Influence of the trematode *Aphalloïdes cœlomicola* Dollfus, Chabaud & Golvan, 1957 on the fecundity and survival of *Pomatoschistus microps* (Krøyer, 1838) (Teleostei: Gobiidæ)

C. PAMPOULIE[1]*, S. MORAND[2], A. LAMBERT[3], E. ROSECCHI[1], J. L. BOUCHEREAU[4] *and* A. J. CRIVELLI[1]

[1] *Station Biologique de la Tour du Valat, Le Sambuc, F-13200 Arles, France*
[2] *Laboratoire de Biologie animale, Université de Perpignan, Centre de Biologie et d'Ecologie Tropicale et Méditerranéenne, Avenue de Villeneuve, F-66860 Perpignan Cedex*
[3] *Laboratoire de Parasitologie Comparée, and* [4] *Laboratoire d'Hydrobiologie Marine et Continentale, Université Montpellier II, Sciences et Techniques du Languedoc, Place E. Bataillon, F-34095 Montpellier Cedex 5, France*

SUMMARY

Monthly data collected during 1993 in the Vaccarès lagoon (Rhône Delta, France) were used to investigate the influence of the trematode *Aphalloïdes cœlomicola* on the survival of the common goby *Pomatoschistus microps*, which acts both as second intermediate and definitive host. Prevalence of the parasite did not increase with fish size in either sex. Mean abundance and variance to mean abundance ratio gave evidence that the trematode could have an impact on host mortality. Gonad weight of parasitized females was reduced and the parasite seemed to provoke a gonadal regression above a certain parasitic biomass. Fecundity and egg diameters did not differ between parasitized and unparasitized females, we thus hypothesize that the energy content of eggs (composition in lipids and proteins) was probably modified as an adaptation to energy losses. Prevalence of infection in males compared to females and influence of the parasite on host survival and reproductive effort are discussed.

Key words: digenean, Gobiidæ, host mortality, gonadal regression.

INTRODUCTION

The influence of parasites on the demographic structure of communities (Dobson & Hudson, 1986; Scott, 1988), fecundity (Møller, 1993; Thomas *et al.* 1995*a*), predation (Maillard, 1976; Lafferty & Morris, 1996; Barber, 1997) and mortality of the host (Thomas *et al.* 1995*b*) has been demonstrated in several species. It is often suggested that parasites participate in regulating the host population (Combes, 1995) and that demographic studies must include the influence of parasites (Thomas *et al.* 1997).

With the exception of Schistosomatidæ and Sanguinicolidæ, Digeneans in particular are generally mesoparasites, whose life-cycle takes place within 2 or 3 hosts. In the definitive host, they release their eggs by natural routes. Maillard (1973) found an exception with *Aphalloïdes cœlomicola*. The cycle of this parasite involves only 2 hosts: *Hydrobia ventrosa* (Gastropoda: Hydrobiidæ) as first inter-

mediate host and *Pomatoschistus microps* (Teleostei: Gobiidae) as second and definitive host (Maillard, 1973). The metacercariæ of *A. cœlomicola* migrate towards the abdominal cavity of the goby where they turn into adults. Thus, the adults are trapped into the abdominal cavity of fishes without any evidence of regulation of their number. Maillard (1976) counted up to 52 adult parasites in the body cavity of a fish (15% of the fish weight). The impact of this parasite on the biology of its host has never been studied and was not considered in biological studies of the common goby (Vaes, 1978; Joyeux, 1991; Bouchereau, 1994). As the adults of *A. cœlomicola* are stuck in the abdominal cavity of the common goby, their eggs cannot be released in the normal way. Given such an important mass, Maillard (1976) thought that the life-cycle of this trematode probably involved the death of its host, but was not able to demonstrate that the parasite induced it. This would therefore be an example of a 'Diplomat-Killer' host–parasite system (Renaud & De Meeüs, 1991) in which the parasite was aggressive. This parasite probably has an impact on life-history traits of its host. The accumulation of a huge quantity of adult parasites in the abdominal cavity of the fish probably

* Corresponding author: Station Biologique de la Tour du Valat, Le Sambuc, F-13200 Arles, France. Tel: +33 (0)4 90 97 20 13. Fax number: +33 (0)4 90 97 20 19. E-mail: pampoulie@tour-du-valat.com

exerts physical pressure on the gonads of females in particular, which generally develop and occupy up to 80 % of the body cavity in the reproductive season.

