Density Dependence in the Host-Parasite Dynamic of *Gyrodactylus salaris on Different Stocks of Salmon, Salmo Salar*

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Introduction

Gyrodactylus is a genus of ectoparasitic platyhelmiths within the class Monogenea that parasitizes fish externally on different parts of the host (Malmberg 1993). The gyrodactylus genus has been known for over 180 years and was described by von Nordmann (1832) (Bakke et al. 2007). These parasites are non swimming, free floating flukes in the water, attaching themselves to suitable hosts when they come in contact with it. They are furthermore unique in the light of having no specific transmission stage, but rather four major methods of transmission between hosts: (1) random contact between an uninfected and an infected host, (2) uninfected host comes in contact with detached parasites on the substrate, (3) uninfected host comes in contact with infected dead host, (4) uninfected host comes in contact with detached parasites drifting in the water column (Bakke et al. 1992: Soleng et al. 1999).

These flatworms are among the smallest monogeneans and possess a fusiform body bearing a posterior opisthaptor with marginal hooks and two anterior cephalic processes with adhesive glands and spiked sensilla for attachment and sensing the surroundings (Bakke et al. 2007). The primary mode of attachment is the posterior opisthaptor armed with 16 peripheral marginal hooks possessing articulated blades, which is capable of considerable mobility as each hook has the potential to move independently from the others. This enables the gyrodatylids to attach themselves firmly to the surface of the fish host and stay put, and represent the major mode of attachment in most gyrodactylids in existence today (Bakke et al. 2007). The gyrodactylids also show a variance in site specificity on the host body, where some species show a marked specificity, whereas others do not. The majority infects the fins and the skin, and some are shown to prefer attachment on and around the gills (Bakke et al. 2007).

When these gyrodactylid flawtorms feed, they position themselves so the anterior end lies flat with the host epidermis and use the opisthaptor to firmly stay in place. Then the pharynx is extended onto the epidermis and a slight pumping motion commences as it feeds on the epidermic cells while minimizing the damage to the host epithelium (Bakke et al. 2007). In spite of having a feeding behavior selected for minimizing skin damage, the feeding may still create small lesions or feeding pits in the skin of the host made by the pharynx (Harris 1982:

Cable et al. 2002), and/or by the marginal hooks digging into the epidermis of the host, which again may lead to secondary bacterial or fungal infections in the skin (Malmberg & Malmberg 1993). Such infections may be seen as negligible when the burden of a gyrodactylid infection is small, but when the amount of parasites reaches high numbers, particularly on susceptible hosts, the outcome of these small bacterial or fungal infections may prove significantly harmful, and in many cases deadly (Shäperclaus 1991: Malmberg 1993). Another source of harm to the host due to lesions in the skin comes from the potential loss of osmoregulatory abilities, where the small tears, if numerous enough, can cause the host to lose body fluids and electrolytes to the environment causing potential death in the event of a severe enough parasite infection (Pettersen et al. 2012).

Furthermore, gyrodactylids are also unique in that the genus contains multiple viviparous species, as well as oviparous species which possess a reproductive system similar to that of most other monogeneans. Viviparous gyrodactylids are also found to be highly progenetic, being able to reproduce early in life. This combination makes these gyrodactylids some of the most successful parasites within the monogeneans (Bakke et al. 2007), in that they can reproduce at a very young age, and the time from birth to first reproduction is very short. This population dynamic resembles that of microparasites more than the typical population dynamic of their fellow parasitic helmiths (Anderson & May 1979: Cable & Harris 2002).

This kind of reproduction follows a highly specific pattern, where the first daughter always arises asexually, and following daughters arises only days later from oocytes that enter the uterus after the previous daughter has been born (Cable & Harris 2002). The viviparous gyrodactylids are furthermore protogynous hemaphrodites where the female reproductive system matures before the male, who develops its reproductive system only after the first asexual birth (Bakke et al. 2007).

Another astounding feature of reproduction in viviparous gyrodactylids is that before the first daughter is born, a second embryo develops within the daughter *in utero* like a "Russian doll" (Fig 1) (Cable & Harris 2002). This adaptation allows gyrodactylids to have the potential for an especially explosive and rapid population growth, which help to make it an especially effective and potentially devastating parasite capable of rapid colonization of its host species.

Figure 1: Gravid G.salaris, showing the fully developed embryo, with its own embryo. Photograph from Bakke et al. 2007.

It is also these adaptations that have made the gyrodactylids able to radiate with such success, infecting teleosts, amphibians, and cephalopods in a variety of different environments (Bakke et al. 1992: Cable & Harris 2002). Because of this astounding ability to adapt to new hosts, the gyrodacylids are now one of the richest genera of species in the lower monogenea, with over 400 potentially valid species on nearly as many hosts, something that may very well indicate a significantly higher number of gyrodactylid species in regard to the amount of fish species in existence (Harris et al. 2004). As there currently are some ~25 000 teleost species, the actual number of the hyperviviparous gyrodactylus species are thought to be in the range of 23 000 (Bakke et al. 2002). The high number of actual and potential hosts for these flatworms are in large part due to the fact that gyrodactylids show a high degree of host specificity, where a staggering 73.7% of 319 species studied by Bakke et al. (1992) only existed on single hosts, and a mere 4.1% on more than four host species (Bakke et al. 1992: Bakke et al. 2002). This high degree of host specificity in gyrodactylids may very well owe to the two major forms of speciation normally found in parasites.

The first one is co-evolution with the host, where gradual divergence of the host species can lead to isolation and thereby create a new species of parasite adapted only to one, or a few different host species. The second method of speciation entails host switching from one host species to another, unrelated, species in the same habitat called ecological transfer or host switching.

As many gyrodactylids possess a very short generation time and hyperviviparity, they are thought to be ideal for such a mechanism as host switching (Cable & Harris 2002). Furthermore, these modes of speciation in gyrodactylid flatworms are assumed to have

been facilitated in significant degree by Pleistocene glaciation events in northern Europe, where host switching and/or co-evolution is thought to have occurred by respectively species mixing and/or species isolation in relatively small ice-free refugia which is thought to have forced association between parasite and host. The fact that teleost species with a relatively wider distribution, and therefore more likely to be present in a larger number of refugia during the Pleistocene ice-age, like the minnow, *Phoxinus phoxinus*, today is infected by no less than 14 gyrodactylid species from 7 families, lends support to this hypothesis (Bakke et al. 2002).

However, there are some taxonomic uncertainties hailing from the fact that gyrodactylid morphology is quite conserved, with features such as body plan being highly simplified causing taxonomic and phylogenetic identification of species to be difficult (Cable et al. 1999). This makes the biological species concept as well as the morphological species concept (Mayr 1963) extremely difficult to use, when trying to distinguish separate species within the gyrodactylus genus because of the use of asexual reproduction and extremely similar morphology. The morphological features traditionally used for differentiating gyrodactylus species, the attachment hooks, comprising of dorsal bars, ventral bars, hamuli and marginal hooks, possess so minute differences between species that separating them with significant certainty is no easy matter (Shinn et al. 1996). It has been suggested by multiple authors that the use of sensory structures could be a valid alternative for taxonomically determining species (Bakke et al. 2007), but the traditional use of attachment structures coupled with modern statistical methods, like multivariate analysis, makes it possible to study morphometric variables in taxonomically close species with more certainty (Shinn et al. 1996).

Gyrodactylus salaris

Within this genus of *Gyrodactylus*, the hypervivparous species *Gyrodactylus salaris* (Fig1) has shown to be a epidemic on East Atlantic salmon, Salmo Salar, in northern Europe, especially Norway (Bakke et al. 2004). The species was first described by Malmberg in 1957 from fins and skin of salmon in a hatchery in Sweden (Johnsen & Jensen 1986). By 1975, the parasite had spread westward to Norway and was detected at what is now the Institute for

Aquaculture in Sunndalsøra (Johnsen 1978: Hansen et al. 2003). As initial research on the parasite was quite slow, the parasite continued to spread, mainly through introduction from Sunndalsøra, to other rivers by different means, and by 1995 the parasite had spread to a variety of locations within Norway (Bakke et al. 2007). During the next decade, *G.salaris* had been observed in as many as 46 rivers (10 % of the rivers in Norway) and 39 salmon farms, of which, by 2007, about 20 rivers remained infected despite 20 years of combating the spreading (Bakke et al 2007). So where did this flatworm currently wrecking havoc on Norwegian salmon populations come from? The consensus is that the parasite was introduced into Norway and to the east Atlantic stocks of salmon, from the Baltic region. Baltic stocks of salmon seem to be relatively resistant to *G.salaris*, unlike stocks from Norway which when tested were highly susceptible (Bakke et al., 1990, 2007). These experiments originally compared salmon stocks from the river Neva in Russia with Norwegian stocks from the rivers Alta and Lone, respectively in northern and western Norway (Bakke et al 1990).

Figure 2: Attached G.salaris attached to a host. Photograph obtained from www.fluefiske.net

This kind of introduction of an evasive species into an ecosystem that has no evolutionary history with the "invaders" often causes exponential population growth and subsequently severe damage to the ecosystem in question. As with the invasion of G. salaris in Norway, there are widespread examples of such occurrences worldwide, like the Brown Tree Snake in Guam, the Caulerpa Seaweed in the Mediterranean, or the Rosy Wolfsnail on islands in the Pacific and Indian Ocean (Lowe et al. 2000).

The economic implications for such a devastating fish parasite on Norwegian salmon is no less than profound, and it is estimated that *G.salaris* causes economic losses of about 200-250 million Norwegian Kroner (NOK) annually (www.lakseelver.no). The parasite has exterminated the salmon population in multiple Norwegian rivers and severely reduced the density in others (Bakke et al. 2007). Per 2007, the annual loss of salmon was estimated to have reached a staggering 250-500 tons, and the methods of controlling the spread of the epidemic using the poison rotenone ($C_{23}H_{22}O_6$) or acidified aluminium, have proven unsuccessful in many cases where the parasite can reappear, even though this types of treatments kill everything living in the stretches of river where it is administered. Another economic drain due to the *G.salaris* parasite lies in the cost of these treatments, adding to the total cost due to this parasitic invader (Bakke et al. 2007).

G.salaris, as with multiple other species of hyperviviparous, progenetic gyrodactylid flatworms, are furthermore not necessarily restricted to live on only one host species such as the east Atlantic salmon in Norway, but has the ability to infect a range of other teleost species as well, something that may have played an important role in the spreading of the parasite throughout Norwegian rivers.

G.salaris in Norway have been shown to use both anadromous and resident Arctic charr, *Salvelinus alpinus*, as a host in absence of salmon, with varying effect on mortality within populations, sometimes causing mortality and in other instances existing in very limited numbers on the hosts (Bakke et al. 1996, Winger et al. 2008, Kristoffersen et al 2005). *G.salaris* may also infect Brown Trout, *Salmo Trutta*, (Jansen & Bakke 1995). Although the Brown Trout shows low susceptibility and innate resistance to *G.salaris* growth and reproduction, it still serves as a possible dispersal tool rather than host species to the parasite, facilitating transport to other suitable hosts.

Rainbow trout, *Onrorhynchus mykiss,* have also been shown to be somewhat susceptible *G.salaris* infections (Jørgensen et al. 2007), and is thought to be the main long distance transport host for the parasite, proving important for the spreading of *G.salaris* (Bakke et al. 1992). Other such temporary "transport hosts" that coexist with salmon include lampreys, roach, perch minnow, flounder, stickleback and eel (Bakke et al. 2001).

The difference observed in susceptibility between east Atlantic and Baltic stocks of salmon to *G.salaris* has shown to be quite profound although the two stocks of salmon are geographically close. Bakke et al. (1990), showed a significant difference in susceptibility to infection by comparing salmon stocks from river Neva in Russia with Norwegian stocks from river Alta and Lone, respectively in northern and western Norway, where the Norwegian stocks showed little resistance to infection compared with Baltic salmon, which were able to eliminate the infection after some time. Also, *G.salaris* placed on salmon from the Norwegian rivers of Lierelva and river Alta have shown a higher fecundity and lower mortality compared to the same strain of *G.salaris* on salmon from the Baltic Neva stock. Here, the parasites gave birth significantly faster on the Norwegian stocks (2.3 days after infection in the Baltic stock compared to 1.8 days) in addition to only giving birth twice while on the Baltic salmon, compared to third and fourth births on the Norwegian stocks (Cable et al. 2000). Experiments on Finnish salmon, being part of the Baltic stocks, also seem to show an innate resistance, or at least significantly less susceptibility, to *G.salaris* infections compared to east Atlantic stocks in Norway, lending further support to this pattern of different susceptibility between Baltic and east Atlantic salmon (Rentamaki-Kinnunen & Valtonen 1996). During the course of experimental *G.salaris* infections on these stocks of salmon, one particular common pattern seems to stand out.

In a report ordered by the Norwegian Institute for Water Research (www.niva.no), Bakke et al. showed that the intensity and growth of infection on both the Baltic Neva stock, and the east Atlantic Lone and Alta stocks of salmon, looks to have a somewhat similar trajectory during the first few weeks. After this initial period though, the infection on the east Atlantic stocks wound take off exponentially, while the infections on the Baltic stocks were kept under control and eventually declined to the point of elimination (Bakke et al. 1990). This lends further support to an innate resistance within the Baltic stocks of salmon, which makes them able to control infections and coexist with the parasite.

In addition to Norwegian stocks of east Atlantic salmon, other stocks located further west, like the Scottish salmon, also show a generally high susceptibility to *G.salaris* (Bakke & MacKenzie 1993).

However, there are some deviations from the paradigm of differing susceptibility. Hybrids of susceptible salmon and innately resistant brown trout, which co-exist naturally in coastal rivers, have been tested in the lab, showing individuals possessing an intermediate susceptibility to *G.salaris*. This further indicates a genetic component for susceptibility to *G.salaris* in salmon (Bakke et al. 1999).

Furthermore, salmon from the Swedish river Indalselv were found to be almost as susceptible to infection from a Norwegian strain of *G.salaris*, as the Norwegian salmon stocks. Apart from a few individuals in the experiment, the Swedish indalselv stock showed a susceptibility to Norwegian strain of *G.salaris* similar to some of the highly susceptible east Atlantic stocks (Bakke et al. 2004).

Host-Parasite Dynamic

Even though most east Atlantic salmon stocks show high susceptibility to *G.salaris*, and are generally unable to control or mount any significant response to an infection, A few individuals apparently have the capability respond and control infections, if they survive the initial exponential growth of the parasite population. According to Bakke et al. (2004), salmon from the Norwegian river Lierelva in south eastern Norway seemed to be somewhat able to control the infection at the end of their experiment, indicating that some Norwegian salmon may mount some sort of a response to infection after all. The experiments on Scottish salmon by Bakke & MacKenzie (1993), also show that a very few individuals have the ability to survive the initial exponential growth of infection and control their parasite burden.

This dynamic of a gyrodactylid population being controlled on a host has been studied in length by Lester & Adams (1974), which investigated factors controlling the rise and fall in parasite number on the host, not including environmental factors, which had been widely relied upon until then. Rather, the rates of reproduction and mortality of the parasite while on the host, mortality and reattachment while off the host, and the rate of parasite shedding by the host were investigated. In their experiments using *Gyrodactylus alexanderi* on three-spined sticklebacks, *Gasterosteus aculeatus,* the population dynamics of gyrodactylid growth

on isolated fish showed a clear trend of infected fish shedding their infections, if they survived for around the first 2 weeks of infection. Those who did not, died from osmotic stress when the parasite burden reached about 150-400 parasites, the stress being hypothesized to hinder any response from the host. The ones that mounted a response by shedding their infections, showed a loss of susceptibility to further infections if they retained a few parasites after responding, while those fish that had shed all parasites got re-infected and started the cycle over again. The experiments also showed that parasites were lost by the shedding of the cuticle by the host, thought to be a possible mechanism of the response to infection at least in the species studied. This experiment sheds light on how population growth of gyrodactylids can be controlled by the host after an amount of time has passed, given they survive the initial couple of weeks of infection, and the infection does not become too big so that the stress experienced hinders a response.

Furthermore, Anderson & Scott (1984) showed that without any addition of new, previously uninfected, and therefore susceptible hosts to a population of fish infected by gyrodactylids, the host population has the ability to control and eliminate infection. This free-running experiment was carried out using *Gyrodactylus bullatarudis* Turnbull 1956, on the guppy *Poecilia reticulata* Peters, and lends support to the trend of successful control of infection given that the hosts survive the initial time period. Once the individuals that became too heavily infected died, the rest of the population, having mounted a response, eliminated the parasites altogether. Also, the fact that some hosts die, while others survive, lends more support to the presence of a genetic factor influencing resistance, not just previous exposure.

The parasite-host interaction shown in these experiments, paints a picture of a possible mechanism of time-dependence in host response, where the control of a gyrodactylid infection is induced after an amount of time has passed post infection. Along with this possible factor of host response, density-dependence of response to the parasite burden, where the host starts responding to infection when the parasite population reaches a certain density, could also be thought of as a possible cue for host response. However, density-dependence in hosts to gyrodactylids is poorly studied and there is currently no consensus on it. If such a density-dependence exists, the growth rate of the parasite population will

have a significant negative relationship with the increase in parasite burden, or possibly a threshold of the size of the infection.

