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Predicting the potential for natural recovery of Atlantic salmon (*Salmo salar* L.) populations following the introduction of *Gyrodactylus salaris* Malmberg, 1957 (Monogenea)

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1 **Predicting the potential for natural recovery of Atlantic salmon**
2 **(*Salmo salar* L.) populations following the introduction of**
3 ***Gyrodactylus salaris* Malmberg, 1957 (Monogenea)**

4
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17 **Abstract**

18 *Gyrodactylus salaris* (Monogenea, Platyhelminthes) is a notifiable freshwater pathogen
19 responsible for causing catastrophic damage to wild Atlantic salmon stocks, most notably in
20 Norway. In some strains of Baltic salmon (*e.g.*, from the river Neva) however, the impact is
21 greatly reduced due to some form of innate resistance that regulates parasite numbers,
22 resulting in fewer host mortalities. *Gyrodactylus salaris* is known from 17 European states; its
23 status in a further 35 states remains unknown; the UK, the Republic of Ireland and certain
24 watersheds in Finland are free of the parasite. Thus, the parasite poses a serious threat if it
25 emerges in Atlantic salmon rearing regions throughout Europe. At present, infections are
26 generally controlled via extreme measures such as the treatment of entire river catchments
27 with the biocide rotenone, in order to remove all hosts, before restocking with the original
28 genetic stock. The use of rotenone in this way in EU countries is unlikely as it would be in
29 contravention of the Water Framework Directive. Not only are such treatments economically
30 and environmentally costly, they also eradicate the potential for any host/parasite evolutionary
31 process to occur. Based on previous studies, UK salmon stocks have been shown to be highly
32 susceptible to infection, analogous to Norwegian stocks. The present study investigates the
33 impact of a *G. salaris* outbreak within a naïve salmon population in order to determine long-
34 term consequences of infection and the likelihood of coexistence. Simulation of the salmon/ *G.*
35 *salaris* system was carried out via a deterministic mathematical modelling approach to examine
36 the dynamics of host-pathogen interactions. Results indicated that in order for highly
37 susceptible Atlantic strains to evolve a resistance, both a moderate-strong deceleratingly costly
38 trade-off on birth rate and a lower overall cost of the immune response are required. The
39 present study provides insights into the potential long term impact of *G. salaris* if introduced
40 into *G. salaris*-free territories and suggests that in the absence of external controls salmon
41 populations are likely to recover to high densities nearing 90% of that observed pre-infection.

42

43 Introduction

44 *Gyrodactylus salaris* Malmberg, 1957 is a viviparous (*i.e.*, live-bearing) freshwater ecto-
45 parasite that infects both wild and farmed populations of Atlantic salmon (*Salmo salar* L.),
46 potentially resulting in juvenile host mortality. It is an Office International des Epizooties (OIE)
47 listed pathogen that was first described from the fins and skin of a Baltic Atlantic salmon strain
48 from a hatchery in Sweden located near the Indalsälvs river [1]. The parasite is believed to be
49 native to the waters of northern Russia, western Sweden and northern Finland [2], but is now
50 known to be widely distributed throughout Europe [3–10] and recently confirmed in Romania
51 [11]. In Norway, the parasite has caused catastrophic damage to wild populations of Atlantic
52 salmon parr since it was first observed in the mid-1970s after a period of mass salmon mortality
53 [12–15]. Moreover, this parasite is known to have been introduced to Norway on at least three
54 separate occasions [16] and can reduce salmon stock in rivers by approximately 85% on
55 average [10]. Within 5 years of initial introduction to a susceptible host population reductions
56 in outbound smolts can be as high as 98% [10,12,17]. This has caused severe damage to the
57 Norwegian economy and to wild salmon fisheries. Although infections in salmon hatcheries
58 have been reported, such infections are more readily controlled, however, if left untreated
59 salmon mortality can reach 100% [10]. In the years post introduction, *G. salaris* has been
60 reported from 50 rivers, 13 Atlantic salmon hatcheries and 26 rainbow trout (*Oncorhynchus*
61 *mykiss* Walbaum) hatcheries in Norway and subsequently managed through coordinated
62 intervention [18]. Subsequent losses to the Norwegian salmon industry up until 2004 exceeded
63 US\$ 655m [19]. The last time loss figures were estimated annual loss of wild juvenile salmon
64 was suggested to be in the region of 250 - 500 metric tonnes as a consequence of parasitic
65 infection reducing the average density of salmon parr in infected rivers [19]. Such annual loss
66 costs the Norwegian economy over US\$ 55m per annum through surveillance and eradication
67 (circa US\$ 23m per annum) along with losses incurred by fisheries, associated industries and

68 tourism (circa US\$ 34m per annum) [14]. Hence, *G. salaris* poses a serious threat if it establishes
69 in territories that are currently *G. salaris* free [9].

