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Reproductive compensation in female *Palaemonetes argentinus* (Decapoda: Natantia) due to *Microphallus szidati* (Trematoda) infection

M. Parietti[1], M.J. Merlo[1], M. Natal[2] and M.A. Méndez Casariego[1]

[1]Laboratorio de Parasitología. Instituto de Investigaciones en Producción, Sanidad y Ambiente (IIPROSAM), Facultad de Ciencias Exactas y Naturales, Universidad Nacional de Mar del Plata, Juan B. Justo 2550 (7600), Mar del Plata, Argentina and [2]Centro Marplatense de Investigaciones Matemáticas (CEMIM), Facultad de Ciencias Exactas y Naturales, Universidad Nacional de Mar del Plata, Funes 3250 (7600), Mar del Plata, Argentina

Research Paper

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Author for correspondence:
M. Parietti,
E-mail: manuelaparietti@hotmail.com

Abstract

Parasites may affect host demographic characteristics because they can directly or indirectly cause the death of their hosts and/or influence their reproduction. Parasitism is therefore recognized as a factor that influences the composition and structure of populations and communities. One of these behaviours is the compensatory response: the host can compensate for the parasite losses effect, modifying the reproductive effort to enhance fitness. Ovigerus female *Palaemonetes argentinus* was collected and sorted into two groups according to the degree of development of their embryos: newly spawned embryos and embryos ready to hatch. The number of embryos and their dry weight for each female were determined. All parts of the female body were checked for parasites. The females of *P. argentinus* were parasitized by *Microphallus szidati*. We found that parasitized females produce more embryos but had more egg loss during development and the percentage of embryonic loss was higher in the parasitized females than in non-parasitized. Parasitized females produced lighter eggs than those from uninfected females. This supports the compensatory reproduction hypothesis suggested for this species. Parasitism can change life history traits in a way that fecundity can be compensated; this co-evolution between host and parasites will be population or context dependent. Parasites are a functional part of any ecosystem and as our results show, deleting parasites in life history traits and reproduction studies in free living organisms could lead to an incomplete picture of the true processes that happen in nature.

Introduction

When a host meets a parasite, it has two strategies: resistance or tolerance. In an evolutionary context, tolerance is the ability to limit the negative fitness effects of a given parasite load (Best *et al.*, 2008) without controlling the infection. If the host can maintain reproductive fitness while being infected, and if resistance results in lower reproductive output, this would select for hosts that can tolerate infections instead of resisting them. A more resistant host that is better able to reduce parasite load could be less successful than a host with a relatively high parasite load with a high tolerance. Because tolerance does not directly affect the parasite load, its effect on parasite prevalence depends on whether the host evolves mortality or fecundity tolerance (Kutzer & Armitage, 2016). If hosts live longer (mortality tolerance), the parasite's infectious period is prolonged and parasite prevalence increases in a population, leading to a positive feedback loop (Best *et al.*, 2008). In contrast, fecundity tolerance has a more complex outcome due to the broader scenarios in host reproduction. According to Kutzer & Armitage (2016) fecundity tolerance could be neutral in terms of fitness, but if it comes at the cost of a reduced host lifespan, the parasite's infectious period will be reduced, affecting, in the end, parasite prevalence.

From a parasite point of view, parasites can manipulate host reproduction using methods that include energetic drain or mechanical or neurochemical disruption (Forbes, 1993). Since energetic drain affects overall host energy budget, infections may alter host life history, changing reproductive investment (absolute amount of energy allocated to reproduction) and reproductive effort (proportion of available energy allocated to reproduction). A reduction in reproductive effort may ensure more energy available to the parasite. From the host point of view, a host may respond to the presence of some parasites by decreasing/increasing its investment into current reproduction. For example, the host may increase reproductive effort in the face of infection and loss of body condition (Schultz *et al.*, 2006). This compensatory behaviour to an adverse environment is especially common for invertebrate hosts (Minchella, 1985; Lafferty, 1993; Krist, 2001; Merlo *et al.*, 2016). Considering parasites as

