ANTAGONISM OF ECHINOSTOMA REVOLUTUM AGAINST SCHISTOSOMA MANSONI IN THE SNAIL BIOMPHALARIA ALEXANDRINA

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Abstract. Antagonism of rediae of Echinostoma revolutum against sporocysts of Schistosoma mansoni was tested in the natural intermediate host Biomphalaria alexandrina. The modes of double-infecting snails of the individual experimental groups with miracidia of E. revolutum and S. mansoni were these: a) simultaneously; b) superinfection of S. mansoni on E. revolutum at intervals of 10 and 20 days; c) superinfection of S. mansoni on E. revolutum producing cercariae. The results disclosed a marked dominance of E. revolutum in all combinations. During its development in the snail intermediate host, E. revolutum suppressed noticeably the development of S. mansoni sporocysts, or inhibited cercarial production. The marked pathogenic effect of rediae and, evidently, also metacercariae of E. revolutum on the organism of B. alexandrina was indicated by the high rate of snail mortality in all experimental groups. Experimental demonstration of marked antagonistic interactions between E. revolutum and S. mansoni during their development in the natural snail intermediate host suggested that this phenomenon may be utilized in the biological control of schistosomiasis of man caused by the fluke S. mansoni in Egypt.

Experimental studies on interspecific antagonism between different species of larval trematodes in a shared snail host have been the subject of numerous papers published within the last 10 years. The possibility to use this phenomenon, i.e., direct redial antagonism (predation), and also indirect antagonism and retardation in the biological control of human schistosomes in hyperendemic areas has been investigated in the laboratory in a combination of echinostome or strigeid flukes and S. mansoni (Lieu 1967, 1968; Basch et al. 1969; Heyneman et al. 1972).

The selection of a suitable trematode species to act as the parasite-predator has to be based on conditions prevailing in the areas of hyperendemic occurrence of human schistosomes. The results of studies by Ryšavý et al. (1973, 1974a, b), Moravec et al. (1974a) confirmed the presence of E. revolutum in Egypt (environments of Cairo). In this area, the fluke was found to develop in the snail Biomphalaria alexandrina, the intermediate host of Schistosoma mansoni Sambon, 1907. Experimental trials with rediae of E. revolutum (Froelich, 1802) = E. liei Jeyarasasingam, Heyneman, Lim et Mansour, 1972 (of the same origin as in our material) against sporocysts of S. mansoni have been made only in the laboratories of the Hooper Foundation, University of California, San Francisco. Heyneman et al. (1972) used the snail Biomphalaria glabrata (laboratory albino strain) as the intermediate host of both trematode species (and also of Paraphysostomum segregatum Dietz, 1909) in a series of experiments. They studied antagonism in this intermediate host both in a superinfection of E. revolutum (= E. liei) on S. mansoni, and a superinfection of S. mansoni on E. revolutum.
Our series of experiments was conducted in order to demonstrate intramolluscan antagonism between the two fluke species (*E. revolutum* and *S. mansoni*), partly by way of exposing the snails to simultaneous miracidial infection, partly to superinfection of *S. mansoni* on *E. revolutum*. It is of importance for the practice that antagonism between *E. revolutum* and *S. mansoni* in the natural snail intermediate host in Egypt, i.e., in *Biomphalaria alexandrina*, has been demonstrated for the first time in these experiments.

**MATERIAL AND METHODS**

Eggs for culture of *E. revolutum* miracidia were obtained from the faeces of laboratory animals (hamsters and white mice) with artificial infection. The methods used for the egg culture, activation of miracidia and their concentration are described in the paper by Moravee et al. (1974a). Eggs of *S. mansoni* were obtained from the liver of experimentally infected mice.* Hatchling of miracidia and their concentration by utilizing their marked positive phototaxis were performed with methods commonly used in work with this species.

In all experiments the infective dose was 20 miracidia per fluke and per snail. In a double infection the snails were exposed individually to either a mixture of 20 *E. revolutum* and 20 *S. mansoni* miracidia, or exposed first to *E. revolutum* and later re-exposed to the same dose of *S. mansoni* miracidia. Miracidia used for infection were not more than 2 hr old. The mode of preparation of the infective dose and of infection were essentially those described by Moravee et al. (1974b).

