

Original article

Host sex and parasite genetic diversity

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Abstract

Is the genetic diversity of parasites infecting male and female hosts equal or different? This is the question we address in this paper by studying the neutral genetic variability of the plathyhelminth trematode *Schistosoma mansoni* within males and females of its natural murine host *Rattus rattus* in the marshy forest focus of Guadeloupe (French West Indies). Using seven microsatellite markers, we demonstrate that parasites from male hosts are genetically more diversified than parasites from female hosts. Three hypotheses are discussed that could explain this pattern: 1) a host sex-specific duration of cercariae recruitment; 2) a difference in the behaviour of male and female hosts that would lead to the exposure of males to a greater diversity of parasites; and 3) a host sex-biased immunocompetence that would lead to the selection of more genetically diversified individuals in male than in female rats. This finding is the first empirical evidence that each host sex may play different roles in the maintenance of parasite genetic diversity and so in their evolutionary dynamics and epidemiology.

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1. Introduction

Males and females can constitute different environments for parasites. One difference between sexes is their immunocompetence, that is their ability to resist parasites [1]. Males are, in general, less immunocompetent than females [1]. In addition to immunocompetence, body size [2], lifespan [3], patterns of foraging [4], social behaviour [5], mobility, home range and dispersal rates [6] can also differ between sexes and hence render male and female hosts different habitats for parasites with their own constraints and characteristics.

The sexual dimorphism of those traits may affect population dynamics of parasites. Parasite infrapopulation size [1] (an infrapopulation being defined as the group of parasites

of the same species present within one individual host) as well as demographic traits such as transmission rates often differ between host sexes [7,8]. Theoretically, host sex could also influence parasite genetics. This could arise, for instance, in host species displaying sex-biased dispersal rates (the rate of dispersal is higher in one sex than in the other), which is frequent in vertebrates [6]. Let's consider a population of parasites which infective forms are geographically and genetically structured, and a population of hosts with sex-specific dispersal. Individuals of the dispersing sex will sample parasites from a larger area than individuals of the philopatric sex and will therefore harbour parasite infrapopulations with higher genetic diversity.

The effect of host sex on the genetic variability of parasites has never been investigated (but see ref. [9]), although this question is important from both evolutionary and epidemiological points of view. This is especially true when attempting to understand how traits such as resistance to anti-parasitic agents arise and spread through a parasite population, since

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parasites from male and female hosts could potentially play different roles in the dynamic and diffusion of such resistance genes. Male and female hosts can also play different roles in the maintenance of the parasite genetic diversity and so in its evolutionary potential.

All these elements led us to ask a simple question, namely, is the genetic diversity of parasites infecting male and female hosts equal (different)? In this paper, we address this question by studying the neutral genetic variability of the plathyhelminth trematode *Schistosoma mansoni* within males and females of its natural murine host *Rattus rattus* in the marshy forest focus of Guadeloupe (French West Indies). Using seven microsatellite loci, we demonstrate that parasites from male rats are genetically more diverse than parasites infecting female rats. Hypotheses related to host sex-biased dispersal, home ranges, duration of cercariae recruitment and immunocompetence are discussed to interpret this result.

2. Materials and methods

2.1. Parasite life cycle

Schistosoma mansoni is a gonochoric trematode. Adult worms (males and females) typically reside in the mesenteric venules of the vertebrate host. They sexually reproduce and the eggs break out of the venules into the intestinal lumen, to be discharged into the faeces of the host. Faecal contamination of water results in eggs hatching into miracidia. The miracidia then infect the intermediate host (a gastropod mollusc) present in the water and transform into sporocysts. The sporocysts undergo several rounds of asexual reproduction that lead to the production of thousands of cercariae, a second free living motile larval stage, infective for the definitive vertebrate host.