The aims of this work are as follows. (1) To compare the male and female infection rates (prevalence and abundance). We can expect males to be more affected than females, as is known for some parasites (Folstad *et al.* 1989). (2) To examine the effect of parasitism on host survival, which is difficult to estimate in field studies. Here, this was assessed using the mean abundance and the variance to mean abundance ratio. (3) To determine the effect of parasitism on host reproductive effort, as the parasite is liable to exert physical or energetic pressure on its host.

MATERIALS AND METHODS

In 1993, monthly sampling took place from March to December in the Vaccarès lagoon (Rhône Delta, France), with a fyke net (6-mm mesh size) set for 5 consecutive days per month. The Vaccarès lagoon is a Mediterranean lagoon of 6400 hectares, with an average depth of 1·5 m and average salinity of 13·6 g/l in 1993.

In this lagoon, *P. microps* is an annual species and a multiple spawner, which reproduces in spring. Throughout the year, the size distribution of this fish indicates that only one cohort is present in the population. In July and August, adults die while recruitment of young of the year occurs (Pampoulie, unpublished data). Thus, the study of the year 1993 data will correspond to the monitoring of the impact of the parasite on one age group.

Common gobies were preserved in 10 % formalin. To make all monthly samples comparable in total length (TL), fish were chosen over the same size range and distributed into 2 mm size class. Several fish were dissected per size class (minimum 5). A total of 383 females and 338 males were thus examined. Total length in mm and eviscerated carcass weight in mg (EW) of these fishes were recorded. The gonads of females and males were weighed (wet weight) to the nearest mg in order to calculate the gonado-somatic index (GSI = $100 \times$ Gonad weight/Eviscerated carcass weight). As *P. microps* is a multiple spawner, the ovarian fecundity per spawning act (*Fov*) was determined by counting the number of oocytes in the most advanced batch (leading clutch) in mature females (stage IV). The egg diameters of the leading clutch were measured on 30 eggs taken at complete random. The leading clutch volume was calculated by multiplying the mean egg diameter by the individual fecundity ($V = D_m \times Fov$).

The stage of maturity of the females' gonads was defined according to the scale established by Bouchereau (1994), based on his observations and those of Miller (1963), Lee (1974) and Webb (1980):

Stage I – Immaturity: the ovaries are separated, filiform to cylindrical, transparent and colourless. Stage II – Beginning of maturation: the ovaries are separated, becoming cylindrical, they are translucent yellow. Oocyte development is just perceptible. Stage III – Advance maturation: the ovaries start to be swollen, firmer, close together or in contact along the posterior part, the surface becomes granular. They vary from yellow to golden yellow colour. Oocytes are of a similar colour as the ovary. Stage IV – End of maturation: the ovaries are swollen, in contact along a major part of their length, the surface is completely granular. Oocytes and ovaries are of a similar golden yellow colour. Stage V – Prespawning: the ovaries are swollen, vascularized, translucent, yellow to orangey-red. The ovarian membrane is very fine. Mature oocytes are transparent, colourless, some show minute red lipid globules at one pole. Oocytes are expelled with slightest pressure on the abdomen.

Body size effects were removed from biological traits (gonad weight and fecundity) by using residuals from the trait-body size regression (log transform when necessary).

Size at maturation was estimated from the size at which 50 % of common goby were mature. The minimum length at maturation of fish was also recorded.

For each fish, the number and the total mass of *A cœlomicola* were recorded. Body size effect on prevalence of the parasite was tested using χ^2 test; when this test was significant, logistic regression was used to determine trends of prevalence in largest fish (values taken from the mode to the largest size class). The effect of the parasite on the fish mortality was estimated by the method described by Margolis *et al.* (1982) and Lester (1984). We calculated the mean abundance of the parasite per fish size class (number of parasites divided by number of hosts per 2-mm size class) and the variance to mean abundance ratio, which represents the degree of aggregation of the parasite in the host. These 2 parameters were recorded for males and females separately. To determine the relation between these 2 parameters and host age, we tested linear and non-linear regressions and chose the regression that explained the highest percentage of variation.

