

# 1 **Rapid sex-specific evolution of age at maturity is** 2 **shaped by genetic architecture in Atlantic salmon**

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## 10 **Abstract**

11 Understanding the mechanisms by which populations adapt to their environments is a  
12 fundamental aim in biology. However, it remains challenging to identify the genetic basis of  
13 traits, provide evidence of genetic changes and quantify the phenotypic response. Age at  
14 maturity in Atlantic salmon represents an ideal trait to study contemporary adaptive evolution  
15 as it has been associated with a single locus in the *vgll3* region, and has also strongly changed  
16 in recent decades. Here, we provide an empirical example of contemporary adaptive  
17 evolution of a large effect locus driving contrasting sex-specific evolutionary responses at the  
18 phenotypic level. We identified an 18% decrease in the *vgll3* allele associated with late  
19 maturity (*L*) in a large and diverse salmon population over 36 years, induced by sex-specific  
20 selection during the sea migration. Those genetic changes resulted in a significant  
21 evolutionary response in males only, due to sex-specific dominance patterns and *vgll3* allelic  
22 effects. Our study highlights the importance of knowledge of genetic architecture to better  
23 understand fitness trait evolution and phenotypic diversity. It also emphasizes the potential  
24 role of adaptive evolution in the trend toward earlier maturation observed in numerous  
25 Atlantic salmon populations worldwide.  
26

## 27 **Introduction**

28 Understanding the mechanisms by which populations adapt to their environments is a  
29 fundamental aim in biology<sup>1,2</sup>. Such mechanisms may represent the only way for certain  
30 populations to persist in the face of strong human pressures and accelerated rates of climate  
31 change altering their environment. Temporal monitoring has documented recent and rapid  
32 phenotypic changes in wild populations in many species<sup>e.g. 3,4</sup>. However, whether or not such  
33 phenotypic changes are adaptive often remains unclear<sup>5,6</sup>. Obtaining evidence of adaptive  
34 evolution requires knowledge of the genetic basis of traits and subsequent demonstration that  
35 natural selection induces changes in this genetic component<sup>6</sup>. Although the ideal strategy for  
36 demonstrating adaptive evolution is to study the genes directly controlling the traits under

37 selection, such examples are extremely scarce <sup>6,7</sup>. Despite the increased availability of  
38 genomic data, the identification of large-effect loci controlling phenotypes of ecological  
39 significance and understanding how contemporary selection affects the allele frequency of  
40 such genes remains indeed challenging <sup>8</sup>. In cases where the genetic architecture of a trait is  
41 well characterized e.g. when a large-effect locus has been identified, retrospective genetic  
42 analyses of archived material for the gene(s) controlling the trait in question can be  
43 performed and provide detailed information about its evolutionary dynamics <sup>e.g. 9</sup>.

44 Age at maturity in Atlantic salmon, defined here as the number of years spent at sea prior to  
45 maturation, has recently been shown to be controlled by a single large-effect locus with sex-  
46 specific effects, located within a narrow (<100kb) region around the *vgll3* gene <sup>10</sup>. The same  
47 locus has also recently been linked with gender-biased auto-immune diseases in humans <sup>11</sup>. In  
48 Atlantic salmon, age at maturity reflects a classic evolutionary trade-off, as larger, later-  
49 maturing individuals typically have higher reproductive success, but run a greater risk of  
50 mortality before first reproduction. Sex-specific selection optima may exist for this trait <sup>10</sup>.  
51 Males generally mature earlier and at smaller size, whereas females mature later and have a  
52 stronger correlation between body size and reproductive success compared with males <sup>12</sup>. It  
53 was suggested that the sex-dependent dominance observed at *vgll3* potentially resolves this  
54 sexual conflict <sup>10,13</sup>. Furthermore, the age structure of many populations has changed  
55 worldwide in recent decades, generally towards an increasing proportion of smaller, earlier  
56 maturing individuals <sup>e.g. 14,15</sup> but see <sup>16</sup>. However, the reasons for this, and whether it is an  
57 adaptive change, remain unknown <sup>17</sup>. Therefore, age at maturity in Atlantic salmon provides a  
58 rare opportunity to investigate the contemporary change of a phenotypic trait directly at the  
59 genetic level.

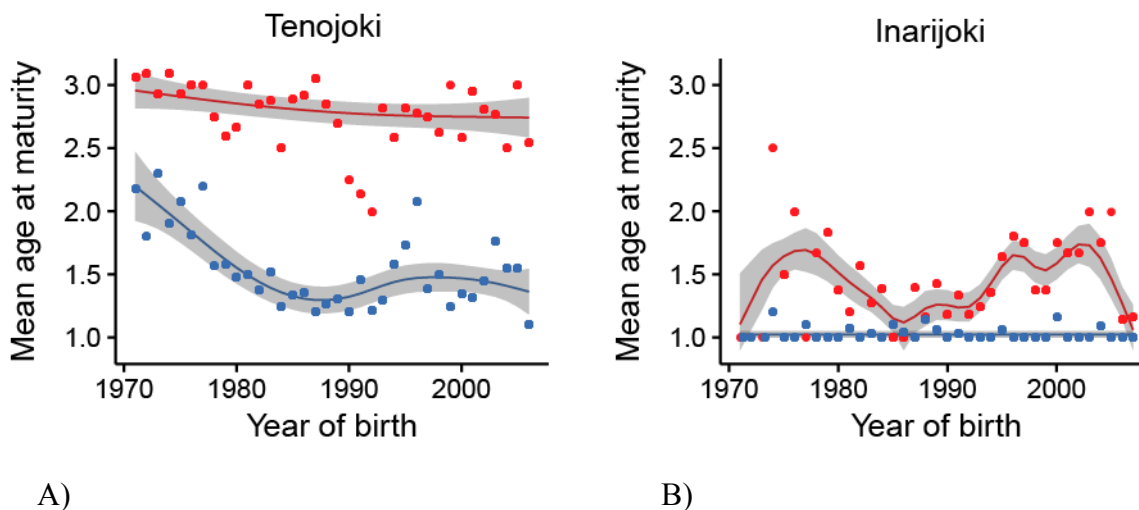
60 We studied two closely related Atlantic salmon populations from northern Europe with  
61 contrasting maturation age structure. Despite a low level of genetic divergence between them  
62 ( $F_{ST} = 0.012$ )<sup>18</sup>, the Tenojoki population displays a high level of life-history diversity  
63 including a high proportion of large, later maturing individuals in both sexes, whereas the  
64 Inarijoki population consists primarily of individuals of younger maturation ages, and with  
65 less life-history variation, particularly in males <sup>15</sup>. Here, we utilized a 40 year time series to  
66 detect potential signs of adaptive evolution in age at maturity by contrasting allele frequency  
67 changes at the maturation gene *vgll3* with life-history phenotypes in 2500 samples from the  
68 two populations. We also used the genetic data to investigate the occurrence of sex- and  
69 population-specific genetic architecture and selection, potentially explaining the observed  
70 diversity variation in age at maturity.

