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Population dynamics of host-parasite interactions in a cockroach-oxyuroid system

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Host-parasite interactions of an urban cockroach, *Blattella germanica*, and its oxyuroid parasite, *Blatticola blattae*, were investigated. Life history data of host and parasites were collected under laboratory conditions. These data were used to model the effect of the parasite on the population dynamics of the host in order to understand the parasite's impact on the host population. The aggregation of parasites within a host was under-dispersed. Hosts normally were found to be infected with only one male and one female and rarely two or three. However, the primary sex ratio after hatching was 1.1 (males/females). Female parasite longevity equalled the life span of its host. *B. blattae* had a significant impact on the survival rate of the cockroach larvae and their time to reach maturity, but no effect on the survival rate of the adults. Infected host females produced fewer first oothecae than uninfected ones. Using the population parameters a simple model was developed to estimate the parasite's effect on the population dynamics of its host. According to the model the parasite suppresses the cockroach populations by ca 11%. Hence, the effect of the parasite does not appear strong enough to be used as a biological control agent by itself.

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Over the last decade, the impact of parasites on host populations has been an area of intense research and debate (Anderson and May 1978, 1982, May and Anderson 1978, Dobson and Hudson 1986, Scott and Dobson 1989, Begon et al. 1990, Minchella and Scott 1991). Parasites are implicated in the control of populations (Anderson and May 1978, May and Anderson 1978, Smith 1994) and several theoretical studies have demonstrated the potential impact of parasites on host populations (Anderson and May 1978, May and Anderson 1978, Roberts et al. 1995). Many studies have evidenced correlations between parasite burden and mortality or morbidity in host populations (Esch and Fernández 1993, Grenfell and Gulland 1995). Though experimental studies have shown that parasites are able to regulate host populations (Scott 1987, 1990, Gregory 1991) evidence from natural populations is still circumstantial, since it is very difficult to disentangle ultimate and proximate causes of regulation or limitation of host populations (Holmes 1982, Grenfell and Gulland 1995). However, even if parasites are not the ultimate cause of population regulation or limitation, they are a factor which can be important in the dynamics of host populations (Scott and Dobson 1989). Theoretical studies

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have revealed that, at medium to low levels of pathogenicity, parasites could have an important effect on host population density (Anderson 1979, 1982, Anderson and May 1979, McCallum 1994) perhaps even more important than that of highly virulent parasites.

Mathematical models show that a highly pathogenic parasite is likely to cause a short-term epidemic but then to die out. In contrast, when most of the members of a population are endemically infected with a mild parasitic disease, a long-lasting effect on host density is expected (McCallum 1994) at equilibrium. Similar trends are observed for macroparasitic infections, but in addition density dependence of the parasite, imposing constraints on parasite populations within individual hosts, is included in models of these systems. At low levels of density dependence, macroparasites with low pathogenicity have the highest impact on their host population at equilibrium, but as the intensity of density dependence increases, maximum depression of host populations occurs at slightly higher pathogenicities (McCallum 1994).

The implication of parasites in the population dynamics of their hosts has made them interesting tools for the control of pest populations (Fenner 1994). Models can predict the potential effect of parasites on the regulation of host populations. Hence, before launching costly tests, models can help in the decision-making process if a certain organism can be used as a control agent and elucidate certain interactions (Waage and Mills 1992). Once promising results have been obtained, model predictions can then be tested under natural conditions. Before embarking on a model or control programme, the epidemiology and biology of host and parasite need to be thoroughly understood.

The cockroach *Blattella germanica* is a worldwide pest which is a nuisance in human habitations. They are omnivorous and live on organic human waste. We studied the interaction of *B. germanica* with one of its nematode parasites, *Blatticola blattae*. In the first part of this paper, we describe the results of observations and experiments on the population dynamics of this parasite in its natural host. We collected life history data for this parasite as well as for its host and for the epidemiology of the parasite in order to develop a model and to estimate parameters for the model. In the second part of this paper, these results are incorporated into a model to study the impact of this parasite on the population dynamics of its host.

Material and methods

Host biology and life cycle

Blattella germanica (L.), the German cockroach (Dictyoptera: Blattellidae), is one of the smallest of the well-known cockroach species, measuring 12 to 15 mm long as an adult. It lives worldwide in any human habitat where it can find food, water and shelter and prefers a temperature of around 25°C. Therefore, it can often be found in kitchens and bathrooms. *B. germanica* is hemimetabolous and has 6 larval stages lasting approximately 7 weeks altogether (Morand and Rivault 1992). The adult life span is estimated to last 3 months under natural conditions (Rivault 1989) and can reach 4 months under laboratory conditions (Cornwell 1968). According to the living conditions, a female is able to produce between 2 and 7 oothecae during her life. Each ootheca is carried until maturation for about 20 d by the female and contains 40 embryos on average. Cockroaches live in large aggregates containing all developmental stages. They stay in the same area as long as all their required living conditions are satisfied.