RESULTS

Epidemiological results

The length of *P. microps* caught varied from 11 to 53 mm. The smallest parasitized individual was 21 mm long (TL) and the largest 46 mm. The maximum number of parasites observed in the abdominal cavity was 25 in a 35 mm female. The maximum parasite mass observed was 38·4 mg in a 30 mm female weighing 251·2 mg (15·29 %).
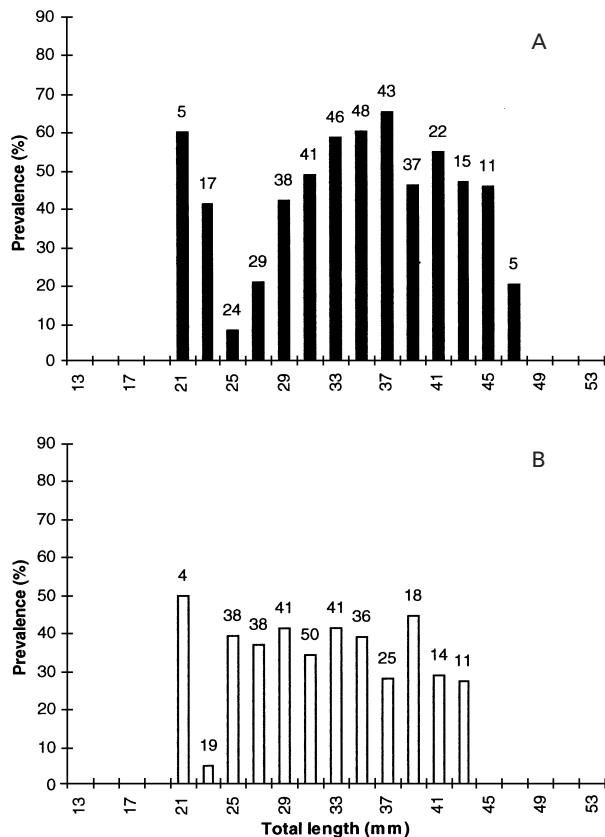
Fig. 1. Frequency of parasitized females (A) and males (B) per 2-mm size class. The number of hosts examined in each length class is indicated above each histogram.

A total of 383 females and 338 males was examined; 181 and 119 respectively were found to be parasitized. The prevalence of the parasite (Fig. 1) did not depend on the host size in males ($\chi^2 = 6.91$, D.F. = 5, N.S.) contrary to females ($\chi^2 = 31.18$, D.F. = 6, $P < 0.001$). No significant prevalence trends were found in the largest (TL > 36 mm) females (logistic regression, $r = 0.14$, $P = 0.11$, $n = 133$).

The frequency distribution of *A. cœlomicola* within the host was fitted to a negative binomial distribution (Kolmogorov Smirnov test, $P > 0.05$) for males and females. The values of the aggregation coefficient were $k = 0.30$ and $k = 0.36$ and the means were $m = 0.96$ and $m = 1.77$ for males and females, respectively.

Mean abundance and variance to mean abundance ratio

The mean abundance (Fig. 2) and the variance to mean abundance ratio (Fig. 3) did not increase with size and were fitted to a polynomial regression in males ($r = 0.75$, $P < 0.01$, $n = 11$; $r = 0.40$, $P > 0.05$, $n = 11$) and in females ($r = 0.86$, $P < 0.01$, $n = 14$; $r = 0.57$, $P < 0.05$, $n = 14$). For the variance to mean abundance ratio, if the highest data point was removed from the analysis, the best regression model was still polynomial ($y = -0.01\,x^2 + 1.06\,x - 16.61$,

$r = 0.50$, $P > 0.05$, $n = 10$ for males; $y = -0.02\,x^2 + 1.52\,x - 21.44$, $r = 0.71$, $P < 0.01$, $n = 13$ for females).

Impact on the life-history traits of the host

Size at maturation did not differ between parasitized (28.58 ± 2.80, $n = 21$) and unparasitized (28.48 ± 2.88, $n = 42$) females (*t*-test, $t = 0.13$ D.F. = 61), and parasitized (27.46 ± 3.64, $n = 12$) and unparasitized (28.01 ± 2.67, $n = 34$) males (*t*-test, $t = 0.56$, D.F. = 44). The minimum length at maturation was 23 mm for parasitized and 24 mm for unparasitized individuals in both sexes. The gonad weight increased with the size of males ($r = 0.61$, $P = 0.0001$, $n = 339$) and females ($r = 0.51$, $P = 0.0001$, $n = 382$). In males, there was no correlation between the relative gonad weight measured as residuals of the regression of gonad weight on size, with parasite numbers ($r = 0.09$, $P = 0.1016$, $n = 339$) or mass ($r = 0.08$, $P = 0.1072$, $n = 339$). In females, the relative gonad weight on size significantly decreased with parasite numbers ($r = -0.10$, $P = 0.0438$, $n = 382$) and mass ($r = -0.16$, $P = 0.0015$, $n = 382$). There was no difference between the gonado-somatic index of the parasitized and unparasitized females whatever the period (*t*-test, $t = 0.35$, N.S. for the spawning period; $t = 1.64$, N.S. for the non-reproductive period). For the males, a small advantage was observed for the unparasitized fish (Student-Newman-Keuls *t*-value = 5.33, $P < 0.001$) during the non-reproductive season. The gonado-somatic index of the females increased with the maturation stages of the gonad whereas the average weight of the parasite decreased (Fig. 4 A).

