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Host-Parasite Relationship of *S. mansoni* and *B. glabrata*

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Abstract: Experiments were conducted to study the host-parasite compatibility of various isolates of *Biomphalaria glabrata* snail and *Schistosoma mansoni* parasite isolates. A series of experiments conducted on 12 *S. mansoni* isolates have shown a range of infectivity potential for *B. glabrata* snail and 9 isolates of *B. glabrata* were found differentially susceptible to infection with *S. mansoni trematode* parasite.

Key words: *S. mansoni*, *B. glabrata*, host-parasite relationship

INTRODUCTION

The trematode-snail relationship represents a dynamic and intimate interplay between two metazoan species. The complexity of host-parasite relationship between trematodes and molluscs makes it difficult to generalize about the infectivity of parasite for a species of snail or about the susceptibility of a snail species to a particular parasite isolate. Investigators studying host-parasite relationship between *B. glabrata* and *S. mansoni* have reported differences in susceptibility to infection between various geographic stocks and hybrids of *B. glabrata* and differences in infectivity between various geographic strains and hybrids of *S. mansoni* (Files and Cram, 1949) with an Egyptian strain and hybrids of *S. mansoni* (Barbosa and Barreto, 1960) with several Brazilian stocks of *B. glabrata*.

Susceptibility and infectivity can be defined only with reference to strains of parasite and host and the overall relationship must be thought of as compatibility. A particular strain of parasite may show a high level of infectivity for certain populations of its normal snail host, a lower level of other populations and others may be completely resistant to that strain of parasite but susceptible to other strain (Wright, 1971).

Susceptible intermediate host has been described as a snail which provides a suitable habitat and are supportive of the metabolic needs of the parasite and susceptibility can be defined as the ability of snail to attract a miracidia and to allow penetration and establishment of a patent infection. However individual of same species show remarkable variation in their level of susceptibility to schistosome parasites. It has been shown that not all individuals of the compatible species of snail are susceptible to infection, hence susceptibility to infection is thought to be an inherited character; and it is largely regulated by genetic factors (Newton, 1953).

Many research workers have demonstrated that susceptibility of the intermediate host to infection is influenced by number of factors. These include the parasite's infectivity genes, in combination with the genes of the snail that determine susceptibility, the host age and the environment (Malek, 1961). The reproductive state of the snail at the time of infection also determines infection success (Raymond, 1991). Therefore, a number of factors may be operating at once to determine the susceptibility of the snail. These factors could be more easily described as forming two barriers to infection; firstly a barrier to penetration and secondly a developmental barrier (Lo, 1972). However, in addition to molluscan susceptibility, when an actively swimming miracidium comes into contact with a potential host, it's ability to produce a viable infection in the snail will also depend upon the parasite's own infectivity potential (Fryer and Bayne, 1990).

Whatever the governing factors affecting susceptibility to infection, be it age, size or reproductive status, a main consideration of such investigation must be how susceptibility should be measured?. Most workers record percentage infection in a group and record cercarial output, though a further indication of how susceptible a snail is to infection is also it's ability to support a long-term mature infection (Frandson, 1979). Indeed long-term infection even with a low daily output of cercariae may be

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very important in the successful passage of the parasite to a new host. It would also have important implications in the epidemiology and control of the disease.

MATERIALS AND METHODS

While this study commenced, there were 9 *B. glabrata* snail and 12 *S. mansoni* parasite isolates in laboratory culture. The main aim was to study the host-parasite relationship by testing the ability of different *S. mansoni* isolates to infect a range of *B. glabrata* snail lines. The test included those on some parasite lines which had been passaged exclusively in a single snail isolate for many years. The availability of these host-parasite pairs, which might be considered as experimental equivalent of sympatric combinations, allowed to test whether a parasite isolate which has been continuously passaged in a single snail line was better able to infect snails of that particular type than snails of any other isolated line.

The infection of snail host and screening for cercarial counting was performed as described by Arijo *et al.* (2001).