Rediae of *E. revolutum* were tested against sporocysts of *S. mansoni* in the natural snail intermediate host of this fluke species in Egypt, i.e., *Biomphalaria alexandrina*. Since all snails collected earlier in the locality Lmawwa Drain close to the village of Warak El Arab were found to be free of infection upon inspection with a compressorium, we collected in March 1973 a large number of snails in this locality and used these in our experiments. In order to adapt the snails to the new conditions, they were kept in the laboratory for one month and then exposed to the sun for the demonstration of cercarial production. Upon obtaining negative results, the snails were divided into groups according to size, and only snails measuring 5–8 mm were transferred to the control and experimental groups.

We arranged a total of four control groups (group 1—snails without infection; group 2—single infection with *E. revolutum*; group 3—single infection with *S. mansoni*; group 8—snails producing cercariae of *E. revolutum*—single infection), and four experimental groups (group 4—simultaneous infection with miracidia of both fluke species; group 5—superinfection of *S. mansoni* on *E. revolutum* at a 10-day interval between exposures; group 6—direct at a 20-day interval between exposures; group 7—superinfection of *S. mansoni* on *E. revolutum* producing cercariae). All experimental groups were kept under uniform conditions of temperature and light (average temperature of the water 24.5 °C, 5 l of filtered Nile water). The water was changed every other day; the snails were fed fresh lettuce leaves (*ad libitum*). The rate of snail mortality in the individual groups was recorded from the first to the last day of the experiment. From day 15 p.i. onwards, the development of cercarial production in snails with a single and double infection (groups 2, 3, 4, 5, 6) was tested by exposing each snail to the sun. In group 7 and 8, cercarial production was tested regularly from the first day of setting up these groups. The shells of snails shedding cercariae were marked with acetone stains (blue for *E. revolutum*, red for *S. mansoni*). Infection rates were calculated from the actual number of snails shedding the pertinent cercarial species.

**RESULTS**

Our present experiments were conducted for the purpose of demonstrating antagonistic interaction between *E. revolutum* and *S. mansoni* in the natural snail intermediate host *B. alexandrina*, partly in simultaneous infection with miracidia of both species, partly in superinfection of *S. mansoni* on *E. revolutum*. Four of the eight groups in our experiments were controls.

**Group 1** (snails without infection): This group was started with 175 snails of the species *B. alexandrina*. The rate of mortality in this group enabled a comparison with this rate.

*) Our thanks are due to Mrs. Soad Sherif M.Sc., Mrs. Aisha Metwalli M.Sc. and Dr. Noshy S. Mansour from Cairo for kindly providing the initial material of the Egyptian strain of *S. mansoni*.  

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Fig. 1. Rate of mortality in control groups of *B. alexandrina* snails. (---) group 1, snails without infection; (-----) group 2, snails with a single infection with *E. revolutum*; (-----) group 3 (snails with a single infection with *S. mansoni*). Arrows mark start of cercarial production of the individual trematode species.

Fig. 2. Infection rates in group 4 (snails with simultaneous mixed infection with miracidia of *E. revolutum* and *S. mansoni*). (-----) snails producing *E. revolutum* cercariae; (-----) snails simultaneously producing *E. revolutum* and *S. mansoni* cercariae; (-----) infection rates in groups 3 (single infection with *S. mansoni*) given for comparison.
in the experimental groups. In group 1 this rate was lower than in the experimental groups (2, 3, 4, 5, 6) — (Fig. 1), its curve was fairly even; 9 snails, i.e., 5.1% of the initial number of snails in the group were alive after the termination of the experiment (on day 162).

**Group 2** (single infection with *E. revolutum*): This group numbered originally 134 specimens of *B. alexandrina*. Each snail was exposed to a dose of 20 miracidia of *E. revolutum*. From day 15 p.i. onwards, the snails were exposed individually to the sun. The first cercariae appeared on day 36 p.i., they were shed by 60 out of the total of 118 snails. The maximum number of snails (95 out of 108) shedding simultaneously cercariae was recorded on day 45 p.i., i.e., 9 days after their first appearance. Infection rate values varied from 50 to 88% during the course of the experiment (Fig. 4).