When length–fecundity regressions for parasitized ($Fov = 18.43$ TL $- 98.67$, $r = 0.45$, $P = 0.01$, $n = 31$) and unparasitized ($Fov = 25.37$ TL $- 220.93$, $r = 0.49$, $P = 0.03$, $n = 21$) females were compared, no significant difference was found between slopes (ANCOVA, F = 0.34, N.S.) or between intercepts (F = 1.30, N.S.). Batch fecundity (Fig. 4B) was therefore calculated for 31 unparasitized and 21 parasitized females pooled, and was found to increase with female size ($Fov = 21.46$ TL $- 194.12$; $r = 0.48$, $P = 0.0003$, $n = 52$). The relative fecundity measured as residuals of the regression of fecundity on size, was not correlated either with parasite numbers ($r = 0.19$, $P = 0.1892$, $n = 53$) or parasite mass ($r = 0.08$, $P = 0.5601$, $n = 53$).

The egg diameter increased significantly with female size in both the unparasitized ($r = 0.63$, $P = 0.0027$, $n = 20$) and parasitized individuals ($r = 0.59$, $P = 0.0070$, $n = 20$). When the regressions of fish length against egg diameter for parasitized and unparasitized females were compared, no significant difference was found between slopes (ANCOVA, F = 0.68, N.S.) or between intercepts (F = 0.87, N.S.). There was no difference between the leading
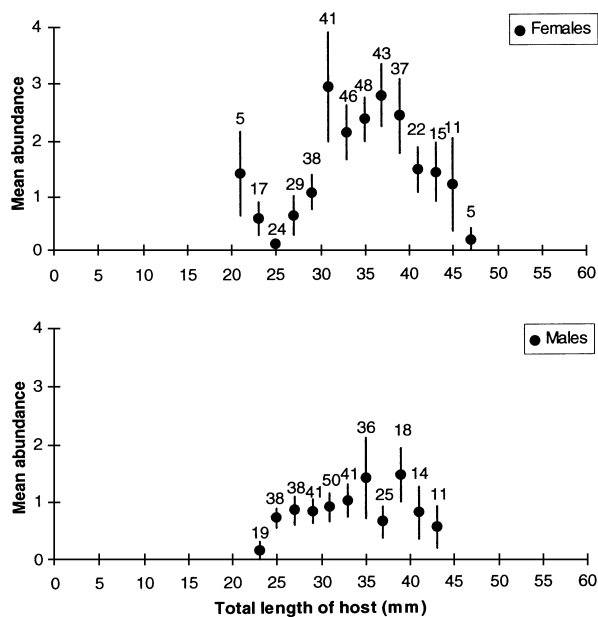
Fig. 2. Changes in mean abundance for *Aphalloides cœlomicola* with size of female and male *Pomatoschistus microps* (2-mm size class). The best regression models were polynomial ($y = -0.002\,x^2 + 1.79\,x - 26.60$ for females; $y = -0.001\,x^2 + 0.53\,x - 7.17$ for males). The number of hosts examined in each length class is indicated above each data point. The vertical bars represent the standard error of the mean.
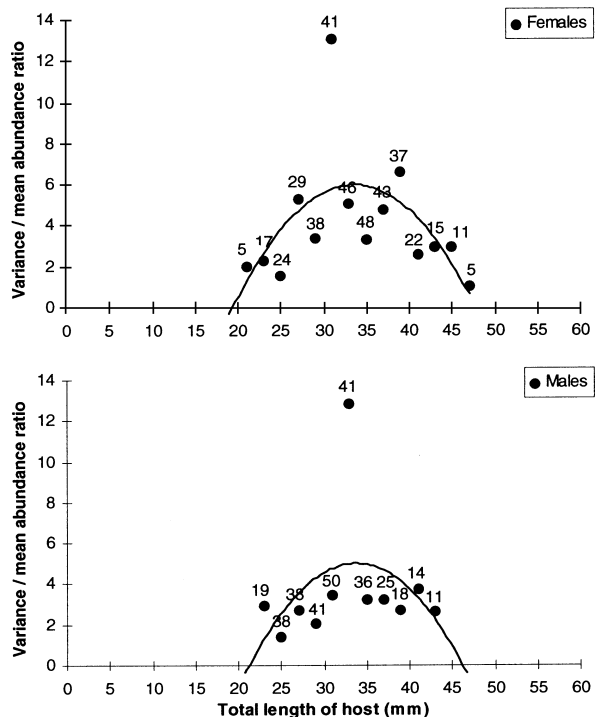