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an adverse environmental factor, if hosts are unable to resist infection by other means (e.g. immunity) they can compensate for the parasite losses effect, modifying their reproductive effort to enhance fitness. For example, increased egg-laying rates by young *Biomphalaria glabrata* snails, exposed to infective stages of schistosomes, may represent a host-mediated change created to compensate for the reduction in fecundity expected following the proliferation of trematodes (Minchella & LoVerde, 1981; Thornhill *et al.*, 1986). Others may enhance courtship behaviour (Drosophila: Polak & Starmer, 1998; amphipods: McCurdy *et al.*, 2000): several taxa increase offspring number and/or size (snails: Krist, 2001; Daphnia: Chadwick & Little, 2005; mice: Schwanz, 2008, fishes: Heins, 2012) and/or greater parental effort (Schwanz, 2008). In these cases, fecundity compensation may be viewed as a tolerance mechanism, by increasing host fitness via mechanisms that do not act directly on reducing parasite densities (see Vale & Little, 2012 for discussion).

Therefore, fecundity compensation could be one of the host's responses to maintain fitness in the presence of parasites. Nevertheless, individuals who have been exposed to parasites could be expected to have distinct life history traits from individuals who have not; and, depending on their plasticity, they may not have the same results in all environments. According to their plasticity, changes could be observed in different features of its life history or different compensatory responses according to the co-evolutionary history in which it is found. The natantian shrimp *Palaemonetes argentinus* Nobili, 1901 is a very good species model to discuss this scenario. It has been reported in Argentina as the second intermediate host for two digenean species belonging to the family Microphallidae: *Microphallus szidati* Martorelli, 1986, and *Levinseniella cruzi* Travassos, 1920 (Martorelli, 1986, 1988; Parietti *et al.*, 2015). A previous work (Merlo *et al.*, 2016) showed that parasitized *P. argentinus* females laid more eggs and have more embryos ready to hatch than uninfected ones of the same size, despite parasitized shrimps losing more embryos through development. Given that parasitized shrimp females were analysed in one shallow lake, and non-parasitized ones in another, we wanted to test the generality of these results and see if parasitized females laid more eggs as a compensatory response due to the higher egg loss. We evaluated female fecundity in *P. argentinus* in La Brava (LB) shallow lake, in which there are parasitized and non-parasitized individuals, unlike the lake previously studied in Merlo *et al.* (2016) in which 100% of the population was parasitized.

Materials and methods

Study design

The study site is located in Buenos Aires Province, Argentina. LB is a shallow-water lake (37°52′S, 57°58′W), surrounded by a serrano cord and has a surface area of about 400 ha and a mean depth of 3.5 m. Shrimps were collected in October and December 2016 to maximize the chances of finding shrimps with embryos in the early stages and advanced stages of development respectively (Ituarte *et al.*, 2007). Ovigerous female shrimps only were taken with a hand net (1 mm mesh size) and immediately transported alive to the laboratory with water from sampling sites. Specimens were always caught along the lakeside with submerged vegetation (depth *c.* 0.5 m). The net was dragged several times until *c.* 200 individuals were collected each sampling. In the laboratory, ovigerous females were sorted into two groups according to the degree of development of their embryos. The two stages of embryonic development used corresponded to those described by Ituarte *et al.* (2007): newly spawned embryos (SI) and embryos ready to hatch (SIII). Realized fecundity (N SI) was defined as the number of SI embryos per female and actual fecundity (*N* SIII) as the number of SIII embryos per female (Ituarte *et al.*, 2007). Only females with SI embryos were used from the first sampling (October), and even when we caught SI embryos in the second sampling (December) only SIII females were used. In this way, the same cohort of ovigerous females was compared. Dry weight of SI embryos was determined on a H54 Mettler AR balance to the nearest 0.01 mg. Embryos were transferred to pre-weighed capsules of aluminium foil and dried at 90°C to constant weight (at least 24 h). The size of females was estimated as the carapace length (CL, mm), from the posterior orbital margin to the dorso-posterior border of the carapace, using an Olympus SZ40 stereomicroscopy.

Females were kept alive in individual plastic beakers (100 ml) until dissection, usually within 24 h of collection. All parts of the female body were checked for parasites, including the appendages. After dissection, parasites were identified and the number of metacercarial cysts was counted.