Parasite life cycle

Blatticola blattae (order: Oxyurida, Chabaud 1974; family: Thelastomatoidea) is a parasite found in *B. germanica*. The parasite has a direct life cycle and a haplodiploid mode of reproduction, i.e. the females can produce parthenogenetically haploid males, but females develop from fertilized eggs and are diploid. The parasites live in the posterior part of the intestines of cockroaches and feed presumably on bacteria (Adamson 1994), but possibly also on other matter in the lumen (Hominick and Davey 1973). Parasite eggs are excreted with cockroach faeces (McCallister and Schmid 1981). Infection occurs orally via contaminated food. Eggs are sensitive to desiccation. They can survive up to 120 d in the external environment like *Blatticola manandros*, a related species (Zervos 1988a). The larvae undergo one or more moults before reaching the infective stage (Cali and Mai 1965, Adamson 1994) (Fig. 1A, B).

Experimental set-up

Cockroaches were kept in boxes containing cardboard shelters with free access to food (dog food pellets) and water under a constant room temperature of 25°C with a 12:12 L:D photoperiod. These experimental conditions take the conditions in the natural environment into account. Uninfected cockroaches were obtained by separating mature oothecae manually from females. Oothecae were then washed in 10% ethanol and left to hatch in clean vials. Under these conditions, larvae were presumed to have not had any contact with any source of contamination and were hence supposed to be uninfected.

After hatching the larvae were divided into two groups: the larvae in one group remained uninfected and the larvae in the second group were infected by

giving them pieces of apple contaminated with the eggs of the parasite. Successful infection was confirmed by control dissection of some individuals shortly afterwards. To estimate the impact of the parasite on the mortality and longevity five boxes with around 100 larvae each were prepared for the two groups. Regular dissections were carried out to check the presence or the absence of parasites under the two breeding conditions. All adults were dissected at the end of the experiment. Every 15 d, 30 individuals were dissected and the presence or absence of parasites was checked. Time of imaginal moult was recorded. Newly emerged adults were removed from the experimental boxes of larvae and kept in different clean boxes. Within one experimental group, individuals which had moulted at the same time were allowed to mate freely within their respective experimental group. To measure the effects of the parasite on fecundity, uninfected individuals were hatched and then divided into two groups, one of which was subsequently infected. For this study females were isolated after mating and kept in individual containers. Mature oothecae were isolated and the number of larvae that hatched from each ootheca was recorded.

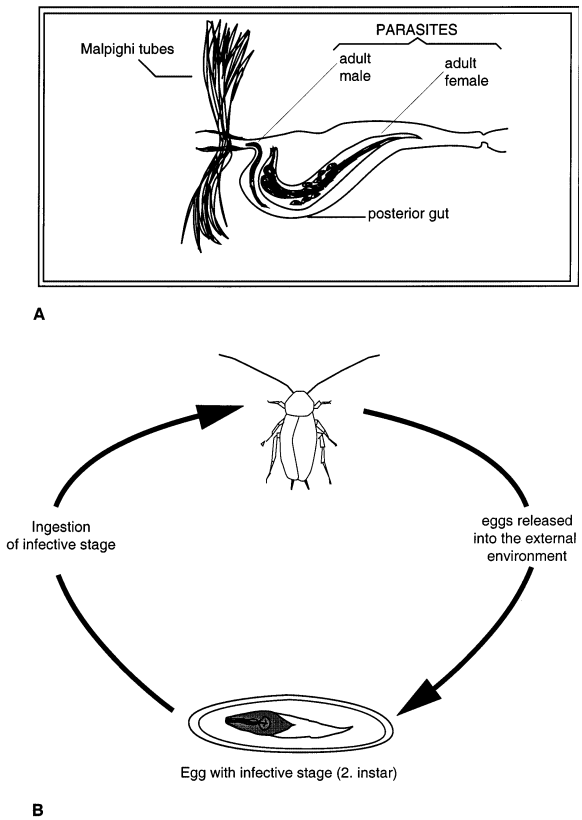

Fig. 1. A) Position of the parasites in the host. B) Life cycle of the *Blatticola blattae* parasite of the cockroach *Blattella germanica*.

Biometrical measures were made on emerging adults and larvae from their first ootheca. The width of the head and the length of the femur of their first right leg were measured on cockroaches from the two experimental groups.