Fig. 3. Changes in variance to mean abundance ratio for *Aphalloides cœlomicola* with size of female and male *Pomatoschistus microps* (2-mm size class). The best regression models were polynomial ($y = -0.03\,x^2 + 1.98\,x - 27.33$ for females; $y = -0.03\,x^2 + 1.93\,x - 30.20$ for males). The number of hosts examined in each length class is indicated above each data point.
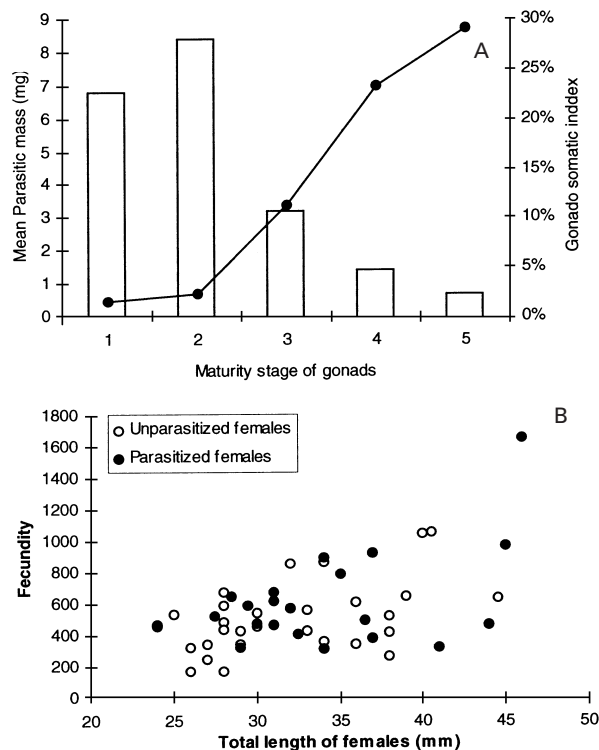

Fig. 4. Effect of the parasite on female reproductive effort. (A) Changes in mean parasite weight (□) and gonado-somatic index (●—●) in relation to the stage of maturity of female gonads. (B) Individual fecundity of parasitized and unparasitized females.

clutch volume (*t*-test, $t = 2.67$, N.S. for the size class 25–35 mm; $t = 0.83$, N.S. for the size class 35–45 mm) of parasitized and unparasitized females.

DISCUSSION

Male versus female infection rates

Males are known to be usually more sensitive to parasites than females due to testosterone synthesis which may exert a cost, decreasing immune competence (Folstad *et al.* 1989; Zuk, 1990; Folstad & Karter, 1992; Zuk, Johnsen & MacLarty, 1995). Display of secondary sexual characters (dependent of testosterone) and courtship are known in male common gobies (Vestergaard, 1976; Miller, 1984) which let us expect higher prevalence and abundance of parasites in males. On the contrary these 2 indices showed that females seemed to be more affected by *A. cœlomicola* than males, revealing that this sex difference could be inverted (Poulin, 1996). Those data suggest that males, showing nesting behaviour are less mobile than females (who continually search for suitable nests to spawn) and are thus less liable to encounter cercariae of this parasite.

Impact of the parasite on the host reproductive effort

As adult parasites are trapped in the abdominal cavity of fishes and the host cannot expel them or prevent infection by cercariae, the greater the

parasite load, the more the female decreases its investment in gonad weight. As neither fecundity nor egg diameter were reduced in parasitized females, we may infer that egg energy content (composition in lipids or proteins) is reduced to decrease the expenditure of energy. No impact of the parasite was observed on gonad weight in males.