Data analysis

Two quantitative descriptors of parasite populations were used: the intensity of infection (number of individuals of a particular parasite species in a single infected host), and mean intensity (total number of parasites of a particular species/the number of hosts infected with that parasites) (Bush *et al.*, 1997). The difference in mean intensities between stages of embryonic development (SI vs. SIII) in parasitized females was tested by Student *t*-tests.

In the first instance, the differences in CL were analysed using a two-way analysis of variance (main factors: embryonic stage and parasitized). The results of this analysis made it possible to determine whether CL should be used as a covariate in subsequent tests.

To evaluate if parasitism negatively affects egg production and the egg development from *N* SI to *N* SIII, a parametric analysis of covariance (ANCOVA) of two fixed factors with interaction was designed. An ANCOVA model was performed using the following model: egg production ~ CL + embryonic stage × parasitized. The fixed factors are the embryonic stage with two factors (SI–SIII) and parasitized with two factors (yes–no). CL was used as the covariate.

To incorporate the qualitative predictor within the framework, qualitative predictors need to be coded using dummy variables. For a k-level predictor, k-1 dummy variables are needed for the representation. Compliance with the model assumptions were verified using the residuals. The Tukey honestly significant difference test was used to test differences among individuals.

The brood loss was calculated in both female groups as a percentage using equation modified from Ituarte *et al.* (2007). The difference in dry weight of SI embryos between parasitized and non-parasitized females was tested by a Student *t*-test. CL was not used as the covariate because sizes between parasitized and non-parasitized females showed no differences (see Results). Analyses were done using the statistical programming language R, version 2.15.2. (R Development Core Team, 2018).

Results

A total of 199 bearing females were examined: 50 non-parasitized females and 50 parasitized females had newly spawned embryos

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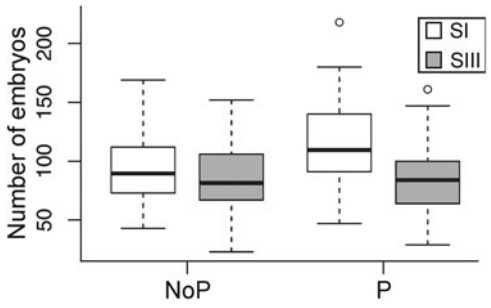

Fig. 1. Total number of newly spawned embryos (SI) and embryos ready to hatch (SIII) in parasitized (P) and non-parasitized (NoP) females of *Palaemonetes argentinus* from La Brava lake.

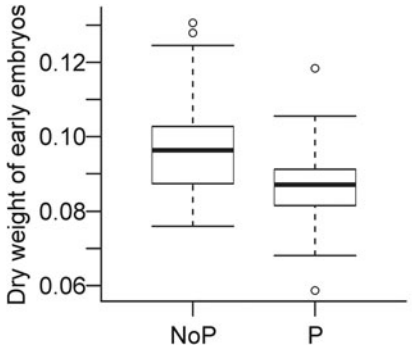

Fig. 2. Dry weight of newly spawned embryos (SI) in parasitized (P) and non-parasitized (NoP) females of *Palaemonetes argentinus* from La Brava lake.

(SI) and 50 non-parasitized females and 49 parasitized females had their embryos in an advanced developmental stage (SIII). The intensity of infection ranged from 1 to 54 cysts in shrimps with SI embryos (mean value 11.4 cysts per host, $n = 50$, SD = 13.1) and from 1 to 73 cysts in shrimps with SIII embryos (mean value 7.43 cysts per host, $n = 49$, SD = 14.3). The mean intensity was not significantly different between females with advanced (SIII) than newly spawned embryos (SI) (Student's t-test: $t = 1.43$, $P = 0.1572$).

CL ranged from 4.81 to 9.46 mm and differences between embryonic stage was detected ($F_{1;195} = 61.08$, $P < 0.001$), but no differences between parasitized vs. non-parasitized females were found ($F_{1;195} = 0.24$, $P = 0.62$), nor in the interaction between factors ($F_{1;195} = 3.84$, $P = 0.06$).