2.2. Study sites

Sampling was performed in the marshy forest focus of Grande Terre Island, Guadeloupe. Rats were captured during the wet season in December 1996 using traps baited with coconut and deployed during five consecutive nights in two transmission sites (Dans Fond and Dubelloy). Rats were anaesthetized following international principles regarding the care and use of vertebrates. For each rat, adult schistosomes were recovered by a standard perfusion technique [10], carefully washed in physiological saline solution and stored in 70% ethanol after isolation of male and female worms. Ten rats (five males and five females) from Dans Fond and six rats (three males and three females) from Dubelloy carried a sufficient number of schistosomes (around 20 parasites of each sex) to perform meaningful genetic analyses.

2.3. Genotyping

DNA from a total of 808 worms collected from the 16 rats (see Table 1 for details about samples) was extracted using the method of Durand et al. [11]. Seven microsatellite loci were genotyped (Genbank accession number: *AF202965*,

Table 1

Host sex (SR), parasitic load of male (Lm) and female (Lf) schistosomes, number of male (Ngm) and female (Ngf) schistosomes genotyped in each infrapopulation at Dans Fond and Dubelloy and genetic diversity (*Hs*) measured for male (Hsm) and female (Hsf) schistosomes

	SR	Lm	Ngm	Hsm	Lf	Ngf	Hsf
Dans Fond							
R2	F	245	27 (21)	0.49	157	27 (19)	0.48
R5	M	71	27 (17)	0.54	47	27 (13)	0.52
R7	M	127	27 (21)	0.53	90	27 (16)	0.52
R8	F	255	27 (13)	0.51	124	27 (15)	0.48
R11	M	381	27 (15)	0.51	161	27 (18)	0.50
R13	F	285	27 (16)	0.47	123	27 (11)	0.43
R14	M	217	27 (7)	0.53	52	27 (10)	0.49
R16	F	76	27 (20)	0.49	67	27 (11)	0.44
R18	M	154	27 (12)	0.52	85	27 (16)	0.49
R19	F	132	27 (21)	0.45	73	27 (25)	0.49
Dubelloy							
R33	M	37	24 (12)	0.50	28	24 (19)	0.47
R34	F	296	24 (14)	0.45	97	24 (19)	0.47
R35	M	25	24 (13)	0.56	35	24 (13)	0.51
R37	M	161	24 (18)	0.50	68	24 (16)	0.45
R39	F	19	19 (9)	0.47	36	24 (12)	0.50
R40	F	17	17 (7)	0.57	17	16 (6)	0.43

Numbers between parentheses indicate the number of different multilocus genotypes present within each infrapopulation and kept for genetic analyses. R2 to R40 refer to analysed rats. F: Female; M: Male.

AF202966, *AF202967*, *AF202968*, *L46951*, *AF325695*, *AF325697*). Amplification was performed following Durand et al. [11] for loci *AF202965*, *AF202966*, *AF202967*, *AF202968*, *L46951* and Curtis et al. [12] for loci *AF325695* and *AF325697*. PCR amplification of each locus was conducted separately. All PCR products were run on an ABI Prism 310 Genetic Analyzer (Applied Biosystems, Foster City, California, USA).

2.4. Genetical analyses

Multiple infection of rats by several identical schistosome genotypes is possible [13] (Table 1), due to the intense clonal reproduction occurring at the sporocyst stage. This may affect estimates of genetic diversity [14]. In particular, we expect clonality to increase the variance of genetic diversity among infrapopulations and therefore to reduce our capacity and power to detect a difference between parasites from male and female rats given our sample size (only 8 male and 8 female rats).

As expected, no difference of genetic diversity was detected between samples with and without clones (Wilcoxon rank sum test, p -value = 0.56) but we observed a significant increase of variance in samples with clones (Bartlett's test of variance comparison, p -value < 10^{-4}). Therefore, the genetic diversity of schistosome infrapopulations was investigated once repeated multilocus genotypes were reduced to single copies within each infrapopulation. Before deleting multiple identical genotypes, we nevertheless ensured that our seven microsatellite loci were sufficiently discriminating to identify clones. For this purpose, we simply computed the multinomial probability of occurrence by panmictic sexual reproduction of

each multilocus genotype (MLG), given the allelic frequencies within each infrapopulation (P_{MLG}). From this can be derived the probability P of observing two or more individuals of a given genotype in a sample of size N using the following formula $P = 1 - (1 - P_{MLG})^N \approx N \cdot P_{MLG}$.