Fifty to seventy snails of each of the *B. glabrata* isolates were harvested from breeding tanks when 6-8 mm diameter and kept in 12 L of water in plastic aquarium tanks. When harvested, snails of the pigmented lines (i.e., the majority) were mixed with an approximately equal number of albino coloured Bg-PR snail line. When the mixed snails had grown to 10-12 mm they were mass-infected twice with miracidia hatched from liver eggs of heavily infected mice, each snail being exposed to approximately 50-100 miracidia (Table 1).

Approximately 35 days after infection the snails were screened for patency. The number of snails shedding cercariae was scored as a percentage of the total number of snails screened, with results for pigmented and albino snails being scored separately.

The mixing of albino Bg-PR snails with batches of other snails that were pigmented allowed the former snails to be used as a standard or marker population against which performance of the pigmented snail isolate could be compared under the same conditions of infection and laboratory mice. Pigmented Bg-Br snails were similarly used as a within tank control to monitor the performance of Bg-Cmp snail line. More than 800 batches of snails were processed, with nearly all the snail lines having been infected with each of the parasite isolate at least three times.

Table 1: The susceptibility of snail isolates and the infectivity of parasite isolates

Parasite isolates	Snail isolates: Bg-swan parasite	Bg-PR	Bg-BH	Bg-Gp	Bg-Br	Bg-Sen	Bg-Eg	Bg-Abrd	Bg-Cmp	Mean
Sm-Eg	3	22	3	8	8	3	9	3	5	
	96±5	94±8	92±4	90±9	86±10	88±4	97±3	77±24	16±20	82
Sm-Br	4	26	3	7	10	4	5	4	4	
	97±5	95±6	86±5	94±6	91±8	94±4	89±11	79±15	5±3	81
Sm-PR	3	46	10	15	20	8	7	4	13	
	98±2	97±5	96±6	94±6	94±5	91±6	89±11	68±38	11±17	81
Sm-Ken A	3	31	3	8	7	3	5	4	3	
	85±24	97±5	91±2	91±7	94±3	86±6	93±3	87±9	7±12	81
Sm-Gp	4	32	3	40	18	5	9	6	11	
	88±7	95±10	75±28	93±6	95±6	81±23	92±3	68±16	4±4	77
Sm-Sen245	3	19	3	9	8	4	4	4	4	
	96±4	96±8	73±18	80±11	89±5	93±3	82±23	67±7	8±8	76
Sm-Sen-old	3	39	4	10	7	17	4	3	3	
	84±16	94±8	77±16	78±13	88±12	85±13	91±7	69±31	3±3	74
Sm-Cmp	3	15	3	6	10	5	3	3	11	
	88±5	82±23	91±4	90±4	87±10	72±8	52±27	67±18	16±17	72
Sm-Abrd	4	21	3	7	3	3	3	8	3	
	94±10	82±11	72±13	63±16	59±15	53±8	65±17	61±13	3±5	61
Sm-Sen 47	3	19	4	7	3	5	3	3	3	
	97±4	88±15	69±20	58±26	48±28	59±38	58±25	58±15	1±2	60
Sm-BH	3	25	12	9	4	3	5	3	3	
	93±2	78±26	58±20	59±16	59±25	64±20	46±27	55±28	2±3	57
Sm-Ken-B	5	21	4	9	6	5	4	4	3	
	45±25	49±28	51±30	32±18	35±17	26±33	26±32	27±13	1±2	32
Mean	88	87	78	77	77	75	73	65	6	

RESULTS

Table 1 shows the mean percentage±SD of the snails with patent infections in the 9 snail lines which had been exposed to miracidia of each of the 12 *S. mansoni* isolates. The snail lines are arranged in columns across the Table 1 with the snails showing the highest susceptibility to *S. mansoni* infection (in terms of mean percentage patency) to the left. Similarly, the parasite lines are arranged in rows, with these showing the highest mean infectivity at the top and lowest infectivity at the bottom. The results in this table suggest that this group of snail lines can be ranked in order of their susceptibility to schistosome infection and that the parasites can similarly be ranked in order of their infectivity for the snails. It is apparent that the Bg-Cmp snail line was significantly resistant ($p<0.000$) to infection by any of these parasite lines when compared with other snail lines. The Sm-Ken B parasite however, was found consistently less infective than all other 12 parasite lines.