Realized and actual fecundity differed within parasitized and non-parasitized females and an effect of female sizes was detected (ANCOVA Parasitized × Stage: $t_{1,194} = -2.070$, $P = 0.039$; CL: $t_{1,194} = 4.786$, $P < 0.001$) (fig. 1). Realized fecundity in parasitized females was 115.8 (±36.5) eggs and in non-parasitized ones was 92.9 (±29.7) eggs. While the actual fecundity in parasitized females was 85.5 (±29.7) eggs and in non-parasitized ones was 85.6 (±29.4) eggs. The ANCOVA model residuals were adjusted to a normal distribution, $P = 0.84$ corresponding to the Shapiro Wilks test. A check on the diagnostics of the ANCOVA model revealed no particular problems. Non-parasitized females present no differences between realized and actual fecundity ($P = 0.064$), but parasitized females presented a significant egg loss during embryonic development ($P < 0.001$). Also, differences were detected NSI between the two groups of females ($P = 0.002$) but no for NSIII ($P = 0.99$). The percentage of embryonic loss was higher in the parasitized females than non-parasitized (26.16% vs. 7.79%).

Dry weight of early embryos (SI) differed between parasitized and non-parasitized females (Student's t-test: $t = 4.54$, $P < 0.01$). Parasitized females produced lighter eggs (0.086 mg dry weight per embryo, $n = 50$, SD = 0.009) than those from uninfected females (0.097 mg, $n = 50$, SD = 0.01) (fig. 2).

Discussion

In this work, we showed that egg loss is higher in parasitized populations of *Palaemonetes argentinus*. Nevertheless, parasitized females produce more embryos than non-parasitized ones. This indicates that parasitized females produce more embryos, but had more egg loss during development. This supports the compensatory reproduction hypothesis suggested for this species.

In response to adverse environmental conditions, many species have evolved phenotypic plastic responses that can modify life history traits to improve fitness costs. One of these compensatory responses is a shift towards greater reproductive effort earlier in life (i.e. fecundity compensation) when the expectation of future reproduction decreases (Minchella & LoVerde, 1981; Forbes, 1993; Lefevre *et al.*, 2008). Considering parasites as an adverse environmental factor, hosts compensate for the parasite losses effect producing more eggs to compensate for the reduction in fecundity expected later as we showed here with *P. argentinus*. Reproductive compensation in response to parasites is considered as a non-immunological defence response in the framework of evolutionary processes and has been studied in different taxa (Parker *et al.*, 2011; Duffield *et al.*, 2017). Examples in crustaceans are scarce (see Duffield *et al.*, 2017), but compensation mechanisms have been observed in *Daphnia magna*, which produces eggs earlier in life if exposed to microsporidian parasite (Chadwick & Little, 2005), and in *P. argentines*, which produces more eggs if they are infected with trematodes (Merlo *et al.*, 2016). All different scenarios assume a trade-off between energy drain due to the parasite, and the subsequent response to allocate the remaining energy to future reproduction or growth. One important issue about what to expect is to consider the life span of the host and the possible reproductive outcomes. *Palaemonetes argentinus* completes its life cycle in 15 months and some females can live up to 2 years. In Argentina, the first ovigerous females can be found in September (Spivak, 1997; Ituarte, 2008). The reproductive season ends in February–March, and a single female can produce at least two broods in a single reproductive season (Ituarte *et al.*, 2010), but only a few females produced a second hatch in the season (Spivak, 1997). Since egg loss is unavoidable, the female shrimp should produce a greater number of lower quality (smaller) offspring during the early season to compensate for egg losses as a result of being parasitized.

The higher egg loss throughout embryonic development in parasitized *P. argentinus* females is most likely a negative effect of infections. Metacercarial cysts of the parasite *M. szidati* usually encyst in shrimp abdominal muscles (Martorelli *et al.*, 2006; Parietti *et al.*, 2015). A larger number of metacercariae in the abdominal muscles could interfere with the care activities of ovigerous females, interfering with ventilation and/or cleaning of embryos. Decreasing care activity that protects against hypoxia and pathogens could, therefore, be related to higher egg loss during embryonic development. Moreover, care activities in most decapods increase through the end of the embryonic development (e.g. Baeza & Fernández, 2002; Giovagnolli *et al.*, 2014).