The genetic diversity of schistosomes was computed using Nei's unbiased mean heterozygosity (H_s) [15]. The unbiased estimator f of Wright's F_{IS} [16] (which measures the departure from panmixia within infrapopulation) was also computed over all loci according to ref. [17]. The hypothesis that f significantly differed from 0 (panmixia) was tested using a procedure of random permutations of alleles between individuals (15 000 permutations). The estimator θ of F_{ST} [17], which measures the difference in allelic frequencies between infrapopulations, was calculated over all loci in each sample (Dans Fond and Dubelloy). Genetic differentiation ($\theta \neq 0$) was tested using the G -based test after 15 000 permutations of individuals between infrapopulations. Estimates of 95% confidence intervals of F_{ST} values were obtained by bootstrapping over loci [18]. Linkage disequilibrium between all pairs of loci within each sample was also tested using random permutation of genotypes between individuals. Bonferroni corrections were applied to correct for multiple tests [19]. All parameter estimates and tests were performed using Fstat V. 2.9.3 (freely downloadable at <http://www.unil.ch/izea/softwares/fstat.html>; updated from ref. [18]).

2.5. Comparison of schistosome genetic diversity in male and female rats from 1996

2.5.1. Parasite and host sex effect on schistosome genetic diversity and deviations from Hardy–Weinberg: generalised linear models

Two generalized linear models with gaussian error and identity link function were fitted to test the effect of host sex (Sr) on parasite infrapopulation genetic diversity (H_s) and on deviation from Hardy–Weinberg expectations (F_{IS}) using SPLUS 2000 (Mathsoft, Inc. 1999) (One-sample Kolmogorov–Smirnov Test of Normality on residuals: for H_s : p -value = 0.92; for F_{IS} : p -value = 0.93). Since male and female schistosomes have been reported to display different genetic diversity within infrapopulations [13], we added schistosome sex (Ss) as a supplementary explanatory variable. The age of the host was also a variable to control for. Since it is difficult to estimate on wild-caught rats, we incorporated to the model the parasitic load (PL). This covariate is indeed likely to be positively correlated with the age of the host at least for the first years of infection [20] (note that H_s is unbiased, hence independent on sample size). At last, we included sample site (O) in the model, in order to investigate site-related effects. The initial complete model was therefore: H_s (or F_{IS}) \sim Sr + Ss + PL + O + first order interactions. A stepwise procedure was then used to select for the most parsimonious model. This stepwise process was undertaken by successively deleting and adding each variable from the general model using Akaike Information Criterion (AIC) to retain or exclude variables (SPLUS-Guide to

statistics, Mathsoft Inc, 1999). In the minimal model, the significance of the different variables was tested using F -tests (SPLUS 2000 guide to statistics, Mathsoft, Inc. 1999).

To ensure that the observed differences in heterozygosity (or F_{IS}) between infrapopulations from male and female rats (or between male and female schistosomes) did not result from the effect of only one or a subset of loci, another linear model was considered. In this second model we let the genetic diversity (or F_{IS}) observed at each of the seven loci be the response variable and the identity of the locus (Loc) be an additional explanatory variable. A locus-specific effect should translate into the interactions Sr: Loc or Ss: Loc to be retained and significant.

Parasitic load is likely to vary between host and parasite sexes. Such effects would be useful to interpret the results of the previous models. We thus investigated the relationships between the sex of the rat, its parasitic load and its parasite sex ratio (defined here as number of male parasites divided by number of females) using generalised linear models in which the response variable was successively the parasitic load and the parasitic sex ratio.

