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Life history and virulence are linked in the ectoparasitic salmon louse *Lepeophtheirus salmonis*

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virulence evolution.

Abstract

Models of virulence evolution for horizontally transmitted parasites often assume that transmission rate (the probability that an infected host infects a susceptible host) and virulence (the increase in host mortality due to infection) are positively correlated, because higher rates of production of propagules may cause more damages to the host. However, empirical support for this assumption is scant and limited to microparasites. To fill this gap, we explored the relationships between parasite life history and virulence in the salmon louse, *Lepeophtheirus salmonis*, a horizontally transmitted copepod ectoparasite on Atlantic salmon *Salmo salar*. In the laboratory, we infected juvenile salmon hosts with equal doses of infective *L. salmonis* larvae and monitored parasite age at first reproduction, parasite fecundity, area of damage caused on the skin of the host, and host weight and length gain. We found that earlier onset of parasite reproduction was associated with higher parasite fecundity. Moreover, higher parasite fecundity (a proxy for transmission rate, as infection probability increases with higher numbers of parasite larvae released to the water) was associated with lower host weight gain (correlated with lower survival in juvenile salmon), supporting the presence of a virulence–transmission trade-off. Our results are relevant in the context of increasing intensive farming, where frequent anti-parasite drug use and increased host density may have selected for faster production of parasite transmission stages, via earlier reproduction and increased early fecundity. Our study highlights that salmon lice, therefore, are a good model for studying how human activity may affect the evolution of parasite virulence.

Introduction

Natural selection tends to maximize the lifetime reproductive success of individuals by optimizing combinations of life history traits such as survival, age at maturity and fecundity. Nevertheless trade-offs between these traits constrain their evolution. For example, in a wide range of taxa, longer time to maturity is associated with increased lifespan, but reduced fecundity (Read & Harvey, 1989; Promislow & Harvey, 1990; Stearns, 1992; Roff, 2002). Opposite patterns have been observed in other taxa (e.g. nematodes), with life history strategies varying from slow-maturing, but highly fecund, species to fast-maturing species with low fecundity (e.g. Skorping *et al.*, 1991; Morand, 1996). Parasitic organisms face the additional constraint of needing to exploit their hosts for their own reproduction and therefore display a certain level of virulence (i.e. parasite-induced increase in host mortality), which may in turn affect their own fitness. Increasing the rate of host exploitation may allow parasites to grow faster (and hence achieve earlier maturity) and/or produce more propagules, but at the assumed cost of increasing the mortality rate of their host, on which they rely for their own survival and transmission. The vast majority of models of virulence evolution are based on the assumption of such a trade-off between parasite transmission and survival (the higher

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the virulence, the shorter the host, and in turn parasite longevity). For saturating trade-off curves, that is, when virulence increases quicker than transmission, models allow to predict the evolution of an intermediate optimal level of virulence that can be modulated by ecological factors such as host background mortality (e.g. due to predation or culling), host population density and the rate of multiple infections (May & Anderson, 1983; Ebert & Herre, 1996). This 'trade-off model' provides a general and intuitive framework for explaining within-species variation in parasite virulence (reviewed in Alizon *et al.*, 2009; see also Schmid-Hempel, 2011 pp. 312–352). However, empirical support for a relationship between parasite transmission and virulence is scant (e.g. Lipsitch & Moxon, 1997; Jensen *et al.*, 2006; de Roode *et al.*, 2008), and the examples listed in recent reviews (e.g. Alizon *et al.*, 2009; Schmid-Hempel, 2011) are limited to microparasites (viruses, bacteria, protozoans). Therefore, more experimental studies – in particular using macroparasites – on the correlation between virulence and transmission have been called for (Ebert & Bull, 2003; Gandon & Day, 2003).

This lack of studies on a central assumption in the theory of virulence evolution may partly be due to methodological and ethical difficulties in measuring transmission and virulence in experimental systems. In observational studies, confounding factors cannot be excluded and unavoidable variation among hosts, for example in immunity, can blur the results (Alizon *et al.*, 2009). Additionally, the strength of such a relationship depends on the type of parasite considered. Macroparasites (e.g. helminths and arthropods) were suggested to generally show lower levels of virulence than microparasites (Hudson & Dobson, 1995), making relationships between virulence and transmission in such parasites harder to detect.

In the current study, we explored the relationships between parasite age at first reproduction (as a proxy for parasite within-host growth rate), parasite fecundity (as a proxy for transmission potential) and disease severity (as a proxy for virulence), using the crustacean copepod *Lepeophtheirus salmonis* (Kroyer, 1837) ectoparasitic on Atlantic salmon *Salmo salar* L. We had the double aim of testing whether transmission and virulence are linked in this system and of investigating the potential for virulence to constrain the evolution of life history traits in *L. salmonis*.