In my thesis, I therefore aim to investigate whether the parasite growth rates in east Atlantic salmon could show evidence of density dependence, time-dependence, or both. I will also investigate if any of these mechanisms for parasite population growth, given their existence, could be linked to host response to-, and subsequent control of infection on east Atlantic stocks of salmon viewed highly susceptible by the classic paradigm. In order to do so, i have re-analyzed data from infection experiments done over the last 20 years on different stocks of both Norwegian and Scottish salmon, both viewed as highly susceptible. In addition, i use data from my own experiments done in the past 2 years. For comparison, i use data from the same type of experiments on the generally resistant stocks of Baltic salmon from river Neva in Russia and river Indalselv in Sweden.

Materials & Methods

For an overview of datasets used, see table 1, and for geographical overview of rivers, see figure 3 and 4. The different datasets used have furthermore been given short names based on location and replicate number for increased simplicity when plotted. These origin of the datasets used in the current study in addition to my own, which can be found in the Appendix, are listed in table 1

Atlantic host stocks:

From northern Norway, I used data from three experiments done on fish from river Alta (Cable et al. 2000, Bakke et al. 1999) which is located in the lower part of the Alta-Kautokeino watercourse, emptying out in the Altafjord in the western part of Finnmark county.

From western Norway, I used a dataset on salmon from Batnfjord (unpublished, Bakke et al. 2001, Fig.6) which is located in Møre og Romsdal county. The river runs from the lake Botnvatnet and empties out in the Batnfjord.

From southeastern Norway, i used two datasets each from Lierelva salmon (Bakke & MacKenzie 1993, unpublished, Bakke et al. 2001, Fig.7) and river Numedalslågen. The Lierelva river is located in Buskerud county and runs from Sylling village in the north, down to the Drammensfjord in the south where it empties out. The river Numedalslågen runs through Buskerud and Vestfold county. It starts at the Hardanger plateau and empties out in the Skagerrak Sea in the town of Larvik, Vestfold about 250 km away.

From southwestern Norway, i used one dataset from salmon in the collected from the Aquatic Research Station Ims (unpublished, Bakke et al 2001, Fig.14), a part of the Norwegian institute for Nature Research, near the city of Stavanger in Rogaland county.

From Scotland, I used two datasets. One from an experiment done on salmon in the river Conon and one of salmon from the river Shin (Bakke & MacKenzie 1993). River Conon is located in the Highlands of Scotland, starting in Loch Luichart, emptying out in the North Sea. River Shin starts in the North West Highlands of Scotland and runs from Loch Shin to the North Sea.

Baltic host stocks:

From Russia, I used datasets from four experiments (Cable et al. 2000, Bakke et al. 1990), all on salmon from river Neva in northwest Russia. It runs from Lake Ladoga, emptying out in the Gulf of Finland in the Baltic Sea.

From Sweden, i used one dataset from salmon from the river Indalselv (Bakke et al 2004), which runs from Jämtland and the lake Storsjön, emptying out in the Baltic sea.

Experimental procedure

Every dataset used are based on similar, common garden, experimental methods using isolated salmon fry 0+ infected with *G.salaris,* regularly observed for change in parasite burdens.

My own experiments on salmon from both river Neva in one replicate, and river Numedalslågen in two replicates, done in the fall of 2011 and the spring of 2012 respectively, follow the same general methodology as Bakke et al. (1990). The host populations of 0+ salmon fry, previously uninfected by *G.salaris,* were kept in grey plastic tanks (1mx1mx1m) with opaque lids and a continuous flow of normal Oslo tap water around the clock. The fish were further individually separated by enclosures approx 20cmX7cm with a wire mesh bottom to allow water to flow through. The temperature was kept steady at 12° C, and light conditions were kept dim continuously. The fish were fed equal amounts at a standard time interval. All variables were kept equal for each individual in both experiments, which took place in the basement aquarium at the Museum of Natural History in Oslo (NHM), Zoological department.

Table 1: Overview of datasets used in current study.

| Region	Stock	Host species	Parasite	# Fish	Time (days)	Mortality %	Original Research
N.Norway	Alta 1	*Salmo Salar*	*G.salaris*	12	51	58.3	Cable et al. 2000
	Alta 2	*Salmo Salar*	*G.salaris*	12	42	0	Bakke et al. 1999
	Alta 3	*Salmo Salar*	*G.salaris*	12	42	8.3	Bakke et al. 1999
W.Norway	Batnfjord	*Salmo Salar*	*G.salaris*	23	36	60.8	Unpublished, Bakke et al. 2001, Fig.6
SE.Norway	Lier 1	*Salmo Salar*	*G.salaris*	24	36	54.1	Unpublished, Bakke et al. 2001, Fig.7
	Lier 2	*Salmo Salar*	*G.salaris*	24	50	50	Bakke & MacKenzie 1993
	Numedals 1	*Salmo Salar*	*G.salaris*	9	55	11.1	Own research
	Numedals 2	*Salmo Salar*	*G.salaris*	9	49	44.4	Own research
SW.Norway	Imsa	*Salmo Salar*	*G.salaris*	18	35	88.9	Cable et al. 2000
Russia	Neva 1	*Salmo Salar*	*G.salaris*	22	35		Cable et al. 2000
	Neva 2	*Salmo Salar*	*G.salaris*	12	51	8.3	Bakke et al. 1990
	Neva 3	*Salmo Salar*	*G.salaris*	16	51		Bakke et al. 1990
	Neva 4	*Salmo Salar*	*G.salaris*	18	21	5.5	Own research
Scotland	Shin	*Salmo Salar*	*G.salaris*	24	49	33.3	Bakke & MacKenzie 1993
	Conon	*Salmo Salar*	*G.salaris*	24	49	54.1	Bakke & MacKenzie 1993
Sweden	Indals	*Salmo Salar*	*G.salaris*	24	50	87.5	Bakke et al. 2004
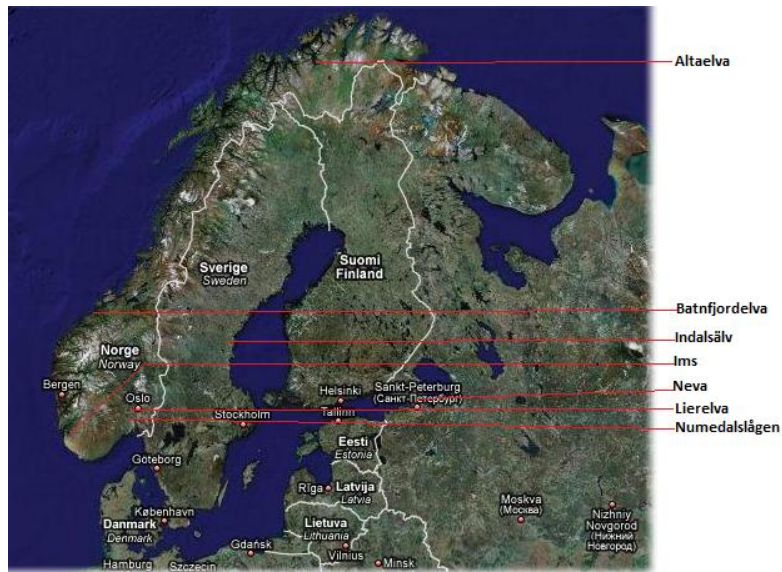
Figure 3: Geographical distribution of rivers in Scandinavia and western Russia where salmon stocks have been collected for the various datasets used. Photograph obtained from Google Maps (maps.google.no)
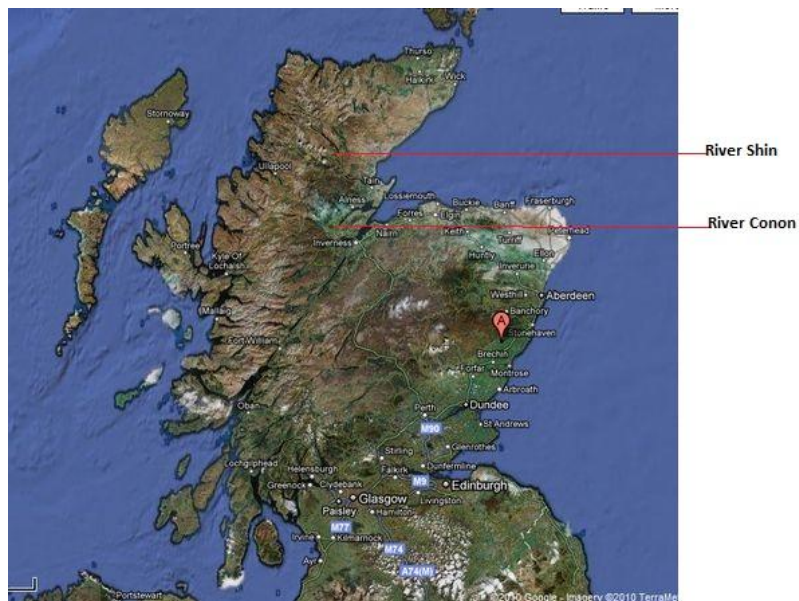

Figure 4: Geographical distribution of Scottish rivers where salmon stocks have been collected for various datasets used. Photograph obtained from Google Maps (maps.google.no)

For the experiment on salmon from Numedalslågen, starting infections of *G.salaris* were administered by obtaining fin clippings of already heavily infected individuals from other stocks kept in the aquarium, and transporting 5 worms, one by one, from these fin clippings

directly onto the anesthetized host individual by use of a pin needle under a stereo microscope, placing them on the caudal fin of the host. In the experiment on Neva salmon, a variable degree of initial infection was administered, using either pin needle to place one and five infections, or by letting host individuals swim with heavily infected fin clippings for 24 hours. After the administering of parasites had been completed, each fish were then divided amongst the separate enclosures within the tank, keeping them isolated from each other. The first day after infections in both experiments, the fish were anesthetized and the infections controlled to ensure that none failed to establish. All host individuals in both experiments were checked once per week throughout the duration of the experiment, at about the same time of day.

The hosts were anesthetized using a 0.05% solution of Chlorobutanol (trichloro-2-methyl-2-propanol) in water. When checking parasite burden, each fish was carefully removed from its enclosure in the tank and put in the bucket containing anesthesia until the individual was anesthetized sufficiently to be examined under stereo microscope in a tray of water. The whole fish was then carefully examined, counting every parasite as fast and effectively as possible so not to put too much stress on the host fish, without overlooking any worms. If the anesthesia proved to be too light and the fish became active during counting, it was simply placed back in the bucket containing the anesthesia solution for a short time until calm again, and the remaining counting completed. When the counting was finished, the host was put over in another bucket containing fresh tap water gathered from the same source as the tank housing the experiment population, and transported back to its respective enclosure within the tank. The dynamic of infection, and growth rates, was calculated on the basis on these weekly counts.

Graphics

To investigate density- and time-dependence in parasite growth rates, and possible host responses in all datasets used, several plots were constructed from each dataset using the graphics tools within Microsoft excel.

The increase in parasite population was plotted in a scatter plot against time to demonstrate the population dynamics for the parasite burden on each host during the course of the experiment

To investigate the possibility of parasite growth rate being dependent on parasite population size, the growth rate of the parasite population for each census point was plotted in a scatter plot against the explanatory variable being intensity of parasite burden counted for each of the corresponding census points. To investigate possible time-dependence of the parasite growth rate, it was plotted in a scatter plot with time as the explanatory variable. Further, parasite growth rate was plotted against the natural logarithm of the recorded parasite burden for the previous census point. As this plot shows the trend between the parasite growth rate and the parasite population density for the previous week, a statistically significant relationship between these variables will indicate, at least in some part, parasite growth rate having some significant relationship with earlier parasite population density. This was done separately for each dataset used.

In addition, to investigate if density- or time-dependence could be seen as possible cues for a host response to parasite infection, a second set of plots, using straight line scatter plots, were made using the mean values only of hosts surviving to the end of the experiment. This was done to correct for any possible skewing of the trajectories due to parasites lost from host death, and to make the trends easier to observe. The trends showing mean parasite population densities are separated according to region of the salmon used, to easier compare trends, and the mean parasite growth rate are plotted against both parasite population density and time, in separate plots for each replicate used from each experiment.

Statistical analysis

To check if these relationships in the scatter plots created indeed were statistically significant, the statistical open-source software R (www.r-project.org) was used to analyze each dataset. To check for statistical significance, all variable interactions used were modeled using general linear modeling (GLM), used to test hypotheses in statistical experiments, and factor in known quantities, estimates, and noise, or other sources of error. The p-values obtained from this modeling were used to determine if the patterns and seeming relationships observed between variables, proves significant enough to draw a conclusion of an active relationship.

Results

(For all GLM values from interaction between variables, see Appendix)

Northern Norway: Alta stock

G.salaris infections became established on every fish in all 3 datasets of salmon from the river Alta the first days post infection, with mean intensities of 5.83, 41.6 and 67.4 parasites per host respectively, and continued to increase until the population reached its peak (Fig 5c). This occurred after between about 30 to 40 days in all 3 datasets on Alta salmon (Alta 1 ~40 days, Alta 2~35 days and Alta 3~35 days). The maximum number of parasites reached on a single host was quite uniform within each population with the peak parasite burden remaining below 500 in Alta 1, having one distinct outlier with a burden of 670 parasites after 44 days. Alta 2 had a maximum parasite burden for most of the hosts at just below 400, with two outliers having 545 and 743 parasites at day 35 and 42 respectively, while the Alta 3 population had a parasite density peak just below 500 for the majority, with two outliers having 565 and 563 parasites at 28 and 35 days respectively. The parasite growth rates were highest the after the first week in all 3 datasets, then declined throughout the experiment, showing a significant negative relationship with time, eventually hitting its minimum at about 30 to 40 days post infection in all 3 datasets, not changing significantly beyond that point (Alta 1~37 days, Alta 2~42 days, Alta 3~28 days)(Fig 5b). The first replicate was the only one showing a statistically significant relationship between parasite growth rate and parasite population density (Fig 5a). At the end of the experiments, both Alta 2 and 3 showed multiple hosts with parasite growth rates below 0.

When parasite growth rate was plotted against the natural logarithm of the number at the previous census date, the result was a significant negative relationship in all replicates (Fig 5d). Not all salmon survived to the end of the experiment and 2 out of the 3 experiment populations of Alta salmon showed mortality. In the first replicate, 7 out of the 12 fish died before experiment end (58%). In the third replicate experiment using Alta stock fish, only one died out of 12 (8.3%). In the second, there was no mortality.
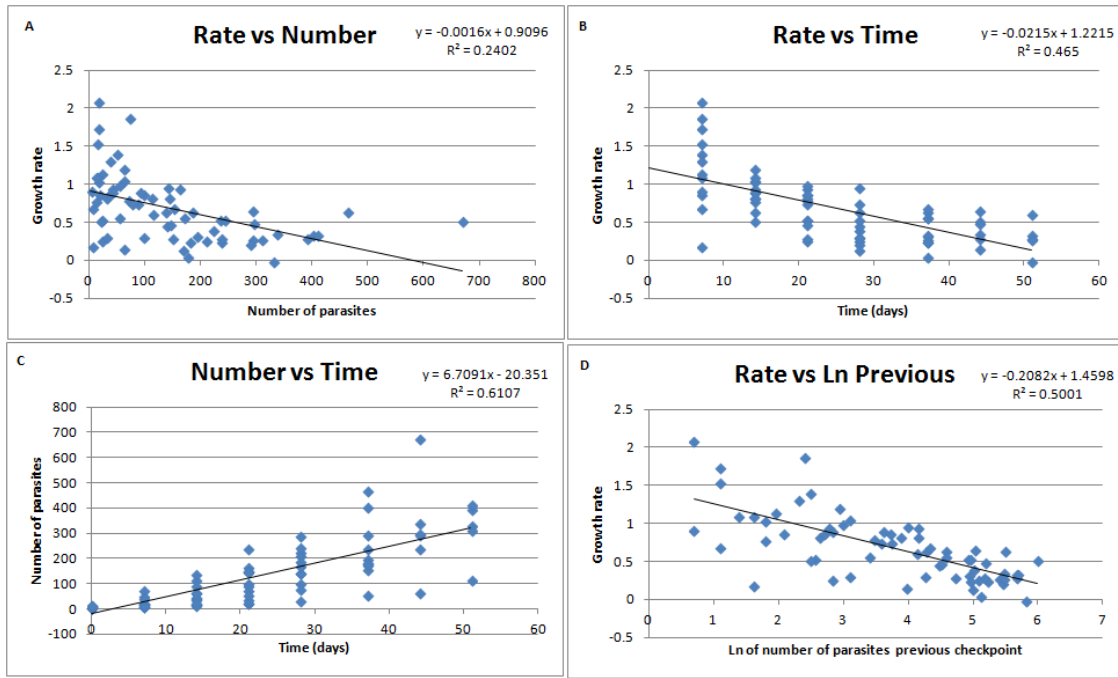
Figure 5: Graphic representation of variables tested in the first dataset on Alta salmon. A) Parasite growth rate against parasite density, B) parasite growth rate against time factor, C) parasite density against time factor, D) parasite growth rate against Ln of previous parasite density.

