscos não apresentam reação alguma ao parasita, enquanto que nos moluscos parcialmente resistentes, a maioria dos esporocistos é destruída pela ação fagocitária dos amebócitos.

A suscetibilidade dos moluscos à infecção por *S. mansoni* é bastante variável. Sabe-se que os esquistossomos humanos se têm diferenciado em cepas geográficas e que, nestes últimos anos, ficou demonstrado que a suscetibilidade dos caramujos à infecção por *S. mansoni* é regulada genèticamente. Sabe-se, ainda, que tais diferenças podem ser de ordem intra-específica. Pequenas diferenças genéticas foram verificadas entre duas cepas brasileiras de *Australorbis glabratus* que divergiam quanto a suscetibilidade à infecção por *S. mansoni*.

A capacidade do vector em transmitir *S. mansoni* depende do equilíbrio entre o parasita e seus hospedeiros intermediários. Diversos aspectos do equilíbrio hospedeiro-parasita são comentados, muito particularmente a capacidade que têm os vectores de conservar e transmitir a esquistossomose mansônica.

O tropismo de penetração próprio aos miracídios de *S. mansoni* não é específico. Os

miracídios penetram em moluscos d'água doce pertencentes a diversas famílias como *Physidae* e *Ampullariidae*. Além disso, os miracídios atacam àvidamente e penetram através o tegumento de girinos.

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