70

71 Though *G. salaris* has had a huge impact in Norway, some Baltic strains of Atlantic
72 salmon appear to be more resistant to the parasite than the Atlantic strains [19]. Bakke *et al.*
73 [20] was the first study to show a difference in the immune response between two strains of
74 salmon. In particular, they showed that parasite numbers grew exponentially on individual fish
75 from an Atlantic strain of Atlantic salmon from the rivers Lone and Alta (Norway), whereas on a
76 Baltic strain of Atlantic salmon from the river Neva (Russia) there was some initial growth in
77 parasite numbers, but those numbers peaked and then generally decreased to zero. This clearly
78 demonstrated some differences in susceptibility of these salmon strains to *G. salaris* through the
79 ability of the some Baltic strains to exhibit some form of resistance or immune response [19–
80 23]. It has been highlighted that the resistance observed in some Baltic salmon strains, such as
81 those from the Neva river, is due to the presence of the parasite in the Baltic watershed since
82 the last glacial period allowing an evolutionary selection process within the host [22]. This
83 supports the hypothesis that *G. salaris* is a recent (c. 40 years) introduction to Norwegian rivers
84 and potentially explains why Norwegian Atlantic salmon are particularly susceptible to the
85 parasite.

86 Due to the impact of *G. salaris* on Norwegian salmon, extreme measures have been taken
87 to try and control and eradicate the parasite. These measures include the treatment of entire
88 river catchments with the biocide rotenone [24] to remove all hosts (and hence, *G. salaris*),
89 before restocking with the original genetic stock [12,14,25,26]. The use of rotenone in this way
90 in EU countries is unlikely as it would be in breach of the Water Framework Directive [27]. Not
91 only are such treatments economically and environmentally costly, they also eradicate the
92 potential for any host/parasite evolutionary process to occur.

93

94 Currently the only European countries recognised as free from *G. salaris* infection are
95 the United Kingdom [28,29], the Republic of Ireland [9,30,31], and some areas of Finland [9,32].
96 Other countries such as Portugal, Spain and France, where *G. salaris* has been previously
97 recorded, are believed to be misidentifications with a morphologically similar species
98 *Gyrodactylus teuchis* Lautraite, Blanc, Thiery, Daniel et Vigneulle, 1999 [32,33]. The collection of
99 further material from these states is required to determine their current *G. salaris* status.
100 Recently, however, it was proposed that *G. salaris* and *G. thymalli* Žitňan, 1960, another
101 morphologically similar and closely-related, but benign parasite of grayling, *Thymallus*
102 *thymallus* L., may represent a single species of *Gyrodactylus* that comprises several pathogenic
103 and non-pathogenic strains on a number of primary hosts [34]. The study [34] analysed
104 microRNA loci from a small number of populations of *Gyrodactylus* from Atlantic salmon and
105 grayling hosts and made the proposal that the two species should be synonymised, however,
106 this has not yet been formally accepted by the OIE and as such this synonymisation is yet to be
107 accepted by the scientific community [11].

108 Despite the fact *G. salaris* is not present in the UK but *G. thymalli* is, it has been
109 demonstrated that UK salmon populations have similar levels of susceptibility to infection as
110 those in Norway [15,23,35,36]. Due to this, *G. salaris* is regarded to pose a serious disease
111 threat to the UK's valuable wild and farmed salmon populations [37]; a report to the Scottish
112 Government advised if *G. salaris* were introduced into Scotland, as an example of potential
113 impact, then the potential losses would be estimated at £44.8 million per annum to the Scottish
114 economy, £34.5 million to Scottish household income each year and 1,996 full time equivalent
115 jobs lost in Scottish employment [38]. It is also likely that *G. salaris*, if introduced, would spread
116 within and between UK rivers before it is detected [2]. Due to this, contingency plans were
117 drawn up setting out a series of actions to follow in the event of an outbreak [37]. Using
118 mathematical modelling approaches based on the existing knowledge of *G. salaris*, the present
119 study aims to simulate salmon/*G. salaris* interaction dynamics in order to investigate the

120 potential for natural recovery of susceptible salmon populations post introduction of *G. salaris*
121 infection.