To estimate the aggregation level of the parasite in its host, 200 newly hatched uninfected cockroaches were left in a box with five infected adult males. Their presence in the box was enough to infect the larvae through deposited faeces. Every 15 d, 30 individuals were dissected and the number of parasites in each cockroach was counted. To estimate parasite life history traits, ten hosts were given a single parasite egg deposited on a piece of apple. Therefore nematode eggs in the faeces of one cockroach contained only unfertilized eggs as each cockroach housed only one parasite. Hosts' faeces were examined every day to obtain information about the periodicity of the parasite's egg laying. If the female *B. blattae* stopped laying eggs, the host was dissected after a few days to confirm that the parasite was dead. If the host died before, host life span was also recorded as parasite life span.

The primary sex ratio was determined by infecting 100 cockroaches each with a randomly chosen single egg and dissecting them after 15 d. Since preliminary results and previous studies of related oxyurids (Zervos 1988a, b, c) suggested that male-male competition took place, this was studied infecting 15 hosts with five male eggs each. Male eggs were obtained from cockroaches infected with a single female. Standard statistical methods were employed including a test of two proportions (Zar 1984).

Results

Dynamics of infection and aggregation

The dynamics of parasite infection and aggregation are shown in Fig. 2A and B. Only 1 male per host was observed and the maximum number of females found was 3, but on average only 1.14. The distribution of parasites, males and females, was found to be underdispersed with a variance close to zero.

Life history data

Table 1 presents the total number of eggs laid by each *B. blattae* female, the daily average of eggs laid by one female and the longevity of the female parasites. All except two *B. blattae* females were still alive when their host died; they seem to be long-lived. The number of eggs laid by individual females was high – as compared to other oxyuroid parasites of the same size – but no obvious egg laying pattern could be noted in relation to time (Fig. 3).

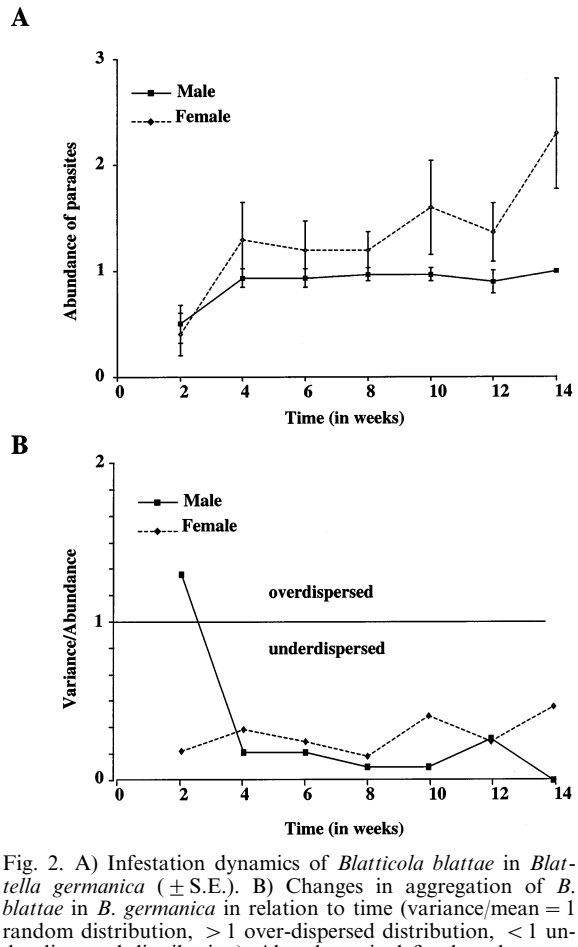

Fig. 2. A) Infestation dynamics of *Blatticola blattae* in *Blattella germanica* ($\pm$S.E.). B) Changes in aggregation of *B. blattae* in *B. germanica* in relation to time (variance/mean = 1 random distribution, > 1 over-dispersed distribution, < 1 under-dispersed distribution). Abundance is defined as the mean number of parasites found in all the individuals.

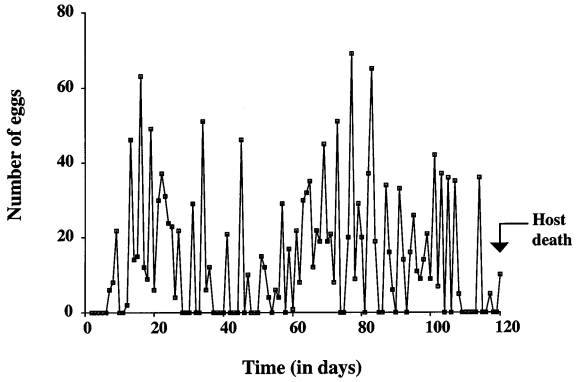

Fig. 3. Daily egg output laid by a single female.