The rate of snail mortality was relatively low during the first half of the experiment. It increased shortly before the first appearance of cercariae and, particularly, during the time of fully developed cercarial production (Fig. 1). The number of snails decreased speedily in this experimental group; on day 70 p.i. it consisted of five snails only, of which four produced cercariae of *E. revolutum*. The last snail of the group died on day 77 p.i. Cercariae were shed by snails of this group for 41 days. In addition to the marked pathogenic effect of *E. revolutum* rediae on the organism of *B. alexandrina*, the factor participating in the vehement increase of the rate of snail mortality was the re-entrance of cercariae into the snail's body, in which they encysted in masses in the pericardiac sac, kidney, and frequently in other organs. The number of cercaria-shedding snails was higher in this group (Table 1) than in groups 4, 5, 6, and this, evidently, accounted for the shortening of the life span of snails in this group by 3 to 7 days.

**Group 3** (single infection with *S. mansoni*): The group comprised 100 snails of which each was exposed to a dose of 20 miracidia of *S. mansoni*. The first cercariae of this species appeared on day 41 p.i. and were shed by two out of the 56 snails. From that day onwards, the number of snails shedding cercariae increased and attained its maximum 12 days later, i.e., on day 53 p.i. (26 out of a total of 46 snails). The number of snails in this experimental group continued to decrease as a result of snail mortality (Fig. 1) under a simultaneous increase in the number of cercariae-shedding snails and, consequently, an increase in the values of infection rates (Fig. 2). On day 64 of cercarial production, i.e., on day 105 p.i., the group consisted of 8 snails only, and all these were shedding *S. mansoni* cercariae. The last living snail stopped producing cercariae on day 118 p.i. and died 5 days later. The experiment lasted 123 days, out of which cercariae were shed for 77 days.

The rate of snail mortality in this group displayed higher values in the early developmental phases of *S. mansoni*. The curve of the rate of mortality was more or less balanced during the course of the experiment and did not increase noticeably after the beginning of cercarial production (Fig. 1). A comparison of death rates disclosed that, within the first 50 days p.i., these were higher in group 3 (single infection with *S. mansoni*) than those recorded for the same period for group 2 (single infection with *E. revolutum*) and groups 4, 5, 6 (double infection). In our opinion this may be ascribed to the fact that the penetrating *S. mansoni* miracidia are larger than those of *E. revolutum* and, together with the sporocysts, are apparently more pathogenic to the snails within the first stages of development than are sporocysts of *E. revolutum*. Since in a single-worm infection the development of *S. mansoni* sporocysts was not inhibited by antagonistic interaction with sporocysts and, particularly rediae of *E. revolutum*, the mortality of snails in this group (no. 3) may have been caused by this full-scale development. In the groups of snails with double infection, antagonistic interactions between the two species inhibit or arrest the development of sporocysts of *S. mansoni* and destroy them. The high pathogenic effect on the snails reflected in the high rate of mortality,
was recorded at the time of development of *E. revolutum* rediae, after the beginning of cercarial production and, later, during the encystment of *E. revolutum* cercariae in vital organs of the snail.

**Group 4** (simultaneous infection with *E. revolutum* and *S. mansoni*): The original number of snails in this group was 74. Each snail was exposed to a mixture of 20 miracidia of *E. revolutum* and 20 miracidia of *S. mansoni*. The first cercariae of *E. revolutum* appeared

![Graph showing mortality rate over time.](image)

**Fig. 3.** Mortality rate in group 4 (——) snails double infected with miracidia of *E. revolutum* and *S. mansoni*; mortality in group 2 (----) snails with a single *E. revolutum* infection given for comparison. Simple arrow marks start of production of *E. revolutum* cercariae; double arrow marks start of double production (*E. revolutum* and *S. mansoni*).

on day 33 p.i., they were shed by 5 out of a total of 54 snails. The number of cercariae-shedding snails increased gradually up to its maximum. This was attained on day 38 p.i., i.e., 5 days after the first appearance of cercariae. On this day, cercariae were released by 23 out of a total of 52 surviving snails.