122 The majority of previous mathematical modelling work concerning the salmon/*G. salaris*
123 system has been centred on risk and statistical analysis highlighting areas such as routes of
124 infection, transmission and risk of introduction [2,39–43]. Some work has been carried out to
125 study the effects of *G. salaris* on different stages of the salmon life-cycle [44] as well as the effect
126 of other gyrodactylid species such as *Gyrodactylus turnbulli* Harris, 1986 on guppies, *Poecilia*
127 *reticulata* Peters, 1859 [45,46]. More recently stochastic models have become popular in
128 studying *G. salaris* infections in salmon and modelling techniques such as Leslie matrix
129 population models and individual based models have also been employed [47–49]. Though a
130 great deal of effort has been placed on understanding the risks and routes by which the parasite
131 may be introduced, little has been done to predict its long-term impact. Moreover, not much is
132 known about what may happen should control efforts similar to those employed in Norway not
133 be possible.

134 In the present study a series of host-macroparasite models are developed, first
135 considering a single fish host and incorporating that into a population model. The effects that an
136 increased immune response has on the host and parasite populations are analysed
137 demonstrating the difference in susceptibility between a highly susceptible salmon strain and a
138 resistant strain. Finally, some mutation and replacement is incorporated to determine how
139 strong an immune response the hosts develop and what types of trade-offs and parameter
140 values are required to allow a fully susceptible host to evolve into a primarily resistant host.

141 **Methods**

142

143 **Individual fish model**

144 To model parasite numbers on an individual host a deterministic ordinary differential
145 equation (ODE) approach is taken. For the number of parasites, P , a simple exponential growth
146 model is assumed, with replication rate μ , death rate ε and dislodgement rate λ . In addition, we
147 include an immune response, I , exhibited by the host which activates at rate m as parasite
148 numbers grow; this in turn increases the parasite death rate by a rate ρI . Finally, the immune
149 response decays at a continuous rate ξ . The equations for these are shown in equation (1)
150 below:

151

$$\begin{aligned}\frac{dP}{dt} &= P(\mu - \varepsilon - \rho I - \lambda) \\ \frac{dI}{dt} &= mP - \xi I\end{aligned}$$

152 (1)

153

154 **Full salmon population model**

155 The individual fish host model was expanded by scaling up the equations in (1), to a
156 population of hosts and parasites. Here the host population, H , is assumed to follow a logistic
157 growth function, a being the birth rate, b the natural death rate and s representing density-
158 dependent competition, with an additional death rate dependent on parasite burden, αM . The
159 equations for average parasite burden, $M = P/H$, or density of parasite per host (where P is the
160 total on-host parasite density), and immune response, I , are taken from equation (1), but
161 expanded in that the parasite burden decreases due to deaths of the host due to infection, α , and
162 birth of new (initially parasite-free) hosts. The on-host parasite distribution is assumed to
163 follow a Poisson distribution across the host population, which is taken into account in the
164 parasite-induced death rate, α . Both Poisson and negative binomial distributions were

165 considered with each giving similar results, the Poisson however, simplified the model
 166 significantly and thus was chosen. The off-host parasite density, W , is assumed to increase as the
 167 parasites leave the host (either by choice or host death) and decrease due to parasite death, σ ,
 168 or parasite latching on to hosts at a rate β , which in turn increases parasite burden. It is
 169 important to note that actual parasite death rates are highly dependent on many factors such as
 170 environmental conditions (*e.g.* temperature), water quality, salinity, *etc.* [50,51]. In the present
 171 study, however, we consider a simplified worst case scenario such that we have a highly
 172 pathogenic strain of parasite and a highly susceptible Atlantic salmon strain.

173 The dynamics for the model take the form in (2). Further details of the model's
 174 derivation are presented in the Supplementary Information (Appendix S1). Parameter values
 175 used in all models are given in Table 1. Parameter values regarding the UK were used where
 176 available.

$$\frac{dH}{dt} = (a - b - sH)H - \alpha MH$$

$$\frac{dM}{dt} = (\mu - \varepsilon - \rho I - \lambda - \alpha - a)M + \beta W$$

$$\frac{dI}{dt} = mM - \xi I$$

$$\frac{dW}{dt} = MH[\lambda + b + sH + \alpha(1 + M)] - \sigma W - \beta WH$$

177 (2)

178

179 **Table 1. List of parameter values used to inform salmon/*G. salaris* host parasite models.**

