Variations in infectivity for *Biomphalaria glabrata* in strains of *Schistosoma mansoni* from the same geographical area

CHARLES S. RICHARDS [1]

Abstract

Genetic differences in infectivity for intermediate snail hosts in two strains of S. mansoni *from the same geographical area and within a single isolated parasite population are reported. The importance of recognizing the potential for such genetic variation is stressed.*

Host–parasite specificity is one of the basic concepts in studies on host–parasite relationships. Owing to various factors, the parasite is typically limited to one or a few host species. Furthermore, it is becoming increasingly evident that, owing to intraspecific genetic variations in parasite infectivity and host susceptibility, the successful host–parasite relationship may be limited at a subspecific level (*6*).

Variations in infectivity for *Biomphalaria glabrata* in strains of *Schistosoma mansoni* from different geographical locations have been reported by several investigators (*2*, *3*, *4*). Anderson & Cheever (*1*) reviewed other reported variations in *S. mansoni* strains. There has been a tendency to consider each geographical strain of *S. mansoni*, such as " Puerto Rican *S. mansoni* ", as a distinct strain typical of that geographical area. The purpose of this note is to stress the importance of defining host and parasite strains more precisely and considering the potential for genetic variations in analysing experimental results.

Three strains of *S. mansoni* and two substrains are compared here as examples of variability. A Puerto Rican strain, designated as NIH-Sm-PR-1, has been maintained at the National Institutes of Health (NIH) for over 30 years with numerous generations alternating between mice and *B. glabrata*. Infectivity for the NIH-Bg-BPR-M colony of *B. glabrata* has been relatively consistent throughout this period. A strain of *S. mansoni* of Puerto Rican origin, isolated from the stool of a patient at NIH in 1975, is designated as NIH-Sm-PR-2. A strain of *S. mansoni* obtained in 1967 (*5*) from St Lucia—NIH-Sm-L-1—has since been maintained in the laboratory at NIH. The results of exposures of genetically selected stocks of *B. glabrata* to this *S. mansoni* strain suggested that it was heterogenic for infectivity. Selection through several generations resulted in two substrains: NIH-Sm-Lt-1—more infective—and NIH-Sm-Lc-1, which is less infective than the parent strain (*6*). Infectivity differences in strains of *S. mansoni* for clonal stocks of *B. glabrata* differing genetically in susceptibility (*6*) are indicated in Table 1. Exposures of juvenile and adult *B. glabrata* to the NIH-Sm-PR-1 strain of *S. mansoni*, with selection and controlled matings, resulted in clonal snail stocks of four different susceptibility types, arbitrarily designated as: I, refractory at any age; II, juvenile susceptible/adult refractory; III, susceptible at any age; and IV, juvenile susceptible/adult variable in susceptibility. When snails of these four types were exposed to miracidia of *S. mansoni* strains differing genetically in infectivity from the NIH-Sm-PR-1 strain, additional differences in *B. glabrata* susceptibility were revealed. Table 1 concerns snails of nine susceptibility subtypes, as differentiated by their susceptibility combinations.

Although the separation of two substrains differing genetically in infectivity from a single strain of St Lucian origin was achieved by experimental selection, this suggests that a change in infectivity may occur, in either a laboratory or a field parasite population, as the result of a shift in gene frequencies. The two Puerto Rican strains represent an extreme difference in infectivity in two isolations from the same general geographical area. More extensive sampling will be required to determine if this reflects a difference in strains from various localities in Puerto Rico or a more general change with time.

When one host species population is exposed to one parasite species population, with negative results, the conclusion that the host species is unsuitable is not necessarily valid. Yet the literature on host–parasite relations includes many such conclusions.

[1] Senior Investigator, Laboratory of Parasitic Diseases, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, MD 20014, USA.

Table 1. Infectivity of 3 strains and 2 substrains of *S. mansoni* for juvenile *B. glabrata* of 9 genetic susceptibility types

B. glabrata susceptibility types tested [a]	Number of clonal stocks	*S. mansoni* strains and substrains				
		NIH-Sm-PR-1	NIH-Sm-Lt-1	NIH-Sm-L-1	NIH-Sm-Lc-1	NIH-Sm-PR-2
I	2	0%	0%	0%	0%	0%
II	2	+ [b]	65%	6%	0%	0%
IIIa	1	+	60%	6%	0%	0%
IIIb	2	+	+	+	0%	0%
IIIc	1	+	(+) [c]	+	+	0%
III	4	+	+	+	+	+
IVb	2	+	(+)	+	0%	0%
IVc	2	+	(+)	+	+	0%
IV	1	+	(+)	+	+	+

[a] Snails exposed individually to 5 miracidia per snail.

[b] + indicates 80–100% infection (these host–parasite combinations represent essentially 100% infection potentials).

[c] (+) indicates 80–100% infections, suggested by limited data.

The potential for intraspecific genetic variation should be considered. Furthermore, the results obtained by different investigators on host–parasite relations can be expected to be at variance unless the host and parasite strains used are defined more precisely than on the basis of species and geographical origin.

ACKNOWLEDGEMENTS

The technical assistance of Mr Paul Shade and Mr Thomas Hallack is gratefully acknowledged.

REFERENCES

1. ANDERSON, L. A. & CHEEVER, A. W. *Bulletin of the World Health Organization*, **46**: 233-242 (1972).
2. BARBOSA, F. S. & BARRETO, A. C. *Experimental parasitology*, **9**: 137-140 (1960).
3. FILES, V. S. *Parasitology*, **41**: 264-269 (1951).
4. FILES, V. S. & CRAM, E. B. *Journal of parasitology*, **35**: 555-560 (1949).
5. LEE, H. G. ET AL. *Bulletin of the World Health Organization*, **45**: 147-155 (1971).
6. RICHARDS, C. S. *Symposia of the British Society of Parasitology*, **14**: 45-54 (1976).