## 71 **Results:**

### 72 **Temporal changes in age at maturity**

73 We first quantified temporal phenotypic changes in both populations. There was a non-linear  
74 decrease in the age at maturity of Tenojoki individuals, with the mean maturation age of  
75 males and females declining by >40% (from 2.2 to 1.3 years;  $edf = 3.87$ ,  $F = 5.11$ ,  $P < 0.001$ )  
76 and 8.1% (from 3.0 to 2.7 years;  $edf = 1.27$ ,  $F = 0.57$ ,  $P = 0.02$ ), respectively, during the 36

77 year time period (Figure 1A). In Tenojoki males, the decrease occurred primarily between  
78 1971 and 1987 before stabilization, while in females, age at maturity gradually decreased  
79 over the 36 year study period, explained best by a slightly nonlinear slope (Figure 1A). In  
80 comparison, Inarijoki males were virtually devoid of variation in age at maturity, with almost  
81 all males having spent one year at sea before maturing ( $edf = 0.00$ ,  $F=0.00$ ,  $P = 0.731$ ),  
82 whereas mean age at maturity in females fluctuated cyclically over the 37 years ( $edf = 10.14$ ,  
83  $F = 6.035$ ,  $P < 0.001$ , Figure 1B), but with no indication of a decrease in average maturation  
84 age (Figure 1B).



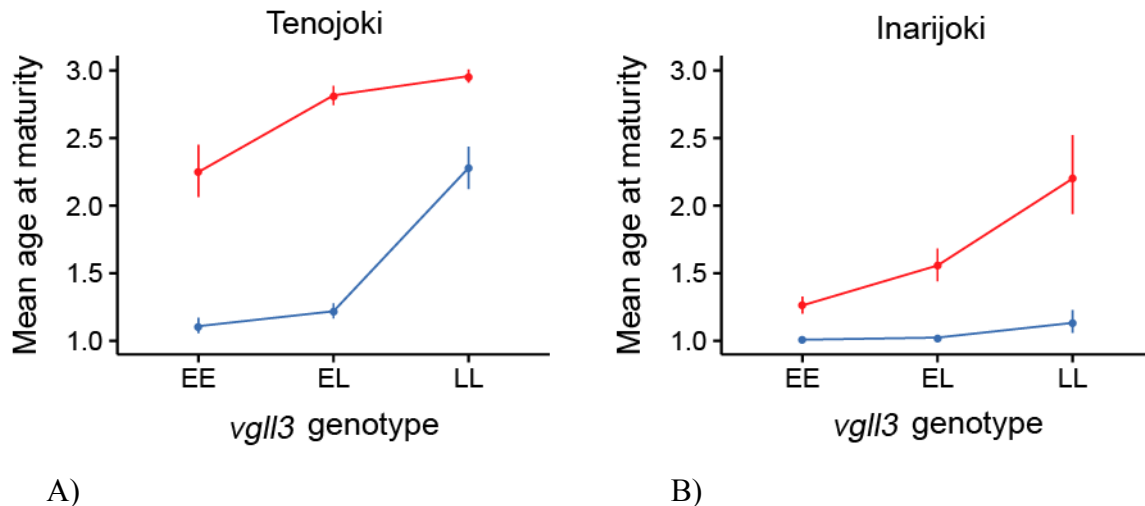
**Figure 1: Change in mean age at maturity in the A) Tenojoki and B) Inarijoki populations.**

Females are in red ( $N$  Tenojoki = 467,  $N$  Inarijoki = 261) and males in blue ( $N$  Tenojoki = 699,  $N$  Inarijoki = 570). Lines represent fitted values from the GAM  $\pm 1.96$  SE, points are observed annual means.

## 85 Genetic architecture of age at maturity

86 We hereafter use genetic architecture to refer to the additive and dominance effects of *vgll3*  
87 on the age at maturity. The *vgll3* genotypes had a sex-specific effect on the probability to  
88 observe the different ages at maturity in the Tenojoki population ( $\chi^2_{(6)} = 27.58$ ,  $P < 0.001$ ). A  
89 sex-specific dominance pattern was observed in this population; heterozygote males had a  
90 mean age at maturity closer to homozygote *EE* (estimated dominance  $\delta_M = 0.09$ , see Method)  
91 whereas heterozygote females had a phenotype closer to homozygotes *LL* (estimated  
92 dominance  $\delta_F = 0.80$ ; Figure 2). In the Inarijoki population, the *vgll3* genotypes were  
93 significantly associated with the probability to observe the different age at maturity groups  
94 ( $\chi^2_{(4)} = 56.41$ ,  $P < 0.001$ ) but not in a sex-specific manner ( $\chi^2_{(4)} = 8.27$ ,  $P = 0.08$ ). Differences  
95 in mean age at maturity between homozygotes varied depending on the sexes and populations  
96 (i.e. additive or allelic effect: effect of the substitution of one allele for the other). In the  
97 Tenojoki population, the relative difference in mean age at maturity between alternative *vgll3*  
98 homozygotes was three times higher in males (+100% for *LL*) than in females (+32% for *LL*).  
99 This pattern was inverted in Inarijoki, with the difference in mean age at maturity between  
100 female homozygotes being about six times larger (+74% for *LL*) than in males (+12% for *LL*,

101 Figure 2). These results imply that selection during the sea migration, defined as the relative  
102 difference in survival between genotypes, is likely to vary between sexes and populations.  
103 There was no statistically significant change in the effect size of *vgll3* on maturation age over  
104 time in either population (Tenojoki:  $\chi^2_{(6)} = 6.07$ ,  $P = 0.42$ ; Inarijoki:  $\chi^2_{(4)} = 4.41$ ,  $P = 0.35$ ).