Materials and methods

Study system

Lepeophtheirus salmonis, commonly named salmon louse, is a natural marine, exclusively horizontally transmitted ectoparasite of salmonids that has now become a major pest in both farmed and wild salmon throughout the Northern Hemisphere (Pike, 1989; Pike & Wadsworth, 2000). Its life cycle consists of ten successive developmental stages. Maturity is reached approximately 60 days post-infection at 10 °C. Pre-adult and adult salmon lice are mobile on the fish host and actively graze on mucus, skin and blood. Highly infested salmon often have severe skin wounds causing osmoregulatory stress, altered feeding behaviour, reduced growth and increased risk of secondary infections (e.g. Dawson *et al.*, 1998, 1999). Soon after mating, adult female lice start extruding fertilized eggs enclosed in a matrix that binds the eggs together in egg strings. Egg strings remain attached to the female until hatching while being physiologically independent (reviewed in Pike & Wadsworth, 2000). Female lice thereafter keep producing egg strings (up to 11 successive pairs of egg strings have been documented) at regular, temperature-dependent intervals for the rest of their life (e.g. every 10 days on average at 10 °C, Heuch *et al.*, 2000). Egg strings can easily be detached from the female and measured under the microscope.

Experimental set-up

Maintenance of the fish

In this experiment, we used 29 Atlantic salmon smolts (approximately 80–100 g) coming from the same cohort (Industry Laboratory, Bergen, Norway) and kept individually in 30-L tanks supplied with a constant flow of filtered, UV-treated seawater (flow rate, 6 L min^{-1}; temperature, 8.6–9.5 °C; salinity, 35 ppm) and 12 h daylight. The fish were fed twice a day with a standard commercial 3-mm pellet diet (1 g/day per fish).

Infection of the fish

To increase variability in fecundity and virulence, *L. salmonis* from two different locations (origin) were used in this experiment, the first from Austevoll, near Bergen (western Norway), the second from the Oslo fjord (eastern Norway). Eggs from the two origins were collected within the same week (15–21 October 2009). Lice from both origins were grown for three generations prior to the start of the experiment (in 500-L tanks containing 20 fish each) to remove effects caused by different environments. The pool of copepodid larvae used to infest the fish came from mothers that had been checked to be free of *Paranucleospora theridion*, a microsporidian parasite of salmon known to be vectored by salmon lice (PCR detection methods described in Nylund *et al.*, 2010).

Prior to infection with *L. salmonis*, salmon smolts were anesthetized in a solution of metacain (80 mg L^{-1}), measured for weight and length and placed in their respective tanks for recovery. The same day they were infected with *L. salmonis* copepodid larvae using a method derived from Glover *et al.* (2001). The water flow was stopped and water was lowered to one-third of the original level. Forty 2- to 5-day-old copepodids were poured into each tank. After 1 h, water flow was started again and water rose up to a normal level. Air was

delivered directly into the tanks for the whole duration of the infection process. Initially, 15 fish were infected with copepodids of the Bergen origin and 14 fish with copepodids of the Oslo origin. Two of the 15 fish infected with lice from Bergen carried only adult males. Therefore, this study was performed on 13 fish carrying lice from Bergen and 14 fish carrying lice from Oslo.

Monitoring and handling of fish and lice

From day 40 post-infection onwards, fish were inspected daily and the number of gravid and nongravid female lice per fish recorded. For each fish, female lice produced egg strings rather synchronously (< 8 days between first and last egg string produced), which enabled us to wait until all female lice on a given fish were gravid before anaesthetizing the fish, measuring it and collecting the lice. Immediately after the measurements, the fish were placed back in their tanks for recovery. Egg strings were detached from female lice genitalia by gently pulling them with a fine forceps. Pictures of egg strings, female and male lice were taken using Leica Application Suite connected to a Leica Z16APOA microscope (Leica Microsystems). Adult females and males were put back on the same fish until production of the next pair of egg strings. The development of infections was monitored for 135 days, after which the experiment was terminated and the fish killed using a concentrated anaesthetic solution.