Parameter	Description	Estimate/day	Source
a	Maximum salmon birth rate	0.02	Assumed
b	Salmon natural death rate	0.00057	[52]
K	Salmon carrying capacity	0.125	[52]
s	Density dependent constraint	0.000155	Estimated using K for 1000 m ²
μ	<i>G. salaris</i> birth rate (Norway)	0.1825	[20]
	<i>G. salaris</i> birth rate (UK)*	0.1708	[15]
ϵ	<i>G. salaris</i> on-host death rate	0.08	[50]
σ	<i>G. salaris</i> off-host death rate	0.14-0.17	[42]
λ	Rate the parasites leave the hosts	0.06	Assumed
β	Parasites attach rate to hosts	0.0585	Assumed
α	Parasite induced death rate of host	0.02	[45]
m	Rate hosts develop an immune response	0 – 0.0175	Assumed
ξ	Decay rate of immune response	0.0055	Assumed
ρ	Rate of increase in parasite mortality due to resistance	1	Adjusted in values of m

180 * parameter value used in this study

181

182 With macro-parasite models, such as those used in the present study, fish-to-fish
183 transmission is not shown explicitly in the model, but is rather an implicit feature modelled

184 through the distribution of parasites across the fish population. This is due to the fact that P
185 gives the total number of on-host parasites which remains unchanged as parasites switch
186 between fish hosts, and due to the large number of parasites involved in these systems, the
187 effect on the distribution of parasites is negligible.

188

189 **Results**

190

191 **Single host model**

192 Using the single host model, equation (1), two different cases were considered (Fig 1):
193 firstly, a highly susceptible Atlantic salmon strain with no immunity, $m \approx 0$; secondly, a resistant
194 salmon strain, $m > 0$ ($m = 0.0175$). Model simulations showed parasite numbers grew
195 exponentially on the susceptible host, whereas on the resistant host parasite numbers decayed
196 to zero. In the case of the resistant host initial parasite growth over the first 7 days was similar
197 to the highly susceptible host, however, parasite population growth slowed thereafter, peaking
198 at around 20 days, before decreasing to zero/low levels. These behaviours approximately follow
199 the experimental results observed by Bakke *et al.* [20] at water temperatures of 12°C on Atlantic
200 Lone and Baltic Neva salmon hosts.

201

202 **Fig 1:** Output from the model in (1) for parasite numbers, with $m = 0$ (susceptible salmon strain
203 – solid line) and $m > 0$ (resistant salmon strain – dashed line).

204

205 **Full salmon population model**

206 Firstly, the model in (2) was simulated to consider a fully susceptible host with a
207 negligible immune response, *i.e.* $m \approx 0$. Here, following the introduction of the parasite into the
208 system the model shows a fast drop in the number of hosts. This mirrors the results in the field,
209 *e.g.*, in Norway where the parasite can reduce the salmon parr population by up to 98% within 5
210 years [12]. As host extinction has not been witnessed, and the average reduction in salmon is
211 86% (and sometimes lower), we can assume that although $m \neq 0$, it must be very small. As we
212 increase the amount of immune response, m (Fig 2A), we very quickly see that the host
213 (equilibrium) population recovers and the average parasite burden decreases. In fact, only
214 negligible values for m produces a reduction approaching 100%, and even a small amount of
215 resistance significantly improves host population size. Moreover, host numbers approach their
216 pre-infection levels, and parasite burden approaches zero, as m gets large. Interestingly the
217 greatest effect on host and parasite numbers occurs at lower increases in immune response m ,
218 with only marginal effects for larger m .

219

220 **Fig 2:** Plot of host (equilibrium) population H (solid line) and parasite burden M (dashed line).
221 (A) with no trade-off; (B) with a linear trade-off on host birth rate. The dotted line represents
222 the (fully susceptible) host population before the parasite outbreak.

223

224 **The trade-off**

225 So far we have assumed that the immune response mounted by the host is cost free.
226 This, however, has been shown not to be the case. One prime example of this is a study of
227 furunculosis in brook trout, *Salvelinus fontinalis* (Mitchill) [53], in which it was shown that an
228 increase in immunity had a negative effect on the host's birth rate; they observed approximately
229 a 7 to 12% decrease in the birth rate of the trout that exhibited resistance to infection. Although

230 there is no evidence to support or deny that a similar trade-off exists in salmon, for the
231 remainder of this study we hypothesise there is a cost of the immune response. In particular, we
232 take a trade-off such that the development of an effective immune response, as measured here
233 by m , can have a significant negative effect on host birth rate a , such that $a = a(m)$ with $a'(m) < 0$.
234 Although the form of $a(m)$ is unknown, we make two assumptions: i) when $m = 0$, $a = 0.02$
235 (maximum birth rate) representing a highly susceptible salmon strain, and ii) when $m = 0.0175$
236 (our maximum resistance), birth rate a is reduced by 10% representing a resistant salmon
237 strain. We initially take a linear trade-off (straight line) passing through these two points to
238 allow us to interpolate a for intermediate m .