Primary sex ratio and male-male competition

We obtained a success rate of 82% when infecting 100 *B. germanica* with a single parasite egg each. Among these 82 infections, we found 43 (CI 42%–63%) with a male and 39 (CI 37%–58%) with a female parasite. That allows us to estimate the primary sex ratio after hatching at 1.1 (males/females). However, in each of the 15 cockroaches, which had been infected with five male eggs, we only found one single male.

Effect of the parasite on its host

Only 62.3% of the infected larvae survived to adulthood, whereas 71.8% of the uninfected larvae reached maturity (Table 2). *B. blattae* had a significant impact on the juvenile survival rate of its host *B. germanica* ($P < 0.001$; $Z = 0.095$; S.E. = 0.011). *B. germanica* larvae with parasites took significantly longer to mature (79.95 ± 6.23 d) than uninfected cockroach larvae (72.92 ± 6.79 d) (ANOVA, $F_{1,531} = 145.2$, $P < 0.0001$) (Fig. 4).

The number of adult females which produced a first ootheca was much higher for the uninfected group than for the infected one ($P < 0.05$; $Z = 0.071$; S.E. = 0.062, Table 3). However, once an ootheca had been produced, the parasite had no impact on the number of viable larvae (38.75 ± 4.70 viable larvae of uninfected cockroaches versus 39.50 ± 6.11 viable larvae of infected cockroaches (t-test, $t = 0.43$, d.f. = 118, $P = 0.43$)). Nematode parasitism did not significantly modify cockroach mortality according to sex (G-test with Yates correction, $P > 0.05$, $G = 0.12$) and host sex ratio remained close to 50% (Table 4).

Table 1. Total clutch size of ten single *B. blattae* females and longevity of the host/parasite system. Hosts were infected with one female each; eggs were therefore not fertilized.

Individual	Total clutch size	Mean number of eggs laid per day ($\pm$ sd)	Female longevity (d)
1	1287	17.63 ± 12.02	73
2	2435	21.55 ± 27.57	113*
3	1408	13.67 ± 15.16	103
4	1069	9.63 ± 10.07	111
5	1700	14.05 ± 16.56	121
6	231	8.88 ± 7.16	26
7	1135	18.61 ± 13.56	61
8	2175	20.10 ± 23.10	108*
9	1731	16.49 ± 19.72	105
10	1547	13.00 ± 17.49	119

* case where the female parasite died before the host.

Table 2. Influence of *B. blattae* on the survival rate of the juvenile stages of *B. germanica*.

	Infected hosts	Uninfected hosts
Adults	645	686
L1 (initial density)	1035	956
Survival rate (%)	62.3	71.8

Morphological measurements of head and femur were used to check whether parasitism exerted an influence on size and development. The presence of *B. blattae* had no influence on the morphological development (head length, femur length) of cockroaches (*t*-test for all comparisons, $P > 0.05$).

Model

Data from the experiments and observations presented above set the stage for a model exploring the dynamics of the parasite and its possible effects on the population of its host. The life history data obtained for host and parasite are used as parameters in the model.

Once a cockroach is infected with *B. blattae*, the infection is life-long. The life expectancy of the host and female parasites was similar (Table 1). Hosts never eliminated their parasite even after a long fast. Therefore, we did not include in this model an immune response as expressed by a recovery rate or by a reduction of the number of individuals in the host.

As one infected host usually housed one male and one female parasite – the presence of two or three females was rarely observed – the following assumption was made: one cockroach host is parasitized by one male and one female nematode only. Strong competition for resources is assumed within a single host (Morand and Rivault 1992). Mean abundance (or mean intensity) (and its variance) of parasites was taken from

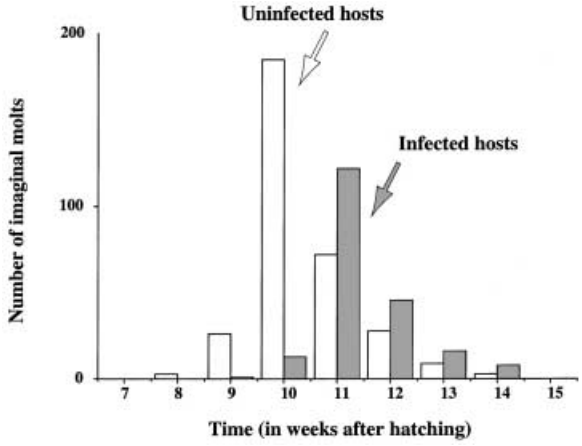

Fig. 4. Weekly number of imaginal moults for infested and non-infested cockroaches. Time in weeks since hatching.