A) B)  
**Figure 2: Mean age at maturity as a function of *vgll3* genotype in the A) Tenojoki and B) Inarijoki populations.**

Females are in red ( $N$  Tenojoki = 522,  $N$  Inarijoki = 286) and males in blue ( $N$  Tenojoki = 804,  $N$  Inarijoki = 612). Means are calculated from multinomial models fitted values, averaged over years. Error bars represents 95% bootstrap confidence intervals based on 1000 replicates. Estimated dominances from mean ages are 0.13 and 0.32 in Inarijoki males and females, respectively.

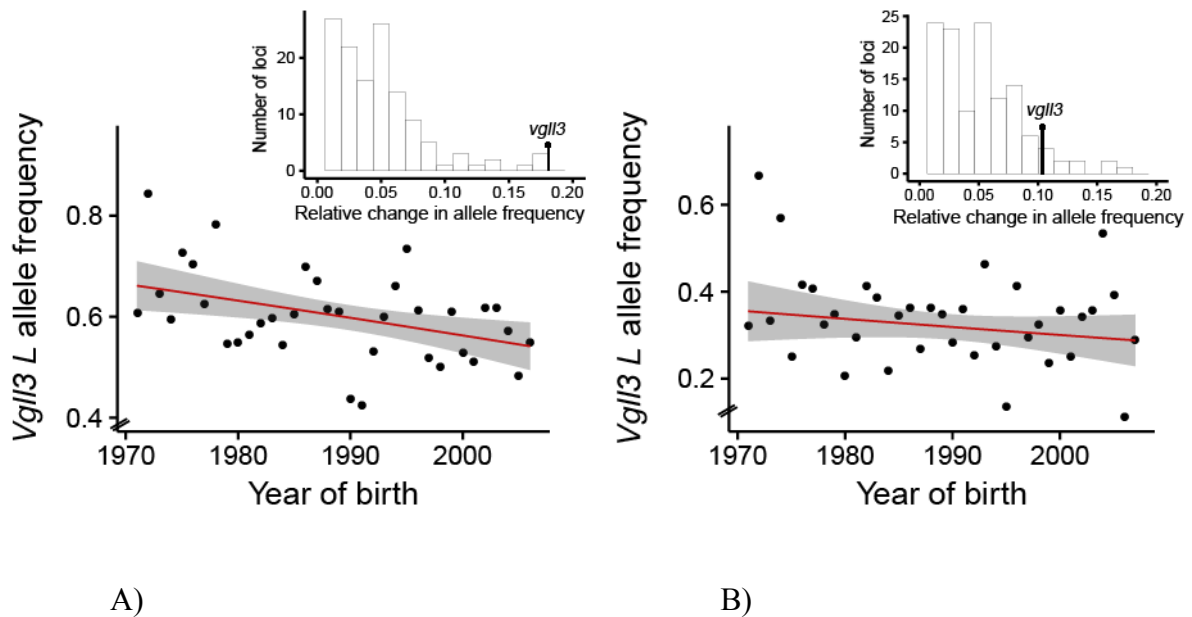
## 105 Evolution of *vgll3* and signals of selection

106 The *vgll3* late maturing (*L*) allele frequency decreased significantly, from 0.66 to 0.54 (18%)  
107 in 36 years, in the Tenojoki population ( $F_{(1)} = 7.80$ ,  $P = 0.009$ ; log-odd slope = -0.014 (95%  
108  $CI_{95} = [-0.004, -0.024]$ ; Figure 3A). This allele frequency change was the highest of all the  
109 144 genome-wide SNPs assessed (Figure 3A). This observation provides strong support for  
110 natural selection acting against the *vgll3* *L* allele in the Tenojoki population as allele  
111 frequency changes of similar or greater size would be expected in other putatively neutral loci  
112 if such changes were due to genetic drift. In the Inarijoki population, the trend in the *vgll3* *L*  
113 allele frequency was also negative (slope = -0.0086,  $CI_{95} = [-0.0232, 0.0060]$  but not  
114 significant ( $F_{(1)} = 1.29$ ,  $P = 0.26$ , Figure 3B). About 10% of the 134 genome-wide SNPs  
115 assessed had a steeper linear trend in allele frequency than *vgll3* in this sub-population  
116 (Figure 3B).

117 To further quantify the strength of selection driving changes in *vgll3* allele frequency, a  
118 Bayesian model was developed to estimate selection coefficients whilst accounting for  
119 genetic drift, similar to a Wright-Fisher model (see Methods). The selection coefficient in the  
120 Tenojoki population was large and significantly higher than zero, albeit with large credibility  
121 intervals ( $-s = 0.33$  (95% credibility interval = [0.01, 0.77]). In Inarijoki, there was no  
122 evidence for significant selection ( $s = 0.25$ ,  $CI_{95} = [-0.07, 0.49]$ ).