Figure 6: Graphic representation of variables tested in the second dataset on Alta salmon. A) Parasite growth rate against parasite density, B) parasite growth rate against time factor, C) parasite density against time factor, D) parasite growth rate against Ln of previous parasite density.

Figure 7: Graphic representation of variables tested in the third dataset on Alta salmon. A) Parasite growth rate against parasite density, B) parasite growth rate against time factor, C) parasite density against time factor, D) parasite growth rate against Ln of previous parasite density.
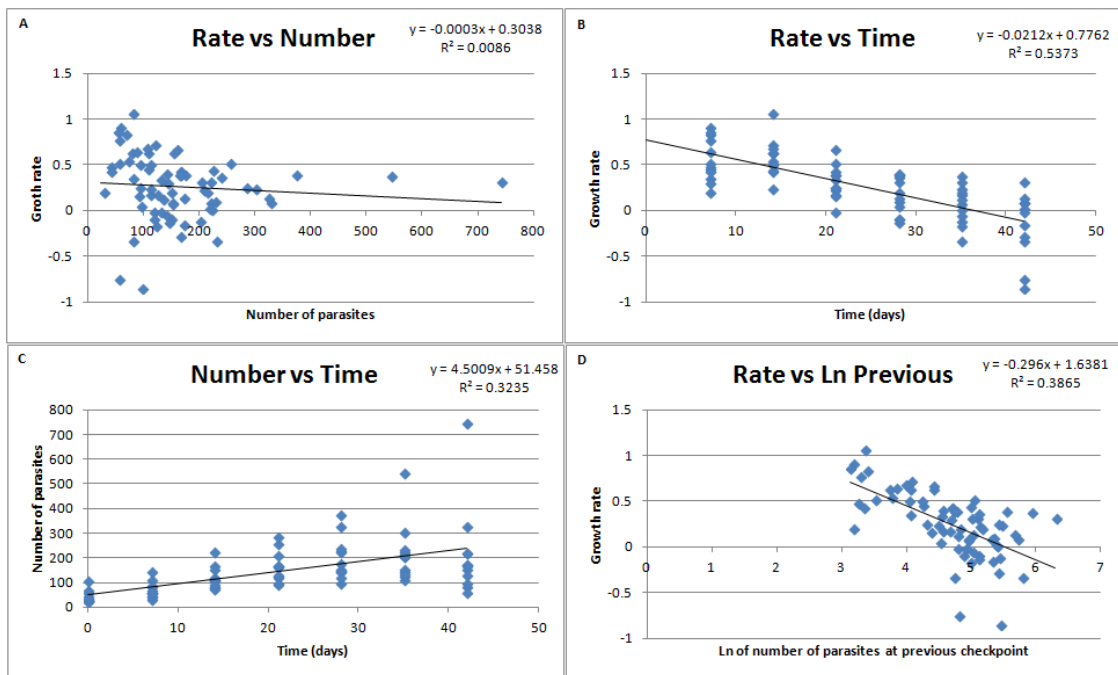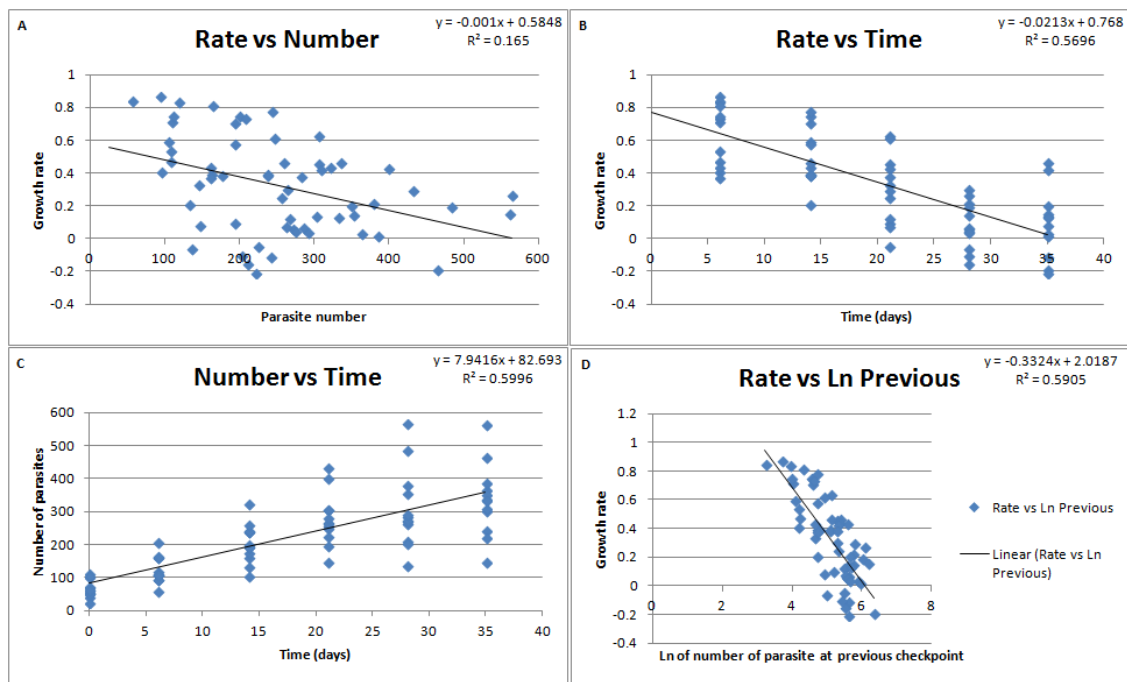
Western Norway: Batnfjord stock

Infections became quickly established on the entire experimental population from Batnfjord first few days post infection with a mean intensity of 84.1 parasites per host. The infection continued to grow on all individuals until the end of the experiment, 36 days post first infection (Fig 8c). The maximum parasite population size was close to 2000 parasites, with 3 outliers at 2500 parasites after 28 days, 2400 after 36 days, and one host with 2500 after 36 days. The parasitic growth rates were highest at the beginning of the experiment, then continued to decline towards the end of the experiment (Fig 8b), showing a significant linear relationship with the time factor. The growth rate also show a similar negative relationship with the increase of parasite population (Fig 8a), declining towards the end of the experiment, with the sharpest drop up to an infection burden of about 500 parasites. The rate plotted against the natural logarithm of parasite population at the previous census point (fig 8d) also shows a significant relationship. During the course of this experiment, 14 out of the 23 fish (60.8%) died before the end of the experiment from gyrodactylosis.
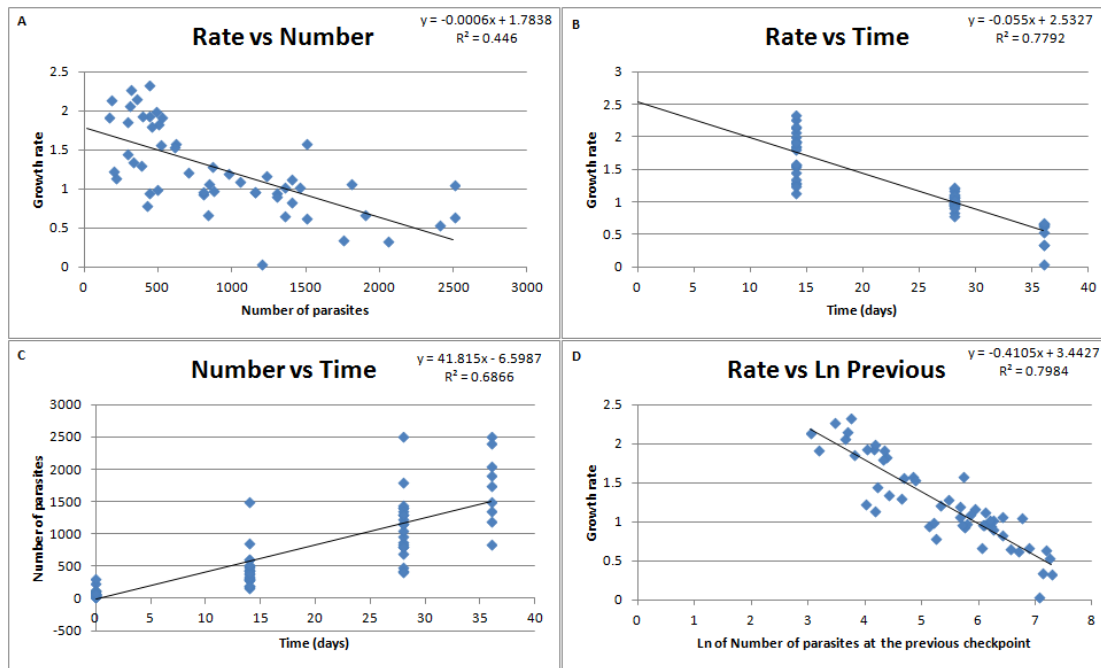
Figure 8: Graphic representation of variables tested in the dataset on Batnfjord salmon. A) Parasite growth rate against parasite density, B) parasite growth rate against time factor, C) parasite density against time factor, D) parasite growth rate against Ln of previous parasite density.

Southeastern Norway: Lierelva and Numedalslågen

In both experiments done using fish from the river Lier, infections became established quickly within the first week post infection with a mean of 128.2 and 77.5 parasites per host respectively, then grew steeply in both experimental populations, before peaking and leveling out after c. 28 days in the first replicate (Fig 9c). In the second replicate, the growth of infections halted after c. 38 days, and then proceeded to decline until the end of the experiment (Fig 10c). In the first replicate, no fish sustained infections above 1500 parasites (apart from 3 outliers with 2500, 1750 and 1750 parasites after 28 days). In the second replicate, the infections reached as high as 1500-2000 parasites on several individuals, although the majority of infections remained below 1500 worms. The growth rate showed a significant decline over the course of the experiment in the first replicate (Fig 9b), with the sharpest decline 28 days post infection. In the second replicate, the growth rate remained approximately constant the first 20 days, and then declined until day 42. After this, it rose again (Fig 10b), while still showing a significant relationship with time. Parasite growth rate also show a significant decreasing relationship with the increase of the parasite population in Lier 1 (Fig 9a), but showed no significant relationship in the second replicate (Fig 10a). The rate plotted against the natural logarithm of parasite number on the previous census date

showed significant relationships in both experimental populations (Fig 9-10d). During the course of the experiments on Lier salmon, 13 out of 24 salmon (54.1%) died in the first replicate, while in the second, 12 out of 24 (50%) died before the end of the experiment.

Figure 9: Graphic representation of variables tested in the dataset on the first replicate of Lier salmon. A) Parasite growth rate against parasite density, B) parasite growth rate against time factor, C) parasite density against time factor, D) parasite growth rate against Ln of previous parasite density.
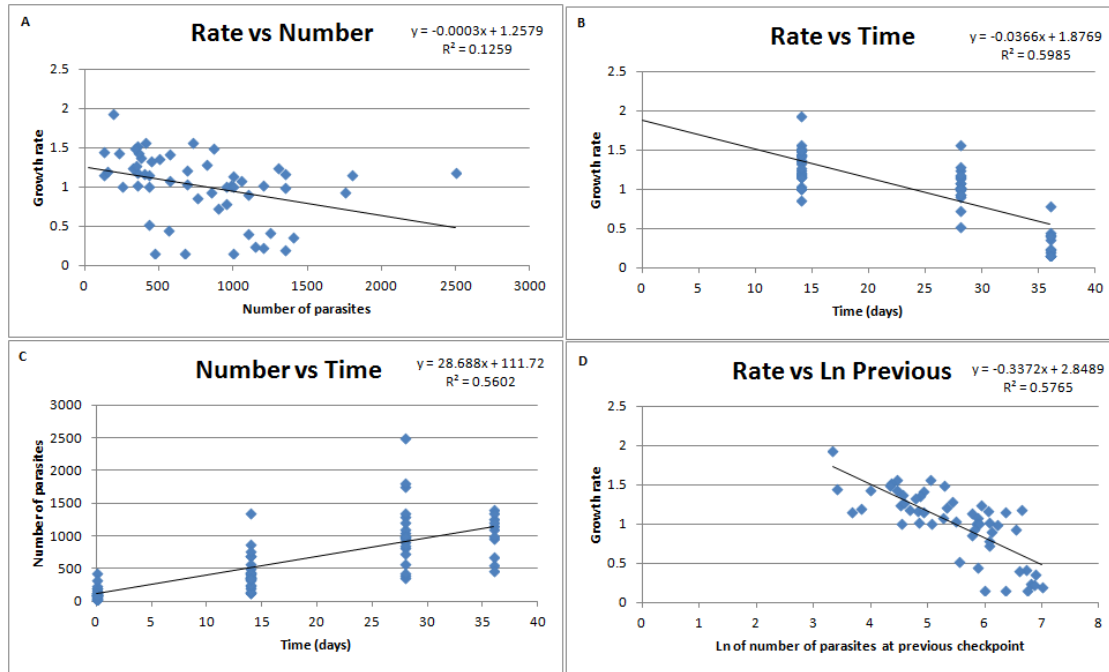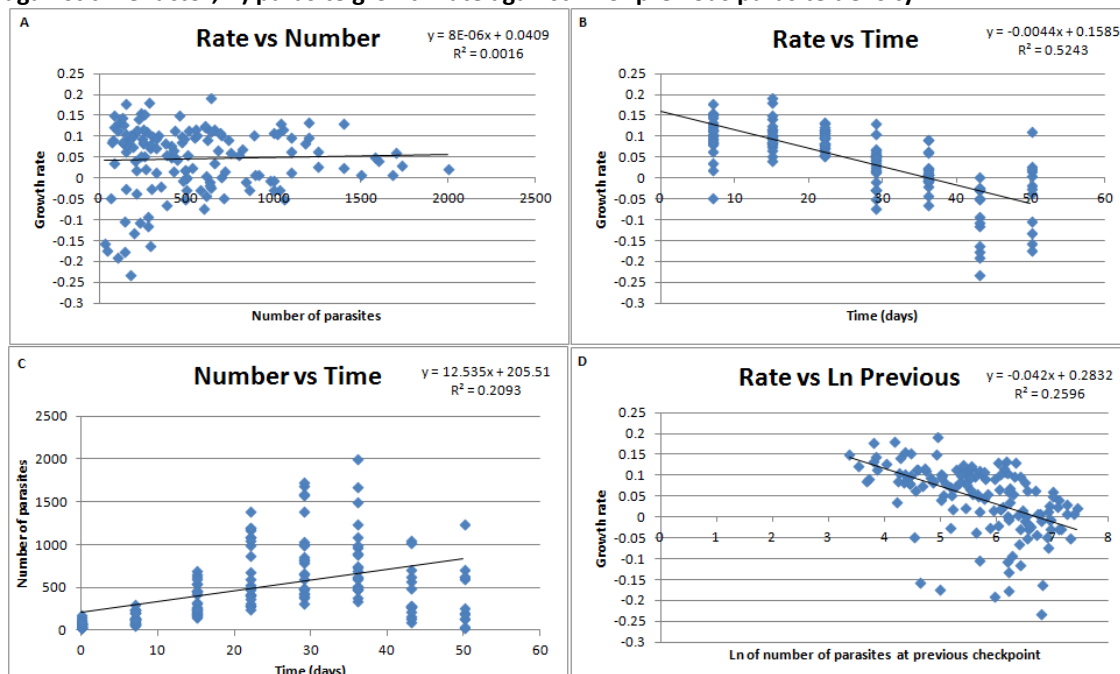

Figure 10: Graphic representation of variables tested in the dataset on the second replicate of Lier salmon. A) Parasite growth rate against parasite density, B) parasite growth rate against time factor, C) parasite density against time factor, D) parasite growth rate against Ln of previous parasite density.

All the hosts in both populations from Numedalslågen showed an established parasite population shortly after experiment start with 5 parasites on each host. In both experiments, the infections grew slowly the first 20-30 days, and then grew at a substantially increased rate until the experiments ended (Fig 11-12c) In the first replicate, the parasite burdens ranging from 300 to almost 800 parasites per fish were achieved by the end of the experiment (55 and 49 days respectively), while in the second replicate, no host achieved an infection of more than 400 parasites. In neither replicate was there a significant relationship between parasite growth rate and time (Fig 11-12b), and neither was there a relationship between parasite growth rate and parasite population growth (Fig 11-12a). When plotting growth rate against the natural logarithm of parasite number at the previous census date, no significant relationship was seen (Fig 11d-12d). During the course of the experiments, 1 out of 9 fish (11.1%) died in the first replicate population, while 4 out of the 9 fish (44.4%) died in the second.