The concurrent release of cercariae of both trematode species occurred in this experimental group only (Fig. 2). One snail shedding originally cercariae of *E. revolutum* only, started to shed also *S. mansoni* cercariae on day 43 p.i. At this time, cercarial production was developed in 22 out of a total of 46 snails. The simultaneous release of
both cercaria species was recorded for three subsequent days. On the fourth day, the snail ceased to shed cercariae of *S. mansoni*, but continued to shed those of *E. revolutum.* Seven days later the snail died. The simultaneous release of cercariae of both species was recorded from two other snails on day 50 p.i. (at this time, 19 out of a total of 38 snails shed cercariae of *E. revolutum*). One of these snails died after two days of producing simultaneously cercariae of both species, the other snail continued to shed cercariae of both trematode species for eight days, and then released only cercariae of *E. revolutum.* It died 6 days later.

![Graph](image)

**Fig. 4.** Infection rates in group 2, 5, 6. (— — —) group 2 (snails with single *E. revolutum* infection); (— — —) group 5 (re-infection of *S. mansoni* on *E. revolutum*—10-day interval); (— — —) group 6 (re-infection of *S. mansoni* on *E. revolutum*—20-day interval).

According to our results, the period for which cercariae of both species were produced simultaneously in the individual snails did not surpass eight days, and the total period of simultaneous release of both cercaria species lasted 15 days only. An important finding was the marked dominance of *E. revolutum* in the snail host shedding simultaneously cercariae of the two trematode species. Tests performed by exposure of the snails to the sun disclosed considerable differences in the number of cercariae in the individual hosts produced by the two trematode species. The average number of *S. mansoni* cercariae shed by the snails ranged from 2 to 8 specimens, that of *E. revolutum* shed by the same snails ranged from 28 to 75 specimens. After suppressing the production of schistosome cercariae, these snails shed only *E. revolutum* cercariae in large numbers. Snails with a double infection had a limited life span (Fig. 3). Infection rate
values for *E. revolutum* surpassed mostly 40% at the time of cercarial production (Fig. 2).

On day 71 p.i., three snails only survived in the group and of these one was shedding *E. revolutum* cercariae. The last snail died on day 80 p.i. In this group, cercariae of *E. revolutum* were shed for a period of 39 days, the concurrent release of both species of cercariae lasted 15 days only. The period for which cercariae of *S. mansoni* only were produced has not been recorded in this group.

**Group 5** (superinfection of *S. mansoni* on *E. revolutum* — with a 10-day interval between exposures): The group was started with 90 snails, of which each was exposed first to 20 miracidia of *E. revolutum* and, 10 days later, re-exposed individually to 20 miracidia of *S. mansoni*. Cercarial production of *E. revolutum* started on day 37 p.i. (from the beginning of the experiment). Out of a total of 70 snails, cercariae of this species were shed by 13 snails. The number of snails shedding *E. revolutum* cercariae increased and attained its maximum on day 45 p.i. (56 out of a total of 63 snails). The rate of snail mortality increased shortly before the production of *E. revolutum* cercariae, and during cercarial production, and the number of snails in the group was reduced speedily (Fig. 5); infection rate values surpassed mostly 90% (Fig. 4). On day 82 of infection with *E. revolutum*, the group consisted of 3 snails only (one of these was shedding *E. revolutum* cercariae). Two days later, all snails were dead. In this experiment which lasted for 84 days, cercariae of *E. revolutum* were produced for 45 days. None of the snails of this group released cercariae of *S. mansoni* although, theoretically, the period of infection with this fluke was long enough (74 days) to enable development to cercarial production.