Measures of lice fecundity, skin damage and fish growth

At each anaesthesia, the fish were measured for body length to the nearest 0.5 cm and weighed to the nearest gram. A transparent plastic film was applied on the fish, and lesions on the body surface caused by *L. salmonis* were traced using a permanent marker. Drawings were scanned, and total skin damage area was measured to the nearest mm^2 in Image J v. 1.43 for Windows (http://rsbweb.nih.gov/ij). The total length of each egg string was measured to the nearest 0.1 mm from pictures taken with a low magnification (3.5 ×). The total number of eggs in each egg string was estimated from five counts made using pictures taken with a higher magnification (20 ×) at five distinct places along the egg string. 'Total egg production' was defined as the total number of lice eggs produced per fish from the start of the experiment. Specific weight (or length) gains were calculated as the weight (or length) gain from the start of the experiment divided by the initial weight (or length) of the fish.

Statistical analysis

Age at first reproduction and fecundity

The number of eggs produced is lower and more variable in the first and second clutches and seems to stabilize afterwards (Heuch *et al.*, 2000, A. Mennerat, unpublished). In addition, the number of eggs produced in the first clutch is not correlated to the number of eggs produced in subsequent clutches (A. Mennerat, unpublished). We therefore considered fecundity over the three first reproductive events as an appropriate estimate of individual fecundity of salmon lice. However, lice could not be individually marked, and it was therefore impossible for most individual females to track them over three consecutive reproductive events. For the same reason, we could not use average values of fecundity per fish because some lice were lost before completing their third clutch, and it was impossible to identify them. As a consequence, we could only obtain reliable, individual measures of fecundity over the three first clutches from 13 female lice from 12 different hosts. The relationship between fecundity and age at first reproduction in those 13 female lice was tested using linear regression in the R software (R Development Core Team 2011).

Fecundity and virulence

The final data set included 80 female lice from 27 fish. We used repeated measures of total area of skin damage, specific weight gain and specific length gain as indicators of salmon lice virulence. Area of skin damage was square-root-transformed to meet requirements for homogeneity and normality of residuals. Summary statistics of the variables used here are presented in Table 1.

Some female lice were lost during the course of the experiment. To account for the decrease in the number of female lice per fish over time, we defined infestation intensity as the average number of female lice per fish from the start of the experiment. Total lice egg production (number of eggs produced from the start of the experiment) was positively correlated with both infestation intensity ($R^2 = 0.16$, $P < 10^{-4}$) and time ($R^2 = 0.44$, $P < 10^{-4}$). To avoid multicollinearity among explanatory variables, in this analysis we defined 'fecundity' as the residuals of linear regressions of total egg production, first on infestation intensity and then on date post-infection.

The relationship between fecundity and virulence was tested using mixed-effects models with virulence

Table 1 Summary statistics of the variables used in the mixed-effects models. Total fecundity is the total number of lice eggs having been produced per fish at the end of the experiment. Initial fish weight and length were measured on day of infection. A total number of 80 female salmon lice carried by 27 fish (Bergen origin: 13 fish; Oslo origin: 14 fish) were used in this study.

	Min	Max	Mean	SD
Number of female lice per fish	0	6	2.0	1.2
Lice age at reproduction (days P.I.)	50	59	53.4	2.0
Total lice fecundity (number of eggs)	829	13 611	5610	3132
Area of fish skin damage (mm^2)	0	671	164.7	122.6
Initial fish weight (g)	80	170	123.2	20.6
Final fish weight (g)	153	301	236.4	35.7
Initial fish length (cm)	21	26.5	24.1	1.2
Final fish length (cm)	25.5	32	29.2	1.6

measures as dependent variables and fish as random effect factor. In all initial models, date post-infection, infestation intensity and fecundity (as defined above) were included as covariates. To account for potential differences in virulence between the two different origins of lice used in this experiment, we included origin as a fixed effect and its interaction with fecundity in all initial models. The initial number of female lice per fish, which may be considered as a measure of infectivity, did not differ between the two origins (Bergen: $N = 13$, Oslo: $N = 14$, $t_{22} = 1.5$, $P = 0.15$).

Because virulence was measured repeatedly over time, we expected to find some degree of temporal autocorrelation (i.e. values measured at a certain point in time were not independent from previously measured values). We therefore included first-level temporal autocorrelation using the corCAR1 correlation structure in all models and tested whether models were significantly improved by comparing their AIC with those of the former models. All models were significantly improved (all $P < 0.001$), and therefore we kept this structure in subsequent analyses.

From initial models, we performed backwards model selection based on AIC values. We tested the significance of each variable in final models using likelihood-ratio tests and validated the models by checking for normality and homogeneity of residuals. All analyses were performed using the NLME package (Pinheiro *et al.* 2011) in the R software (R Development Core Team 2011).

