SHORT REPORT: SCHISTOSOMA MANSONI MIRACIDIA ARE KILLED BY THE DEFENSE SYSTEM OF AN ARGENTINE STRAIN OF BIOMPHALARIA STRAMINEA

LUBIANA GRASSI, MARTÍN TORRES JORDÁ, ZILTON ANDRADE, AND STELLA MARIS GONZÁLEZ CAPPA
Departamento de Microbiología, Parasitología e Inmunología, Facultad de Medicina, Universidad de Buenos Aires, Buenos Aires, Argentina; Centro de Pesquisas Gonçalo Moniz-Fiocruz, Salvador, Bahia, Brasil

Abstract. Biomphalaria straminea snails from Argentina fail to shed cercariae even if exposed to high doses of Schistosoma mansoni EC miracidia. Alternative explanations for this failure are that miracidia are unable to penetrate the snail’s epithelium or that the miracidia are killed by the snail’s defense system. To discriminate between these 2 possibilities, B. straminea snails were individually exposed to increasing doses of miracidia. Susceptible B. glabrata were used as controls. Exposed snails were fixed 12 hr after exposure, and histological sections of the whole specimens were examined. Miracidia were seen to penetrate the epithelium of B. straminea and B. glabrata at similar rates (14.7%), independent of the exposure level. Regardless of the miracidial dose, 94% of the penetrating miracidia appeared encapsulated by the B. straminea defense system, whereas in B. glabrata, only 42% of the miracidia underwent encapsulation. These results show that resistance of B. straminea to S. mansoni EC strain is due to an efficient defense system that destroys miracidia once they have penetrated.

The intermediate hosts of Schistosoma mansoni in America are, in order of increasing susceptibility, Biomphalaria straminea, Biomphalaria tenagophila, and Biomphalaria glabrata. Differences in susceptibility of these snail species are strongly related to the capacity of their defense system to encapsulate and destroy the parasites. Even though B. straminea and B. tenagophila are present in Argentina, there are no reports on the presence of S. mansoni in this country so far. In our laboratory, we maintain a colony of B. straminea from the locality of San Miguel, Corrientes province, Argentina. Previous attempts to infect these snails with various doses of S. mansoni EC strain miracidia have failed. The EC strain was isolated in October 1981 by Lobato Paraisense from the feces of a resident of Picos, Piauí state, Brazil, where the vector of S. mansoni is B. straminea. Experimental infections of B. straminea collected in this locality

![Figure 1](image-url)

**Figure 1.** Number of penetrating miracidia detected from sections of the whole snails at 12 hr after exposure as a function of miracidial dose. Points represent counts from individual snails. Differences between the regressions of B. glabrata and B. straminea were not significant.
Location of penetrating miracidia and miracidia with cellular response at 12 hr after exposure

<table>
<thead>
<tr>
<th>Snail species (n)</th>
<th>n</th>
<th>Penetrating</th>
<th>Encapsulated</th>
<th>Foot</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biomphalaria straminea</td>
<td>1,450</td>
<td>210 (14.5)</td>
<td>197 (93.7)†</td>
<td>180 (86)‡</td>
<td>30 (14)</td>
</tr>
<tr>
<td>Biomphalaria glabrata</td>
<td>1,050</td>
<td>156 (14.9)</td>
<td>66 (42.3)</td>
<td>83 (53)</td>
<td>73 (47)</td>
</tr>
</tbody>
</table>

* For each snail species, pooled data from all miracidial doses are shown.
† P < 0.01.
‡ P = 0.032.

Table 2

<table>
<thead>
<tr>
<th>Miracidial dose</th>
<th>Biomphalaria straminea</th>
<th>Biomphalaria glabrata</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>s</td>
<td>s²</td>
</tr>
<tr>
<td>10</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>40</td>
<td>6.4</td>
<td>9.3</td>
</tr>
<tr>
<td>80</td>
<td>10.4</td>
<td>15.3</td>
</tr>
<tr>
<td>160</td>
<td>24.2</td>
<td>48.7</td>
</tr>
<tr>
<td>Overall U*</td>
<td>2.05†</td>
<td>6.70‡</td>
</tr>
</tbody>
</table>

* Values of the U statistic greater than zero indicate that actual variance is higher than the one expected from the binomial distribution. The U statistic follows a standard normal distribution under the null hypothesis of binomial distribution. ‡ ND = not tested.
† P < 0.05.
‡ P < 0.001.
fense system that stops any progress of miracidia once they have penetrated. The independence of the penetration rate and the miracial dose indicates that the barrier effect of the snail epithelium was not affected by the rate of miracidial penetration.

Penetrating miracidia were overdispersed among snails of both species ($R > 1$), providing indirect evidence that snails were heterogeneous with respect to the probability of miracidial penetration. This heterogeneity may be associated with either the activity of different snails during exposure to infections or with individual variability in the barrier effect of the epithelium. Miracidia of *S. mansoni* attained deeper locations in the organs of *B. glabrata* than in those of *B. straminea*. It is apparent that in *B. straminea* encapsulation of miracidia soon after penetration prevents them from moving further into the snail’s organs.

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Authors’ addresses: Lubiana Grassi, Martín Torres Jordá, and Stella Maris González Cappa, Departamento de Microbiología Parasitología e Inmunología, Facultad de Medicina, Universidad de Buenos Aires, Paraguay 2155, Piso 13, (1121) Buenos Aires, Argentina. Zilton Andrade, Centro de Pesquisas Gonçalo Moniz-Fiocruz, Rua Valdemar Falcao 121, 40295-001 Salvador, BA, Brasil.

Reprint requests: Lubiana Grassi, Departamento de Microbiología Parasitología e Inmunología, Facultad de Medicina, Universidad de Buenos Aires, Paraguay 2155, Piso 13, (1121) Buenos Aires, Argentina, Telephone and fax: 54-11-49637078 (e-mail: sngcappa@fmed.uba.ar).

REFERENCES