**Group 6** (superinfection of *S. mansoni* on *E. revolutum* — with a 20-day interval between exposures): The original number of snails in this group was 80 specimens. Each snail was exposed to 20 miracidia of *E. revolutum* and, 20 days later, re-exposed to 20 miracidia of *S. mansoni*. *E. revolutum* developed to cercarial production in 11 out of the 63 surviving snails on day 37 p.i. (after the start of the experiment). The number of cercaria-shedding snails increased in the following days and attained its maximum on day 49 p.i. (46 out of a total of 56 snails). The rate of snail mortality increased after the first appearance of *E. revolutum* cercariae; at that time, the values of infection rates surpassed mostly 80% (Fig. 4). In view of the high rate of mortality, the number of snails in the group was quickly reduced. On day 77 p.i., four snails only remained and three of them released cercariae. The last snail died on day 81 p.i. (Fig. 5). The release of cercariae of *E. revolutum* from the snails of this group was recorded for a period of 40 days.

Although, theoretically, cercarial production of *S. mansoni* could have developed during the 61 days postexposure to miracidia of this species, none of the snails in the group was found to release cercariae of this species.

**Group 7** (superinfection of *S. mansoni* on *E. revolutum* at the time of cercarial production): We set up a group consisting of 233 *B. alexandrina* snails and exposed each snail to infection with 20 miracidia of *E. revolutum*. On day 47 p.i., we selected snails which had started to shed cercariae of *E. revolutum* and of these we formed two groups. In group no. 7, we placed 60 snails. On the same day, i.e., on day 47 p.i. with *E. revolutum*, these were re-exposed individually to 20 miracidia of *S. mansoni*. Development of infection in the snails was tested by exposure of the snails to the sun every other day (similar to the other groups). The values of infection rates were 100% for the duration of this experimental group.

Snail mortality was extremely high (Fig. 6), and the last snail died on day 37 following re-infection with *S. mansoni*, i.e., on day 80 p.i. with *E. revolutum*. None of the snails released cercariae of *S. mansoni* but, judging from the data obtained in group 3, *S. mansoni* could not have developed to cercarial production during this period and at the given temperature.
Group 8 (single infection with *E. revolutum*—at the time of cercarial production): This group consisting originally of 60 snails was used as control of group 7 (double infection). All snails of this group produced *E. revolutum* cercariae on day 47 p.i. Infection rate values persisted at 100% during the time of observation of this group. Snail mortality was high. The last snail died 35 days after the setting up of this group, i.e., on day 78 of infection with *E. revolutum*. The curve indicating the rate of snail mortality was similar in both groups (7 and 8) and confirmed the marked pathogenic effect of cercariae and rediae of *E. revolutum* on the organism of *B. alexandrina*. A more marked increase in the rate of snail mortality in group 7 (Fig. 6) was recorded only within the first 15 days of re-infection with *S. mansoni*. After this period, snail mortality in both groups was so high that any more marked differences between the two groups were practically wiped out. This may, evidently, be ascribed to the pathogenic effect of *E. revolutum* rediae, and also to the re-entrance of cercariae into the snails and their encystment in the organs.

**DISCUSSION**

It has been clearly indicated by the results of studies by Lie (1966, 1967, 1969) on antagonistic interaction between *P. segregatum* and other echinostome trematodes that the development of *S. mansoni* can be inhibited by the rediae of these flukes, and their

![Graph showing mortality rate](image.png)

**Fig. 5.** Mortality rate in groups 5 and 6. (---) group 5 (re-infection of *S. mansoni* on *E. revolutum*-10-day interval); (-----) group 6 (re-infection of *S. mansoni* on *E. revolutum*-20-day interval). Arrows mark start of cercarial production of *E. revolutum*.