2.5.2. Parasite and host sex effect on schistosome genetic diversity and deviations from Hardy–Weinberg: randomisation approaches

In order to confirm results obtained using the generalised linear models, we used randomisation procedures, which do not assume a particular distribution of data. As no difference was found either between the average genetic diversity of parasites or F_{IS} from Dans Fond and Dubelloy (see Section 3), randomizations were performed over the entire dataset to ensure maximum statistical power.

The effect of host sex on the infrapopulation genetic variability of *S. mansoni* and deviation from Hardy–Weinberg (F_{IS}) was tested using a permutation procedure that allows comparing the genetic structure of different groups of infrapopulations. Under the null hypothesis of no host sex effect, *S. mansoni* H_s (or F_{IS}) computed within infrapopulations of male hosts (denoted H_s^M and F_{IS}^M) is expected to be equal to H_s (or F_{IS}) computed within infrapopulations of female hosts (denoted H_s^F and F_{IS}^F). Therefore, the difference between the observed statistics of each group (e.g. $H_s^M - H_s^F$ or $F_{IS}^M - F_{IS}^F$) was first computed. Then infrapopulations were permuted between groups a large number of times (here 5000 times), thus randomly creating new groups of infrapopulations. For each of the 5000 randomized dataset, the difference between the statistics of each group was computed. The p -value of the test was derived as the proportion of randomized data sets giving an equal or larger difference than the observed one. These randomization procedures were implemented using Fstat.V.2.9.3.

2.6. Comparison of schistosome genetic diversity and deviations from Hardy–Weinberg in male and female rats from 1997

To ensure that the observations made in 1996 were not artefactual, we re-analysed the dataset used in ref. [13] where 3

male and 3 female rats were sampled in DFO in 1997. Analyses were made on male and female schistosomes separately. Given the low sample size, generalised linear models could not be used. The difference of genetic diversity (and F_{IS}) between schistosomes from male and female rats was therefore tested using a Mann–Whitney U -test.

3. Results

3.1. Multilocus genotypes and probability of occurrence

The probability that two multilocus genotypes might be identical after sexual reproduction was always very low, ranging between $P_{MLG} = 0.0004$ to $P_{MLG} = 10^{-11}$ depending on the rat considered (mean = 2.5×10^{-7}). $P \approx N \cdot P_{MLG}$ was then low enough to consider that parasites displaying the same multilocus genotype within the same infrapopulation were the result of clonal reproduction. All the analyses presented below were therefore undertaken once repeated multilocus genotypes were reduced to a single copy within each infrapopulation.

3.2. Polymorphism and genetic structure

Genetic diversity was moderate for microsatellite markers with values of 0.49 in Dans Fond and 0.48 in Dubelloy. The number of alleles per locus varied between 2 and 13 alleles (mean = 6.57). In schistosomes, female is the heterogametic sex and male the homogametic one. The patterns displayed by our seven microsatellite loci were incompatible with loci being situated on a sex chromosome. Overall differentiation between parasite infrapopulations (i.e. between hosts) is highly significant in both Dans Fond (θ [+95% confidence interval] = 0.013 [0.004–0.023], p -value < 10^{-4}) and Dubelloy (θ [+95% confidence interval] = 0.034 [0.022–0.044], p -value < 10^{-4}). Thus, different hosts harbour genetically differentiated schistosome infrapopulations. Deviation from Hardy–Weinberg expectations was found neither in Dans Fond ($f = 0.018$ [–0.005–0.039], p -value = 0.14) nor in Dubelloy ($f = -0.013$ [–0.091–0.077], $p = 0.74$). No linkage disequilibrium between any pair of loci was found after Bonferroni correction for multiple tests.