Table 3. Impact of the parasite *B. blattae* on the oothecae production of *B. germanica*.

	Infected cockroaches	Uninfected cockroaches
No. of females which produced a first ootheca	93	158
Total no. of females	107	168
% of the reproducing females	86.9	94.0

our observations (Fig. 2 and Morand and Rivault 1992). An unusual trait of this macroparasite was that it is under-dispersed (variance close to zero). However, this distribution simplified the model as no changes in parasite aggregation had to be taken into account. This parameter is usually included as k in models of macroparasite dynamics (Anderson and May 1978, May and Anderson 1978, Smith 1994). Here, cockroaches were either infected or not. Therefore, we are using a model which is indistinguishable from a microparasite model. Infected cockroaches all contributed equally to the infection of susceptible uninfected hosts.

The parasites reduced the survival of the cockroach larvae, slowed down their development and reduced female fecundity. However, they did not seem to affect survival of adult hosts. We also assumed that the infection could become established equally well in larvae and in adult hosts. However, taking into account the maturation time of the parasite, we assumed that only adult hosts contributed to the infection of new hosts considered as susceptible hosts because they were the only ones that harboured adult parasites ready to produce eggs.

Values given by our experimental results were used for the parameters in the model (Table 5). The competition among adult hosts α_a and the competition among juvenile hosts α_j were estimated. We assumed that competition among adult cockroaches for food is an order of magnitude higher than among the juveniles because, being larger, they probably require more food. This estimate is further investigated carrying out a sensitivity analysis of varying both parameters mediating density dependence further below.

The model, outlined in Fig. 5, is given by four equations. The density of the uninfected adult cockroaches changes according to

Table 4. Impact of *B. blattae* on the mean number of viable larvae – according to sex – per *B. germanica* ootheca.

	Infected hosts	Uninfected hosts
No. of males	341	324
No. of females	686	645
Sex-ratio (M/F)	0.497	0.502

Table 5. Values of the parameters used in the model.

	Parameters	Values
μ_a	per capita mortality of adult cockroaches	0.01 d^{-1}
μ_j	per capita mortality of juvenile cockroaches	0.005 d^{-1}
τ	maturation time of cockroaches	73 d
ε	delay of maturation time caused by the parasite	7 d
α_a	competition among adult hosts	0.01 (estimated value)
α_j	competition among juvenile hosts	0.001 (estimated value)
λ_s	per capita fecundity of healthy females	0.61 d^{-1}
λ_i	per capita fecundity of infected females	0.57 d^{-1}
δ_j	parasite-induced juvenile mortality	0.002 d^{-1}
β	transmission efficiency	0.14 d^{-1}

$$\frac{dA_u}{dt} = \frac{1}{\tau} J_u - (\mu_a + \alpha_a(A_u + A_i))A_u - \beta A_u A_i \quad (1)$$

where the number of uninfected adults (A_u) is described by the maturation of juveniles (J_u/τ), the adult mortality (μ_a) and competition amongst adults ($\alpha_a(A_u + A_i)$), and infection through encounters with infected adults ($\beta A_u A_i$), determined by β, the transmission rate (infected juveniles are not infective).

To prevent unchecked growth of the cockroach population in the absence of the parasite, we have to introduce density dependence. Depending on the details of cockroach dynamics, any life history parameter could be density dependent, be it reproduction (λ_u and λ_i), development time (τ) or mortality (μ_a and μ_i). Here, we will assume that mortality is density dependent. Density dependence is considered the same for infected and uninfected individuals. According to previous observations (Rivault and Cloarec 1992a, b), aggressive interactions on food sources mainly occur between cockroaches in the same age group and competition between adults and larvae is reduced, since for instance smaller animals managed to reach food by crawling under larger animals (Rivault and Cloarec 1992a). The mortality rates of juvenile and adult cockroaches in absence of competition are μ_j and μ_a, and α_j and α_a represent the intensity of within-age-class competition. In the model, there is no among-age-class competition, but the results do not change significantly if this is included (results not shown). Even though there are a few interactions between the largest larvae (instar 6) and the adults, this is further dependent on group structure and would lead to an inclusion of six stages of larvae and a parameter for group structure into the model, making it less transparent.