Table 2 shows the infection of Bg-Cmp snail and pigmented *B. glabrata* control snails with 12 *S. mansoni* isolates. Bg-Cmp snail line was found significantly different ($p<0.0001$) form their pigmented control snails in terms of mean percentage of snail infected and mean number of cercariae per infected snail except for a non-significant difference in mean number of cercariae when infected with Sm-Ken A.

Table 3 shows the infection of Bg-Abrd snail line when mixed with Bg-PR as in-tank control. Infection with 12 *S. mansoni* isolates shows that when infected with Sm-Gp, Sm-Sen-245, Sm-Abrd and Sm-Sen-47 parasite isolates, Bg-Abrd snail line was significantly different ($p<0.0003$, 0.0086, 0.0001 and 0.0115, respectively) from their Bg-PR control snail line in terms of percentage snail infected. However a significant difference was seen in terms of mean number of cercariae per infected snail when Bg-Abrd snail line was compared with Bg-PR control line after infection with Sm-Eg, Sm-Gp, Sm-Sen Old and Sm-Abrd parasite isolates. The Table 3 shows that Bg-Abrd snail was more susceptible to most of the parasite isolates except for its own sympatric isolate.

Table 4 shows that of 12 *S. mansoni* isolates tested, Bg-BH snail line was significantly different ($p<0.05$) from its Bg-PR control snail in terms of % infected snail after infection with Sm-Ken A parasite isolate and a significant difference ($p<0.18$) was seen in terms of mean number of cercariae per infected snail when infected with Sm-Gp

Table 2: The infection Bg-Cmp snail together with intake pigment controls

		Bg-Cmp					Pigmented control		
Parasites isolate	No. of beachs	Mean No. of snail	Infected snail (%)	p	Mean cercriae/ infected snail	p	Mean No. of snail	Infected snail (%)	Mean cercriae/ infected snail
Sm-Eg	5	48±4	16±20	0.0001	302±311	0.0033	42±7	88±12	2127±937
Sm-Br	4	35±4	5±3	0.0001	138±103	0.0164	32±8	89±10	3086±1784
Sm-Pr	13	36±7	11±17	0.0001	182±289	0.0001	38±10	88±18	2776±1784
Sm-Ken A	3	34±3	7±12	0.0003	200±1346	0.0784	35±11	94±3	2101±387
Sm-Gp	11	33±7	4±4	0.0001	474±705	0.0001	34±10	94±7	2994±1384
Sm-Gp 245	4	36±15	8±8	0.0001	1246±1382	0.0437	28±5	90±5	6277±3801
Sm-San old	3	39±2	3±3	0.0005	100±0	0.0001	39±19	84±18	761±3801
Sm-Cmp	12	35±11	16±17	0.0001	769±751	0.0034	33±5	77±18	2515±1682
Sm-Abrd	3	28±6	3±5	0.0001	33±58	0.0001	40±2	63±10	2272±884
Sm-Sen 47	3	39±16	1±2	0.0001	33±58	0.0001	34±6	60±32	2080±871
Sm-BH	3	38±17	2±3	0.0001	58±10	0.0001	42±14	42±14	1396±845
Sm-Ken-B	3	38±10	1±2	0.0001	33±58	0.0001	27±5	43±17	977±712

Table 3: The infection of Bg-Abrd snail together with intake Bg-PR controls snails