3.3. Host and parasite sex effect on genetic diversity (H_s and F_{IS})

For samples from 1996 and H_s , the AIC-based model selection led to a minimal model where only variables host sex (Sr; $r^2 = 21\%$, p -value = 0.003) and schistosome sex (Ss; $r^2 = 16.5\%$, p -value = 0.01) explained variations in mean H_s overall loci: male schistosomes were, on average, more genetically diversified than female schistosomes and male rats harboured higher parasite genetic diversity than female rats (Fig. 1). Neither parasitic load, nor schistosome locality nor interactions between variables were significant. The effect of the host sex (Sr) as well as of the schistosome sex (Ss) on genetic diversity were not locus-specific as none of the

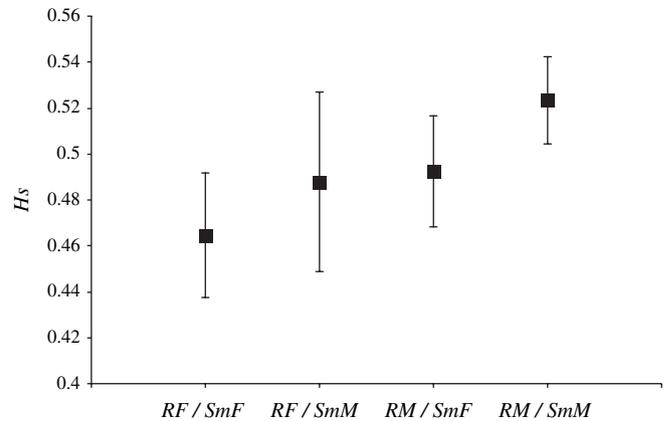


Fig. 1. Mean unbiased expected heterozygosity ($H_s \pm$ standard deviation overall infrapopulations) computed in male and female schistosomes in male and female rats sampled in 1996. RF/SmF: Female rats, Female schistosomes; RF/SmM: Female rats, Male schistosomes; RM/SmF: Male rats, Female schistosomes, RM/SmM: Male rats, Male schistosomes.

interactions Sr: Loc and Ss: Loc were retained after model simplification. Neither the sex ratio of schistosomes (p -value = 0.22) nor the parasitic load (p -value = 0.25) differed between male and female rats. The effect of host sex was confirmed using randomisation approaches (difference between H_s^M and H_s^F : p -value = 0.009; male schistosomes only: p -value = 0.007; female schistosomes only: p -value = 0.07). For F_{IS} , no difference among host sexes was found both using parametric and randomisation approaches (for male schistosomes: mean f in male rats = 0.0018; mean f in female rats = 0.012; for female schistosomes: mean f in male rats = 0.048; mean f in female rats = –0.0076).

The results of the analyses performed on samples from 1997 were consistent with those obtained on sample from 1996. Despite the low 1997 sample size, the average diversity of male schistosomes in male rats was significantly higher than the one in female rats (for male schistosomes: mean H_s male rats = 0.55; mean H_s female rats = 0.52; p -value = 0.05; for female schistosomes: mean H_s male rats = 0.54; mean H_s female rats = 0.52; p -value = 0.50). No difference was observed for F_{IS} (for male schistosomes: mean f male rats = –0.065; mean f female rats = –0.019; Mann–Whitney test: p -value = 0.35; for female schistosomes: mean f male rats = 0.014; mean f female rats = –0.009; p -value = 0.35).

4. Discussion

We have analysed the infrapopulation genetic diversity of the trematode *S. mansoni* in male and female individuals of the rodent host *R. rattus*, in the marshy forest focus of Guadeloupe (French West Indies). Results previously obtained with another set of parasites sampled in Dans Fond in 1997 are confirmed: within hosts, male schistosomes are more genetically diversified than female schistosomes (see ref. [13] for detailed discussion of this pattern). We now specifically demonstrate for the first time a strong influence of the sex of the host on

the genetic variability of their parasites: whatever their sex, schistosomes are more genetically diverse in male hosts than in female hosts (Fig. 1). This pattern is found both in samples made in 1996 and 1997.