123

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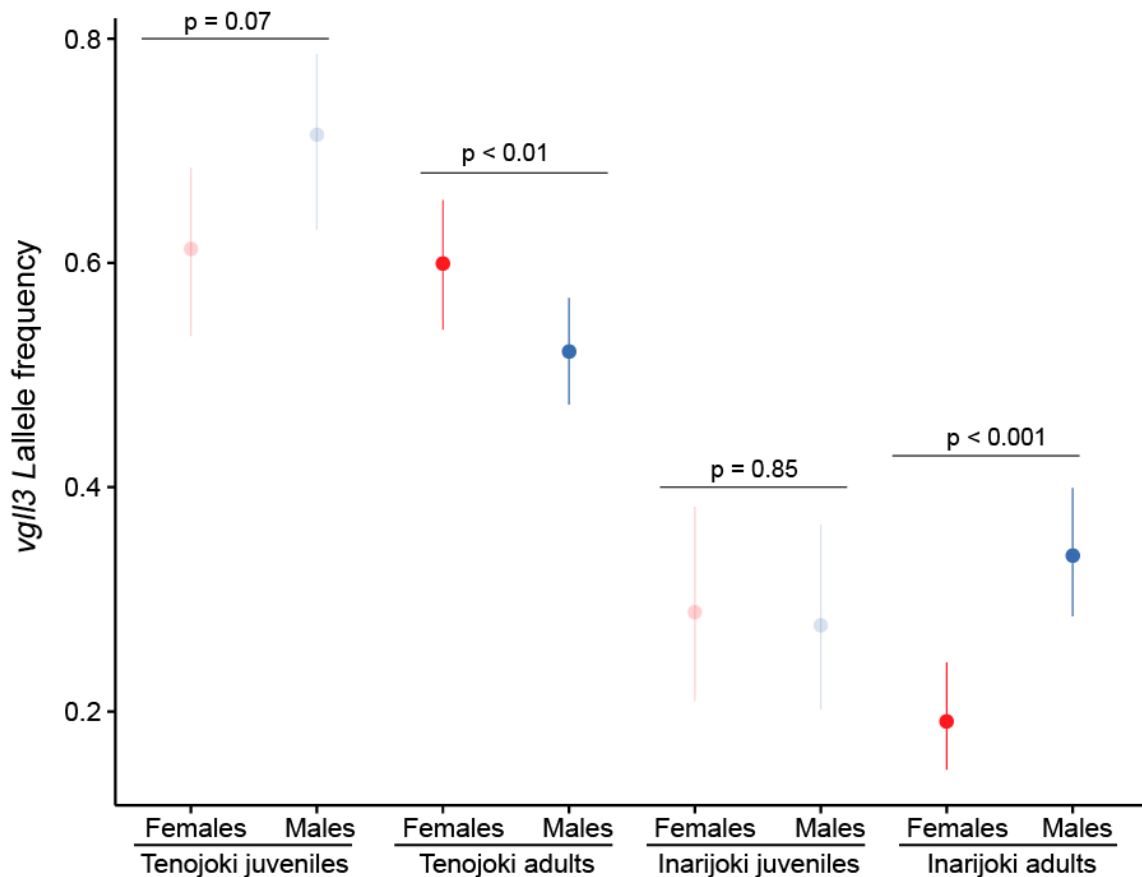


**Figure 3: Temporal changes in *vgl3* L allele frequency associated with late maturation in the A) Tenojoki and B) Inarijoki populations.**

The lines represent fitted values from the quasibinomial model with  $\pm 1.96$  SE ( $N$  Tenojoki = 1166,  $N$  Inarijoki = 765). Panels compare the percentages of change in allele frequencies among loci over the studied period.

125 The *vgl3* L allele frequency differed between sexes in a contrasting manner in the two  
126 populations. The odds of possessing an L allele was 37% higher in females than in males in  
127 the Tenojoki population ( $CI_{95} = [0.12, 0.69]$ ,  $F_{(1)} = 8.72$ ,  $P < 0.01$ , Figure 4) but 53% lower in  
128 Inarijoki ( $CI_{95} = [0.40, 0.65]$ ,  $F_{(1)} = 36.51$ ,  $P < 0.001$ , Figure 4). This could be the result of  
129 either sex- and genotype-specific sex ratios at fertilization or juvenile mortality in freshwater,  
130 or alternatively, *vgl3* genotype-specific selection differing between the sexes during sea  
131 migration, prior to returning to reproduce. In order to distinguish between these alternatives,  
132 we genotyped 143 and 108 juveniles of various ages collected from the same freshwater  
133 locations in Tenojoki and Inarijoki (1-3 years old, see Methods), respectively. Juvenile sex  
134 ratios were close to parity and the *vgl3* L allele frequency was similar in both sexes in both  
135 populations ( $\chi^2_{(1)} = 3.27$ ,  $P = 0.07$  in Tenojoki,  $\chi^2_{(1)} = 0.04$ ,  $P = 0.85$  in Inarijoki, Figure 4).  
136 This provides support for the notion that selection is acting on the L allele in a sex-specific  
137 manner during the marine life-history phase, as opposed to during the freshwater juvenile  
138 phase. Such sex-specific allele frequency patterns may be reinforced by sex specific  
139 dominance (Supplementary Figure 7).

140



**Figure 4: Model predicted mean *vgll3* L allele frequency as a function of the sex and reproductive status in Tenojoki and Inarijoki.**

Error bars indicates 95% confidence intervals. The years 2006 and 2007 were used as reference years for adults allele frequency in dark red (females) and dark blue (males) in respectively the Tenojoki and inarijoki populations.

#### 141 Sex-specific evolutionary response

142 In Tenojoki, the sex-specific genetic architecture drove contrasting evolutionary responses in  
143 the two sexes. Temporal changes in genotypes explained about 50% of the non-linear  
144 decrease in male age at maturity (0.46 years,  $edf = 3.95$ ,  $F = 3.51$ ,  $P < 0.001$ ; Supplementary  
145 Figure 8) but didn't explain the temporal changes in female age at maturity ( $edf = 0$ ,  $F = 0.00$ ,  
146  $P = 0.54$ ). Both the sex-specific dominance patterns and *vgll3* effect sizes potentially  
147 contribute to the contrasting genotype-phenotype link between the sexes, despite similar  
148 linear trends in genotype frequencies ( $\chi^2_{(2)} = 2.78$ ,  $P = 0.25$ ). For example in females, the  
149 dominance of the late maturation L allele means that a decrease in the frequency of this allele  
150 does not necessarily result in a decrease in age at maturity, as a decrease in LL genotype  
151 frequency would be partially compensated by the increase in the EL genotype frequency,  
152 which results in a similar phenotype distribution due to dominance effect (Figure 2,  
153 Supplementary Figure 9). Hence, temporal life-history change would mostly stem from the  
154 increase in the proportion of individuals with the EE genotypes, maturing about 0.71 years  
155 earlier than LL individuals. In contrast, in males, the recessivity of the *vgll3* L allele ( $\delta_M =$

156 0.09) would favor a larger decrease in age at maturity when proportion of heterozygotes *EL* is  
157 increasing as the age at maturity decline is a result of the combined increase in *EE* and *EL*  
158 genotype frequencies, which mature on average 1.12 year earlier than *LL* individuals. Most of  
159 the female age at maturity decline could be explained by the spawning year (0.20 year,  $edf =$   
160 2.16,  $F=21.61$ ,  $P < 0.001$ ). The spawning year would induce in parallel up to 0.15 year  
161 decrease in male maturation age ( $edf = 2.126$ ,  $F = 12.41$ ,  $P < 0.001$ ).