Results

Age at first reproduction was negatively related to fecundity in individual female lice ($N = 13$, $F_{1,11} = 14.42$, $P = 0.003$, Fig. 1). The final model explaining variation in skin damage area included time, origin and infestation intensity as explanatory variables. Skin damage area decreased over time ($P < 10^{-4}$) and increased with infestation intensity ($P < 10^{-4}$). Skin damage was larger on fish infested with lice from the Bergen area as compared to the Oslo lice ($P = 0.02$) (Table 2).

Final models explaining specific weight and length gains included both time and fecundity as explanatory variables. Specific weight and length gains both increased with time ($P < 10^{-4}$). Specific weight gain decreased significantly with increasing fecundity ($P < 10^{-3}$, Fig. 2), but not specific length gain ($P = 0.10$) (Table 2). The relationship between specific weight gain and fecundity remained significant ($P < 10^{-3}$) after removing the data point with the very highest average lice fecundity from the data (point furthest right in Fig. 2).

Discussion

In this study, we found that parasite age at first reproduction and early fecundity, two important life history traits for an ectoparasite such as *L. salmonis*, are strongly correlated. Earlier reproducing lice are also the more fecund, which contrasts with patterns observed in comparative studies of other parasitic taxa, especially nematodes (Skorping *et al.*, 1991; Morand, 1996). This may be explained by the fact that, unlike what is observed in nematodes, adult body size in salmon lice is related neither to age at maturity nor to fecundity (A.Mennerat, unpublished). Our results were obtained in laboratory conditions after standardized breeding of lice for three generations, suggesting a genetic correlation between the two traits. Arguably, the observed negative correlation might be driven by other third factors, for example differences in immunocompetence among the fish hosts. Higher levels of host immune response might both slow down development and reduce fecundity of

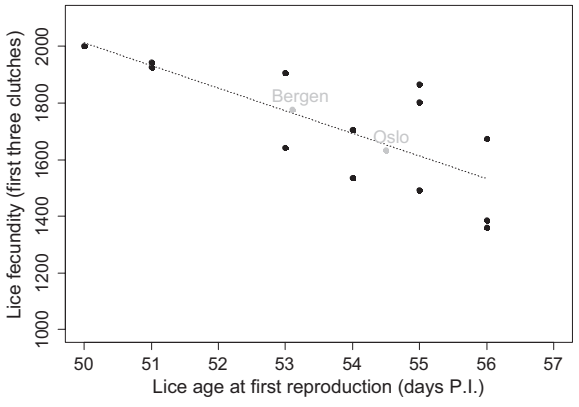

Fig. 1 Regression of lice fecundity (as measured over the first three clutches) on lice age at first reproduction. Black dots represent individual adult female *Lepeophtheirus salmonis*. $F_{1,11} = 14.42$, $P = 0.003$. Grey dots indicate average values for the two different origins of lice.

Table 2 Effects of time (days post-infection), origin of lice, infestation intensity (number of female lice per fish) and average fecundity of lice (total lice egg production per fish corrected for infestation intensity and time) on repeated measures of disease severity in salmon. Final models resulting from backwards selection based on AIC values. Residual degrees of freedom for each model are indicated in brackets. All three models are mixed-effects models including fish as random effect and accounting for first-level temporal autocorrelation (see Methods). LR, likelihood ratio.

	d.f.	LR	*P*
Skin damage	(151)		
Time	1	15.70	10^{-4}
Origin	1	5.95	0.02
Infestation intensity	1	40.90	$< 10^{-4}$
Specific weight gain	(152)		
Time	1	448.87	$< 10^{-4}$
Fecundity	1	13.14	$< 10^{-3}$
Specific length gain	(152)		
Time	1	286.07	$< 10^{-4}$
Fecundity	1	2.74	0.10

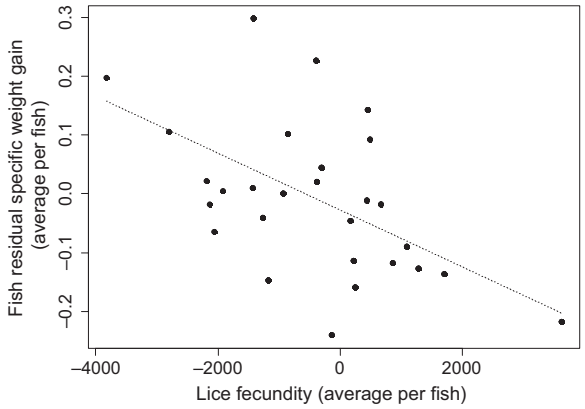

Fig. 2 Relationship between specific salmon weight gain (corrected for time) and lice fecundity (total number of lice eggs produced per fish corrected for both infestation intensity and time). Dots represent average values per fish (up to nine repeated measures). Linear regression: $F_{1,180} = 61.86$, $P < 10^{-4}$. Mixed-effects model: $P < 10^{-3}$ (see Methods and Table 2).