![Graph showing rate of mortality](image.png)

**Fig. 6.** Rate of mortality in groups 7 and 8. (---) group 7 (re-infection of *S. mansoni* on snails producing cercariae of *E. revolutum*); (-----) group 8 (control—snails producing cercariae of *E. revolutum* from a single infection).
sporocysts attacked, destroyed and consumed by them. Although Heyneman et al. (1972) considered it possible to introduce these echinostome fluke species to endemic areas of schistosomiasis of man, the use of natural parasites of the snail intermediate hosts of schistosomes in the areas under consideration appeared to these authors to be better suited for the purpose of biological control. Therefore, these authors selected the species *E. revolutum* (= *E. liei*) and employed it in a number of experiments and in various combinations with *S. mansonii* in the snail *Biomphalaria glabrata*. Since *E. revolutum* is a species which can easily be maintained and multiplied in the laboratory, this facilitates its introduction to foci of schistosomiasis, because it is also a natural parasite of *B. alexandrina*, the snail intermediate host of *S. mansonii*. Recent studies on the biology of *E. revolutum* (Jeyaraseasingam et al. 1972; Moravec et al. 1974a) cleared a number of problems essential for laboratory—and field trials with this species.

The results of our experiments performed in various combinations (i.e., simultaneous infection of snails with miracidia of *E. revolutum* and *S. mansonii*; superinfection of *S. mansonii* on *E. revolutum* at various developmental stages) demonstrated that antagonistic interactions developed between the two trematode species in the natural intermediate host (*B. alexandrina*), whereby *E. revolutum* was always the dominant species. An evaluation of a simultaneous double infection of snails (group 4) showed that the miracidia of both species (*E. revolutum* and *S. mansonii*) can enter the snail simultaneously and develop in it. This is consistent with the results obtained, e.g., by Basch and Lie (1966a, b). Rodiae of *E. revolutum* displayed a marked aggressiveness against sporocysts of *S. mansonii* and shortened remarkably the period of production of *S. mansonii* cercariae, reduced the number of produced cercariae, arrested their production and led, finally, to the death of the intermediate snail host. A remarkable shortening of the period of cercarial production in *S. mansonii* was observed in snails with double infection (Fig. 2). This showed up particularly well, in a comparison with control group 3 (single infection with *S. mansonii*) in which cercariae were shed for a period of 77 days, while snails with a double infection released cercariae for 15 days only. We should like to recall that the period of production of *S. mansonii* cercariae did never surpass 8 days in the individual snails. Strong evidence on the dominant effect of *E. revolutum* was obtained also from a comparison of infection rate values in group 4 and control group 3 (Fig. 2). Another important fact is the high pathogenicity of *E. revolutum* to the organism of the snail in both a double and single infection, as this has been pointed out in an earlier paper by Heyneman et al. (1972) for the intermediate host *B. glabrata*. According to our observations, *E. revolutum* has a similar effect on the organism of *B. alexandrina*. The life span of snails with a single and double infection (Figs. 1, 3, 5) was considerably shorter than that of the controls (group 1—snails without infection; group 3—with *S. mansonii* infection). In groups 4, 5, 6, 7 (double infection) the rate of mortality was considerably higher particularly during the early phases of development of *E. revolutum*, than that recorded for control group 2 (single infection with *E. revolutum*). The final result, however, disclosed no marked difference in the longevity of snails of the experimental groups and that in control group 2. The snails of all these groups died within 84 days of infection with *E. revolutum*, cercarial production lasted from 39 to 45 days. In control group 3 (single infection with *S. mansonii*), the snails survived for 123 days, in group 1 (without infection), 5.1% of the initial number of snails in this group survived past the termination of this observation (162 days). Heyneman et al. (1972) demonstrated in double infected *B. glabrata* snails (*E. liei = [E. revolutum]* and *S. mansonii*) that the development of *E. revolutum* was prolonged in these snails. This manifestation of indirect antagonism has not been confirmed in our experiments with *B. alexandrina* (Table 1).
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<th>Original no. of snails</th>
<th>Maximum no. of snails shedding cercariae simultaneously</th>
<th>Maximum no. of snails shedding cercariae simultaneously expressed in % (out of original no. of snails in a group)</th>
<th>Days p.i. at which the maximum no. of snails shed simultaneously cercariae</th>
<th>No. of days up to the start of cercaria production</th>
<th>Total period of cercaria production in days</th>
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<tr>
<td>Single infection with <em>E. revolutum</em>—group 2</td>
<td>134</td>
<td>95</td>
<td>70.8</td>
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<td>Single infection with <em>S. mansoni</em>—group 3</td>
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<td>26.0</td>
<td>53</td>
<td>41</td>
<td>77</td>
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<td>Simultaneous infection with <em>S. mansoni</em> and <em>E. revolutum</em>—group 4</td>
<td>74</td>
<td><em>Echinostoma</em> 23</td>
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<td>39</td>
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<td>43</td>
<td>15</td>
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<td>90</td>
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<td>Superinfection of <em>S. mansoni</em> on <em>E. revolutum</em> with 20-day interval—group 6</td>
<td>80</td>
<td><em>Echinostoma</em> 46</td>
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The high rate of snail mortality recorded in the later phase of development of *E. revolutum* may be due to the pathogenic effect of cercariae of this species re-entering the snail and encysting in its tissues. It is evident that a small number of metacercariaae does not damage severely the organism of the snail, but if there are several hundred of them, and this is common under experimental conditions, the situation may be quite different. A high concentration of metacercariae of various fluke species in the snails may occur also in nature. While studying the biology of *Echinoparyphium recurvatum*, Rašín (1933) observed in the field that mortality of *e.g.* *Lymnaea peregra* was caused by metacercariae.