## 162 Discussion

163 We provide convincing evidence of rapid adaptive evolution of age at maturity toward small,  
164 early-maturing individuals in a large Atlantic salmon population. This indicates that despite  
165 having a reproductive advantage due to their large size<sup>12,19</sup>, the late maturation life-history  
166 strategy has become increasingly costly and modified the reproductive success vs survival  
167 trade-off such that earlier maturation is increasingly advantageous. Adaptive evolution may  
168 represent a realistic mechanism behind changes in age at maturity observed worldwide in the  
169 last decades in Atlantic salmon<sup>e.g. 14–16,20</sup> and other salmonid fish species<sup>e.g. 21</sup>. What could be  
170 the causes of such rapid evolution of a life-history trait? One explanation is that it could be  
171 linked to recent rapid changes in the marine environment of the Teno salmon populations.  
172 For example, climate change may affect Atlantic salmon growth and/or survival directly<sup>e.g. 22</sup>  
173 or indirectly through changes in Arctic food-webs and ecosystem functioning resulting from  
174 e.g. species range expansions<sup>20,23,24</sup>. Atlantic salmon occupying the northernmost parts of the  
175 globe will be unable to move to a colder climate in response to ocean warming, which would  
176 reinforce the importance of adaptation for population persistence. Another possibility is  
177 human-induced evolution of age at maturity through fishing targeting Atlantic salmon  
178 differentially according to their size<sup>e.g. 25, but see 26</sup> or reducing prey availability<sup>e.g. 27</sup>. It is  
179 important to note however, that natural selection didn't entirely explain the observed  
180 temporal changes in age at maturity in the Tenojoki population. Irrespective of the *vgll3*  
181 genotypes, the probability to mature at younger ages, after one or two years at sea, increased  
182 over time (Supplementary Information). This could be due to adaptive phenotypic plasticity  
183<sup>28</sup>, through changes in maturation probability towards the same direction as selection, or to  
184 changes in allele frequencies of potential minor effect loci. Further investigation is required  
185 to test these hypotheses. Regardless, such changes in population age structure can negatively  
186 affect the population growth rate and/or temporal stability induced with the portfolio effect<sup>e.g.</sup>  
187<sup>29</sup> and also have negative consequences on genetic diversity levels<sup>e.g. 30</sup> and thus are a  
188 concern for future population persistence.

189 Despite common temporal changes in *vgll3* allele frequency between the sexes,  
190 differing genetic architectures, in terms of additive and dominance patterns, resulted in sex-  
191 specific selection strengths and evolutionary responses to selection. We observed sex-specific  
192 differences in *vgll3* allele frequencies in adult salmon that were not present in pre-marine-  
193 migration juveniles from the same populations (Figure 4). Interestingly, the direction of the  
194 sex-specific differences was opposite in the two populations studied. The combined effects of  
195 sex-dependent dominance and sex-specific selection patterns can explain these contrasting  
196 patterns. The relative strength of allelic effects differed dramatically between sexes and these

197 effects were in opposite directions in the two populations: in Inarijoki, the difference in mean  
198 age at maturity between homozygotes is about six times larger in females compared to males  
199 whereas in Tenojoki, the relative difference was three times higher in males (Figure 2).  
200 Therefore, selection against *LL* genotype individuals acts primarily on females in Inarijoki,  
201 but on males in Tenojoki (Figure 4). However, sex-specific dominance also plays a role by  
202 introducing differences in allele frequencies between sexes that are dependent on population  
203 allele frequency (Supplementary Figure 7). Furthermore, sex-specific genetic architectures  
204 induce sex-specific evolutionary responses in Tenojoki, by accelerating the decrease in age at  
205 maturity in males and reducing the temporal variation in females. Sex-specific dominance is  
206 likely to have evolved to reduce intra-locus sexual conflict<sup>10</sup>. However, whether this genetic  
207 architecture is nowadays at its optimum is questionable in light of the quick decrease in *vgll3*  
208 *L* allele frequency and age at maturity. Further studies are necessary to determine whether  
209 sexually antagonist selection in Tenojoki is persisting in ever changing environments.

210           Age at maturity evolved rapidly under sex-specific selection in just 40 years,  
211 equivalent to 7-8 generations in Atlantic salmon. Despite being genetically similar, the two  
212 studied populations had distinctive genetic architectures, sex-specific selection and  
213 consequently *vgll3* allele frequencies variation. This study shows that variability in genetic  
214 architectures can create complex selection and evolution patterns, with responses to  
215 environmental changes or anthropogenic pressures differing according to sex and population.  
216 This highlights the importance of determining the genetic basis of fitness traits in order to  
217 understand their evolution and to explain the phenotypic diversity observed between  
218 populations and species.

## 219 **Material and methods**

### 220 **Study site and sampling**

221 The subarctic Teno River forms the border between Finland and Norway and drains north into  
222 the Barents sea (68 - 70°N, 25-27°E). Genetically distinct salmon populations<sup>31</sup> are  
223 distributed throughout the 16 386 km<sup>2</sup> catchment area. Annual river catches range from about  
224 20,000 to 60,000 individuals, representing up to 20% of the entire riverine Atlantic salmon  
225 harvest in Europe<sup>32</sup>. Atlantic salmon populations from Teno have been monitored since early  
226 1970s with collections of scales and phenotypic information by trained fishers. Scales were  
227 stored in envelopes at room temperature and used to determine individual life-history  
228 characteristics including the number of years spent in the freshwater environment prior to  
229 smoltification (river age), number of years spent in the marine environment prior to  
230 maturation (sea age) and possible previous spawning events, following international  
231 guidelines (ICES 2011). The Teno river Atlantic salmon have diverse life history strategies  
232<sup>15,34</sup>. They can spend from two to eight years in freshwater before smoltifying, from one to  
233 five years at sea before maturing and have up to five breeding attempts. Overall, a total of  
234 120 combinations of river age, sea age at maturity and repeat spawning strategies have been  
235 described<sup>15</sup>. Age at maturity has been declining in Teno salmon over the last 40 years, with



236 proportionally fewer late maturing salmon returning over years. Age at maturity also differs  
237 largely among populations displaying genomic signatures of local adaptation<sup>15,35</sup>.

