Snail susceptibility or trematode infectivity?

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Introduction

The success or failure of a trematode parasite in a potential molluscan host has traditionally been seen in terms of the mollusc's susceptibility to infection. Newton's (1952) investigations on the host–parasite relationships between Biomphalaria glabrata and Schistosoma mansoni showed that in non-susceptible strains of the snail invading miracidia were rapidly attacked by amoebocytes and subsequently encapsulated while no such reaction occurred in susceptible snails. In a later paper Newton (1954) demonstrated that the tendency to produce this tissue reaction was inherited in second generation snails resulting from cross-matings between susceptible and non-susceptible strains. Other workers subsequently confirmed these general findings with different snail hosts and parasites. As a result many malacological taxonomists were encouraged to pursue their searches for readily identifiable characters of snails which could be associated with susceptibility to infection.

In 1961, however, Tripp described the cellular responses of Biomphalaria glabrata to various materials, including biologically inert objects and heterologous grafts of fresh tissue of the planorbid Planorbarius corneus. The tissue responses to some of the biologically inert materials reported by Tripp and those shown by non-susceptible B. glabrata to miracidia of S. mansoni appeared to be very similar. This led Wright (1966) to suggest that the lack of cellular reaction to invading miracidia in successful infections might be due to evasion or inhibition by the parasite of the snail's innate response to foreign material. Wright (1971) explored this idea in more detail and speculated on the mechanisms which might be involved. In the present paper the implications of some current work in this laboratory are discussed briefly.

Tissue responses in Bulinus to Schistosoma species

Little is known about the responses of bulinid snails to invading miracidia apart from Wajdi's (1964) histological study of S. haematobium in various members of the B. forskali species group and Kinoti's (1971) observations on S. mattheei in B. africanus and B. truncatus. A more extended study of the relationships between bulinids and several species of Schistosoma belonging to the groups with terminal spines on their eggs is now being carried out in this laboratory.

Because Kinoti (1971) found that very few miracidia of S. mattheei succeeded in penetrating into B. truncatus, an abnormal host for the parasite, he suggested that such incompatible relationships were due to surface phenomena which
prevented entry of the larvae. Although miracidia of the 'terminal spined' schistosomes are less prone to penetration of unusual hosts than are those of *S. mansoni* they do enter snails which are not suitable for their further development. Kinoti himself described fairly typical cellular reactions to *S. mattheei* miracidia in *B. truncatus* and in the current work such lesions have been found in a number of combinations of parasites and hosts. The situations of particular interest in the present context are those in which one species of parasite invokes a cellular reaction in a snail host while another species of schistosome develops successfully without any apparent host response in the same strain of snail. This is particularly well-illustrated by the relationships of *Bulinus crystallinus* (Morelet), a member of the *B. forskali* complex from the forest areas of Angola, to the Cameroon strains of *Schistosoma haematobium* and *S. intercalatum*. In experimental infections *B. crystallinus* has proved to be a good host for the Cameroon strain of *S. intercalatum* with infection rates of the order of 75% but so far no successful infections of this snail have been obtained with any strain of *S. haematobium*. Histological examination of *B. crystallinus* snails exposed to miracidia of *S. haematobium* has revealed intense tissue reaction with encapsulation of the parasites while snails exposed to larvae of *S. intercalatum* show no apparent cellular responses and the parasites develop normally.

**Snail-host range of hybrid schistosomes**

Most species of schistosomes belonging to the 'terminal-spined' groups are markedly restricted in the range of their snail hosts. Thus *Schistosoma mattheei* will develop successfully only in snails belonging to the *Bulinus africanus* group and not all of the species in this complex are equally good hosts. The Lower Guinea strain of *S. intercalatum* develops only in members of the *B. forskali* group (Wright *et al.*, 1972) and thus neither of these two parasites is able to develop in the snail hosts suited to the other species. However, hybrid miracidia resulting from the cross *S. intercalatum* ♀ × *S. mattheei* ♂ are infective to both