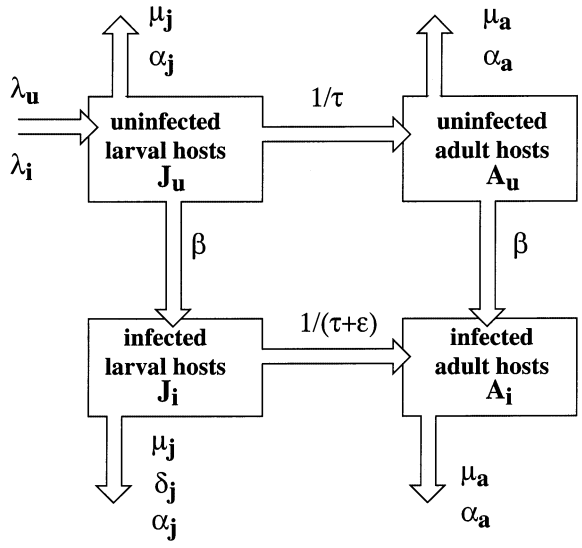

Fig. 5. Flowchart of the model (see Material and methods).

The dynamics of infected adults are given by

$$\frac{dA_i}{dt} = \frac{1}{\tau + \varepsilon} J_i + \beta A_u A_i - (\mu_a + \alpha_a(A_u + A_i))A_i \quad (2)$$

Note that infected adults arise either because of maturation of infected juveniles, which is prolonged due to the effect of the parasite ($1/(\tau + \varepsilon)$), and through infection of uninfected adults ($\beta A_u A_i$); the number of infected individuals decreases due to natural mortality (μ_a) and to competition ($\alpha_a(A_u + A_i)$).

The dynamics of uninfected larvae are given by

$$\frac{dJ_u}{dt} = \frac{\lambda_u A_u + \lambda_i A_i}{2} - \frac{1}{\tau} J_u - (\mu_j + \alpha_j(J_u + J_i))J_u - \beta J_u A_i \quad (3)$$

Here, the factor 2 represents the fact that only half of the population (the females) reproduces (the sex ratio is assumed to be fixed at 0.5). Their density is determined by adult fecundity ($\lambda_u A_u/2 + \lambda_i A_i/2$) and diminished by the maturation rate of larvae, larval mortality (μ_j) and competition ($\alpha_j(J_u + J_i)$) and the number of the larvae which were infected by adults.

Finally, population dynamics of infected juveniles are given by

$$\frac{dJ_i}{dt} = \beta J_u A_i - \frac{1}{\tau + \varepsilon} J_i - (\mu_j + \delta_j + \alpha_j(J_u + J_i))J_i \quad (4)$$

where the number of larvae which became infected is $\beta J_u A_i$, from which are subtracted those which matured ($1/(\tau + \varepsilon)J_i$) and the infected larvae which died either naturally (μ_j), due to a parasite (δ_j) or competition ($\alpha_j(J_u + J_i)$).

When the values of all the parameters are incorporated (Table 5), the result of simulations shows that the density of larvae (Fig. 6A) and adults (Fig. 6B) is reduced by infestation of the parasite, and that the population density of infested adults and larvae is

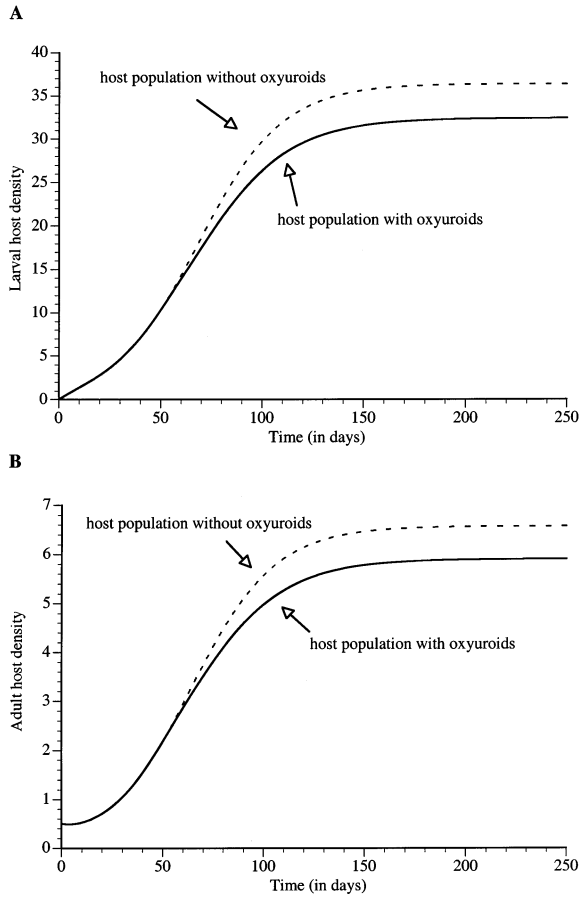

Fig. 6. Results of model simulation. Densities of (A) larval and (B) adult hosts in the presence and absence of the parasite.

depressed compared to that of the uninfested adults and juveniles by around 11% for the whole population of the infected hosts.