As the highest parasitic biomass was observed in the 2 first maturity stages of female gonads and higher parasitic biomasses reduced the gonad weight, *A. cœlomicola* might be able to prevent female common gobies from developing their gonads or to induce gonadal regression, when a certain parasitic biomass is reached. Parasitic castration and gonadal regression due to parasites are well documented for molluscs (Crews & Esch, 1987; Zhokhov & Pugachera, 1995) and crustaceans (Ramirez & Dato, 1989; Amat *et al*. 1991; Thomas *et al*. 1995*a*) but not for fish. Further, the literature on the impact of parasites on fish fecundity (Rosenqvist & Johansson, 1995) and gonadal regression (Gall, McClendon & Schafer, 1972; Wiklund *et al*. 1996) is fairly small. By this phenomenon, *A. cœlomicola* would gain more space in the host to develop and either to produce eggs or increase its number.

Parasite induced host-mortality

The effect of parasitism on host survival is difficult to assess in field studies, we therefore try to use indirect clues. When the parasite is responsible for host death by parasite overloading or energy losses, the graphical plot of the mean abundance and the variance to mean abundance ratio has a convex form (Anderson & Gordon, 1982; Lester, 1984), as observed for *A. cœlomicola*. Moreover, the change in the variance to mean abundance ratio with size confirms that a high parasite load leads to host mortality. As adult parasites are trapped in the abdominal cavity of the fish, the only way for them to evacuate their eggs into the environment is to wait for the death of the fish or to provoke it (Maillard, 1973). It would thus be expected to find host mortality related to high parasitic load, even if this parasite '*a posteriori*' uses a vertical transmission (less aggressive for the host) as defined by Bourguet, Viard & Renaud (1995) and that adverse effects are usually produced by larval stages of parasites (Faliex & Morand, 1994). However, the changes in mean abundance and variance to mean abundance ratio could also be explained by other hypotheses. First, *A. cœlomicola* could reduce and even stop the growth of the host, when the parasitic load reaches a threshold. However, the parasite would gain no direct benefit as this would reduce the fish abdominal cavity volume and thus the available space for the parasite. Secondly, those changes may indicate a density-dependent regulation of parasites as demonstrated for the oxyurid of cockroaches (Morand & Rivault, 1992). Impact of parasites on fish survival was revealed by direct observations of death in some fish species (Delisle, 1972; Bunya *et al*. 1987) but data comparable to ours are scarce (De Meeüs *et al*. 1995). However, the available evidence is inconclusive as our study does not clearly demonstrate the induced host-mortality, although this hypothesis remains the most credible.

Evolution of host life-history traits

Parasites which have adverse effects on the life-history traits of their host, have generally provoked evolution in life-history traits (Møller, 1993). Host adaptations to parasitism that influence gonad development and cause castration can sometimes induce delay in the age of maturity as was shown in fish (Michalakis & Hochberg, 1994) or in marine snails (Lafferty, 1993). As the parasites accumulate in the abdominal cavity of the host, we might expect parasitized female and male gobies to compensate by maturing earlier, which was not the case. However, the impact of parasites on this trait could only be ascertained by comparing sizes at maturation and minimum length at maturation in parasitized and unparasitized populations.

In conclusion, we found that *Aphalloïdes cœlomicola* influences the reproductive effort of female common goby and could even cause gonadal regression above a certain parasite weight. Finally, the durable interaction (Combes, 1995) of this particular host–parasite system is probably ensured by the association of host mortality induced by the parasite and predation.

REFERENCES

AMAT, F., GOZALBO, A., NAVARRO, J. C., HONTORIA, F. & VARO, I. (1991). Some aspects of *Artemia* biology affected by cestode parasitism. *Hydrobiologia* **212**, 39–44.

ANDERSON, R. M. & GORDON, D. M. (1982). Processes influencing the distribution of parasite numbers within host populations with special emphasis on parasite-induced host mortalities. *Parasitology* **85**, 373–398.

BARBER, I. (1997). A non invasive morphometric technique for estimating cestode plerocercoid burden in small freshwater fish. *Journal of Fish Biology* **51**, 654–658.

BOUCHEREAU, J. L. (1994). Bioécologie et tactiques adaptatives d'occupation d'une lagune

méditerranéenne (Mauguio, Languedoc, France) par trois poissons Téléostéens Gobiidés: *Pomatoschistus minutus* (Pallas, 1770), *P. microps* (Krøyer, 1838), *Gobius niger* (Linnaeus, 1758). Doct. d'État, Université de Montpellier II.