Our analysis does not reveal which mechanisms could be responsible for this latter pattern. We will here discuss some potential scenarios explaining the influence of host sex on the genetic variability of schistosomes: 1) a rat sex-specific duration of cercariae infection; 2) a rat sex-specific habitat use that would lead to the exposure of male rats to more genetically diverse parasites; and 3) a host sex-biased immunocompetence that would lead to the selection of more genetically diversified clones in male than in female rats. These scenarios are of course not exclusive and others could be suggested.

4.1. Host sex-specific duration of cercariae recruitment

A difference in the total duration of the recruitment between sexes could have consequences on the genetic diversity of parasites. Older rats may acquire more diverse parasites over time: a) if the total area explored by a rat increases with time; or b) if cercariae of a given place at time t are genetically different from cercariae of the same place at time $t + 1$.

Under this scenario, sampling male and female hosts of different ages could artificially generate such picture, with older rats displaying the highest parasite genetic diversity. In our study, we were not directly able to test this hypothesis since the assessment of the age of the rats was not possible. We tested it indirectly using parasitic load as a surrogate for the age of the rats, by assuming that both variables were positively correlated. Our results did not support the existence of such sampling bias. First, we did not detect any difference in the average parasitic load between male and female rats and second, there was no correlation between the rat parasitic load and the parasite genetic diversity. It seems therefore unlikely that our results were due to variations in the duration of cercariae recruitment in male and female hosts, consecutive, for instance, to a sampling bias relative to their age.

4.2. Rat sex-biased dispersal and home range

Differential migration, mobility patterns or variation of the size of home range between host sexes could explain the higher *S. mansoni* genetic diversity observed in male rats. Indeed, if infecting forms of the parasite are spatially genetically structured, host individuals from the dispersing sex (or the sex with a larger home range) could recruit parasites from a higher number of distinct larval subpopulations, translating into adult parasite infrapopulations being more genetically diverse.

Regarding free-living infective stages (cercariae) within transmission sites (local scale), a previous study in Dans Fond showed very low snail infection rates whatever the season (1% in average) and a strong spatial aggregation of the few infected snails at some particular places in the site [21]. Taking into account the absence of current in this standing water transmission site and the abundant vegetation limiting

cercarial dispersion, this probably also leads to infective cercariae being patchily distributed and genetically differentiated between aggregates. Cercarial patches can be separated by a few meters up to 200 m. Regarding variation in home ranges between male and female rats, previous mark-recapture studies of *R. rattus* in the marshy forest of Guadeloupe (Dubelloy site), over a 1 year period, demonstrated that most rats were highly philopatric. Rats were recaptured, on average, within only 14–27 m of the first capture location [22]. No difference between males and females could be demonstrated [22]. Therefore, a male-biased home range within transmission sites (local scale) does not seem to be a very likely hypothesis to explain the higher genetic variability observed in schistosome infrapopulations from male rats.

At the regional scale, *S. mansoni* transmission sites are geographically structured in the marshy forest of Guadeloupe (Fig. 1) and parasites from these different sites have been demonstrated to be highly genetically differentiated [23,24]. Concerning rats, direct observation of long distance migration events between transmission sites (regional scale) has never been reported. However, Prugnolle et al. [24] demonstrated, using population genetic tools, that long distance dispersal events of *R. rattus* could occur between sites. Genetic data also suggested the existence of patterns of sex-biased dispersal between sub-populations (unpublished data), as the genetic differentiation observed between transmission sites was higher between female than between male rats (mean female $F_{ST} = 0.080$; mean male $F_{ST} = 0.048$). Host sex-biased dispersal rates between transmission sites could therefore be an explanation for the higher schistosome genetic diversity observed in male hosts. Such a phenomenon should however also translate into an observed F_{IS} higher in male rats compared to females, which is not the case. It is also based on the idea that we only sampled immigrant male rats which is unlikely. This hypothesis is therefore, one more time, rather improbable.