A sensitivity analysis of the parameters α_j and α_a – the only parameters in the model which were not experimentally measured – showed that the effect of the parasite on the population was strongly dependent on the intra-species interactions of the host (Fig. 7).

We also calculated the basic reproductive rate R_0 for the model in two versions. The first one is the classical epidemiological type where J and A are the host densities in absence of the parasite and R_0 describes the invasion of the parasite into an uninfected population.

$$R_0 = \frac{\beta}{(\mu_a + \alpha_a A)}\left[A + \frac{J}{(\mu_J + \delta_J + \alpha_J J)(\tau + \varepsilon) + 1}\right] \quad (5)$$

This measure expresses the fact that there are two transmission routes for the parasites: through infecting adults directly (density A) and through juveniles (density J). The latter are not infective directly; only a proportion

$$\frac{\dfrac{1}{\tau + \varepsilon}}{\mu_J + \delta_J + \alpha_J J + \dfrac{1}{\tau + \varepsilon}} = \frac{1}{(\mu_J + \delta_J + \alpha_J J)(\tau + \varepsilon) + 1} \quad (6)$$

of which will make it into adulthood and become infective. Infected adults then transmit the parasite with efficiency β for a period of $(\mu + \alpha_a A)^{-1}$, on average.

The second type is a more evolutionary approach, using the effective reproductive rate (Anderson and May 1991), where the infection in the host population already exists

$$R_0 = \frac{\beta}{(\mu_a + \alpha_a (A_i + A_u))} \times \left[A_u + \frac{J_u}{(\mu_J + \delta_J + \alpha_J (J_i + J_u))(\tau + \varepsilon) + 1}\right] \quad (7)$$

This measure would be needed to infer selection pressure on the parasites in endemic populations.

Control

When the parasite's R_0 exceeds unity, it will spread in the population. Given that it causes damage to its hosts (increases juvenile mortality, prolongs development, and reduces fecundity) it will depress cockroach population densities at least to some extent. But will it be able to reduce the host population to low densities? An indication is given by the host fitness when the force of infection is extremely high. Then, every individual will be infected soon after hatching and hence the parasite will exert the maximum effect on cockroach demography. Comparing the maximum host per-capita fitness (lifetime reproductive success in the absence of density dependence) of uninfected individuals

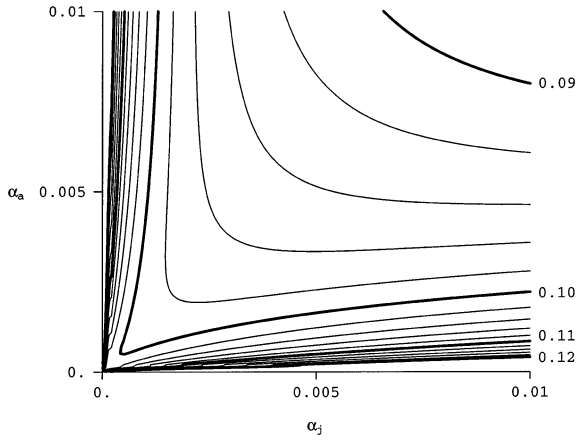

Fig. 7. Depression of cockroach population densities (from carrying capacity) as a function of the intensity of resource competition among juveniles (α_j) and adults (α_a).

$$F_u = \frac{1}{2}\frac{1}{1+\mu_j\tau}\frac{\lambda_u}{\mu_a} \approx 22.3 \quad (8)$$

with the maximum per-capita fitness of infected individuals

$$F_i = \frac{1}{2}\frac{1}{1+(\mu_j+\delta)(\tau+\varepsilon)}\frac{\lambda_i}{\mu_a} \approx 18.3 \quad (9)$$

confirms that the parasite is not very virulent. Furthermore, as the per-capita fitness of infected individuals is over 1, it implies that even under extremely high levels of infection, the cockroach population can continue to grow. In other words, the parasites cannot keep the host population in check in the absence of other density-dependent factors. However, the parasite still has an effect and reduces the growth rate of the host population and the population size at equilibrium in combination with density dependence in the model. This is demonstrated in Fig. 6.

It is difficult to assess the parasite's real capacity to depress host equilibria, as we have no observational data for the intensity of within-host competition and hence do not know carrying capacities in the absence of the infection. A sensitivity analysis shows, however, that the depression factor q varies only from 0.09 to 0.12 in a large range of values for α_j and α_a. So whatever the density dependence, the parasite will reduce the size of the host population.