Figure 11: Graphic representation of variables tested in the dataset on the first replicate of Numedals salmon. A) Parasite growth rate against parasite density, B) parasite growth rate against time factor, C) parasite density against time factor, D) parasite growth rate against Ln of previous parasite density.
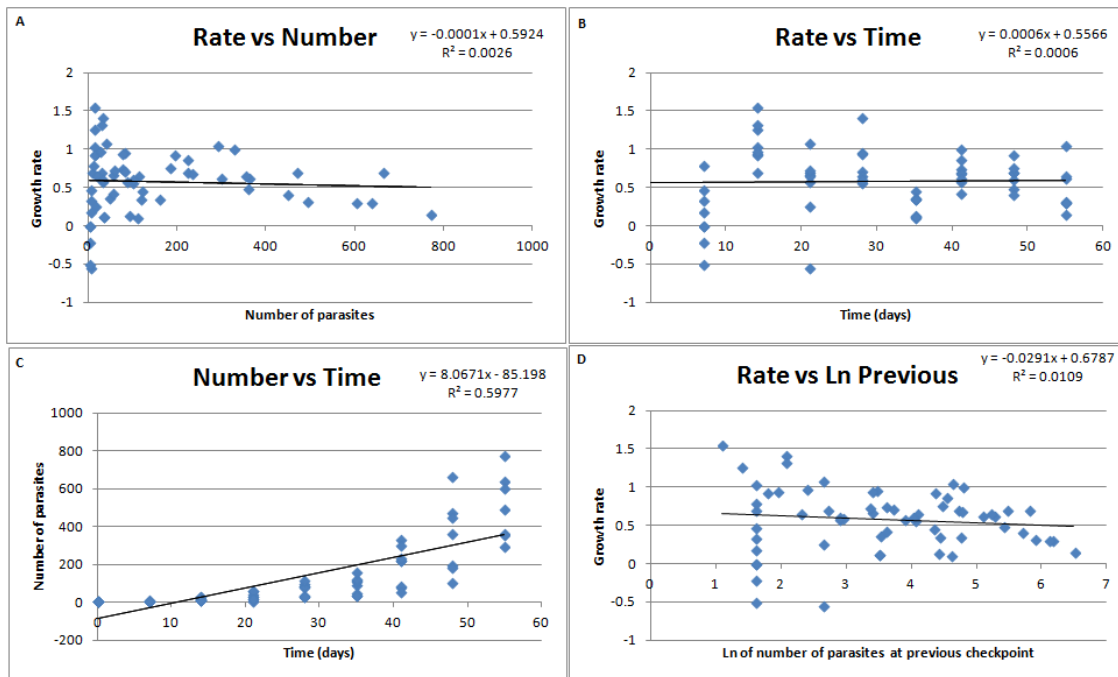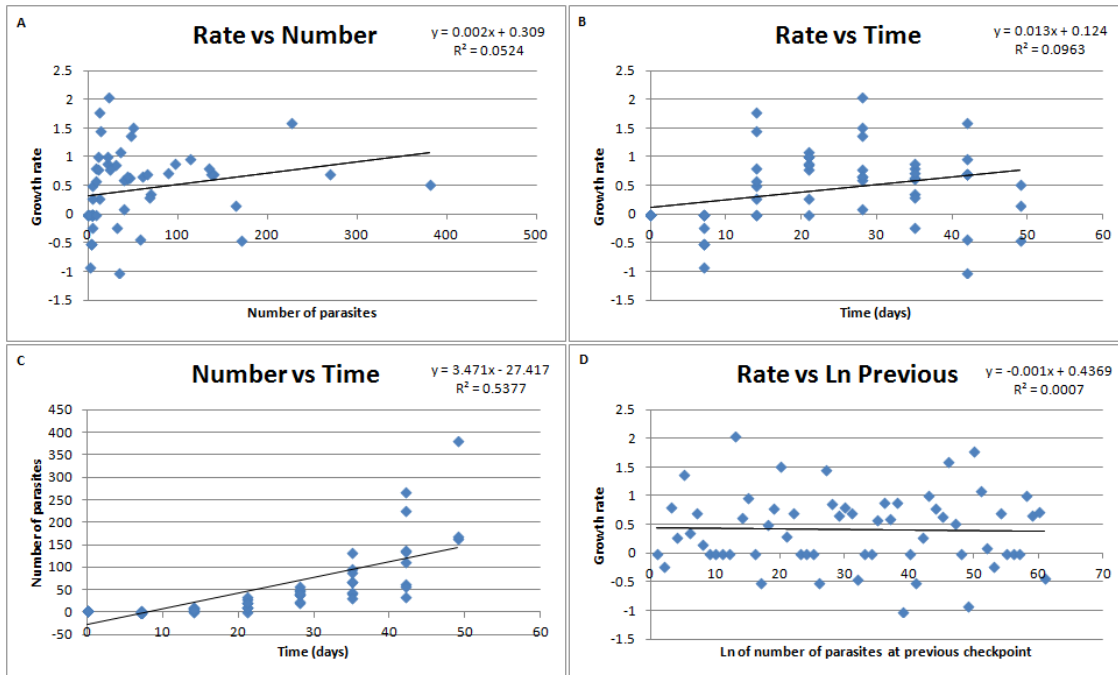
Figure 12: Graphic representation of variables tested in the dataset on the second replicate of Numedals salmon. A) Parasite growth rate against parasite density, B) parasite growth rate against time factor, C) parasite density against time factor, D) parasite growth rate against Ln of previous parasite density.

Southwestern Norway: Ims

In the experiment on salmon from Ims, all infections became established during the first week with a mean infection of 79.3 parasites per host, and continued to grow until approximately day 21, when the parasite population growth peaked and started to decline towards the end of the experiment (Fig 13c). The vast majority of the fish did not experience infection levels above 1000 parasites, apart from three outliers infected with 1200 parasites after 21 days, and 1030 and 1100 parasites after 28 days. For the Ims salmon, the parasite growth rate showed a significant declining relationship with time towards the end of the experiment (35 days) (Fig 13b). This decline in parasite growth rate was also significantly related to the increase in density (Fig 13a), with the steepest decline occurring up to a burden of about 500 parasites. The growth rate also shows a significant negative relationship with the natural logarithm of parasite number at the previous census point (Fig 13d). During this experiment, only 2 of the 18 original fish survived (mortality 88.9%) to the end of the experiment at 36 days.
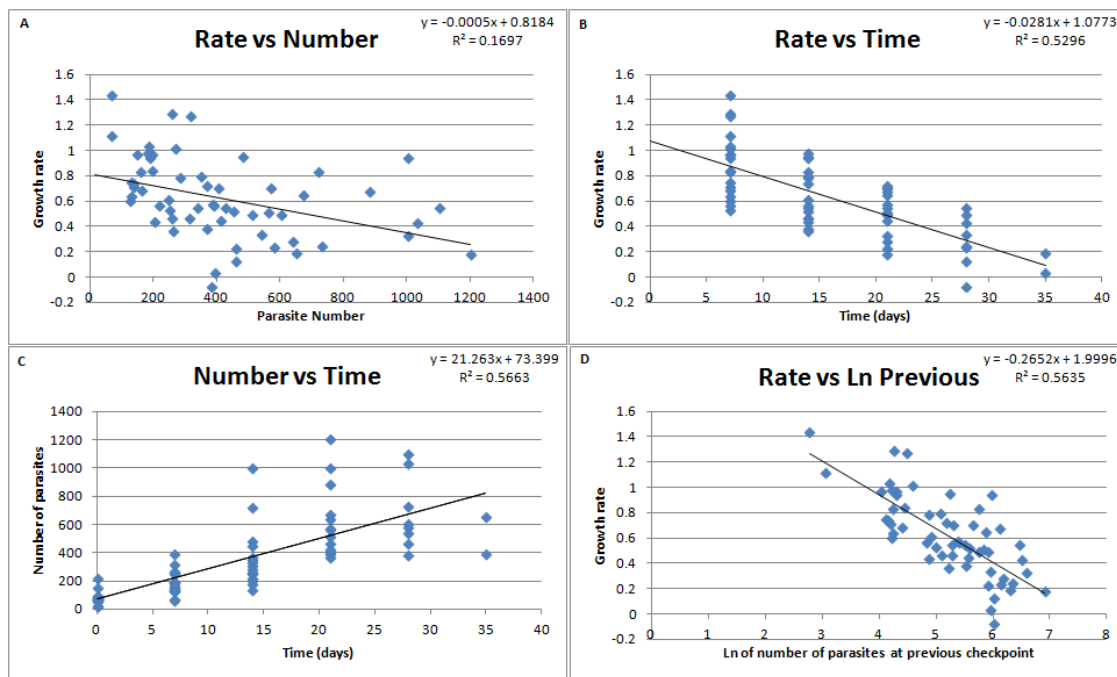
Figure 13: Graphic representation of variables tested in the dataset on the Ims salmon. A) Parasite growth rate against parasite density, B) parasite growth rate against time factor, C) parasite density against time factor, D) parasite growth rate against Ln of previous parasite density.

Scotland: river Conon & river Shin

Both experimental populations of Scottish salmon established a parasite infection on all individuals the first week with a mean intensity of 20.5 parasites per host in Conon, and 91.8 in Shin. These infections continued to increase in size until reaching a peak infection after c. 42 days in Conon, after which it decreased until the end of the experiment (Fig 14c), whereas the parasite burden in the Shin population grew steady up to c. 28 days, after which it declined sharply towards the end of the experiment (Fig 15c). Most of the salmon from the Conon experiment did not experience parasite burdens above 1500, with the exception of 5 outliers experiencing 1700, 3000, 4000, 1650, and 4000 after 21, 28, 35, 35 and 42 days respectively. In the Shinn population, the vast majority of individuals experienced infections below 1400, but some individuals exceeded this and the highest infection reached 1760 parasites. In both experimental populations of Scottish salmon, the parasite growth rate showed a significant negative relationship with time, decreasing until the linear regression predicted a negative growth rate after 42 days in the Conon population (Fig 14b), while the

Shinn population on the other hand showed a linear regression predicting a negative growth rate of parasites after about 35 days (Fig 15b). Neither Scottish salmon population showed a relationship between parasite growth rate and parasite density (Fig 14-15a), but the growth rate plotted against the natural logarithm of parasite population size at the previous census point did show a significant negative relationship (Fig 14-15d). During these 2 experiments, the population in the Conon experiment lost 13 out of 24 fish (54.1%), while the Shinn population lost 8 out of 24 fish (33.3%).

Figure 14: Graphic representation of variables tested in the dataset on the Conon salmon. A) Parasite growth rate against parasite density, B) parasite growth rate against time factor, C) parasite density against time factor, D) parasite growth rate against Ln of previous parasite density.
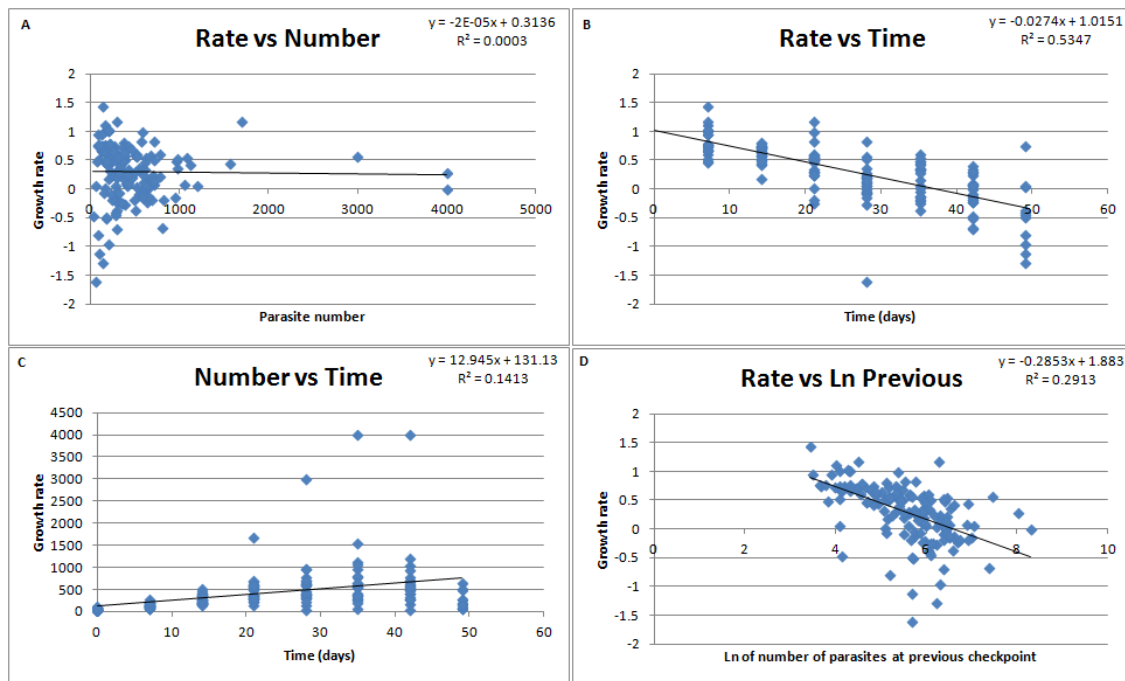
Figure 15: Graphic representation of variables tested in the dataset on the Shin salmon. A) Parasite growth rate against parasite density, B) parasite growth rate against time factor, C) parasite density against time factor, D) parasite growth rate against Ln of previous parasite density.

Baltic Stocks

Russia: river Neva

In all the 4 experiments on salmon from river Neva in Russia, the *G.salaris* infections became established the first week with mean numbers of 27.5, 4.25, 37.3 and 29.5 parasites per host respectively, continuing to grow. In the first, third and fourth replicate, the parasite populations increased until about day 21, after which the population of parasites started to decline until the end of the experiments in replicates 1 and 3 (Fig 16-18c) The fourth replicate ended only after 21 days, and so a decline could not be observed (Fig 19c). In the second replicate, the same trend was observed, but the increase of the parasite population continued slightly longer until c. day 28, before a decrease in parasite population was observed. No Neva salmon experienced parasite burdens of more than 400 parasites, except in the final fourth replicate, where burdens up to c 800 were observed 21 days post infection. All populations of Neva salmon showed a significant decrease in parasite population growth rate over time, with parasite population growth rate declining to zero and becoming negative for most individual fish after 28-35 days (Fig 16-19b). In replicate 2,

parasite growth rate decreased most rapidly during the first 14 days, and then declines more slowly to a negative growth rate throughout the experiment, showing a possible weak tendency to start rising again. In replicate 3, the growth rate also decreased steadily, becoming negative for all individuals after 35 days post infection. The parasite growth rate in the fourth replicate showed little overall change between the first and second week, but then declined towards zero until the end of the experiment. Parasite growth rate showed a significant relationship with increase in parasite population growth in replicates 3 and 4, but no significance in the first and second (Fig 17-19a). In the third replicate, the parasite growth rate seems to mostly start of negative for low numbers of parasite infections, then increase up to a parasite population density of about 50-100 parasites, where the linear regression predicts the mean growth rate becomes positive, then stabilize for higher densities. In the fourth replicate, the growth rate decreases, from relative high variability, slowly with rising parasite population density towards the peak infection number observed in the experiment. Parasite growth rate plotted against the natural logarithm of the number of parasites found at the previous census date, showed a significant relationship in the first, second and fourth replicates, and not in the third. Where significant, the relationship is negative (Fig 16-19d). During the experiments on Neva salmon,

