SHORT COMMUNICATION

Sequential Histological Changes in *Biomphalaria glabrata* during the Course of *Schistosoma mansoni* Infection

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Biomphalaria glabrata, *highly susceptible to* Schistosoma mansoni, *were seen to shed less and less cercariae along the time of infection. Histological examination kept a close correlation with this changing pattern of cercarial shedding, turning an initial picture of no-reaction (tolerance) gradually into one of hemocyte proliferation with formation of focal encapsulating lesions around disintegrating sporocysts and cercariae, a change that became disseminated toward the 142nd day post miracidial exposure. Findings were suggestive of a gradual installation of acquired immunity in snails infected with* S. mansoni.

Key words: *Biomphalaria glabrata* - *Schistosoma mansoni* - histopathology

The evidences of susceptibility or resistance to *Schistosoma mansoni* infection presented by different species of *Biomphalaria* snails may vary considerably (Newton 1952, Pan 1965, Souza et al. 1995). Such variability can even be observed in snails belonging to a same species, from different geographic areas (Correa et al. 1979). The capacity of an infected snail to shed cercariae, is well correlated with histopathology. In highly susceptible *B. glabrata*, for instance, sporocysts and cercariae are observed in great number, simply displacing the structures, in the absence of any host tissue reaction. On the other hand, resistant snails, eliminating few cercariae, exhibit marked focal and diffuse hemocyte proliferation in several organs and tissues, usually resulting in encapsulation of disintegrating sporocysts and cercariae (Lie et al. 1980, Lewis et al. 1993).

We observed that such histological features indeed reflect resistance. However, it is not a fixed characteristic of a given snail strain, but may gradually develop along the course of infection in snails previously presenting a susceptible pattern of cercarial shedding.

A group of highly susceptible *B. glabrata* from Feira de Santana, BA, Brazil, originated from a single egg, were used. A total of 30 snails were submitted to infection with 15 miracidia, during 50 min with a Feira de Santana strain of *S. mansoni* (Andrade & Sadigursky 1985). Later, each of them was individually exposed to bright light during 40 min, for cercarial shedding and counting, starting 30 days post-infection and continuing thereafter at every 15th day, up to the 142nd after miracidium exposure. At each experimental point, two infected snails were selected for histological examination, the one representing the highest number of eliminated cercariae and the other, the lowest number in a particular day. These snails were anesthetized with menthol crystals before being taken out of the shell, and immediately fixed in Bouin' s fluid for 4 h. They were then washed in 70% alcohol, dehydrated in absolute alcohol, cleared in xylol and embedded in paraffin. Sections were stained with hematoxylin and eosin. The curve representing cercarial shedding (Fig. 1) started at the 37th day post-infection and reached the highest peak by

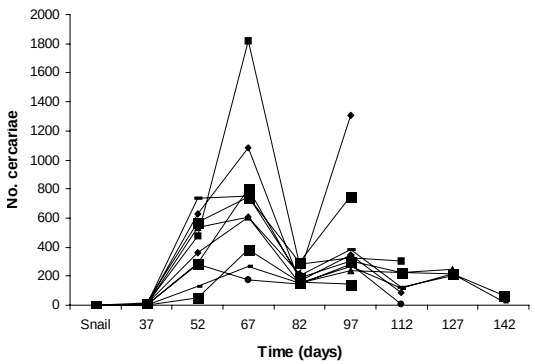

Fig. 1: graphic showing the dynamic of cercarial shedding along time. Only snails surviving the 97th day of infection and over were considered.