Interesting results were obtained with a superinfection of *S. mansoni* on *E. revolutum* at 10- and 20-day intervals between exposures, and superinfection on *E. revolutum* producing cercariae. Our results are in accord with those obtained by Heyneman et al. (1972). Although the miracidia entered readily the organism of the snail, *S. mansoni* did not develop to cercarial production in any of our experimental groups. This finding is very important from the aspect of a possible utilization of *E. revolutum* for the biological control of *S. mansoni*, because it confirms the great predatory activity of the rediae of this echinostome fluke against *S. mansoni* in the natural intermediate host *B. alexandrina*. It supports an important aspect which characterizes *E. revolutum* as the suitable biocontrol candidate of human schistosomiasis caused by *S. mansoni*, in Egypt.

We are fully aware of the fact that other series of laboratory- and field trials will have to be conducted in selected biotopes in which the incidence of infection with *S. mansoni* is different in the snails. In the first phase it may be possible to introduce experimentally an infection with *E. revolutum* and secure a suitable definitive host for this species in these sites. In an area as intensively changed by man-made activities from the times of antiquity to the present days as in that under consideration, the definitive hosts may have become extinct in several biotopes, while in others host populations may not be dense enough to maintain a continuous, uninterrupted circulation of *E. revolutum* and, simultaneously, a high incidence of infection with this fluke in the snail intermediate hosts. A high incidence of infection with *E. revolutum* in the snail intermediate hosts in natural biotopes is the requisite for the development of antagonistic interactions between this fluke and *S. mansoni*. It is possible that the problem of finding a suitable definitive host of *E. revolutum* in the various biotopes may be solved by the introduction of domestic duck breeding along the various irrigation systems as suggested earlier by Ryšavý et al. (1973). The problem of schistosomiasis of man in Egypt, however, cannot be solved by a single method, even if this proves to be most effective, but by a complex of fully developed methods, particularly those concerned with biological control, the application of molluscocides, therapeutic procedures and health education.

АНТАГОНИЗМ МЕЖДУ ECHINOSTOMA REVOLUTUM И SCHISTOSOMA MANSONI В МОЛЛЮСКЕ BIOMPHALARIA ALEXANDRINA

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Резюме. Нами проводились эксперименты по антагонизму между редиами вида *Echinostoma revolutum* и спористами вида *Schistosoma mansoni* в естественном промежуточном хозяине *Biomphalaria alexandrina*. Результаты, полученные из отделных экспериментальных групп моллюсков зараженных мирицидами видов *E. revolutum* и *S. mansoni*, путем одновременной инвазии, путем суперинвазии видом *S. mansoni* после инвазии видом *E. revolutum* 10—20 дней спустя и путем суперинвазии видом *S. mansoni* в моллюсках с продукцией церкарий видов *E. revolutum* показали, что *E. revolutum* является домнирующим видом
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