		Bg-Abrd					Bg-PR		
Parasites isolate	No. of beachs	Mean No. of snail	Infected snail (%)	p	Mean cercriae/ infected snail	p	Mean No. of snail	Infected snail (%)	Mean cercriae/ infected snail
Sm-Eg	3	34±16	77±24	NS	1120±790	NS	29±7	81±7	2055±1360
Sm-Br	4	46±5	79±15	NS	1022±922	NS	39±10	97±3	2738± 1747
Sm-Pr	4	31±9	68±38	NS	918±553	NS	40±10	86±24	2045±1128
Sm-Ken A	4	40±6	87±9	NS	1215±584	NS	29±12	93±9	2435±825
Sm-Gp	6	34±9	68±16	<0.001	1114±595	<0.02	28±5	99±2	2970±1417
Sm-Gp 245	4	45±13	67±7	<0.01	1154±595	NS	27±2	92±11	2553±1139
Sm-San old	3	36±15	69±31	NS	877±104	<0.05	27±2	95±5	1973±596
Sm-Cmp	3	46±12	67±18	NS	1271±884	NS	26±10	92±5	2619±1120
Sm-Abrd	8	39±8	61±13	<0.001	1430±390	<0.05	33±5	88±7	2203±829
Sm-Sen 47	3	43±20	58±15	<0.02	890±453	NS	30±6	97±3	2322±1648
Sm-BH	3	35±4	55±28	NS	726±114	NS	35±2	76±30	2313±1802
Sm-Ken-B	4	49±9	27±13	NS	548±481	NS	33±2	54±21	1316±546

Table 4: The infection of Bg-Abrd snail together with intake Bg-PR controls snails

		Bg-BH					Bg-PR		
Parasites isolate	No. of beachs	Mean No. of snail	Infected snail (%)	p	Mean cercriae/ infected snail	p	Mean No. of snail	Infected snail (%)	Mean cercriae/ infected snail
Sm-Eg	3	48±4	92±4	NS	2238±365	NS	42±7	96±7	2126±513
Sm-Br	2	40±20	85±5	0.01	2564±1055	NS	24±5	96±4	2243±1125
Sm-Pr	10	28±7	96±6	NS	2247±1189	NS	26±8	98±3	2201±1153
Sm-Ken A	3	25±17	91±2	<0.01	2881±1357	NS	26±2	99±2	2523±973
Sm-Gp	3	51±5	75±28	NS	1305±685	<0.01	37±12	99±2	2956±289
Sm-Gp 245	3	43±9	73±18	NS	3572±579	NS	38±25	91±10	3903±1987
Sm-San old	4	42±10	77±16	NS	1436±953	NS	28±14	97±4	2375±1036
Sm-Cmp	3	34±11	91±4	NS	3462±1020	NS	30±6	90±8	2071±290
Sm-Abrd	3	33±3	72±13	NS	2480±903	NS	31±7	87±3	2176±611
Sm-Sen 47	4	46±11	69±23	NS	2686±1987	NS	28±15	90±9	2831±1469
Sm-BH	12	33±2	58±20	<0.05	1703±1039	NS	35±8	77±200	1642±949

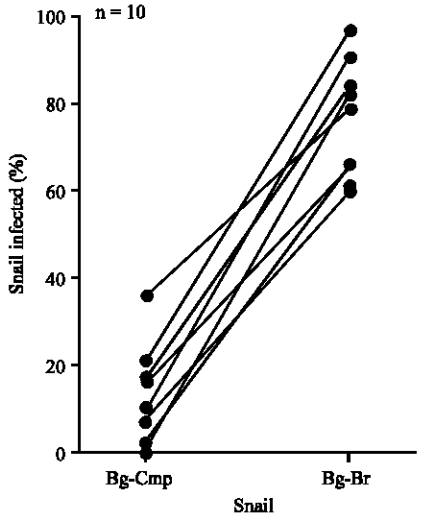

Fig. 1: Susceptibility of Bg-Cmp with its pigments control snails

isolate. It is apparent from this Table that Bg-BH snail line was more susceptible to most of the parasite lines than to its own sympatric snail.