Discussion

The parasite *B. blattae* shows some interesting characteristics. An uncommon trait in macro-parasite life histories is the observed aggregation rate: the parasite was under-dispersed (Anderson and May 1978, May and Anderson 1978, Dobson 1989). In another cockroach-oxyurid system (Zervos 1988a, b, c) an under-dispersed distribution has also been observed. The fact that only one male per host was observed indicates that this is caused by density-dependent regulation. This may be due to competition between males, for instance for resources or females. The longevity of the female parasites was equal to the longevity of their host. According to Morand (1996), Morand et al. (1996) and Sorci et al. (1997), the length of host life is a very important determinant of nematode life histories, especially female life histories, since it has an impact on parasite adult mortality and thus fecundity of the parasite. For the time being, it is not possible to test the duration of potential longevity of females above the life span of their hosts. The fact that the female parasite life span had the same length as that of the cockroach host was surprising, since the longevity of the cockroach pinworms is thus greater than that of vertebrate pinworms (Morand 1996). This result agrees with the observation that fecundity of cockroach pinworms is much greater than that of vertebrate pinworms. For example, *Syphacia muris*, a parasite of the domestic mouse, *Mus musculus*, lays 450 eggs during its life time, while *B. blattae* lays about 1500 eggs. These results comply with previous life history analyses, but the observed tendency gives higher values than expected (Morand 1996). The reason for this tendency remains to be investigated.

The data showed that the presence of parasites had an impact on the rate of development of *B. germanica* larvae, which took longer, and on survival rates, which were reduced in parasitized juvenile cockroaches. However, parasites did not seem to influence the size of their hosts or longevity of adult hosts. In addition, infected females are also less likely to produce oothecae than healthy females. As food shortage stops oothecae production in *B. germanica*, reduced oothecae production in infected females could be due to reduction of available resources by the parasite (Cochran 1983). We do not know if cockroaches detect the parasite's presence in their gut, but they do not seem to compensate any loss by an increase of their food intake (food access was ad libitum in these experiments). Further experiments are necessary to solve this problem. The observed data allowed us to develop a model of the infection. Epidemiological theory predicts that if the parasite has an impact on fecundity and has a high prevalence (the model predicts 94% prevalence for the juveniles and 99.5% for the adults and 94.9% for all; in a field study 75%–90% of host adults were found to be infected (Morand and Rivault 1992)), then it is likely to reduce the size of the population (McCallum 1994). Low survival rates of host larvae can also influence host density. Some of the life history data indicated that *B. blattae* might play a role in the depression of the host population. The subsequent model led to the conclusion that the population dynamics of *B. germanica* can be limited by its parasite *B. blattae*, since its presence suppressed approximately 11% of its host population according to the model. Only 88% of larvae reach adulthood when parasitized. However, the presence of parasites does not alter the population structure (i.e. the ratio of adults to juveniles in the population), possibly because parasites affected survival rates of larvae and did not modify those of adults.

The sensitivity analysis showed that the effect of the parasite was dependent on the strength of the host-host competition: the stronger the competition between the adult cockroaches or the larval cockroaches, the weaker the effect of the parasite. Interesting is also the fact that if juveniles could also transmit the parasite, it could produce up to six times as many descendants (simulations are not shown here). However, even if this were the case, the impact on the overall population would still be small. Presently, the maturation rate of the parasite is too long to allow the occurrence of larval

hosts which are infective. Even though there seems to be selective pressure on the parasite to develop more quickly, this may be counter-balanced by life history constraints and the trade-off between time of maturation and levels of fecundity.

The model suggests that the parasite alone is not able to eradicate a *B. germanica* population. Host-parasite interactions may help to control cockroaches, in addition with other methods of pest control. To use a pathogen to eradicate the host population successfully, the pathogen must induce high mortality. To control a host population, the parasites must suppress this population below the carrying capacity (Jaenike 1998). However, several factors are likely to determine the potential control of the host population by the parasites, not only the effect of the parasite on host mortality and fecundity, but also parasite aggregation and the effect of within-host competition (Jaenike 1998). It has been proposed that within-host parasite competition demands more stringent conditions under which the parasite will limit the host population (Jaenike 1998). Therefore, within-host competition resulting in the under-dispersed distribution may reduce the effect of the parasites on the population.

Our data confirmed that the impact of parasites does not have to be very strong and evident to affect a population, a result predicted by theoretical studies. Highly pathogenic parasites may disequilibrate a population and lead to an epidemic, but may not be able to control it constantly over long periods. Our experimental data, in combination with the model, demonstrate that *B. blattae* in *B. germanica* has the potential to suppress the numbers of its host. However, according to our model, the effect does not appear to be strong enough to use *B. blattae* as a biological control agent against cockroaches on its own.

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