Figure 16: Graphic representation of variables tested in the first dataset on Neva salmon. A) Parasite growth rate against parasite density, B) parasite growth rate against time factor, C) parasite density against time factor, D) parasite growth rate against Ln of previous parasite density.
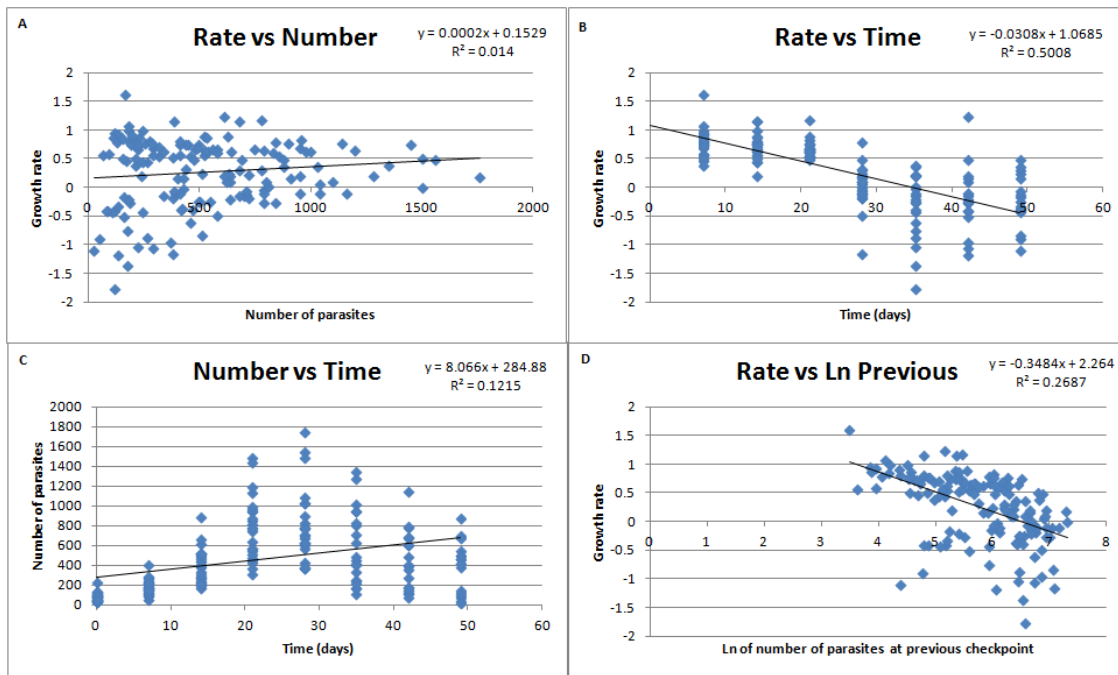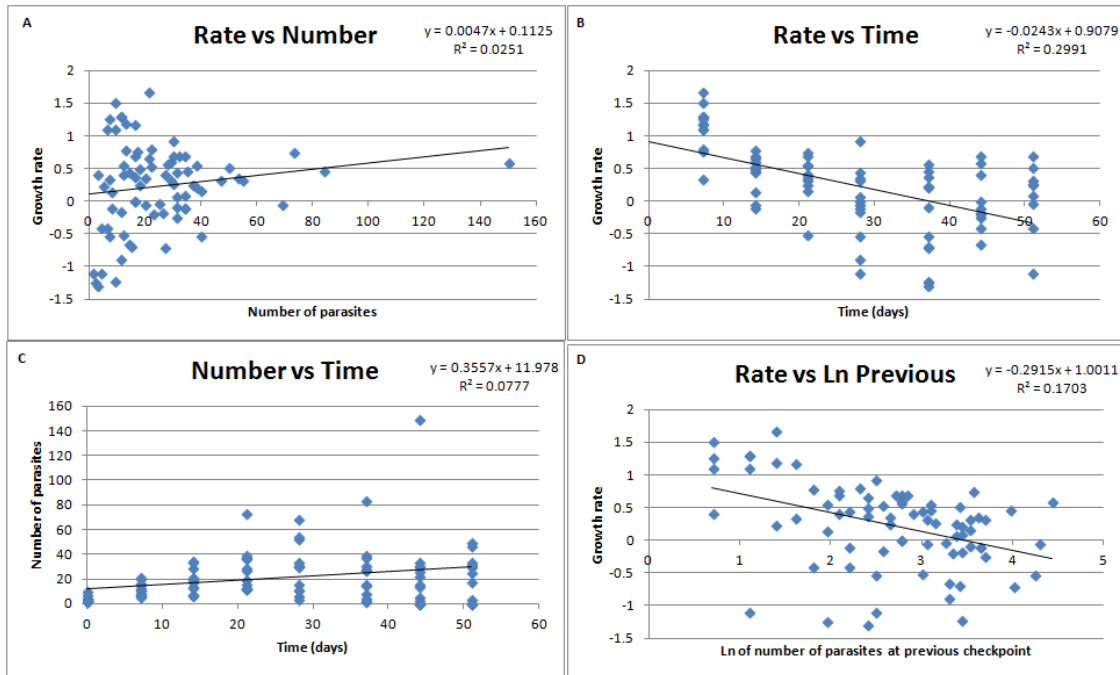
Figure 17: Graphic representation of variables tested in the second dataset on Neva salmon. A) Parasite growth rate against parasite density, B) parasite growth rate against time factor, C) parasite density against time factor, D) parasite growth rate against Ln of previous parasite density.
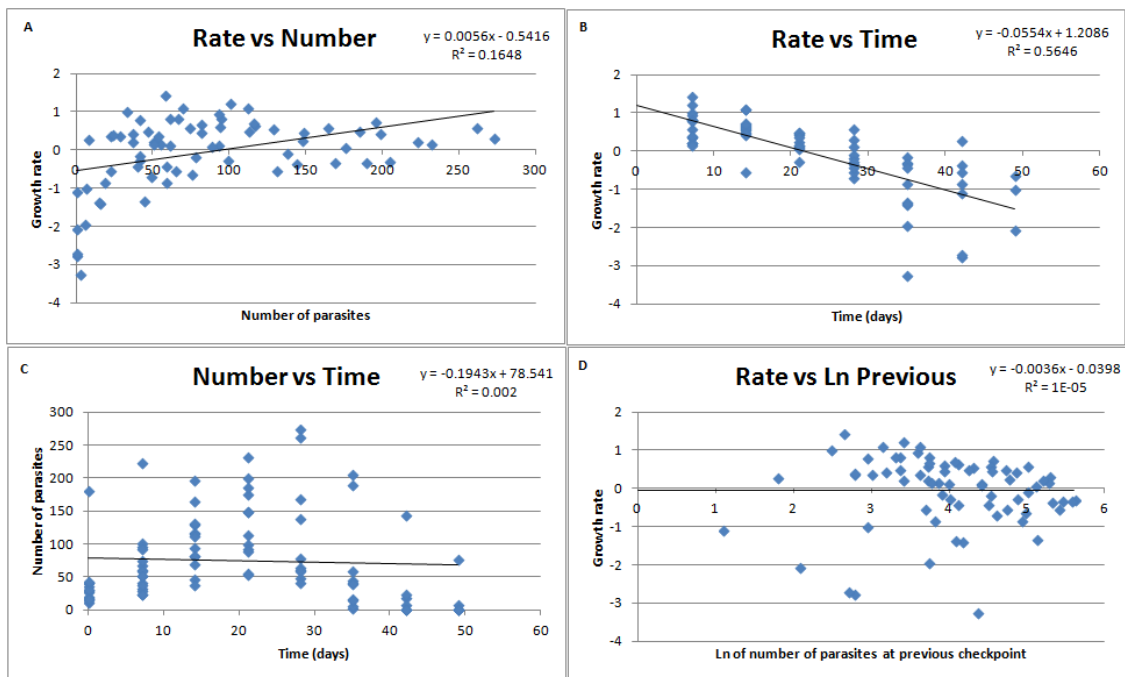
Figure 18: Graphic representation of variables tested in the third dataset on Neva salmon. A) Parasite growth rate against parasite density, B) parasite growth rate against time factor, C) parasite density against time factor, D) parasite growth rate against Ln of previous parasite density.
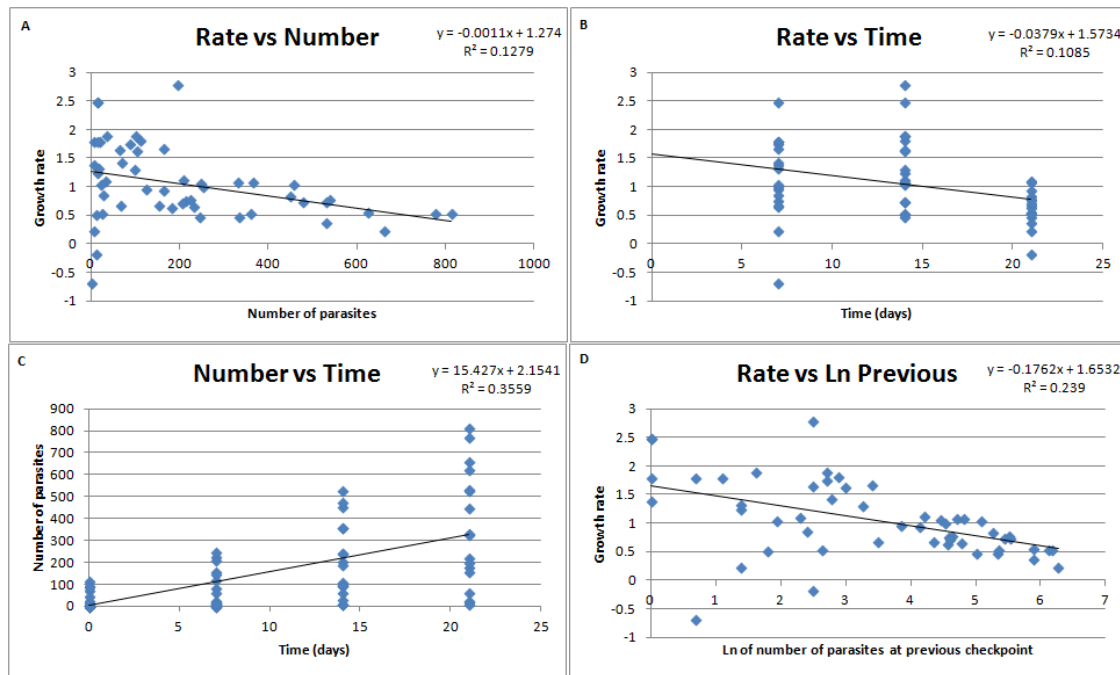
Figure 19: Graphic representation of variables tested in the fourth dataset on Neva salmon. A) Parasite growth rate against parasite density, B) parasite growth rate against time factor, C) parasite density against time factor, D) parasite growth rate against Ln of previous parasite density.

Sweden: Indalsälv

The Swedish population from river Indalsälv showed an establishment of parasites during the first week, with a mean of 115.8 parasites per host, and the infections then grew rapidly in size. The parasite population grew on every host until c. day 35, where on most hosts, parasite population growth began to slow down somewhat (Fig 20c). Most of the host individuals did not experience parasite burdens greater than 1000 parasites, but six reached around 1500, and on two fish, the parasite population was as large as 2000 after 40 days. The parasite growth rate on all hosts showed a significant negative relationship with time, declining the first 14 days, and then stabilizing growth rates around 0-0.1 (Fig 20b). The parasite growth rate shows no significant relationship with parasite density at all (Fig 20a), but the growth rate show a significant negative relationship with the natural logarithm of previous parasite numbers population size at the previous census date. Out of the 24 fish in the experiment, only 3 survived till the end, giving a mortality of 87.5%.
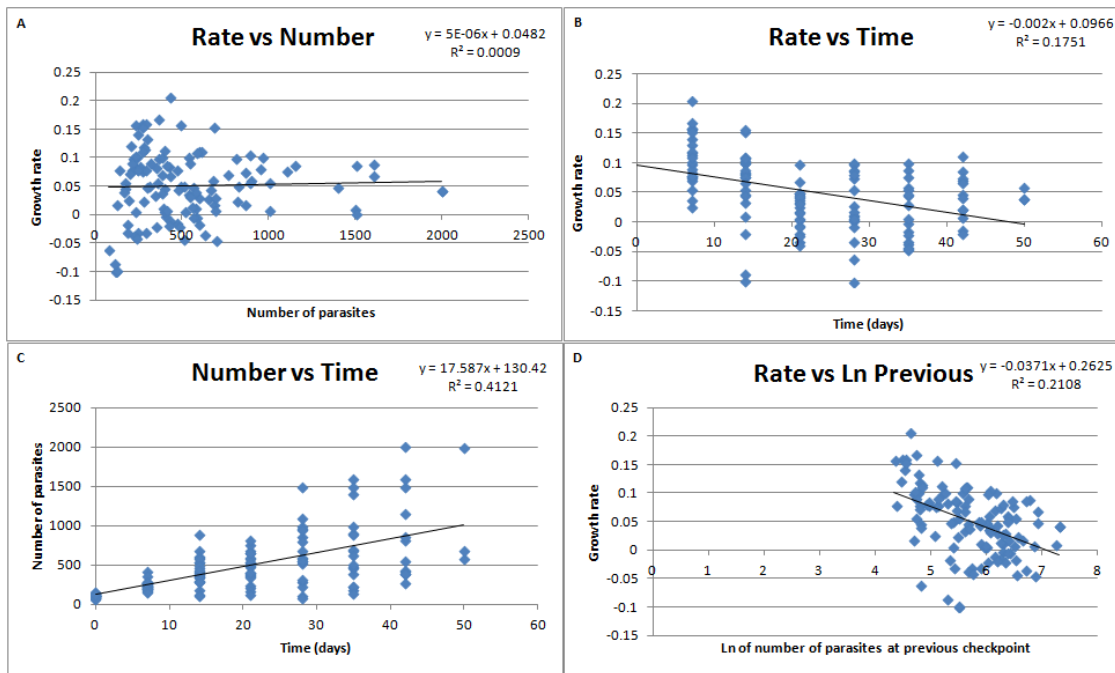
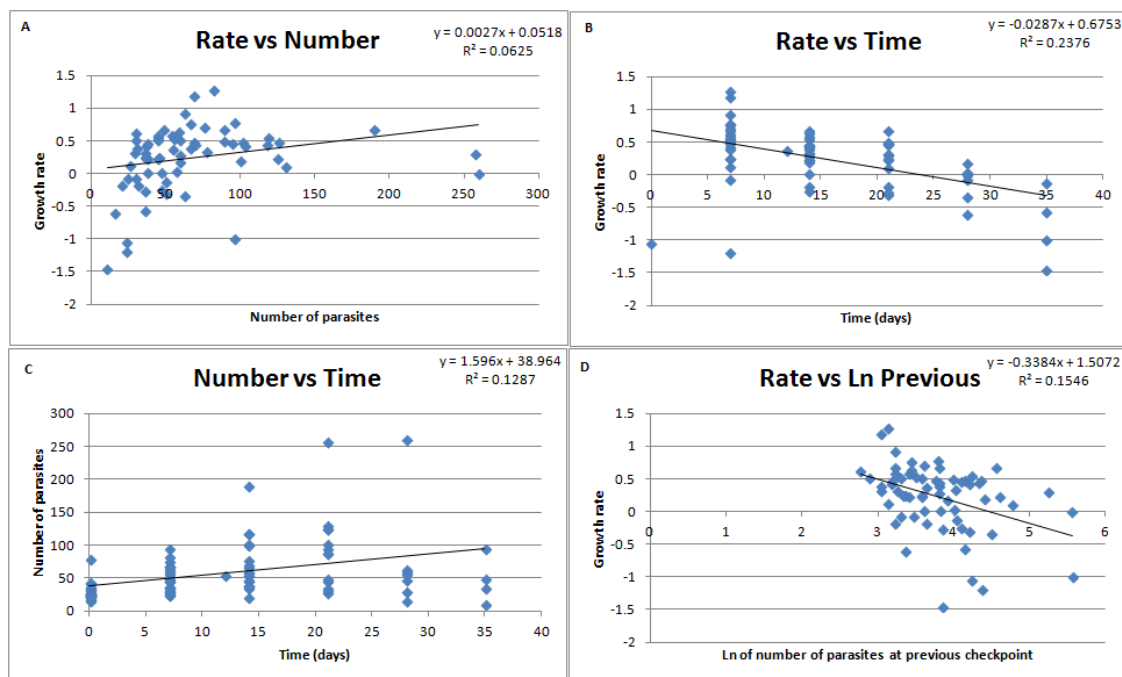**Figure 20: Graphic representation of variables tested in the dataset on Indalsälv salmon. A) Parasite growth rate against parasite density, B) parasite growth rate against time factor, C) parasite density against time factor, D) parasite growth rate against Ln of previous parasite density.**

The plots on the mean parasite population numbers against time, and the mean growth rate against mean density, to correct for any skewed results from a drop in parasite population numbers due to possible host death before end of experiment, show a marked difference in trends of parasite population growth, fitting the results of the plots containing the same variables done on whole datasets, but show more visually pleasing representations of the data (Fig 22). The use of plots only containing values from surviving hosts, makes little to no difference in the strength of the representation of data, as they mach the same type of plots done on the entire population when correcting the means of parasite population number and parasite growth rate in a select few examples plotted (figure, Appendix)

Further analysis of the results

The results obtained in the current study show a significant negative relationship between parasitic population growth rate and parasite population density in 7 of the 16 experimental populations investigated, being the first and third replicate of the Alta stock, the Batnfjord stock, the Ims stock, the first replicate of the Lier stock and the third and fourth replicate of the Neva stock. In these seven experiments, parasite population growth rate is highest when

the parasite population is small, i.e in the first weeks of the experiments. This negative relationship between abundance and population growth rate may be for a number of reasons, for example possibly because population growth of *G.salaris* is fastest when the population age structure is skewed towards young (pre-1st) birth flukes, which is most likely in the first few days and weeks of the infection. This is a result of the asymmetry in parasite reproduction, with the first birth occurring in one third of the time of the second and subsequent births (Cable et al. 2000; Ramirez et al. 2012). This asymmetry is a consequence of the development of embryos within the uterus of the mother, while the mother is itself still an embryo (Bakke et al. 2007), and results in very high initial rates of population growth.

The other 9 datasets, the second replicate of the Alta stock, the second replicate of the Lier stock, both replicates of the Numedals stock, the first and second Neva stock, the Indalsälv stock, and the two Scottish stocks from river Conon and river Shin, show no such relationship between the parasitic growth rate and parasite population density, but rather show only time dependence. Their infections seem to have growth rates unrelated to any trend with parasite population, but rather just time.

When comparing the populations showing a significantly density-dependent parasitic growth rate, to the ones where parasite population growth rate are independent the population density, a distinct pattern is observed.