Figure 1 shows the susceptibility of Bg-Cmp together with its pigmented control snails when infected with its sympatric Sm-Cmp isolate. Figure 2a, b show the susceptibility of Bg-Abrd snail line when infected with its sympatric parasite isolate i.e., Sm-Abrd and Bg-BH when infected with Sm-BH isolate. For both snail lines, Bg-PR served as in-tank control. These figures show that in each of these cases, the sympatric parasite lines could not infect higher percentage of their sympatric snails when compared with their allopatric control snails.

Figure 3 shows that even after 11 successive cycles, the Sm-Cmp parasite could not adapt to its sympatric snail. Of 11 batches, only one batch was found with higher patency. Figure 4 although, shows a slight adaptation of Sm-Abrd parasite isolate to Bg-Abrd snail line, but Fig. 5 shows that Sm-BH parasite isolate could not adapt to its sympatric snail. Figure 6 shows that as

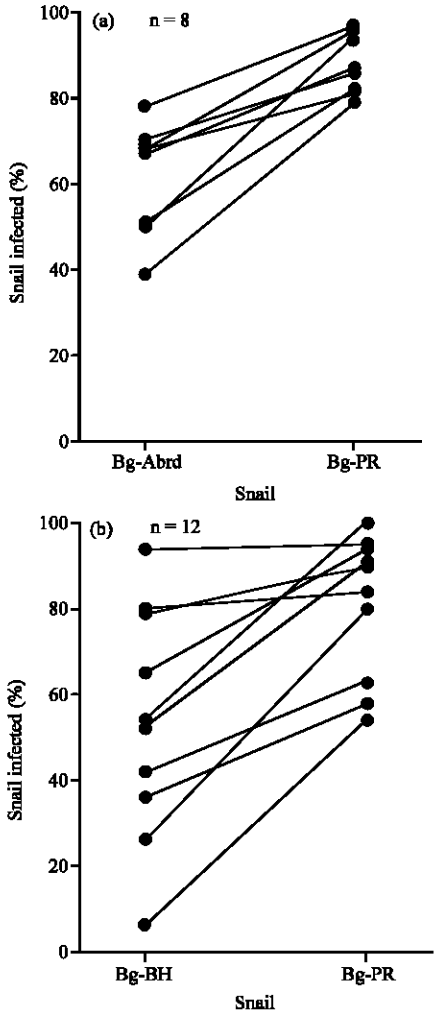

Fig. 2: (a) Susceptibility of Bg-Abrd with pigment control snails and (b) Susceptibility of Bg-BH with pigment control snails

result of 8 successive cycles, Sm-Ken B parasite isolate has adapted to Bg-Gp snail isolate, but this is not a sympatric partnership.