239 The addition of this trade-off has a marked effect on the host population. In particular, at
240 high levels of immune response, m , the cost of a lower birth rate begins to outweigh the benefit
241 of higher immune response (and subsequent lower parasite burden) and the host population
242 begins to decrease (Fig 2B). Here an optimal level of immunity now exists which maximises the
243 host population when $m = 0.010$.

244

245 **Mutation and replacement of hosts**

246 The optimal immune response observed may not, however, represent the level of m that
247 the host species evolve to; this instead would likely be determined by the level of m which
248 optimises the growth rate of the host population. To study the long-term evolution of immune
249 response, we take a mutation and replacement approach, broadly following that of adaptive
250 dynamics [54].

251 Consider a single resident host strain of salmon, with immune response m and
252 population density H existing alone in an environment, with the dynamics as given in equation
253 (2). Now suppose a mutation creates a host with slightly different immune response \hat{m} , with
254 population density \hat{H} . Mutations are generally small, and hence, the difference between m and
255 \hat{m} is small. Here \hat{M} and \hat{I} are the (average) parasite burden and immune response for this

256 mutant host strain. If this new type is initially rare, then we can write down the fitness of this
257 mutant type, *i.e.* the long-term growth rate of this mutant population, as

258

$$r(\hat{m}, m) = a(\hat{m}) - b - sH(m) - \alpha\hat{M}(\hat{m}, W(m))$$

259 (3)

260 Here \hat{M} is the average parasite burden on a mutant host. We make the assumption that
261 parasites will reach their “average” (equilibrium) burden on the new mutant host type \hat{M}
262 quickly, when compared to the natural fish lifespan - a reasonable assumption given the much
263 shorter generation time of the parasite. The full derivation of the fitness is given in the
264 Supplementary Information (Appendix S3). If the fitness is positive, then the mutant host type
265 will increase in number, generally replacing the existing resident host type, whereas if the
266 fitness is negative the mutant will die out. For simplicity, we assume no ‘intermediate strains’
267 due to cross-breeding. The fitness is used to calculate the location of the evolutionary singular
268 point and determine whether it is an evolutionary steady state, ESS, *i.e.* an evolutionary end
269 point.

270 To demonstrate the evolutionary behaviour more clearly, we numerically simulate
271 evolution using a similar mutation and replacement approach, using the full mutant-resident
272 dynamics – details of which are presented in the Supplementary Information (Appendix S3).
273 This has been shown to be a good approximation to the analytical approach using the fitness in
274 (3) and has the benefit of not making the assumption about the parasite burden being at
275 equilibrium. Starting from a highly susceptible salmon strain, we plot how m evolves through
276 time. Fig 3A plots the strains present following each mutation and shows how m evolves over
277 time with a (linear) trade-off. Here ‘time’ means the number of mutation events that occur – as
278 we do not currently know how often mutations occur, we leave time deliberately in terms of
279 these mutation events. In addition, the colouring represents the total host population present.

280 For the first 100 time steps, the system is parasite-free, hence minimal resistance and maximum
281 host population (Fig 3A). At time step 100, however, we introduce a small number of (free-
282 living) parasites. Immediately the population of host drops (Fig 3B). Resistance then begins to
283 be selected for, leading to an increase in m (Fig 3A). This in turn leads to an increase in host
284 population and a lower parasite burden (Fig 3B). The level of immune response eventually
285 settles at an intermediate level, *i.e.* an ESS, with the host population normally distributed about
286 this resistance level (Fig 3A- inset). This is at approx. $m = 0.0075$ here, slightly below the
287 optimal m (≈ 0.010) which maximises the host population.

288

289 **Fig 3:** In (A) we plot how m evolves over time, with a linear trade-off; the colour of the line
290 denotes the total host population at that time. The inset graphs give the distribution of
291 resistance levels in the host population at time=100, just prior to parasite invasion, and at
292 time=300, when the population reaches its ESS. In (B) we plot the host population and parasite
293 burden over time, corresponding to m evolving.

294

295 **Trade-off shape**

296 So far we have only considered a linear trade-off - whereby each benefit (*i.e.* unit
297 increase in immune response, m) always comes at the same cost (*i.e.* same decrease in birth
298 rate, a). We now vary the trade-off shape by means of a parameter θ (see Supplementary
299 Information, Appendix S3, for specific details). Specifically, a positive θ represents an
300 ‘acceleratingly costly trade-off’, whereby each benefit comes at an increasing (accelerating) cost
301 (*i.e.* larger decrease in birth rate, a); with larger θ giving a greater effect. Conversely, a negative
302 θ represents a ‘deceleratingly costly trade-off’, whereby each benefit comes at a decreasing
303 (decelerating) cost (*i.e.* a smaller decrease in birth rate, a). Finally $\theta = 0$ represents a linear
304 trade-off [54].