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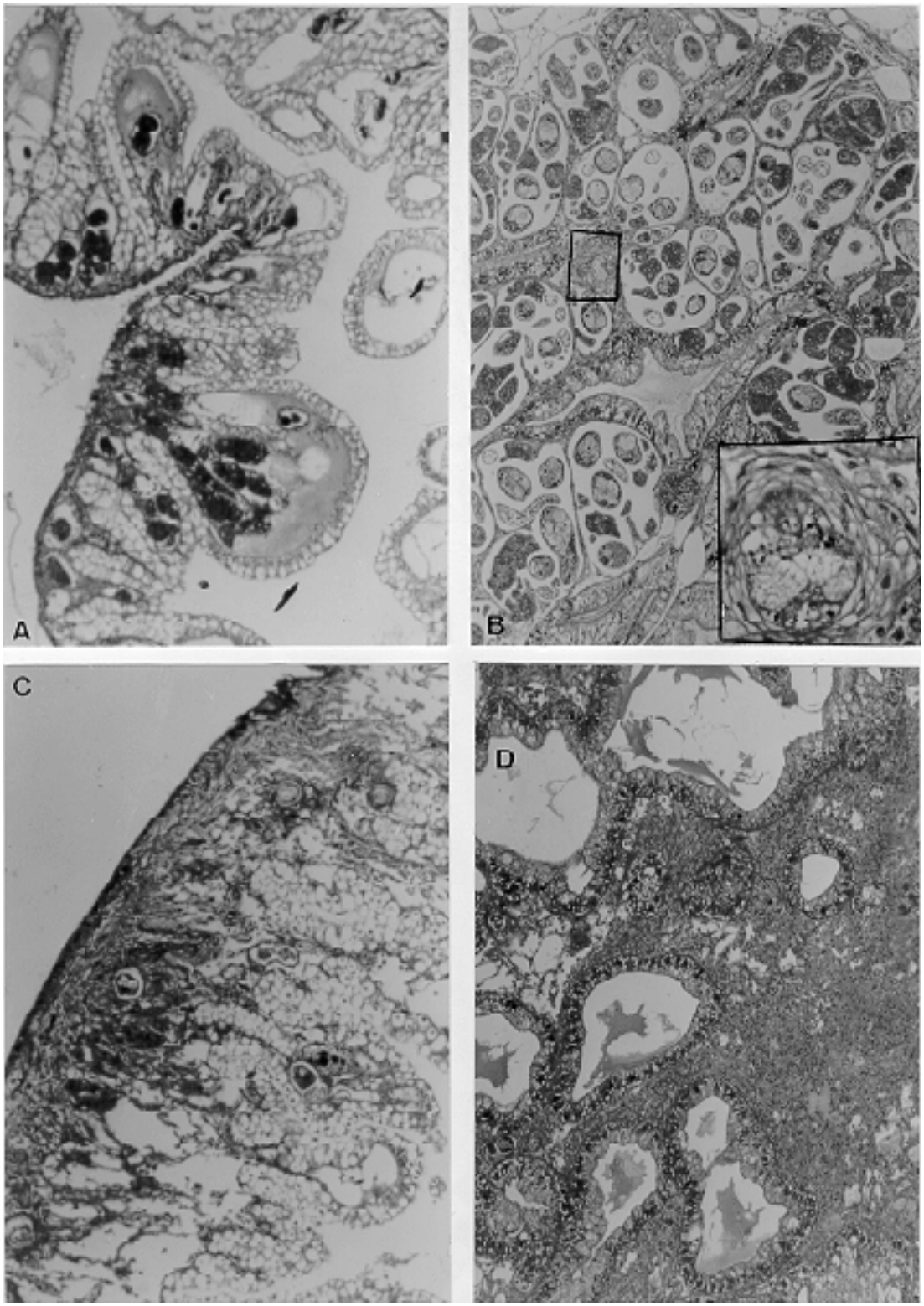

Fig. 2 - A: numerous multiplying sporocyst appear within the tubular portion of the kidney without any tissue reaction; B: snail shedding 1,047 cercarie on the 67th day of infection. Few focal reactions can be noted, as the one amplified in the insert; C and D: extensive hemocyte proliferation is seen around a few desintegrating parasites in the kidney and digestive glands, respectively at late infection. All sections stained with hematoxylin & eosin. Magnification 100X. Insert 400X

approximately the 67-82nd day, gradually decreasing toward the 142nd day. Up to the 30th post-infection day no cercarial shedding occurred, but histological examination disclosed numerous multiplying sporocysts at several locations, without tissue reaction. No cercarial differentiation was noted at that time (Fig. 2A). Up to the 67th day, the histological picture of the two snails examined at a time was similar, although the number of cercariae produced could range from 1,047 to 218. In both cases numerous developing sporocysts and cercariae were seen within several organs and tissues, accompanied by minimal focal accumulations of hemocytes around occasionally disintegrating parasites (Fig. 2B). Tissue reactions became more pronounced after the 82nd day, with the formation of several encapsulating lesions, centered by disintegrating sporocysts and cercariae. Toward the end of the experiment, these lesions had became disseminated, especially involving the renal region (Fig. 2C), digestive glands (Fig. 2D) and ovo-testis, while cercarial shedding proportionally decreased.

It is well recognized that, with time, an infected snail may gradually eliminate less and less cercariae. Two possibilities, not mutually exclusive, may explain this situation. The gradual exhaustion of the multiplication capacity of the parasite or the development of a host mechanism of defense, probably due to acquired immunity. The change in host reactivity during the course of infection may, immunopathologically, be interpreted as the development of a strong immunity against the parasite. Susceptibility or resistance of snails to *S. mansoni* has mainly been claimed to be dependent on host genetic background (Richards 1975a,b) rather than on acquired immunity. Although the capacity to develop acquired immunity may ultimately be related to genetic background, further studies similar to this one, along the lines of infection kinetics in snails susceptible to *S. mansoni*, may help to better define the role played by, and the factors involved with, acquired immunity in host-parasite relationship.

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