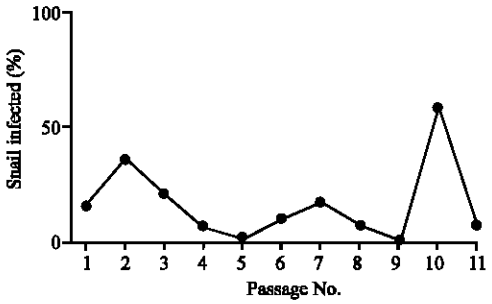

Fig. 3: Successive cycle of Sm-Cmp in Bg-Cmp isolate

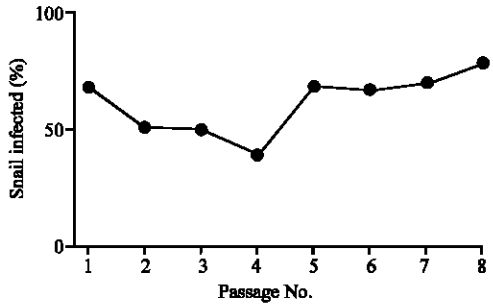

Fig. 4: Successive cycles of Sm-Abrd isolate in Bg-Abrd isolate

Fig. 5: Successive cycles of Sm-Bh isolate in Bg-Bh isolate

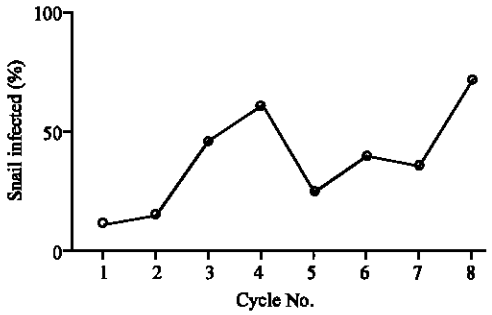

Fig. 6: Infectivity of Sm-Ken B in successive cycles in Bg-GP

DISCUSSION

Bash (1974) and Donges (1974) introduced an idea that snail populations are homogenous with respect to infection by larval trematodes. The ability of miracidia to penetrate a snail host can very greatly depending on the host-parasite combinations. It has been shown that miracidia will infect both compatible and incompatible snails, although they fail to develop further in later.

The results presented herein confirm the genetic heterogeneity in the relationship of *S. mansoni* and *B. glabrata* strains. We have found (Table 1) that snails from different geographical regions were different in terms of their susceptibility to different geographical isolates of *S. mansoni*. Likewise, different isolates of *S mansoni* were different in their infectivity for snail lines under our controlled laboratory culture conditions.

On one hand we found a snail (Bg-Cmp for example) consistently resistant to 12 *S. mansoni* isolates from different geographical locations and a snail (Bg-Abrd for example) consistently displayed intermediate level of resistance and some snails were found highly susceptible (Bg-PR, Bg-Eg and Bg-Br Bg-Gp, Bg-BH and Bg-Sen for example). On the other hand, we found (Table 1) that parasite from different geographical areas differ in their infectivity, some being highly infective (Sm-PR, Sm-Br, Sm-Ken, Sm-Sen, Sm-Sen-245 and Sm-Gp for example) and some less infective (Sm-Ken B, Sm-BH and Sm-Sen-47 for example). This we think is due to variations in the genetics of snail lines and also due to the variations in the environment of the area these snails originally belong to.

The susceptibility of 9 snail isolates to infection with 12 parasite isolates did not appear to change during under study period of laboratory culture, each of the parasite lines similarly displayed relatively unchanging level of infectivity in successive passages.

In particular, the ability of parasite to infect a particular resistant snail host did not appear to increase as a result of successive passages exclusively through that snail during this period of experimentation. Figure 1 shows that the percentage of Bg-Cmp when mixed with pigmented snails and exposed to Sm-Cmp isolate in the same tank, respectively became patently infected in successive passages. Despite cercariae from Bg-Cmp snails being used exclusively to transmit this parasite line into passage mice, the parasite has shown no propensity to become adapted to this snail host. In fact, as Table 1 demonstrate, throughout this period of study, the Sm-Cmp isolate remained more infective for all the other snail lines exposed to it than for the snail in which it was being routinely passaged.

A similar conclusion can be drawn from Table 3 showing patterns of infectivity of the Sm-Abrd line, which was passaged exclusively in Bg-Abrd snails and Table 4 that shows the infection of Bg-BH snail line with Sm-BH parasite. It can be seen from these Tables that most of the parasite lines were more infective to these snails than their sympatric parasite isolates. These results suggest that passage through a sympatric snail line does not result in the parasite becoming more infective for that snail.

There is a belief commonly held by parasitologists that if a parasite isolate has marked pathogenic effect upon its sympatric host, the association between the two is of relatively recent origin and that if there is little or no pathology then the association is more ancient. In any endemic area, the most important thing for the parasite is its survival. A parasite which does little damage to its host is unlikely to provoke much host response, but which interferes at harmful level will either eliminate the host (and therefore itself) or will elicit a more marked host response and will also stimulate selection in favour of resistant host.

Genetic variation in susceptibility of *B. glabrata* for infection with *S. mansoni* has been reported to occur between snail populations in different geographic areas, between snail populations in the same area, between individuals in the same population and at different ages in the same individual (Richards, 1984). Variation in infectivity for *B. glabrata* in strains of *S. mansoni* from different geographical localities have been reported by several investigators (Barbosa and Barreto, 1960; Files and Cram, 1949; Lee *et al*., 1971).