305 In Fig 4 we plot how the evolutionary singular point (ESS) m^* changes as we change the
306 shape of the trade-off (θ values); where the ESS is denoted by the thick black line. The host
307 evolves to increase their resistance level m if currently below the ESS, and evolve to decrease
308 resistance if above. In addition, the contour lines represent the equilibrium host density. We
309 immediately gain two main results from this. Firstly, that the evolutionary singular points are
310 always just below the maximum host density for each specific value of θ , meaning that the
311 optimal value of m which maximises the host population is not the same value of m that
312 maximises host fitness. Secondly, for strong deceleratingly costly trade-offs, as $\theta \rightarrow -1$, the host
313 evolves to maximise the immune response m , whereas for weakly deceleratingly costly or
314 acceleratingly costly trade-offs, the host evolves to an intermediate value of m . This suggests a
315 limited range of trade-offs that allow a highly susceptible salmon host strain to evolve into a
316 highly resistant host strain.

317

318 **Fig 4:** Plot of the evolutionary singular point (ESS - thick black line) for various shapes
319 of trade-off. Here $\theta < 0$ represents a deceleratingly costly trade-off; $\theta > 0$ represents an
320 acceleratingly costly trade-off; and $\theta = 0$ (dashed line) represents a linear trade-off – as taken in
321 Fig 3 simulation. The host evolves such that the immune response m either increases or
322 decreases (vertically on the plot) to the singular point – see Supplementary Information
323 (Appendix S4) for derivation of this line. The thin contour lines represent the total host
324 population size for corresponding values of m and θ . The parameters are as given in Table 1.

325

326 **Virulence**

327 In Fig 5A, we plot the evolutionary singular points (ESS) for varying levels of parasite
328 virulence, in terms of a higher or lower parasite-induced host death rate, α . Higher levels of
329 virulence, common in *G salaris* [15,23,35,36], encourages the evolution of a stronger immune
330 response.

331

332 **Cost of resistance**

333 In Fig 5B, we show the equivalent results for the lower and upper estimates for the cost
334 of resistance, as given by Cipriano *et al.* [53], 7% and 12% respectively (as opposed to the
335 'averaged' 10% initially taken). As would be expected, the location of the evolutionary singular
336 points (ESS) is lowered as the cost of resistance is increased, implying that the hosts evolve a
337 lower immune response, m , if more costly. This suggests that for the host to evolve into a highly
338 resistant strain, the cost of being highly resistant must not be too high.

339

340 **Fig 5:** Plot of the evolutionary singular point (ESS) for various shapes of trade-off: $\theta < 0$
341 deceleratingly costly, $\theta > 0$ acceleratingly costly and $\theta = 0$ (dashed line) linear. The colour of
342 each line is defined by the average host density along that line, as represented on the colour bar.
343 In (A) the virulence of the pathogen is varied, with $\alpha = 0.02$ being the baseline value. In (B) the
344 cost of resistance is varied, with 10% being the baseline.

345 **Discussion**

346 Wild Atlantic salmon populations the world over are currently threatened, with
347 numbers in some regions in decline [55]. The catastrophic impact that infections by *G. salaris*
348 can have on susceptible salmon populations, and the consequential financial implications, have
349 already been witnessed in Norway [12,17,56,57]. In the years post introduction to Norway, *G.*
350 *salaris* has since been reported from many other river systems throughout Europe [3–8,10]. The
351 aim of the present study was to explore the long-term interactions between populations of
352 Atlantic salmon and the monogenean parasite *G. salaris* in order to make predictions on the
353 natural recovery of salmon populations post introducing such an infection into an environment
354 containing susceptible salmon host populations such as the United Kingdom.

355 In the present study models were used to study the possible differences between strains
356 of Atlantic salmon to determine the mechanisms evolved by some Baltic strains in order to be
357 able to beat infection and in some cases coexist with low levels of *G. salaris* infection. Model
358 outcomes have highlighted that simple host-parasite models can show the varying levels of
359 resistance as seen in the Atlantic Lone and Baltic Neva salmon systems, with the addition of an
360 immune response. Models were used to investigate the possibility highly susceptible strains of
361 Atlantic salmon evolving traits and resulting trade-offs to become more like their resistant
362 counterparts.