4.3. Host sex-biased immunocompetence

When infected by a pathogen, vertebrate hosts often develop an immunity against that type of pathogen (concomitant immunity) [25], and in many cases, this immunity is strain-specific (a strain is defined in terms of alleles at loci – antigenic loci – that are involved in host-protective immune responses). When acquired immunity acts against infection stages, previous exposure to a strain reduces the susceptibility of that host to related strains (that share same alleles at antigenic loci [26]). Therefore, strains that are not related to the one already established in the host individual have a higher chance to penetrate and develop in the host. After penetration and successful development, these new strains immunize the host against future infective stages of related strains. This phenomenon of strain-specific acquired immunity acts therefore as a process of diversifying selection since it favours the establishment of different strains in one host individual. Obviously, the more efficient is the immune response the higher is the diversifying selective pressure acting on the antigenic loci. Moreover, if the number of antigenic loci is high and

randomly distributed in the entire genome, the same pattern could be observed at neutral markers as related strains for antigenic loci will also share more alleles at neutral ones.

This effect of the immune response on the genetic variability of parasites was previously suggested to explain the higher genetic diversity of male schistosomes versus females given that the immune response of the host (whatever its sex) is known to be higher against male schistosomes than against females [27]. We now propose it as a potential explanation for the observed differences of parasite genetic diversity between male and female hosts, given that host sexes are generally known to differ in their immunocompetence.

Host sex-differences in immune competence are very well established in vertebrates and, in general, males are less immunocompetent than females [28]. This immunodepression seems, at least in part, mediated by androgens [1,29]. Although males are more susceptible than female conspicuous to many infections, males are not more susceptible to all infectious pathogens. Several studies illustrate that males are actually less susceptible than females to certain pathogens (reviewed in ref. [1]). The cause of reversed sex-differences in response to certain pathogens is not well understood but may also involve differences in host-pathogen interactions that are affected by the endocrine system. This is the case with *S. mansoni*. Nakazawa and colleagues [30] have studied the effect of testosterone on the susceptibility to this parasite, in *Mus musculus*. Paradoxically, they demonstrated that treated individuals with testosterone displayed lower parasitic loads than untreated individuals. Eloisantos et al. [31] similarly demonstrated that male mice were more resistant to schistosome infection than female mice. The effect of testosterone on schistosomes has been demonstrated to be associated with the fact that testosterone inhibited mitochondrial respiratory chain function of the parasite larvae at the schistosomula stage, the stage occurring just after the penetration by cercariae of the vertebrate host skin [32]. Therefore, we propose that testosterone could act in synergy with the immune system, paradoxically making male acquired immunity more efficient in filtering schistosome strains than the female one, even if the global immune response seems higher in female than in male hosts [27]. Infrapopulations of male rats would therefore be more diversified than infrapopulations of female hosts because of a higher immunity-based diversifying selection process acting in male hosts. This hypothesis is obviously fundamentally based on the assumption that the number of antigenic loci recognised by the immune system is sufficiently large to make differences observable at neutral markers and that the immune system act in synergy with testosterone.

4.4. Conclusion

These different scenarios still remain to be explored. An important additional work, both empirical and in the field, is still necessary to get an explanation. As detailed above, the explanation has certainly to be searched in the definitive host ecology because parasite genetic structure is mainly dependent on its life history traits notably movements,

dispersal, territoriality or others. Nothing makes sense in parasite population genetics except in the light of its host ecology and genetics. Therefore, we should particularly focus, in the future, on better understanding rat ecology in the marshy forest focus of Guadeloupe.

The purpose of our study was not to elucidate all the mechanisms leading to the host sex-specific genetic structure of schistosomes, but to point out a possible factor influencing the genetics of parasites: the sex of the host. Differences among host sexes might have different consequences on parasite evolution. Among others, it seems from our results that male and female hosts might play different roles in maintaining parasite genetic diversity, hence their evolutionary potential.

We hope therefore that these findings will encourage similar studies on other host-parasite systems, given its strong implications for the understanding of host-parasite interactions, evolution and epidemiology.

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