BOURGUET, D., VIARD, F. & RENAUD, F. (1995). Les parasites ménagent ils toujours leurs hôtes? *La Recherche* **272**, 90–91.

BUNYA, T., HOSHIAI, G. I., HATAI, K., TAMAI, N. & KUBOTA, S. S. (1987). Mortality of Scorpaneid fish (*Sebastes schlegeli*) fry naturally infected with *Vibrio ordalii* and the histopathology. *Fish Pathology* **22**, 113–114.

COMBES, C. (1995). *Interactions Durables: Écologie et Évolution du Parasitisme*. Masson, Paris.

CREWS, A. E. & ESCH, G. W. (1987). Histopathology of larval trematode infections in the freshwater pulmonate snail, *Helisoma anceps*. *Journal of Invertebrate Pathology* **49**, 76–82.

DELISLE, C. E. (1972). *Henneguya* sp. (Sporozoa: Myxosporida) as a probable cause of death of *Esox lucius* in Brome lake, Québec. *Canadian Journal of Zoology* **50**, 1499–1500.

DE MEEÜS, T., MORAND, S., MAGNAN, N., DO CHI, T. & RENAUD, F. (1995). Comparative host-parasite relationship of two copepods species ectoparasitic on three fish species. *Acta Oecologia* **16**, 361–374.

DOBSON, A. P. & HUDSON, P. J. (1986). Parasites, diseases and the structure of ecological communities. *Trends in Ecology and Evolution* **1**, 11–15.

FALIEX, E. & MORAND, S. (1994). Population dynamics of the metacercarial stage of the bucephalid trematode, *Labratrema minimus* (Stossich, 1887) from Salses-Leucate lagoon (France) during the cercarial shedding period. *Journal of Helminthology* **68**, 35–40.

FOLSTAD, I., NILSSEN, A. C., HALVORSEN, O. & ANDERSON, J. (1989). Why do male reindeer (*Rangifer t. tarandus*) have higher abundance of second and third instar larvae of *Hypoderma tarandi* than females. *Oïkos* **55**, 87–92.

FOLSTAD, I. & KARTER, A. J. (1992). Parasites, bright males and the immunocompetence handicap. *American Naturalist* **139**, 603–622.

GALL, G. A. E., McCLENDON, E. L. & SCHAFER, W. E. (1972). Evidence on the influence of the copepod (*Salmincola californiensis*) on the reproductive performance of a domesticated strain of rainbow trout (*Salmo gardneri*). *Transactions of the American Fisheries Society* **101**, 345–346.

JOYEUX, J. C. (1991). Biologie des populations de *Gobius niger* Linné, 1758, *Pomatoschistus minutus* (Pallas, 1770) et *Pomatoschistus microps* (Krøyer, 1838) (Teleostei, Gobiidæ) dans une lagune nord-méditerranéenne, la lagune de Mauguio-France, Doct. d'État, Université de Montpellier II.

LAFFERTY, K. D. (1993). The marine snail, *Cerithidea californica*, matures at smaller sizes where parasitism is high. *Oïkos* **68**, 3–11.

LAFFERTY, K. D. & MORRIS, A. K. (1996). Altered behavior of parasitized Killifish increases susceptibility to predation by bird final hosts. *Ecology* **77**, 1390–1397.

LEE, S. C. (1974). Biology of the sand goby *Pomatoschistus minutus* (Pallas) (Teleostei: Gobiidæ) in the Plymouth area. Ph.D. thesis, University of Bristol.

LESTER, R. J. G. (1984). A review of methods for estimating mortality due to parasites in wild fish populations. *Helgolander Meeresunters* **37**, 53–64.

MAILLARD, C. (1973). Mise en évidence du cycle évolutif abrégé *d'Aphalloïdes cœlomicola* Dollfus, Chabaud et Golvan, 1957 (trematoda). Notion d'hôte historique. *Compte Rendu de l'Académie des Sciences*, Paris, **277**, série D, 317.

MAILLARD, C. (1976). Diastomatoses des poissons en milieu lagunaire. Doct. d'État. Université de Montpellier II.

MARGOLIS, L., ESCH, G. W., HOLMES, J. C., KURIS, A. M. & SCHAD, G. A. (1982). The use of ecological terms in parasitology (report of an *ad hoc* committee of the American Society of Parasitologists). *Journal of Parasitology* **68**, 131–133.