363 Results from the present study highlight salmon will evolve to a more resistant state and
364 therefore be able to naturally recover from *G. salaris* infection if the salmon immune response is
365 allowed to evolve. This evolution would be subject to a trade-off such that host birth rate is
366 negatively correlated with resistance. Such recovery would result in host coexistence,
367 potentially at relatively high host densities, nearing 90% to that observed in the absence of
368 infection, with low parasite densities. The level of immune response however depends on
369 several factors: In order for a susceptible host to gain the level of resistance witnessed in some
370 Baltic salmon strains, it requires both a moderate-strong deceleratingly costly trade-off (*i.e.*, the
371 host pays a large cost in the creation of the immune response, for low m , and then the additional
372 costs for improving that immune response, increasing m , are less and reducing) and a lower
373 overall cost of the immune response. In addition, the virulence of parasite can play a significant
374 part, with higher virulence rates leading to lower host population sizes but higher resistance
375 levels; conversely, lower virulence rates leads to higher host populations with lower resistance
376 levels. For this reason, the water chemistry can play a crucial part in how salmon evolve as
377 identical strains of parasite can have different virulence rates solely due to environmental
378 factors.

379 In general, mathematical models represent a simplified version of a system, as such,
380 there are always going to be certain limitation. Future studies would do well to build on the
381 models herein and explicitly model the seasonal effects and implications of the salmon and

382 gyrodactylid life-cycles. Salmon spawning, for example, primarily takes place once a year
383 between mid-October and late February [58]. Similarly, salmon do not spend their entire life in
384 a river and in fact spend the majority of their adult life at sea, returning to their natal river to
385 spawn. Though it is possible for some salmon parr to mature sexually in a river without the
386 need to run to sea, and hence, stay to participate in spawning [59]. Such behaviours will have an
387 important impact on the length of time it would take for a population of salmon to recover from
388 *G. salaris* infections due to the time between salmon leaving and returning to infected rivers.

389 Salinity and water temperature are very important in determining *G. salaris* survival.
390 *Gyrodactylus salaris* is a freshwater parasite and survival is only possible in waters with a
391 salinity between 0 – 20ppt at temperatures of 3°C - 20°C [50,51]. The survival of *G. salaris* in low
392 salinity waters has been shown to be negatively correlated with water temperature and hence,
393 parasites can survive longer, both on and off a host, in such waters at lower temperatures [51].
394 Environment can also play an important role; in situations where water velocity is high,
395 detached parasites have the potential to drift further down a river and infect new populations of
396 hosts. Infection may also have an impact on the way in which salmon interact with each other,
397 for example, in populations of guppies, *P. reticulata*, (where individuals are infected with
398 *Gyrodactylus turnbulli*) females have been observed preferring, and selecting, males with low
399 parasite burdens [60]. Furthermore, changes in host feeding behaviour has also been witnessed
400 with feeding response and feeding activity significantly negatively correlated with parasite load
401 [61].

402 Whilst the varying degrees of pathogenicity of the different *G. salaris* strains was not
403 explicitly modelled in the present study, future studies would do well to include such
404 information into predictive models. Different strains of *G. salaris* have been shown to have
405 varying effects on salmon hosts [16]. The three currently known clades of *G. salaris* include *G.*
406 *salaris sensu stricto* - a highly pathogenic strain only found on Atlantic salmon (Clade 1); a strain
407 found on salmon from the river Göta älv in Sweden (Clade 2); and a strain that was found on
408 salmon from the rivers Lærdalselva, Drammenselva and Lierelva in Norway and on rainbow

409 trout from a fish farm in Lake Bullaren, Sweden [16]. A further strain of *G. salaris* has been found
410 on rainbow trout in Denmark [3,4]. This variant of the *G. salaris* parasite shows low virulence
411 towards Atlantic salmon and under experimental conditions, on isolated hosts, this strain
412 showed limited reproduction or no establishment at all [62]. Lindenstrom et al. [63], however,
413 observed high susceptibility to this strain in rainbow trout and noted that this strain of the
414 parasite greatly resembles *G. salaris sensu stricto*.

415 As highlighted earlier, fish-to-fish transmission was modelled through the distribution
416 of parasites across the fish population and not as an explicit feature in the model. The models
417 proposed consider the total densities of a *G. salaris* population within a salmon host population.
418 It would also be interesting to take an approach looking into the density of *G. salaris* populations
419 on individual hosts within a population with particular focus on the impact that fish-to-fish
420 transmission has on the dynamics of infection. It is known that juvenile Atlantic salmon are
421 highly territorial [59,63] and hence have a high chance of becoming infected due to fish-to-fish
422 contact when defending a territory against an infected individual. Moreover, fish-to-fish contact
423 between dead infected hosts and live uninfected hosts as well as live infected hosts and live
424 uninfected hosts also provide important routes for *G. salaris* spread [64,65].