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The production of lighter eggs could be considered also a compensatory response: on one hand, parasitized shrimps have to face an energetic drain. Producing lighter eggs could be a trade-off that allows them to produce more eggs with the same investment. This is a well-known strategy among marine species, ranging from very few but large eggs or lots of small ones (Smith & Fretwell, 1974; Rollinson & Hutchings, 2013). Given that this compensation in reproductive investment comes with some costs, this trade off can be observed in other species: infected female sticklebacks lay more eggs, but these eggs are smaller (Heins, 2012). On the other hand, if parasites affect the care ventilation activity mentioned earlier, lighter eggs could facilitate aeration of embryos. Given that egg volume increases throughout embryonic development, an increment in egg volume may imply a lack of space in the incubation chamber provoking egg loss through embryonic development (Ituarte *et al.*, 2007). During that time, females generate water flow around the embryos by beating their pleopods that could carry away spores. Also, using their chelipeds mother shrimp could also keep embryos free of microbial and sediment particles (Fisher, 1983; Caceci *et al.*, 1996; Bauer, 2004). So, heavier eggs or a larger clutch mass will require more energy to ventilate. In some species, smaller eggs hatch earlier (Einum & Fleming, 2000) and even if we did not evaluate this, it may be another possibility that could explain a trade off in energy. Nevertheless, another possibility is that the body condition of parasitized females would be lower than that of non-parasitized females, affecting the reproductive physiology of infected females. In this case, lighter eggs may reflect only the energy state of the female, leaving the number of eggs as the only compensatory response.

Parasitism can change life history traits in a way that fecundity can be compensated. So, it is reasonable to think that the traits involved in the reproductive compensation will be population or context dependent (Scharsack *et al.*, 2016). A population of hosts that never faced a pathogen will present life history traits very different from other populations. When we compare the results with the previous studies done in Nahuel Rucá lake (NR) (Merlo *et al.*, 2016) we can detect some particularities respect to the female shrimps analysed here. The females at NR had the highest parasitic load (number of metacercariae) and all of them were parasitized (100% prevalence). The intensity of infection triple that observed in females from LB in SI (35.95 vs. 11.4) and was nine times over in SIII (69.44 vs. 7.43) (Merlo *et al.*, 2016). However, these infected females from NR lose fewer eggs throughout development (18.6% egg lose NR vs. 26.16% LB). The tolerance mechanism of parasitism seems to be more intense in NR respect to LB, in which a possible long-term interaction between host and parasite may lead to more plastic responses, avoiding in this way the heavy effect over host reproduction. It is clear here that this species is well adapted to this relationship, with no need to invest more in resistance towards infection.

The dry weight of parasitized females' eggs in both lakes (NR and LB) have the same weight (w = 0.086 mg × egg) while non-parasitized females measured in this work produced heavier eggs (w = 0.097 mg × egg). Particularly, in El Burro lake (EB) no first intermediate host, *Heleobia parchappii*, was registered, and no other molluscs that will act as host (Merlo *et al.*, 2016). For this reason, shrimps have no parasites and, so, it has no compensatory response and the lighter eggs of all (w = 0.081 mg × egg). This lake (EB) could be showing less phenotypic plasticity than other lakes because it never faced strong parasitic pressure.

Parasites play an important role in structuring communities; however, their presence is often ignored. As we saw here, parasites can produce modifications in life history traits of their hosts. These traits (fecundity, reproduction, growth rate and others) are an object of selection, so natural selection should probably act towards the capability of altering those historical traits in face of parasitism. Life history traits works in crustaceans are common (Korpelainen, 1986; Devin *et al.*, 2004; Santangelo *et al.*, 2018), but they are mainly focused on physical parameters, competition, predation and only in a small way, parasites. Parasites are a functional part of any ecosystem and as our results show, removing parasites in life history traits and reproduction studies in free living organisms could lead to an incomplete picture of the true processes that happen in nature.

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Ethical standards. None.

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