MICHALAKIS, Y. & HOCHBERG, M. E. (1994). Parasitic effects on host life-history traits: a review of recent studies. *Parasite* **1**, 291–294.

MILLER, P. J. (1963). Studies on the biology and taxonomy of British gobiid fishes. Ph.D. thesis, University of Liverpool.

MILLER, P. J. (1984). The tokology of Gobioid fishes. In *Fish Reproduction: Strategies and Tactics* (ed. Potts, G. W. & Wootton R. J.), pp. 119–147. Academic Press, London.

MØLLER, A. P. (1993). Parasitism and the evolution of host life history. In *Host–Parasite Evolution: General Principles and Avian Models,* (ed. Clayton, D. H. & Moore, J.), pp. 105–127. Oxford University Press, Oxford.

MORAND, S. & RIVAULT, C. (1992). Infestation dynamics of *Blatticola blattae* Graeffe (Nematoda: Thelastomatidae), a parasite of *Blattella germanica* (Dictyoptera: Blattelidae). *International Journal for Parasitology* **22**, 983–989.

POULIN, R. (1996). Sexual inequalities in helminth infections: a cost of being a male? *American Naturalist* **147**, 287–295.

RAMIREZ, F. C. & DATAO, C. (1989). Observations on parasitism by *Thalassomyces fagei* on three euphausiid species in southern Atlantic waters. *Oceanologica Acta* **12**, 95–97.

RENAUD, F. & DE MEEÜS, T. (1991). A simple model of host parasite evolutionary relationship. Parasitism: compromise or conflict. *Journal of Theoretical Biology* **152**, 319–327.

ROSENQVIST, G. & JOHANSSON, K. (1995). Male avoidance of parasitized females explained by direct benefit in a pipefish. *Animal Behaviour* **49**, 1039–1045.

SCOTT, M. E. (1988). The impact of infection and disease on animal populations: implications for conservation biology. *Conservation Biology* **2**, 40–56.

THOMAS, F., LAMBERT, A., DE MEEÜS, T., CÉZILLY, F. & RENAUD, F. (1995a). Influence of *Microphallus hoffmani* (Trematoda, Microphillidæ) on the survival, sexual selection, and fecundity of *Gammarus aequicauda* (Amphipoda). *Canadian Journal of Zoology* **73**, 1634–1639.

THOMAS, F., RENAUD, F., ROUSSET, F., CÉZILLY, F. & DE MEEÜS, T. (1995b). Differential mortality of two closely related host species induced by one parasite. *Proceedings of the Royal Society of London, B* **260**, 349–352.

THOMAS, F., CÉZILLY, F., DE MEEÜS, T., CRIVELLI, A. J. & RENAUD, F. (1997). Parasitism and ecology of wetlands: a review. *Estuaries* **20**, 646–654.

VAES, M. (1978). Infection of the common goby, *Pomatoschistus microps*, with *Aphalloïdes cœlomicola* (Trematoda Digenea). *Vlaams Diergeneeskundig Tijdschrift* **47**, 274–278.

VESTERGAARD, K. (1976). Nest building behaviour in the common goby *Pomatoschistus microps* (Krøyer) (Pisces, Gobiidæ). *Vidensk Meoldr dansk naturh Foren* **139**, 91–108.

WEBB, C. J. (1980). Systematics of the *Pomatoschistus minutus* complex (Teleostei: Gobioidei). *Philosophical Transactions of the Royal Society of London* **291**, 201–241.

WIKLUND, T., LOUNASHEIMO, L., LOM, J. & BYLUND, G. (1996). Gonadal impairment in roach *Rutilus rutilus* from Finnish coastal areas of the northern Baltic Sea. *Diseases of Aquatic Organisms* **26**, 163–171.

ZHOKHOV, A. E. & PUGACHERA, M. N. (1995). Effect of Trematode invasion on the reproductive function of the pea clam *Pisidium amnicum. Ehkologiya* **2**, 130–135.

ZUK, M. (1990). Reproductive strategies and sex differences in disease susceptibility: an evolutionary viewpoint. *Parasitology Today* **6**, 231–233.

ZUK, M., JOHNSEN, T. S. & MACLARTY, T. (1995). Endocrine-immune interactions, ornaments and mate choice in red jungle fowl. *Proceedings of the Royal Society of London, B* **260**, 205–210.