238 We randomly selected scales from individuals caught by rod between 1972 and 2014 during  
239 the later part of the fishing season, from July 20 to August 31. Most of the Teno salmon are  
240 expected to have reached their home river by late July<sup>36</sup>. Samples came from two different  
241 locations, the middle reaches of the Tenojoki mainstem (hereafter Tenojoki) and a headwater  
242 region Inarijoki (Supplementary Figure 1). These populations host weakly differentiated  
243 salmon populations with contrasting ages at maturity<sup>31,35</sup>. Individuals from the Tenojoki  
244 spend, on average, more time at sea before maturing than individuals from the Inarijoki  
245 population<sup>15,30</sup>. Seventy additional females were selected in Inarijoki over the study period,  
246 by following the same sampling scheme, to increase the sampling size in analyses with sex-  
247 specific estimates. Scale or fin samples were also collected from juvenile salmon from the  
248 Tenojoki (N=143, 2-3 years old) and Inarijoki (N=108, 1-3 years old) populations caught by  
249 electrofishing in the 2012 and 2016, respectively. They were used as the baseline for  
250 population assignment of adults and to determine potential sex-specific *vgll3* allele frequency  
251 differences at the juvenile stage.

## 252 Genotyping

253 DNA extraction from scales, sex determination and genotyping were performed following  
254 Aykanat et al. (2016). In total, 2482 individuals were genotyped at 191 SNPs, including the  
255 SNP the most highly associated with the age at maturity, *vgll3TOP* (vestigial-like family  
256 member 3 gene also called *vgll3*,<sup>10</sup>) and outlier and baseline SNP modules<sup>37</sup>. The outlier  
257 module consisted of 53 SNPs highly differentiated between the Inarijoki and Tenojoki  
258 populations, thus allowing a more powerful assignment of population of origin, between  
259 these two closely related populations<sup>i.e. see 30,37</sup>. The baseline module included 136 putatively  
260 neutral markers in low linkage disequilibrium, distributed over the whole genome  
261 proportionally to chromosome length, previously filtered to have minor allele frequency  
262 >0.05 and heterozygosity >0.2<sup>37</sup>. There were used to estimate the level of differentiation  
263 among populations of the Teno River (Weir and Cockerham's  $F_{ST}$ ) and genetic drift. Mean  
264 genotyping success was on average 0.80 per locus and individual.

## 265 Population assignment

266 The 53 outlier loci were used to determine the optimum number of genetic clusters and assign  
267 the population of origin of adults using the software STRUCTURE. First, an admixture  
268 model with correlated allele frequencies<sup>38</sup> was run on adult and juvenile data for 80,000  
269 MCMC iterations, including a burn-in length of 50,000. The model was replicated six times  
270 for each cluster value K, varying from one to four. The optimal number of clusters was  
271 thereafter estimated using the  $\Delta K$  method described in Evanno, Regnaut, and Goudet (2005)  
272 using STRUCTURE HARVESTER<sup>40</sup>. This allowed us to determine whether juveniles were  
273 correctly assigned to their sampling locations and could thus be used as a baseline for adult  
274 assignment. Then another admixture model with correlated allele frequencies was replicated  
275 six times on adult data using juvenile data as a baseline, with prior migration set to 0. The

276 *fullsearch* algorithm from the CLUMP software (Jakobsson & Rosenberg 2007) was used to  
277 account for across replicate variability in membership coefficients. Finally, the cluster of each  
278 adult was assigned by using the optimum K and membership probability superior or equal to  
279 0.8. The differentiation between populations was tested by calculating the likelihood ratio G-  
280 statistic<sup>41</sup> and comparing it with the G-statistic distribution obtained by permuting 1,000  
281 times individuals between populations.

282 The most likely number of clusters determined with the  $\Delta K$  method was two when juveniles  
283 and adults data were combined (Supplementary Figure 2). Juveniles were assigned  
284 accordingly to their sampling location in more than 96% of cases. Using juvenile data as a  
285 baseline, 90% of adults were classified to one of the two clusters with probabilities equal or  
286 higher than 0.8. Individuals sampled in Tenojoki were assigned to the Inarijoki population in  
287 25% of the cases whereas only 2% of the individuals caught in Inarijoki were assigned to the  
288 Tenojoki population. In total, 1330 and 911 individuals clustered in the Tenojoki and  
289 Inarijoki populations, respectively (Supplementary Figure 4). The two populations were  
290 significantly genetically differentiated ( $F_{ST} = 0.013$ ,  $G = 201.55$ ,  $P < 0.01$ ) and had contrasted  
291 age structures (Supplementary Figure 5).

## 292 **Statistical analyses**

### 293 **Temporal variation in age at maturity and proportion of females**

294 Non-linear temporal variation in age at maturity was estimated separately for each population  
295 using generalized additive models, with the Gaussian family as the residual distribution. Year  
296 of birth was included as an independent variable inside a cubic regression spline for each sex.  
297 The birth year periods were selected according to the generation length spent from 1971 to  
298 2006 in Tenojoki and from 1971 to 2007 in Inarijoki. Sex was also included as an  
299 explanatory variable.

300 The amount of smoothing was determined in each case using the maximum likelihood  
301 method. Automatic smoothness selection was performed by adding a shrinkage term. The  
302 significance of independent variables was assessed using F-tests and an alpha risk of 0.05. All  
303 statistical tests included in this manuscript were two-tailed. The additive models were run with  
304 the R package *mgcv*<sup>42,43</sup>.