lice. In this case, we would expect to find a relationship between age at reproduction of the focal lice and the average age at reproduction of other lice carried by the same fish, which was not the case ($N = 11$, $F_{1,9} = 0.20$, $P = 0.66$). Other ecological explanations might be proposed, for example, that differences in the microhabitat exploited by individual lice could have affected both developmental rates and fecundity. We do not think it is likely in our study system because the average number of lice per fish was low (Table 1) and so that there was little within-host competition. In addition, being mobile on the fish, the lice could use more than one location during the experiment. Finally, consistent with the hypothesis that the negative correlation is based on genetic differences among females is the observation that the average values for the two lice populations used in this study closely follow the correlation line (see Fig. 1).

Genotypes that both mature early and are more fecund should have a selective advantage over slower, less fecund genotypes. The observed substantial variation in these traits, however, indicates that there are costs associated with such a 'fast' life history strategy. Our results suggest that higher virulence of the more fecund salmon lice may be one such cost. We indeed found that higher average fecundity of lice per fish was associated with lower rate of fish growth. This association was strongly significant and none of the other variables included in the initial model could explain the observed variation in fish growth rate. There is of course still a substantial amount of the variation in virulence that could not be explained in our study (see Fig. 2). A better resolution might be observed if lice were followed individually. In our study, we could not measure individual virulence of lice but only the collective harm caused to their host, because lice could not be individually marked, and as they are mobile on the fish, it was not possible to associate each skin lesion with an individual louse. Nevertheless, the observed negative correlation between lice fecundity and fish growth indicates that virulence may be an important factor constraining evolution towards higher fecundity of salmon lice. In addition, other factors such as a trade-off between early and late fecundity in *L. salmonis* or stronger effects of the host's immune response against the 'faster' parasites might also affect their life history evolution. The present study did not allow us to investigate such effects, and more work is therefore needed to better understand how life history and virulence evolve in this system.

As proxies for virulence, we chose to use indices of disease severity that reflect both the direct, short-term effects of lice on the fish (skin damage) and their indirect, long-term costs in terms of fish growth. Post-smolt growth is a good predictor of survival and recruitment rates in wild Atlantic salmon populations (Friedland *et al.*, 2005; Peyronnet *et al.*, 2007) and therefore seems an appropriate proxy for virulence in our system. Our results show contrasting short-term and long-term effects of salmon lice on their hosts. Skin damage area was related to infestation intensity, but not to average fecundity. This may partly be explained by the fish's ability to heal their wounds, as reflected in the significant decrease in skin damage area over the duration of the study. In contrast, we found that higher average fecundity was associated with lower specific weight gain. This is consistent with the view that for ectoparasites virulence, results from a long-lasting cost of infection (i.e. a long-term decrease in host condition) rather than acute pathogenicity (e.g. causing a peak in mortality shortly after infection) and can only be detected by monitoring hosts over a long period of time. There may be two nonexclusive mechanistic explanations to this long-term effect of salmon lice. It may stem from consistently higher rates of host exploitation by more fecund parasites. But it may also reflect a trade-off between growth and immune response in juvenile salmon, as in many other vertebrate taxa (e.g. Sheldon & Verhulst, 1996). Lice with higher fecundity, by exploiting their host more intensely, may elicit a stronger and therefore more costly immune response from the fish, which may translate into lower growth rates.

Our experiment was performed in clean (filtered and UV-treated) seawater and using smolt of known origin that had been raised in clean freshwater prior to transforming into seawater juvenile salmon (smoltification). None of the fish showed any symptom of new infection by any known salmon pathogen during the course of the experiment. We can therefore confidently attribute the effects observed in this study to lice themselves. In natural conditions, these effects are likely to be amplified by secondary infections occurring in hosts with damaged skin.

Salmon host populations have expanded considerably in the last decades due to increasing salmon farming, and concerns have been raised that such an ecological change

might select for faster life histories of their parasites. Models of the evolution of virulence indeed predict that a rapid increase in both the number of susceptible hosts and host population density, combined with an increase in adult parasite mortality rates (due to frequent drug use), should select for earlier and higher production of transmission stages (Mennerat *et al*., 2010). The observed link between early reproduction, increased fecundity and higher virulence of salmon lice suggests that virulence might evolve whenever the demographic conditions for the lice – and hence selection on parasite transmission – change. Salmon lice, therefore, appear as a good model for studying how human activity influences parasite demography and by this affects the evolution of their life history and virulence.

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