425 Aggregation of parasites on hosts also has an important impact on the evolutionary and
426 population dynamics of both parasites and hosts [66,67]. Many studies have been carried out in
427 this area in order to develop our understanding of what causes heterogeneity in the distribution
428 of macroparasites within a host population [68]. Parasite aggregation in the wild is often
429 complex, in macro-parasitic infections the majority of hosts are observed harbouring a low
430 number of parasites with a minority of hosts harbouring a large number [69]. Such skewed
431 aggregations have been shown to follow a negative binomial distribution [66,67,69]. The
432 negative binomial distribution, (defined as $s^2 = m + m^2/k$, where s^2 and m are the variance and
433 mean respectively) quantifies the (inverse) degree of aggregation via the parameter k [70] such
434 that for small k parasite aggregation is increased, whereas for large k aggregation decreases.
435 The negative binomial distribution converges on the logarithmic series as $k \rightarrow 0$ and on the

436 Poisson for $k \gtrsim 20$ [68,71]. Due to the complicated life-cycle of *G. salaris* and its similarities with
437 micro- as well as macro-parasites we used a Poisson (defined as $s^2 = m$) to model parasite
438 aggregation in the present study, thus, allowing parasites to be randomly (and evenly)
439 distributed throughout the host population. This simplified model analyses considerably whilst
440 still allowing for important observations to be made on the dynamics of infection. Previous studies
441 have considered a Poisson distribution when modelling free-living *G. salaris* parasites [72].
442 Moreover, the effect on the distribution of parasites is negligible due to the large number of
443 parasites considered in the present models.

444 Even though the literature concerning *G. salaris* infections in salmon is vast, models
445 would greatly benefit from more accurate and up to date parameter estimates. Experimental
446 studies undertaken exclusively for this reason would be worthwhile in order to obtain estimates
447 for currently unknown parameters. Through our research we have determined that more data
448 are required in order to accurately parameterise the rate at which parasites leave, attach to and
449 kill hosts.

450

451 At present the United Kingdom and Ireland are the only known countries to officially
452 establish complete freedom from *G. salaris* infections [10,28–30,37]. As highlighted earlier,
453 Atlantic salmon populations in the UK are believed to be just as susceptible as those found in
454 Norway [15,35], hence, if *G. salaris* was introduced a similar environmental impact to that of
455 Norway can be expected. Extreme measures have been adopted in an attempt to control and
456 eradicate *G. salaris* infections. While eradication is preferred, this rarely happens and hence
457 “management and control” is what is actually being carried out and alternative methods of
458 treatment such as aluminium have been trialled [73]. It is understandable that survivors are
459 undesirable as we may see the development of resistance in the parasite population with
460 consequentially continued catastrophic effects on the host population, however, we also would
461 like to see the evolutionary process occur where there is adaptation or co-evolution to the
462 extent that parasite and host can co-exist without mortality and parasite numbers are maintained

463 at low levels or are removed by the host. Our results highlight that the current practice of
464 treating entire river catchments with rotenone before restocking with salmon from the original
465 genetic stock [12,14,25,26] may be severely damaging the potential for any evolutionary
466 process to occur.

467

468 Results from the present study have provided evidence that in the absence of
469 intervention salmon populations should naturally recover from *G. salaris* infection, however, the
470 timescale required for this to happen remains unknown. Furthermore, model output suggests
471 susceptible populations would evolve such that they reach a level of resistance required to
472 coexist with the parasite and recover to relatively high densities, nearing 90% of that observed
473 pre-infection. *Gyrodactylus salaris* and its impact on susceptible hosts must continue to be
474 studied in order to aid in contingency planning and defence against introduction and
475 emergence.

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- 674

675 **Supporting Information**

676

677 **S1 Fig. Schematic representation of salmon-*Gs* model**

678 **S2 Fig. The trade-off between host birth rate, α , and the rate hosts mount an immune**
679 **response to the parasite (resistance), m .**

680 **S1 File. Appendix S1 - Derivation of the model**

681 **S2 File. Appendix S2 - Equilibrium and stability analysis of model**

682 **S3 File. Appendix S3 - Derivation of the fitness of mutant type and trade-off**

683 **S4 File. Appendix S4 - Details of evolutionary simulations**

684 **S5 File. Appendix S5 - Supporting Information reference list.**

685