### 305 **Effect size of *vgll3* on age at maturity**

306 To estimate the genetic effect of *vgll3*, age at maturity was also regressed using a multinomial  
307 model separately for each population. In Tenojoki, two individuals having matured after five  
308 years at sea were considered having matured after four years to avoid the estimate of  
309 additional model parameters without data support. The sex, year of capture and *vgll3*  
310 genotype can all influence age at maturity and were included in models as a three-way  
311 interaction. Multinomial models in this study were performed using the R package *nnet*<sup>44</sup>.  
312 Model selection was performed using backward selection with F-tests and by calculating the  
313 AICc of all possible models. The effect of year on the probability to mature was calculated  
314 with the Effect package<sup>45</sup> which averages the effect size across sexes and genotypes. The  
315 mean age at maturity per sex and genotype was calculated from model predicted values. First,

316 predicted age was obtained for each year, sex and age at maturity combination by multiplying  
317 the probabilities of having matured after one, two, three or four years at sea by the  
318 corresponding sea age and taking the sum. Second, the age at maturity was calculated for  
319 each sex and genotype by averaging over years. This process was replicated 1000 times by  
320 randomly sampling with replacement and fitting a new model. A 95% bootstrap confidence  
321 interval was then determined by taking values of the 2.5 and 97.5 percentiles. The *vgll3*  
322 alleles were called *L* and *E* to indicate their association with late and early maturation,  
323 respectively<sup>10</sup>. Dominance for each sex and population was estimated from the mean age at  
324 maturity ( $\mu$ ) following  $\delta = \frac{\mu_{EL} - \mu_{EE}}{\mu_{LL} - \mu_{EE}}$ . The *L* allele is recessive if  $\delta = 0$ , additive if  $\delta = 0.5$  and  
325 dominant if  $\delta = 1$ .

326 To determine how much of the observed changes in age at maturity over time could be  
327 attributed to changes in genotypes and year of capture, a new dataset with the spawning year  
328 held constant at 1975 was created for Tenojoki. The previous multinomial model was used to  
329 predict new maturation probabilities from which model predicted age at maturity were  
330 calculated for each individual, as above. Temporal changes in age at maturity attributed to  
331 genotypes were determined by fitting a generalized additive model using the Gaussian family  
332 and including the individual birth year in a cubic regression spline and the sex as independent  
333 variable. Changes in age at maturity attributed to the year of capture corresponded to the  
334 difference between individual predicted age at maturity calculated from the original dataset  
335 and the one with the year fixed. Another Gaussian generalized additive model was also  
336 performed on those differences, by including the birth year in a cubic regression spline.  
337 Automatic smoothness selection was performed by adding a shrinkage term.

### 338 **Change in allele and genotype frequencies**

339 Temporal variation in allele frequencies was determined for each population and locus using  
340 generalized linear models (*glm*), with the quasibinomial family to account for overdispersion.  
341 Sex-dependent *vgll3* genetic effect on the age at maturity<sup>10</sup> may create sex-specific selection  
342 at sea, leading to differences in *vgll3* allele frequency between male and female spawners  
343 from the same generation. The sex variable can capture this potential intra-generation  
344 variation in allele frequency. Hence, sex and year of birth were included as independent  
345 variables in the *glm*. To keep the potential effect of sex-specific selection on the *vgll3* allele  
346 frequency temporal change, the model was also run without including sex as a covariate. In  
347 order to determine whether sea age related SNPs varied across time more than under the  
348 neutral expectation, model predicted temporal changes in allele frequencies were compared  
349 among loci with individual genotyping success higher than 0.7 (145 and 135 loci for the  
350 Tenojoki and Inarijoki populations, respectively). This threshold was chosen given a trade-off  
351 between increasing the quality and minimal amount of data per locus (average genotyping  
352 success superior to 0.90 in those subsets) and keeping a large number of loci for the  
353 comparison (~25-30% of loci were excluded). The significance of variables was assessed  
354 with F-tests.

355 To determine whether potential differences in *vgll3* allele frequencies between adult males  
356 and females are likely to arise during the sea migration, juvenile allele frequencies were

357 analyzed using a separate *glm* with the binomial family for each population. Sex was  
358 introduced as an independent variable. A backward model selection was performed using  
359 Likelihood-Ratio Tests (LRT) and an alpha risk of 0.05. Confidence intervals were calculated  
360 with the *lsmean* package <sup>46</sup> by taking the years 2006 and 2007 as reference for the Tenojoki  
361 and Inarijoki adults, respectively.

362 To further describe temporal changes in *vgll3* genotypes, a multinomial model was used for  
363 each population. The year of birth and the sex were introduced as independent variables in a  
364 two way interaction. A backward model selection was performed using LRT and an alpha  
365 risk of 0.05.

366 Allele frequencies may differ between sexes in the presence of selection and sex-specific  
367 dominance patterns. The expected sign and magnitude of those differences was determined  
368 for different selection strengths by using the dominance patterns calculated previously for the  
369 Inarijoki and Tenojoki populations. Considering a gene with 2 alleles *A* and *B* with respective  
370 frequencies *p* and *q*, the allele frequency after a selection event corresponds to:

$$p_s = \frac{p^2 W_{AA} + pq W_{AB}}{p^2 W_{AA} + 2pq W_{AB} + q^2 W_{BB}}$$

371 with  $W_{AA}$ ,  $W_{AB}$  and  $W_{BB}$  the relative fitness of each respective genotype:

372 
$$W_{AA} = 1; W_{AB} = 1 - DS \text{ and } W_{BB} = 1 - S.$$

373 where *S* is the selection coefficient common to each sex, varying from 0 to 0.90 by 0.15  
374 intervals. *D* is the dominance coefficient.  $P_s$  was calculated for each sex and population using  
375 the corresponding dominance coefficients previously calculated from phenotypes ( $\delta$ ) and an  
376 initial *p* varying from 0 to 1. The expected difference in allele frequency in Supplementary  
377 Figure 7 corresponds to  $p_s(\text{female}) - p_s(\text{male})$ , calculated for each combination of *S* and *p*.

### 378 **Estimation of selection coefficients**

379 A Bayesian model was developed to estimate selection coefficients by accounting for drift  
380 induced by a limited number of spawners, in a similar way to Wright-Fisher models.

381 First, the linkage disequilibrium method <sup>47</sup> implemented in the software NeEstimator 2.01 <sup>48</sup>  
382 was applied on samples grouped by cohort year to estimate the parental effective number of  
383 breeders (*N<sub>b</sub>*). This approach was favored over the standard temporal method potentially  
384 generating biased effective size estimates when used with temporally close samples from  
385 species with overlapping generations <sup>49</sup> and only providing information about the harmonic  
386 mean of effective sizes. In order to use the linkage disequilibrium *N<sub>b</sub>* values and associated  
387 95% parametric confidence intervals in the Bayesian models, parameters of log-normal  
388 distributions with similar percentiles were assessed using the R package *rriskDistributions* <sup>50</sup>.  
389 Weights of 7, 2 and 1 were respectively assigned to the 2.5, 5.0 and 97.5 percentiles to  
390 increase the approximation precision for lower bounds and medians. The negative or infinite  
391 values were replaced by 5000 or 10 000 for the median and 95% confidence interval upper  
392 bound, respectively. These are realistic maximum breeder numbers in the populations and

393 represent a conservative approach. If the lower bound also displayed infinite values, the  
 394 corresponding distribution had a median of 9 000 and lower and upper bounds of respectively  
 395 8 000 and 10 000.

