RELEVANT PROBLEMS TO THE EPIDEMIOLOGY OF MANSONIC SCHISTOSOMIASIS

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Field observations and laboratory experiments have provided new perspectives to deal with mansonic schistosomiasis and improved knowledge of the biologic cycle of Schistosoma mansoni.

Four problems will be presented on this paper: 1) competitive exclusion between snail species vectors of S. mansoni; 2) inter-specific antagonysm between larvae of digenetic trematodes in the body of vector snails; 3) miraxonal chemotaxis atraction; 4) amoebocytic activity and humoral mechanisms observed in the course of studies of resistance of snail to S. mansoni infections.

- 1 The exclusive competition between snail species has been described in the Brazilian technical literature to an extent that evidences its epidemiological importance. L. A. Magalhães (1966, Folia Clin. Biol., 35: 102-120) studied the introduction of a population of Biomphalaria glabrata originated from Aracaju (Sergipe, Brazil) on grounds of Instituto Oswaldo Cruz (Rio de Janeiro, Brazil). W. L. Paraense (1970, in Cunha, : Esquistossomose mansônica, São Paulo, Editora da USP: 13-20) described a population of B. tenagophila which displaced, in a period of five years, the original B. glabrata population in Belo Horizonte (Minas Gerais, Brazil). Other references to exclusive competition include: R. M. Andrade (1972, Ciência e Cultura, 24: 375); F. S. Barbosa (1973, Malacologia, 14: 401-408); and U. Kawazoe (1980, Rev. Bras. Saúde Publ., São Paulo, 14: 65-87). Since the Brazilian snail vector species show different susceptibility to infection, the importance of the exclusive competition stands out.
- 2 With regard to the inter-specific antagonism between larvae of distinct digenetic trematodes observed in the bodies of snail vectors, there is reference to two distinct types: predation (rediae destroy sporocystes or other rediae) and other indirect mechanisms yet to be explained (P. F. Bash et al., 1969, J. Parasitol., 55: 753-758; H. Lim et al., 1972, Adv. in Parasitol., 10: 191-269). S. M. P. Machado et

- al. (1985, Resumo do XXI Congresso Soc. Bras. Med. Trop., 56: 62) found that snails previously infected with larvae of other digenetic trematodes became, either partially or totally, immune to infection by S. mansoni. On conclusion, one may conceive the use of this antagonism on the control of the endemic.
- 3 Miraxonal chemotaxis has been well studied by several workers. Among them, one can refer to E. Chernin (1970, J. Parasitol., 56: 287-296) and B. C. Brasio et al. (1985, Rev. Saúde Publ., São Paulo, 19: 18-27). Some related phenomena important from the epidemiologist point of view are: a) on two strains of S. mansoni, namely SJ and BH strains, it was observed that the snails atract more intensely the simpatric miracidea; b) on the simultaneous presence of the two vector snail species (B. glabrata and B. tenagophila) the miracidea of the SJ and BH strains lose their ability to discriminate among the host species; in such a case, miracidea of both strains are more atracted by B. glabrata snails; c) SJ strain miracidea atraction to their host (B. tenagophila) is more noticeable in low lighting conditions; the opposite happens with BH strain miracidea.
- 4 Lastly, concerning the amoebocytic activity, J. V. Santana et al. (1985, Resumos do XXI Congresso Soc. Bras. Med. Trop., 58: 63), among other workers, suggest that the phagocytic capability of amoebocytes is activated when snails are infected by S. mansoni. There is evidence, however, that the mechanism of snail resistance to infection is not solely based on the amoebocytic activity: one observes the total destruction of sporocystes of S. mansoni on snails infected concomitantly with other digenetic trematodes, with lack of any amoebocytic reaction (D. S. L. Balan et al., 1986, Resumos da XXXVI Reunião da Soc. Bras. Prog. da Ciência, 991: 25-G.L.9.). These observations reinforce interest on the study of mechanisms of resistance to infection, on the amoebocytic activity and on the humoral mechanisms on vector snails (T. Sminia et al., 1987, Dev. Comp. Imunol., 11: 17-28).