When controlling for host death by using mean parasite burdens from only those hosts surviving to the end of the experiment, the behavior of parasite population growth over time suggests that almost none of the experimental populations in which parasite population growth rate exhibits density dependence are able to control their infections before death or the end of the experiment. Instead they demonstrate exponential growth in parasite population with no threshold. In contrast, in those experimental populations lacking a statistical significant relationship between parasite population density and parasite population growth rate, the infections were controlled and growth of the parasite population halted, and in some cases the population decreased in size (Fig 21, Appendix). There were some exceptions to this general trend. For example the Ims salmon data set, in which a significantly negative relationship between parasite density and parasite population growth rate was noted, appear at first sight able to control their infections by the end of the

experiment. However, as only 2 of the original 18 fish survived to the end of the experiment, this is not representative, and removing these two survivors from the dataset results in an exponential growth curve for the parasites until the experiment ends. The third Neva replicate also showed an unusual trend, with the population showing a significant relationship between parasite growth rate and parasite population size, but this fish still appeared able to control its infection. One explanation might be that in this replicate the fish did not mount a response to infection because the parasite population never increased above a level of c. 80 parasites, with the exception of one outlier reaching 150. Another factor that could explain this deviation from the trend of density dependent parasitic growth rate and the inability to respond to infection, is the possibility of environmental influence on parasite growth rate, or salmon susceptibility. The third replicate with Alta salmon also showed density dependence weakly, but also appeared able to control infections by the end of the experiment.

When parasite growth rate was corrected for host death by excluding fish from the data set which subsequently died, the decline in parasite population growth rate can be fitted using a predicted polynomial regression, and is similar to the corresponding plots done on whole datasets. When fitting polynomial regressions to these plots of mean population growth rates, we can clearly see that the trajectories of the two types of parasite population behavior (density dependent, no host response versus host response, no density dependence) is inverse to each other Fig (22-27 Appendix). Also, when using this polynomial regression, another deviation in the datasets arises. The fourth Neva replicate looks unusual in that it display exponential growth when plotted using mean density against time, and also density dependence, but when plotted using the mean of parasitic growth rate against time, is showed the same trend as the controlling populations (Fig 25 Appendix). Amongst most of the host populations showing significant density-dependence in parasite population growth rates, but without limitation or control of the parasite population, the polynomial regression represents a negative asymptote, with the growth rate declining most rapidly early in the infection or at low parasite population density. On the other hand, for host populations which control their infections and fail to show parasite population density dependence, the rates can best be fitted by a polynomial regression which is a positive asymptote, with the

fastest decline in the rate of parasite population growth occurring late in the infection, when parasite population size is large (Fig 22-27 Appendix).

In some host stocks on which the parasites exhibit density-independent growth and are controlled, the mean parasite population growth rate declines when plotted against parasite population density until it becomes negative, indicating a loss of parasites. It then however tends towards circularity (skewing the polynomial regression lines), indicating that, after the parasite growth rate has become negative, the parasite population can start to grow again (Fig 22-27 in Appendix). Amongst the other host stocks which exhibit a response to parasite abundance, the initial pattern of change in parasite population growth rate when plotted against parasite abundance is similar, but growth rate does not increase at the end of the experiment. It is probable that, had the experiment been prolonged, the same pattern of fluctuating positive/negative parasite population would have become apparent. The dynamic of host-parasite population growth observed here is similar to findings from experiments with *G.alexanderi* on three-spined sticklebacks (*Gasterosteus aculeatus*) performed by Lester & Adams (1974), who showed that if the parasite infection is controlled and begins to decrease, immune hosts may experience a new increase in parasite populations if a few parasites remain after the bulk of the infection has been lost. This cyclic behavior in host resistance could translate to the circular pattern in parasite growth rate changing with parasite population density observed in the current study.

Density- or Time-dependence as cues for host response

 As 43.8% of the experimental populations investigated show significant relationships between parasite growth rate and parasite population density, and all the datasets used show a significant relationship between parasite growth rate and time, one or both of these factors may play a part in the mounting of a host response to infections of *G.salaris* on east Altantic salmon stocks at least, and perhaps Baltic stocks as well.

The trend shared by all the experimental populations capable of mounting a response to infection, and thereby has no significant density dependent relationship between the parasite growth rate and the density of the parasite population, within both east Atlantic and Baltic stocks, seem to show that the response to infection is based on time passed from initial infection. The vast majority of these responding populations (8 out of 10) seem to

start responding to their parasite burdens around the 21st day post infection (Fig 21). This may indicate the host response being somewhat time-dependent. This timing in response to infection furthermore seems to transcend parasite population density, where both Norwegian and Scottish stocks investigated seem to be able to respond at different densities of parasite population, but at the same time period. Baltic stocks investigated from river Neva show the expected high resistance previously shown for these fish (Cable et al. 2000).

At last, the most bizarre and unexplainable results obtained in this study came from the Numedalslågen replicates which warrants a separate interpretation These salmon showed a highly unusual pattern of parasite population growth when compared to the other populations considered here. They show an unusually slow trend in parasite population growth over time, resembling the asymptotic growth trajectory of the parasite populations found in the density dependent populations investigated with the biggest rate of change happening at later time points (Fig 21). During these experiments, many of the hosts nearly lost the infection altogether several times, further indicating high initial resistance to G.salaris in both replicates. However, since both replicates eventually started to show increased parasite population growth towards the end of the experiments, it is hard to determine if they would have mounted a response given enough time, or continued to show the exponential parasite growth fashion present at the end of the experiments. Furthermore, since one of the replicates displayed a mortality of 44.1 % at the end of the experiment, and the other 11.1 %, both control and exponential growth can be viewed as possible outcomes. Why these hosts showed such a high resistance to parasite population growth the majority of the experiment is uncertain. Reasons for this may be many things, biotic or abiotic. The only certain thing is that these two populations were highly unusal for them being Numedals salmon.

Discussion

With its short generation time and high reproductive output, *G.salaris* is truly an excellent model species when investigating possible methods of adaptation. The high number of different species and strains the gyrodactylids shows that these parasites are highly efficient at adapting to new environments, something underlined by the use of different host species for of transport between main hosts. This explains why *G.salaris* established so rapidly on Norwegian salmon which show a high susceptibility to them in contrast to the co-evolved Baltic stocks of salmon. This paradigm however, being somewhat challenged in the last decade by showing high susceptibility in Swedish Indalsälv salmon, and some potential for control in Lier salmon, as I have reiterated in the current study, may, on the background of new research be proven to not be fitting as anymore. Early infection studies investigating host susceptibility to infection, used small parasite population sizes to look for relationships and host response. In these instances, parasite population growth rate may not be such a useful parameter, as the subtle differences in susceptibility and host response are not as evident in small parasite infections (Ramirez et al. 2012). I suspect this also being the case for at least one of my own datasets, the fourth replicate of Neva salmon. This host population showed a significant relationship between parasite population and parasite growth rate, and not seeming to respond to infection, yet being from a salmon stock with historically high resistance. As the experiment used low initial infections of parasites and terminated after only three weeks, earlier than it took the other population showing host response to control their infections, I suspect this would also have been the case if this population had been allowed to continue its infection. The results obtained in the current study can furthermore be used to explain the classic paradigm of host susceptibility, as the host populations are experiencing the steepest decline in parasite growth rate early, and thereby at low population parasite population intensities, start out with fewer parasites the first weeks of infection, all displayed an exponential growth without being able to control infection, concurrent with the general thought of high susceptibility in east Atlantic stocks of salmon.

When looking at the resulting trends from the datasets then, the general pattern observed in parasite growth rate over time, indicate that if the initial parasite growth rate is more stable

at higher levels early in the experiment, and the steepest change in growth rate doesn't occur until higher time points, the parasite burden will grow faster during this early time period. This results in a higher parasite population on the host these first days. This trend is observed in the experimental populations not showing any statistical relationship between the parasite growth rate and the size of the parasite population itself, i.e density dependent growth rate, but being able to respond to- and control their infections. This initial time when the parasite growth rate is at its highest, lasts for the first 14 days based on the asymptotic growth curves, after which the parasitic growth rate starts to decline with increased rate of change towards zero for the rest of the experiment.

In contrast to this, the populations possessing density dependent growth rates, not being able to respond and control their infections, experience the sharpest decline of parasite growth rate during this same initial 14 days, after which it starts to stabilize. This translates to these populations yielding relatively lower parasite populations during these first couple of weeks.

The difference between these two groups then, with respect to host-parasite dynamic in the populations showing statistically significant relationships between parasite growth rate and parasite density, and the populations with no clear density dependence, is apparently not only the latter groups ability to respond to- and control infection. It is also the initial size of the parasite population gained during the first weeks of the experiments that is differing, and this remains the only observable factor varying before a possible response to infection from the start of infection. Therefore, even if the responses mounted by the populations able to control infection, all occur after an equal amount of time, the initial cue for host response seems rather to be influenced by how large the parasite population is allowed to grow during these first few days post infection. It would seem then, that host response to infection is not exclusively dependent on time, but more likely is controlled indirectly by density of the parasite population at an early period of the infection.

To my knowledge this is the first time this method of investigating density dependence in parasitic growth rate have been used, and subsequently have been able to show this form of density dependence within the population dynamic of *G.salaris*, backed up by statistical significance that the growth rate in a *G.salaris* population can be dependent on the size of

the parasite population when the infection is in its youngest stages. Furthermore, the emerging trend that was found, indicating a density controlled host response, have not been previously investigated either, at least not using common garden experiments between and among stocks of both generally resistance and susceptible salmon populations.

However, even though density dependence in parasitic growth rate is currently an area of very limited study on gyrodactylids, other parasitic species have been has been studied, both endo-, and ectoparasittic. The responses by immune systems in Wood mice, *Apodemus sylvaticus*, to variable density of the nematode *Heligmosomoides polygyrus*, as well as the louse *Polyplax serrata*, show a significant relationship between growth rate and parasite density. The mice mounted immune responses more successfully at lower densities of infection by these parasites indicating a possible parasite-density driven immune response (Jackson et al. 2009). Another example of density dependent growth in parasites comes from the cestode species, *Hymenolepis diminuta.* Roberts (1961) showed that this cestode has a relative high growth rate the first 48 hours when population density is low, but then experiences lower growth rate at higher densities, much like the *G.salaris* populations being able to control their infections.

 Nonetheless, the strong relationship observed between the differences in initial parasite population size, and the ability for some host populations to mount successful responses to an infection by *G.salaris* proves that, at least in these populations, the response to infection being controlled by density dependent cues by the parasite population intensity. Even though the classic paradigm of high susceptibility and an inability to respond to infections in all east Atlantic stocks of salmon, have been somewhat disproved, the comparative and statistical evidence for a density dependent mechanism of host response is certainly novel.

In addition, the results from the current study also show a difference in the ability to control infection within stocks of salmon, shown most prominently in the comparisons of Alta salmon. This may very well indicate a genetic factor for density dependent host response. If this is the case, genetic heterogeneity may be no more important than environmental factors or phenotypic plasticity in determining the direction and outcome of a *G.salaris* infection. Furthermore, the results obtained by statistical comparison as well as comparisons

of host-parasite dynamic, does have a good strength to them based on the experimental method used.

Since every dataset investigated in this study came from common garden experiments on individually isolated fish, we can safely rule out such effects of possibly confounding factors, be it differences in feeding success, temperature, proximity to other hosts, contact with the substrate etc, within each dataset. Such experiments are widely used in studies of local adaptation in parasites, where the genetic composition of local host populations is assumed to be the environmental factor essential for parasite adaptation (Kaweki & Ebert 2004). Environmental factors then, which have the ability to influence the time between first infection, rate of parasite population growth, thereby also and a possible host response, should one choose to believe the results found in this study, could cause biases in the data, affecting the outcome of an experiment. Jansen & Bakke (1993a) showed that susceptibility and/or resistance to *G.salaris* infections on east Atlantic salmon from river Glitra, a tributary to river Lierelva, can be influenced by host size, where parasite densities tend to decline faster from peak infections on larger individuals.

Stress may also be central a factor influencing susceptibility/resistance to infection. Suppression of immune response was simulated by Harris et al. (2000) on three species known to act as transportation hosts for *G.salaris* between salmon hosts, being Brook charr, *Salvelinus fontinalis*, Arctic charr, *Salvelinus alpinus*, and Brown trout, *Salmo trutta*. Brook charr and Arctic charr are show initial susceptibility, but are able to eliminate their infections, whereas Brown trout have shown to be highly resistant. To simulate a stress induced suppression of the host immune system, individuals from these species were implanted with hydrocortisone acetate, which mimics a natural hormone released during stress, and then infected with *G.salaris*. Their findings show the innate ability to resist infection was negatively affected in both Brook trout and Brown trout, where both experiments showed higher parasite burdens, as well as longer durations of infection on individuals treated with hydrocortisone acetate compared to the controls. This was also the case for Arctic charr showing variable levels of susceptibility within stocks. These experiments also included challenge infections 6 months later using the same method of suppressing the immune system, which showed the same results of increased susceptibility in treated individuals. Thereby the immune system of s host looks to be a possible

mechanism for controlling resistance to *G.salaris* infections at least in these species, and probably in other salmonid species as well. Such stress affecting the capability of host resistance, may be induced by various sources. Predation risk, resource competition, mate competition etc may induce the release of stress hormone in the wild, causing a possible bias in host resistance.

Furthermore, successful resistance to *G.salaris* also been seen to be influenced by water temperature; parasite population growth rate is positively correlated with temperature, generating a seasonal variation in parasite resistance and susceptibility (Jansen & Bakke 1993b). The increase in parasite population growth rate with increased temperature is attributed to the negative relationship between parasite life-span and temperature, and also generation time and temperature. (Jansen & Bakke 1991). The relationship with temperature may also have an effect on potential host responses to infection.

The results found in the current study also fit well with previous work in host-parasite dynamic such as the model of response generated by Lester & Adams (1974), so the existence of a density dependent mechanism for host-response may not be so surprising after all, even if new. Further research is indeed needed on this subject to locate stronger evidence for more stocks. Elevated research on these findings may furthermore have implications that could add to the research being done on controlling *G.salaris* presence in Norwegian rivers.

The fact that this density dependence is being somewhat masked behind time dependence in the populations not showing a direct link between parasite population size and parasite growth rate, may also explain the amount of statistically significant trends observed then growth rate was plotted against the natural logarithm of parasite number at previous census dates indicating statistically significant density dependence in instances where it was not directly observed. The main trend of statistical significance here, is that the highly resistant Neva salmon show the weakest link to it, as they do not possess neither the indirect density dependence-inducing host response, as they do seldom need to respond to increasing levels of infection, nor the exponential directly density dependent parasitic growth rate. This method of plotting then, may very well be a more useful tool than previously thought, possibly unmasking the density dependence inducing host response. Further research is truly

needed to better understand the dynamic this fascinating parasite has with its hosts, but for now, this study is the only indication that such a mechanism exists for host-parasite dynamic in *G.salaris.*

Acknowledgements

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Appendix

Glm outputs for all datasets:

Alta 1

	Estimate	Std. Error	t value	Pr(>|t|)
(Intercept)	1.221471	0.080746	15.127	< 2e-16 ***
day	-0.021472	0.002835	-7.574	1.53e-10 ***

AIC: 41.313

	Estimate	Std. Error	t value	Pr(>|t|)
(Intercept)	0.9095945	0.0674473	13.486	< 2e-16 ***
No	-0.0015768	0.0003452	-4.568	2.21e-05 ***

AIC: 65.169

	Estimate	Std. Error	t value	Pr(>|t|)
(Intercept)	1.45983	0.10248	14.245	< 2e-16 ***
lnPrev	-0.20821	0.02563	-8.125	1.58e-11 ***

AIC: 36.706

Alta 2

Estimate Std. Error t value Pr(>\|t\|)

	Estimate	Std. Error	t value	Pr(>|t|)
(Intercept)	0.776152	0.064198	12.090	< 2e-16 ***
day	-0.021231	0.002355	-9.016	2.47e-13 ***

AIC: 2.1216

Estimate Std. Error t value Pr(>\|t\|)

	Estimate	Std. Error	t value	Pr(>|t|)
(Intercept)	0.3037506	0.0737876	4.117	0.000104 ***
No	-0.0002924	0.0003747	-0.780	0.437801

AIC: 56.985

```
        Estimate Std. Error t value Pr(>|t|)
(Intercept)  1.63809   0.21062   7.778 4.69e-11 ***
lonPrev    -0.29602   0.04457  -6.641 5.59e-09 ***
---
```

AIC: 22.427

Alta 3

```
Estimate Std. Error t value Pr(>|t|)
(Intercept)  0.741085   0.052781  14.041  < 2e-16 ***
day        -0.019564   0.001958  -9.991 4.84e-15 ***
---
```

AIC: -23.279

```
         Estimate Std. Error t value Pr(>|t|)
(Intercept)  0.4653941  0.0862030   5.399 8.95e-07 ***
No        -0.0007471  0.0003002  -2.489  0.0152 *
---
```

AIC: 34.146

```
        Estimate Std. Error t value Pr(>|t|)
(Intercept)  2.03606   0.20088  10.136 2.67e-15 ***
lonPrev    -0.34041   0.03841  -8.863 5.30e-13 ***
---
```

AIC: -13.717

Batnfjord:

```
         Estimate Std. Error t value Pr(>|t|)
(Intercept)  2.53273   0.10174  24.89  <2e-16 ***
day        -0.05501   0.00410  -13.42  <2e-16 ***
---
```

AIC: 10.016

        Estimate Std. Error t value Pr(>|t|)

(Intercept) 1.784e+00 9.998e-02 17.841 < 2e-16 ***

No   -5.733e-04 8.948e-05 -6.407 4.69e-08 ***

AIC: 58.78

        Estimate Std. Error t value Pr(>|t|)