396 The selection coefficient represents “the reduction in relative fitness, and therefore genetic  
 397 contribution to future generations, of one genotype compared to another”<sup>51</sup>. Selection  
 398 coefficients were estimated using 32 and 33 different spawning years, with corresponding  
 399 birth years, for Tenojoki and Inarijoki, respectively. Considering a SNP with alleles A1 and  
 400 A2 and  $W_y^{11} = 1$ ,  $W_y^{12} = 1 - DS$  and  $W_y^{22} = 1 - S$  being the relative fitness of each  
 401 genotype.  $S$  corresponds to the selection coefficient, following a uniform prior distribution  
 402 ranging from -1 to 1.  $D$  denotes the dominance coefficient, following a uniform prior  
 403 distribution ranging from 0 to 1. The observed number of each genotype  $g$  in spawners of sex  
 404  $s$  in year  $y$  ( $O_{g,s,y}$ ) followed a Dirichlet Multinomial (DM) distribution:

$$O_{g,s,y} \sim DM(T_{s,y}, g_{s,y}^{11} \dots g_{s,y}^{22}, \eta)$$

405 where  $\eta$  is the variation parameter following a uniform distribution ranging from 1 to 2500  
 406 and  $T_{s,y}$  the total number of spawners per sex and year. The spawners genotype frequency for  
 407 each sex ( $g_{s,y}^{11} \dots g_{s,y}^{22}$ ) varied over years according to a hierarchical model,  
 408  $g_{s,y}^{11} \dots g_{s,y}^{22} \sim \text{dirichlet}(\mu g_s^{11} \dots \mu g_s^{22}, \eta)$  and  $\mu g_s^{11} \dots \mu g_s^{22} \sim \text{dirichlet}(1,1,1)$ . The observed number  
 409 of allele A1 ( $n_y$ ) in individuals born in year  $y$  follow a binomial distribution:

$$n_y \sim \text{Binomial}(p_y, 2 N_y)$$

410 with  $N_y$  being the total number of individuals per year and  $p_y$  the population allele frequency.  
 411 The expected allele frequency in the cohort  $y$  depends on genotype frequency in spawners the  
 412 year before as follow:

$$E[p_y] = \frac{g_{y-1}^{11} W^{11} + 0.5 g_{y-1}^{12} W^{12}}{\bar{W}}$$

413 with  $\bar{W}$  being the population mean fitness  $\bar{W} = g_{y-1}^{11} W^{11} + g_{y-1}^{12} W^{12} + g_{y-1}^{22} W^{22}$ .  $g_{y-1}^{11}$ ,  
 414  $g_{y-1}^{12}$  and  $g_{y-1}^{22}$  are the genotypes of spawners averaged across sexes, as each sex contributes  
 415 equally to the next generation despite a potential biased sex-ratio. Genetic drift should be  
 416 taken into account to estimate  $p_y$  from the genotype frequencies of the previous year’s  
 417 spawners. In populations with random mating, it corresponds to drawing randomly  $p_y$  from a  
 418 binomial distribution (Fisher 1930; Wright 1931) with as parameters the expected allele  
 419 frequency  $E[p_y]$  and twice the effective number of spawners, previously estimated with the  
 420 linkage disequilibrium method ( $2 N b_y$ ). Consequently, the expected variance of the allele  
 421 frequency  $p_y$  subject to drift is after one generation  $\text{Var}(p_y) = \frac{p_y(1-p_y)}{2 N b_y}$ . For computing time  
 422 and convergence reasons, a beta distribution with equal mean and variance was used instead:

$$p_y \sim \text{Beta}(\alpha, \beta)$$

423 with

$$424 \alpha = \frac{E[p_y](\text{Var}(p_y) + E[p_y]^2 - E[p_y])}{\text{Var}(p_y)} \quad \text{and} \quad \beta = \frac{(\text{Var}(p_y) + E[p_y]^2 - E[p_y])(E[p_y] - 1)}{\text{Var}(p_y)}$$

425 Priors used in this model were chosen to be as uninformative as possible. For the *vgll3* locus,  
426 the *L* allele was chosen as reference. The “pMCMC” were calculated from the two chains as  
427 following:  $2 * \min(p < 0; 1 - p < 0)$ ,  $p < 0$  being the proportion of values below zero.

428

429 Posterior distributions were approximated using Monte Carlo Markov Chain (MCMC)  
430 methods with the Just Another Gibbs Sampler software (JAGS, Plummer 2017) run in the R  
431 environment<sup>43</sup>. Two MCMC chains were run for 4.5 million iterations, including a burnin  
432 length of 3.5 million. Only one iteration out of 100 was kept to reduce the memory size used.  
433 Gelman and Rubin’s convergence diagnostic<sup>53</sup> was used to assess convergence. Models were  
434 run longer if the potential scale reduction factor (*psrf*) was initially superior to 1.10. Finally,  
435 all models had potential scale reduction factor inferior or equal to 1.10 for all parameters,  
436 except for up to 2  $Nb_y$  parameters in 10 models for Inarijoki, having larger *psrf* (inferior to  
437 1.30).

#### 438 **Data and code availability:**

439 The datasets used during the current study will be uploaded to a public data repository upon  
440 acceptance.

#### 441 **Code availability:**

442 The custom codes used during the current study are available from the corresponding author  
443 on reasonable request.

444

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## 575 **Author contributions**

576 J.E and P.O. coordinate the collection of samples; C.R.P., Y.C., T.A. and J.E. designed the  
577 study; Y.C. analyzed the data; Y.C., C.P. and T.A. wrote the manuscript and all authors  
578 contributed to its revision.

## 579 **Competing interests**

580 The authors declare no competing financial interests.

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