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\begin{align*}
S. \text{intercalatum} & \times S. \text{mattheei} \\
(B. \text{scalaris}) ♀ & \times (B. \text{globosus}) ♂ \\
F_1 & \\
(B. \text{scalaris} & B. \text{globosus}) \\
F_2 & \\
(B. \text{scalaris}, B. \text{senegalensis} & B. \text{globosus}) \\
F_3 & \\
(B. \text{scalaris} & B. \text{globosus}) \\
F_4 & \\
(B. \text{globosus})
\end{align*}
\]
of the parental host snail groups. This ability to develop in either of the parental host snail species has persisted through the F₁, F₂ and F₃ generations, in each case the infections were more successful in terms of cercarial productivity in the original paternal host species and in the F₄ generation this was the only success obtained (table I).

Similar results have been reported by Taylor (1970) with crosses between S. bovis and S. mattheei but they do not illustrate the point so well because the snail hosts of these two species are not as mutually exclusive as those in our experiments. S. bovis normally develops in snails of the B. truncatus group but successful infections in B. africanaus group snails can be achieved and the dual host behaviour of the hybrids is therefore less significant.

Discussion

What inferences can be drawn from these observations? In the first example B. crystallinus produces a typical cellular reaction against the miracidia of S. haematobium, with which it is not compatible, but fails to react against those of S. intercalatum. Since the snail has shown itself to be capable of recognising and responding to the presence of a foreign object it must be assumed that its failure to respond to the miracidia of S. intercalatum is due to some property of these larvae. In the second example the behaviour of the F₁ hybrid miracidia is particularly instructive in that while they might well be expected to develop successfully in the correct snail host for their female parent they are equally successful in snails suited only to their paternal species. This suggests that the failure of the paternal host snails to recognize the hybrid larvae as foreign bodies is due to inheritance by the miracidia of some factor from their male parent.

The evidence of these two examples points strongly towards the ability of successful miracidia to evade the innate cellular responses of their snail hosts but the mechanisms by which this evasion is achieved remain a subject for speculation. It is possible that some initial inhibition of the snail’s response occurs, perhaps by a component of the secretions from the miracidial penetration glands. However, any such inhibition is likely to be rather ephemeral in duration and merely provide some respite while the parasite adopts a more permanent defence or camouflage. This could be by the adsorption of host substances onto the larval teguments in a manner similar to that of adult schistosomes (Smithers et al., 1969), it could be by the production of some ‘immunologically’ inert covering or by the continued synthesis of inhibiting substances. The mechanisms employed may not be the same throughout the Digenea. In species such as Fasciola which shed the ciliated miracidial covering at the time of penetration and replace it by a freshly secreted tegument (Southgate, 1970) it is likely that the process differs from that in the schistosomes where the ciliated plates are retained after entry into the molluscan host.

Postulation of possible different stages in the evasion process allows some flexibility in considering the fate of unsuccessful miracidia. Even in normally compatible host-parasite associations not all miracidia which penetrate the mollusc survive and it may be that these failures result from an inability to proceed to the second stage of camouflage or inhibition. In incompatible associations the failure might occur almost immediately through lack of appropriate inhibitory substances or it may be brought about by some unfavourable
physiological characteristic of the snail such as the presence of substances antagonistic to the parasite or the absence of some essential factor. In all of these cases encapsulation of the miracidium will follow but the time at which this occurs will presumably depend upon the stage at which failure occurs.

The emphasis of this contribution has been on the rôle of the parasite in establishing a successful infection in a snail host and some possible causes of failure have been considered. These considerations lead back to the concept of snail susceptibility which now appears as a less positive attribute in the immunological sense. However, susceptibility implies that the snail is providing a satisfactory biochemical environment for the developing parasite and, like any other environment, this will consist of a number of interacting factors whose presence or absence determine its favourable status. Seen in these terms the inheritance of susceptibility may in some cases be due to a single factor (not necessarily the same in each case) and in others it may be infinitely more complex.

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References