(Intercept) 3.44268   0.15778 21.82 <2e-16 ***

lonPrev   -0.41048   0.02888 -14.21 <2e-16 ***

AIC: 5.2063

lms:

        Estimate Std. Error t value Pr(>|t|)

(Intercept) 1.07731   0.06392 16.854 < 2e-16 ***

day   -0.02811   0.00354 -7.941 9.67e-11 ***

AIC: -8.8907

         Estimate Std. Error t value Pr(>|t|)

(Intercept) 0.8184161 0.0690892 11.846 < 2e-16 ***

No   -0.0004710 0.0001392 -3.383 0.00131 **

AIC: 24.071

        Estimate Std. Error t value Pr(>|t|)

(Intercept) 1.99958   0.16425 12.174 < 2e-16 ***

lonPrev   -0.26518   0.03119 -8.502 1.16e-11 ***

AIC: -13.218

Lier 1

        Estimate Std. Error t value Pr(>|t|)

(Intercept) 1.876937 0.103435 18.146 < 2e-16 ***

day   -0.036632 0.004122 -8.888 4.39e-12 ***

AIC: 15.997

        Estimate Std. Error t value Pr(>|t|)

(Intercept) 1.2579431 0.1025901 12.262 < 2e-16 ***

No   -0.0003103 0.0001123 -2.763 0.00786 **

AIC: 58.781

        Estimate Std. Error t value Pr(>|t|)

(Intercept) 2.8489   0.2190 13.012 < 2e-16 ***

lonPrev   -0.3372   0.0397 -8.494 1.83e-11 ***

AIC: 18.923

Lier 2

        Estimate Std. Error t value Pr(>|t|)

(Intercept) 0.1585148 0.0102994 15.39 <2e-16 ***

day   -0.0043672 0.0003528 -12.38 <2e-16 ***

AIC: -408.66

        Estimate Std. Error t value Pr(>|t|)

(Intercept) 4.086e-02 1.133e-02   3.606 0.000432 ***

Popn   7.826e-06 1.635e-05   0.479 0.632977

AIC: -304.13

        Estimate Std. Error t value Pr(>|t|)

(Intercept) 0.283232 0.034600 8.186 1.56e-13 ***

LonPrev   -0.041978 0.006013 -6.982 1.10e-10 ***

AIC: -346.28

Numedals 1

        Estimate Std. Error t value Pr(>|t|)

(Intercept) 0.5566435 0.1162259   4.789 1.14e-05 ***

days   0.0006292 0.0034033   0.185   0.854

AIC: 70.39

        Estimate Std. Error t value Pr(>|t|)

(Intercept) 0.5923644 0.0670096   8.840 1.84e-12 ***

No   -0.0001111 0.0002800 -0.397   0.693

AIC: 70.263

        Estimate Std. Error t value Pr(>|t|)

(Intercept) 0.67869   0.13693   4.957 6.2e-06 ***

lnPrev   -0.02911   0.03581 -0.813   0.42

AIC: 69.747

Numedals 2:

        Estimate Std. Error t value Pr(>|t|)

(Intercept) -27.417   11.114 -2.467 0.0166 *

days   3.471   0.419   8.284 1.81e-11 ***

AIC: 652.98

       Estimate Std. Error t value Pr(>|t|)

(Intercept) 0.362448   0.237400   1.527   0.134

days   0.006059   0.007905   0.766   0.447

AIC: 101.54

       Estimate Std. Error t value Pr(>|t|)

(Intercept) 0.460081   0.127515   3.608 0.000771 ***

No   0.001099   0.001298   0.847 0.401636

AIC: 101.41

       Estimate Std. Error t value Pr(>|t|)

(Intercept) 0.78071   0.23506   3.321 0.00178 **

LnPrev   -0.08972   0.07560 -1.187 0.24155

AIC: 100.7

Conon

       Estimate Std. Error t value Pr(>|t|)

(Intercept) 1.015060   0.060970   16.65   <2e-16 ***

day   -0.027362   0.002098 -13.04   <2e-16 ***

AIC: 104.79

       Estimate Std. Error t value Pr(>|t|)

(Intercept) 3.136e-01 5.404e-02   5.803 3.82e-08 ***

No   -1.518e-05 7.497e-05 -0.203   0.84

AIC: 219.5

```
        Estimate Std. Error t value Pr(>|t|)
(Intercept) 1.88298   0.20502   9.184 3.43e-16 ***
lonPrev    -0.28526   0.03658  -7.799 1.03e-12 ***
---
```

AIC: 167.91

Shin

```
        Estimate Std. Error t value Pr(>|t|)
(Intercept) 1.06850   0.07494  14.26  <2e-16 ***
day        -0.03083   0.00253 -12.19  <2e-16 ***
---
```

AIC: 171.97

```
         Estimate Std. Error t value Pr(>|t|)
(Intercept) 0.1528982  0.0874462  1.748  0.0825 .
No       0.0002000  0.0001378  1.452  0.1486
---
```

AIC: 274.05

```
        Estimate Std. Error t value Pr(>|t|)
(Intercept) 2.26397   0.27519  8.227 9.10e-14 ***
lonPrev    -0.34841   0.04725 -7.374 1.09e-11 ***
---
```

AIC: 229.24

Neva 1

```
         Estimate Std. Error t value Pr(>|t|)
(Intercept) 0.794951  0.103107  7.710 1.15e-10 ***
```

day   -0.034555 0.005754 -6.005 1.04e-07 ***

AIC: 66.732

```
        Estimate Std. Error t value Pr(>|t|)
```

(Intercept) 0.092966 0.102843 0.904 0.3695

No   0.002322 0.001249 1.858 0.0678 .

AIC: 92.684

```
            Estimate Std. Error t value Pr(>|t|)
```

(Intercept) 1.43702   0.36421   3.946 0.000203 ***

lonPrev   -0.31519   0.09546 -3.302 0.001586 **

AIC: 85.779

Neva 2

```
        Estimate Std. Error t value Pr(>|t|)
```

(Intercept) 0.907950 0.135355 6.708 3.26e-09 ***

day   -0.024344 0.004303 -5.658 2.65e-07 ***

AIC: 130.14

```
        Estimate Std. Error t value Pr(>|t|)
```

(Intercept) 0.112527 0.111634 1.008   0.317

No   0.004687 0.003375 1.389   0.169

AIC: 155.55

```
        Estimate Std. Error t value Pr(>|t|)
```

(Intercept) 1.00112   0.20825   4.807 7.69e-06 ***

lonPrev   -0.29145   0.07428 -3.924 0.000192 ***

AIC: 143.13

Neva 3

```
        Estimate Std. Error t value Pr(>|t|)
(Intercept)  1.208574  0.152830  7.908 2.95e-11 ***
day       -0.055443  0.005862  -9.459 4.40e-14 ***
---
```

AIC: 139.15

```
        Estimate Std. Error t value Pr(>|t|)
(Intercept) -0.541623  0.167539  -3.233 0.001881 **
No        0.005647  0.001530  3.690 0.000443 ***
---
```

AIC: 185.39

```
        Estimate Std. Error t value Pr(>|t|)
(Intercept) -0.039846  0.493797  -0.081   0.936
lonPrev    -0.003565  0.120123  -0.030   0.976
```

AIC: 198.18

Neva 4

```
          Estimate  Std. Error  t value  Pr(>|t|)
(Intercept) 0.06866   0.01982   3.464   0.000914 ***
Week       0.02768   0.01060   2.612   0.011007 *
---
```

AIC: -122.51

```
           Estimate  Std. Error  t value  Pr(>|t|)
(Intercept) 1.104e-01  1.586e-02  6.965  1.44e-09 ***
Infection  -1.632e-06  6.181e-05  -0.026   0.979
```

Signif. codes: 0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

AIC: -115.82

Estimate Std. Error t value Pr(>|t|)

(Intercept) 1.65323   0.17110   9.663 4.1e-13 ***

lonPrev   -0.17623   0.04403 -4.002 0.000204 ***

AIC: 96.966

Plots of mean values of variables:
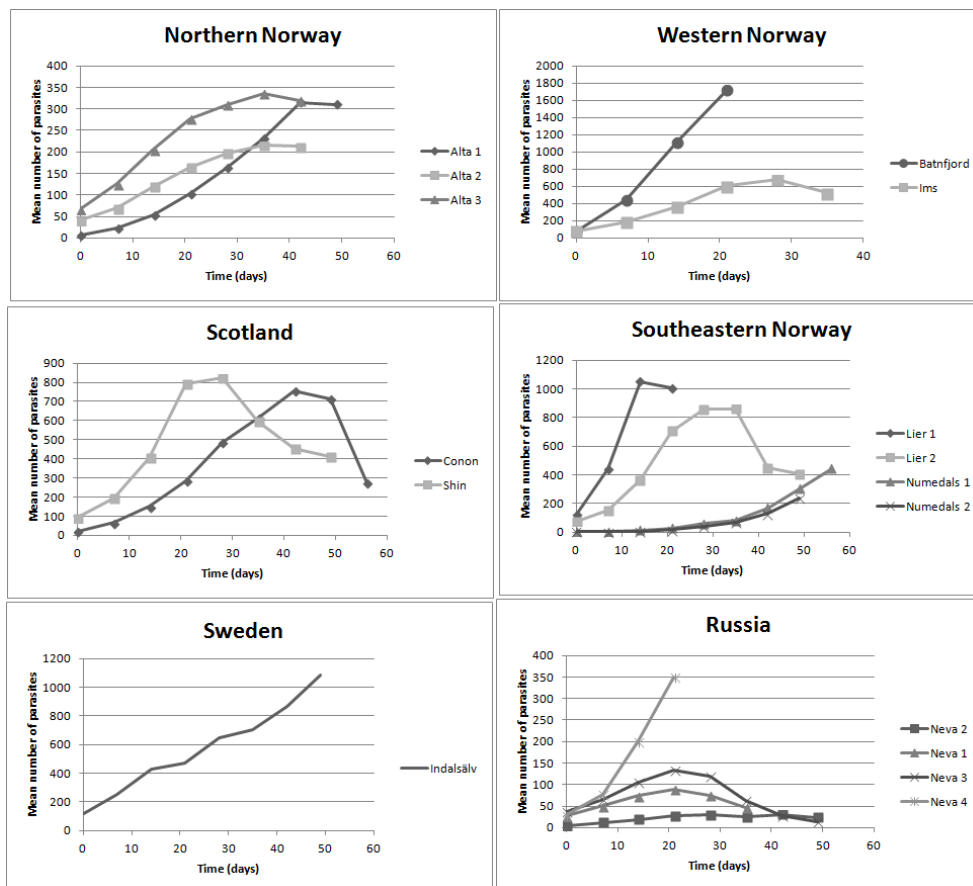
Figure 21: Mean number of parasites in hosts surviving until end of experiment, plotted against time
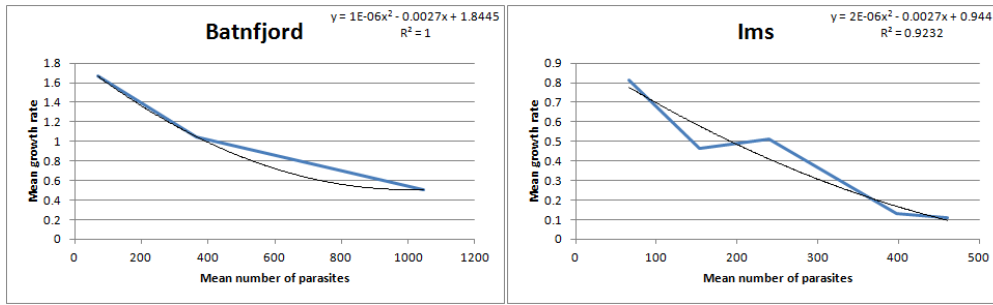
Figure 22: Western Norway. The mean growth rate of the parasite population from surviving hosts plotted against mean number of parasites at the same point in time for each surviving host
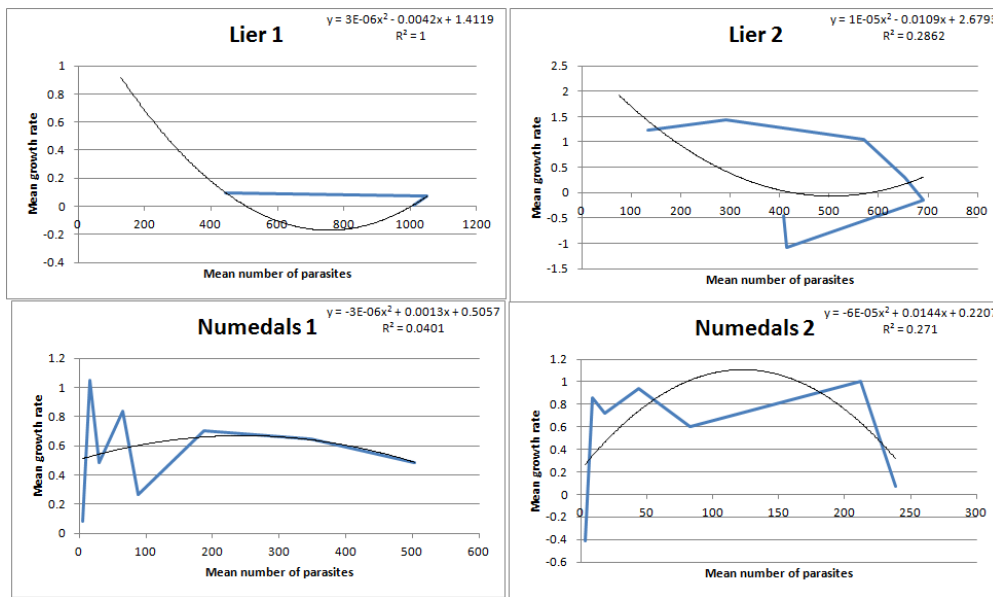
Figure 23: Southeastern Norway. The mean growth rate of the parasite population from surviving hosts plotted against mean number of parasites at the same point in time for each surviving host
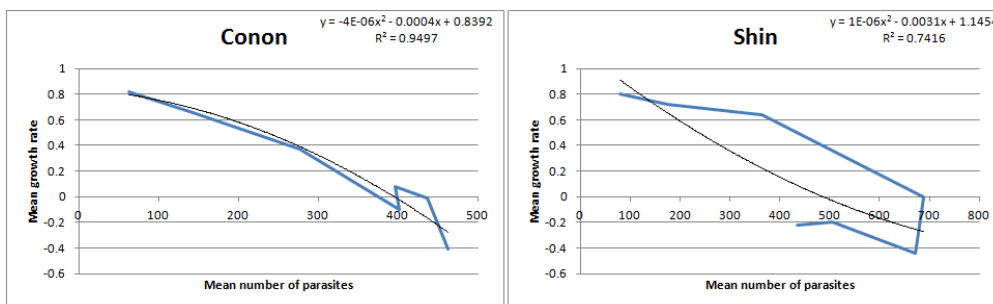
Figure 24: Scotland. The mean growth rate of the parasite population from surviving hosts plotted against mean number of parasites at the same point in time for each surviving host
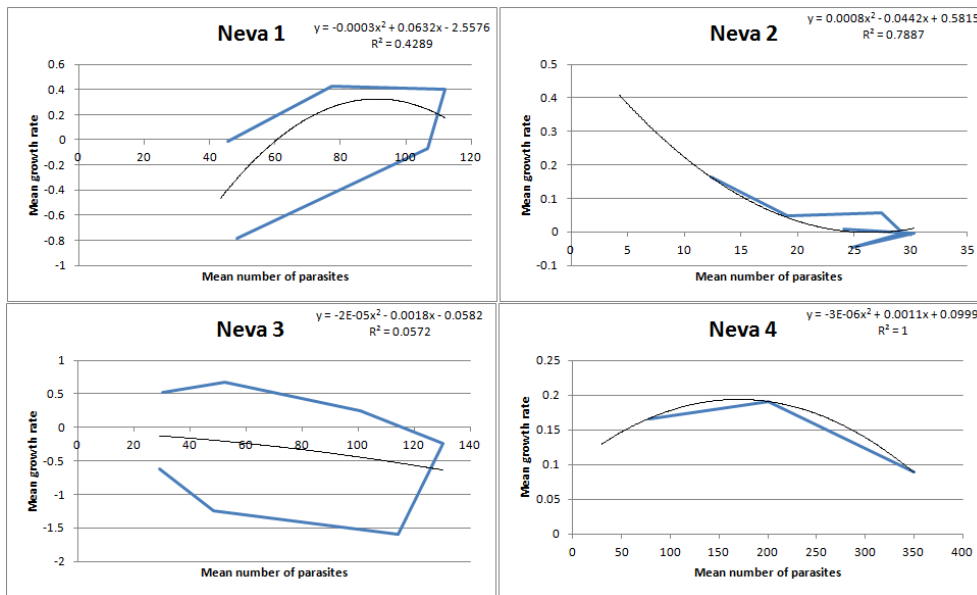
Figure 25: Russia. The mean growth rate of the parasite population from surviving hosts plotted against mean number of parasites at the same point in time for each surviving host
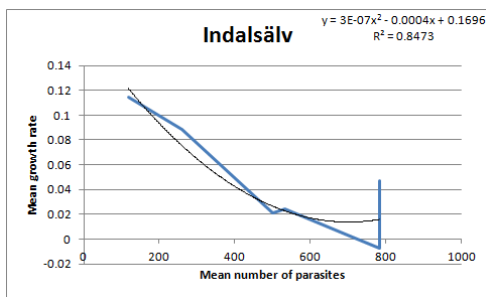
Figure 26: Sweden. The mean growth rate of the parasite population from surviving hosts plotted against mean number of parasites at the same point in time for each surviving host
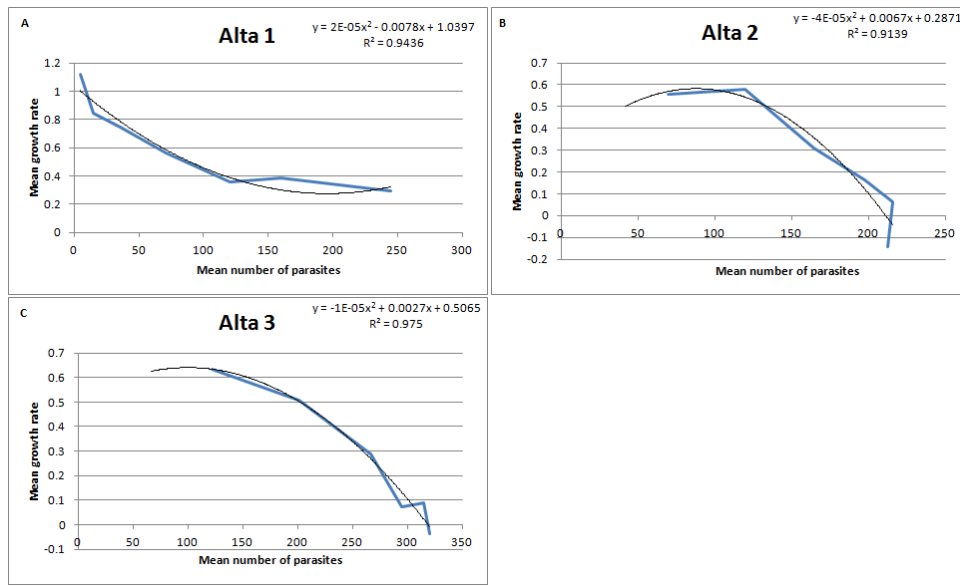
Figure 27: Alta. The mean growth rate of the parasite population from surviving hosts plotted against mean number of parasites at the same point in time for each surviving host