Several different studies have shown that parasites are more infective to sympatric hosts than to allopatric hosts of the same species (Morand *et al*., 1996). The incompatibility between snail and parasites of different endemic areas has been explained on the basis of inter-specific and intra-specific differences between snails in respect of the physiological factors that are responsible for the development of the parasite and physiological differences between strains of the parasites.

The *B. glabrata* from Puerto Rican for example, was found by Files and Cram (1949) to be highly susceptible to Puerto Rican and Brazilian isolate of *S. mansoni* but only 9% susceptible to Egyptian strain of *S. mansoni.* Another snail from Brazil was found susceptible to Brazilian and Puerto Rican isolates of *S. mansoni* but not to the parasites from Venezuela and Egypt.

Present results appear to be contradictory with the above findings of Files and Cram (1949). Our data (Table 1) indicate that a parasites that does not perform well in its sympatric snail, can infect higher percentage of allopatric snail lines. This may be due to availability of relatively more favourable physiological environment for that particular strain of the parasite. Present findings thus are not in agreement with the conclusion of Sturrock and Sturrock (1970) who advocate the idea that a local strain of snail and the local parasite they are infected with, should be better adapted.

We agree with the Frandson (1979) who tested the susceptibility of 7 snail lines to 3 isolates of *S. mansoni* from distant geographical origin and concluded that it is not always the rule that the local combination of *S. mansoni* and *B. glabrata* sp., are the most compatible.

Latest work of Maning *et al.* (1995) using *B. globusus* snails and *S. haematobium* has been published to show that sympatric parasite-host combinations are more compatible than allopatric combinations. But, Vera *et al.* (1990) could not find any better sympatric host-parasite compatibility with *B. trancatus* snail and *S. haematobium* parasite.

Present results (Table 2-4) also are suggestive of the idea that the sympatric association is not always more compatible. It can be seen that Sm-Abrd, Sm-BH and Sm-Cmp parasite lines were more infective to other snail lines than their sympatric snail (Table 1). Present data (Table 1-3 and Fig. 4-6) indicate that the susceptibility of snail lines and infectivity of parasite isolates remain unchanging, however susceptibility of snail and infectivity of parasite may be altered by selective breeding and that it is relatively easy to establish an infection-susceptible snail line from a highly resistant snail such as Bg-Cmp.

The complexity of the host-parasite relationships between the *S. mansoni* isolates and its snail host makes it difficult to generalize about the infectivity of a particular strain of the parasite for a strain of snail or about the susceptibility of a snail strain to a particular parasite isolate. Either of these terms can be defined only with reference to strain of parasite and races of host and the overall relationship must be thought of as compatibility. A particular strain of parasite may show a high level of infectivity for certain populations of its normal snail host, a lower level for other populations and others may be completely resistant to that strain of parasite but susceptible to other strains (Wright, 1971). Even within a suitable host strains, there is known to be variability in the compatibility between the trematode and snail.

This compatibility acts such that in compatible interactions the parasite recognises, penetrates and develops within the snail, giving rise to the parasite's next infective stage, the cercariae. Alternatively, in incompatible interaction, the larval trematode either fails to recognise and penetrate the snail, or penetrates and is

recognized as non-self and is destroyed by the snail's internal defence system (Van der Knapp and Loker, 1990).

Under field conditions, the susceptibility to infection has been reported to be affected by the environmental conditions such as humidity, temperature and differential feeding behaviour etc. The present study was conducted under controlled laboratory conditions and all snail batches were treated with same standard protocol.

Genetic differences in the infectivity of parasite isolates are thought to be the result of geographic adaptation (Files and Cram, 1949; Mulvey and Vrijenhoek, 1982) to snails. As the result of this study, we conclude that the susceptibility and infectivity are governed by two distinct genomes, i.e., those of the host and parasite respectively and the interaction between them. Data in Table 1 suggests that in different snail and parasite isolates, a wide-ranging degree of snail susceptibility to infection and parasite infectivity may exist.

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