Datasets from own research conducted at NHM, UiO.

Neva:

| Fish	Day post infection	Number of parasites	Ln Number of parasites	Ln Numer of parasites Previous date	Parasite growth Rate
1	0	15	2.70805		
1	7	86	4.454347	2.70805	1.746297
1	14	247	5.509388	4.454347	1.055041
1	21	537	6.285998	5.509388	0.77661
2	0	16	2.772589		
2	7	67	4.204693	2.772589	1.432104
2	14	207	5.332719	4.204693	1.128026
2	21	332	5.805135	5.332719	0.472416
3	0	30	3.401197		
3	7	161	5.081404	3.401197	1.680207
3	14	457	6.124683	5.081404	1.043279
3	21	775	6.652863	6.124683	0.52818
4	0	47	3.850148		
4	7	123	4.812184	3.850148	0.962037
4	14	363	5.894403	4.812184	1.082218
4	21	528	6.269096	5.894403	0.374693
5	0	98	4.584967		
5	7	211	5.351858	4.584967	0.766891

5	14	359	5.883322	5.351858	0.531464
5	21	624	6.43615	5.883322	0.552828
6	0	119	4.779123		
6	7	230	5.438079	4.779123	0.658956
6	14	477	6.167516	5.438079	0.729437
6	21	813	6.700731	6.167516	0.533215
7	0	1	0		
7	7	6	1.791759	0	1.791759
7	14	10	2.302585	1.791759	0.510826
7	21	30	3.401197	2.302585	1.098612
8	0	1	0		
8	7	4	1.386294	0	1.386294
8	14	14	2.639057	1.386294	1.252763
8	21	24	3.178054	2.639057	0.538997
9	0	77	4.343805		
9	7	151	5.01728	4.343805	0.673474
9	14	244	5.497168	5.01728	0.479888
10	0	2	0.693147		
10	7	1	0	0.693147	-0.69315
10	14	12	2.484907	0	2.484907
10	21	10	2.302585	2.484907	-0.18232
11	0	4	1.386294		
11	7	5	1.609438	1.386294	0.223144
11	14	33	3.496508	1.609438	1.88707
11	21	65	4.174387	3.496508	0.67788
12	0	93	4.532599		
12	7	251	5.525453	4.532599	0.992853
12	14	529	6.270988	5.525453	0.745535
12	21	660	6.49224	6.270988	0.221251
13	0	3	1.098612		
13	7	18	2.890372	1.098612	1.791759
13	14	111	4.70953	2.890372	1.819158
13	21	329	5.796058	4.70953	1.086528
14	0	11	2.397895		
14	7	26	3.258097	2.397895	0.860201
14	14	96	4.564348	3.258097	1.306252
14	21	181	5.198497	4.564348	0.634149
15	0	1	0		
15	7	12	2.484907	0	2.484907
15	14	193	5.26269	2.484907	2.777784
15	21	449	6.107023	5.26269	0.844333
16	0	7	1.94591		
16	7	20	2.995732	1.94591	1.049822

16	14	103	4.634729	2.995732	1.638997
16	21	222	5.402677	4.634729	0.767948
17	0	4	1.386294		
17	7	15	2.70805	1.386294	1.321756
17	14	99	4.59512	2.70805	1.88707
17	21	204	5.31812	4.59512	0.723
18	0	2	0.693147		
18	7	12	2.484907	0.693147	1.791759
18	14	63	4.143135	2.484907	1.658228
18	21	162	5.087596	4.143135	0.944462

Numedalslågen 1

Fish	Days post infection	Number of parasites	Ln Number of parasites	Ln Number of parasites at Previous date	Parasite growth rate
Fish1	0	5	1.609438		
Fish1	7	4	1.386294	1.609438	-0.22314
Fish1	14	14	2.639057	1.386294	1.252763
Fish1	21	8	2.079442	2.639057	-0.55962
Fish1	28	33	3.496508	2.079442	1.417066
Fish1	35	37	3.610918	3.496508	0.11441
Fish1	41	78	4.356709	3.610918	0.745791
Fish1	48	195	5.273	4.356709	0.916291
Fish1	55	364	5.897154	5.273	0.624154
Fish2	0	5	1.609438		
Fish2	7	7	1.94591	1.609438	0.336472
Fish2	14	18	2.890372	1.94591	0.944462
Fish2	21	32	3.465736	2.890372	0.575364
Fish2	28	83	4.418841	3.465736	0.953105
Fish2	35	94	4.543295	4.418841	0.124454
Fish2	41	224	5.411646	4.543295	0.868351
Fish2	48	361	5.888878	5.411646	0.477232
Fish2	55	495	6.204558	5.888878	0.31568
Fish4	0	5	1.609438		
Fish4	7	5	1.609438	1.609438	0
Fish4	14	14	2.639057	1.609438	1.029619
Fish4	21	41	3.713572	2.639057	1.074515
Fish4	28	84	4.430817	3.713572	0.717245
Fish4	35	119	4.779123	4.430817	0.348307
Fish4	41	236	5.463832	4.779123	0.684708
Fish4	48	472	6.156979	5.463832	0.693147
Fish4	55	638	6.458338	6.156979	0.301359

Fish5	0	5	1.609438		
Fish5	7	6	1.791759	1.609438	0.182322
Fish5	14	15	2.70805	1.791759	0.916291
Fish5	21	30	3.401197	2.70805	0.693147
Fish5	28	77	4.343805	3.401197	0.942608
Fish5	35	121	4.795791	4.343805	0.451985
Fish5	41	330	5.799093	4.795791	1.003302
Fish5	48	665	6.499787	5.799093	0.700694
Fish5	55	772	6.648985	6.499787	0.149198
Fish7	0	5	1.609438		
Fish7	7	5	1.609438	1.609438	0
Fish7	14	10	2.302585	1.609438	0.693147
Fish7	21	19	2.944439	2.302585	0.641854
Fish7	28	34	3.526361	2.944439	0.581922
Fish7	35	49	3.89182	3.526361	0.36546
Fish7	41	87	4.465908	3.89182	0.574088
Fish7	48	186	5.225747	4.465908	0.759839
Fish7	55	356	5.874931	5.225747	0.649184
Fish8	0	5	1.609438		
Fish8	7	11	2.397895	1.609438	0.788457
Fish8	14	29	3.367296	2.397895	0.969401
Fish8	21	60	4.094345	3.367296	0.727049
Fish8	28	115	4.744932	4.094345	0.650588
Fish8	35	162	5.087596	4.744932	0.342664
Fish8	41	300	5.703782	5.087596	0.616186
Fish8	48	449	6.107023	5.703782	0.40324
Fish8	55	604	6.403574	6.107023	0.296551
Fish9	0	5	1.609438		
Fish9	7	3	1.098612	1.609438	-0.51083
Fish9	14	14	2.639057	1.098612	1.540445
Fish9	21	18	2.890372	2.639057	0.251314
Fish9	28	33	3.496508	2.890372	0.606136
Fish9	35	37	3.610918	3.496508	0.11441
Fish9	41	56	4.025352	3.610918	0.414434
Fish9	48	102	4.624973	4.025352	0.599621
Fish9	55	292	5.676754	4.624973	1.051781

Numedalslågen 2

| Fish	Days post infection	Number of parasites	Ln Number of parasites	Ln Number of parasites at Previous date	Parasite growth rate
Fish10	0	5	1.609438	NA	NA
Fish10	7	4	1.386294	1.609438	-0.22314
Fish10	14	9	2.197225	1.386294	0.81093
Fish10	21	12	2.484907	2.197225	0.287682
Fish10	28	48	3.871201	2.484907	1.386294
Fish10	35	69	4.234107	3.871201	0.362905
Fish10	42	140	4.941642	4.234107	0.707536
Fish10	49	164	5.099866	4.941642	0.158224
Fish11	0	5	1.609438	NA	NA
Fish11	7	0	NA	1.609438	NA
Fish11	14	3	1.098612	NA	NA
Fish11	21	3	1.098612	1.098612	0
Fish11	28	23	3.135494	1.098612	2.036882
Fish11	35	43	3.7612	3.135494	0.625706
Fish11	42	114	4.736198	3.7612	0.974998
Fish12	0	5	1.609438	NA	NA
Fish12	7	3	1.098612	1.609438	-0.51083
Fish12	14	5	1.609438	1.098612	0.510826
Fish12	21	11	2.397895	1.609438	0.788457
Fish12	28	50	3.912023	2.397895	1.514128
Fish12	35	68	4.219508	3.912023	0.307485
Fish12	42	137	4.919981	4.219508	0.700473
Fish13	0	5	1.609438	NA	NA
Fish13	7	0	NA	1.609438	NA
Fish14	0	5	1.609438	NA	NA
Fish14	7	3	1.098612	1.609438	-0.51083
Fish14	14	13	2.564949	1.098612	1.466337
Fish14	21	31	3.433987	2.564949	0.869038
Fish14	28	60	4.094345	3.433987	0.660357
Fish14	35	134	4.89784	4.094345	0.803495
Fish14	42	269	5.594711	4.89784	0.696872
Fish14	49	171	5.141664	5.594711	-0.45305
Fish15	0	5	1.609438	NA	NA
Fish15	7	5	1.609438	1.609438	0
Fish15	14	9	2.197225	1.609438	0.587787
Fish15	21	22	3.091042	2.197225	0.893818
Fish15	28	40	3.688879	3.091042	0.597837
Fish15	35	97	4.574711	3.688879	0.885832
Fish15	42	35	3.555348	4.574711	-1.01936
Fish16	0	5	1.609438	NA	NA
Fish16	7	3	1.098612	1.609438	-0.51083
Fish16	14	4	1.386294	1.098612	0.287682
Fish16	21	11	2.397895	1.386294	1.011601
Fish16	28	24	3.178054	2.397895	0.780159
Fish16	35	46	3.828641	3.178054	0.650588

Fish16	42	227	5.42495	3.828641	1.596309
Fish16	49	381	5.942799	5.42495	0.517849
Fish17	0	5	1.609438	NA	NA
Fish17	7	2	0.693147	1.609438	-0.91629
Fish17	14	12	2.484907	0.693147	1.791759
Fish17	21	36	3.583519	2.484907	1.098612
Fish17	28	40	3.688879	3.583519	0.105361
Fish17	35	32	3.465736	3.688879	-0.22314
Fish17	42	65	4.174387	3.465736	0.708651
Fish18	0	5	1.609438	NA	NA
Fish18	7	1	NA	1.609438	NA
Fish18	14	8	2.079442	NA	NA
Fish18	21	22	3.091042	2.079442	1.011601
Fish18	28	43	3.7612	3.091042	0.670158
Fish18	35	89	4.488636	3.7612	0.727436
Fish18	42	58	4.060443	4.488636	-0.42819
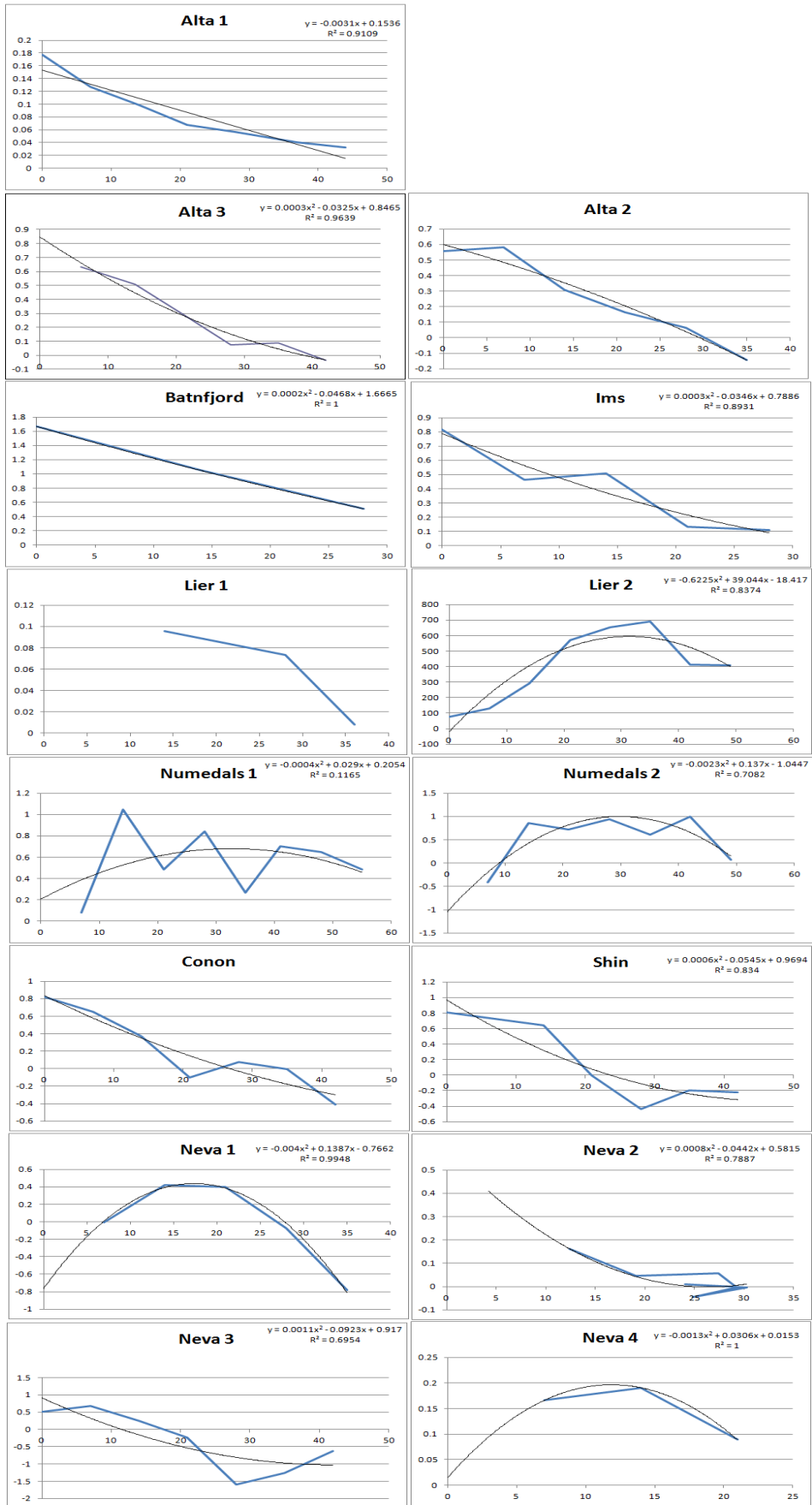
Figur 28: Plots showing mean growth rate plotted against time, indication the inverse trajectories observed.