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1 **Is starvation a cause of overmortality of the Mediterranean sardine?**

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18 **Running title:** Starvation and mortality in fish

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21 **Abstract**

22 Animal mortality is difficult to observe in marine systems, preventing a mechanistic understanding of
23 major drivers of fish population dynamics. In particular, starvation is known to be a major cause of
24 mortality at larval stages, but adult mortality is often unknown. In this study, we used a laboratory
25 food-deprivation experiment, on wild caught sardine *Sardina pilchardus* from the Gulf of Lions. This
26 population is interesting because mean individual phenotype shifted around 2008, becoming
27 dominated by small, young individuals in poor body condition, a phenomenon that may result from
28 declines in energy availability. Continuous monitoring of body mass loss and metabolic rate in 78
29 captive food-deprived individuals revealed that sardines could survive for up to 57 days on body
30 reserves. Sardines submitted to long-term caloric restriction prior to food-deprivation displayed
31 adaptive phenotypic plasticity, reducing metabolic energy expenditure and enduring starvation for
32 longer than sardines that had not been calorie-restricted. Overall, entry into critical fasting phase 3
33 occurred at a body condition of 0.72. Such a degree of leanness has rarely been observed over 34
34 years of wild population monitoring. Still, the proportion of sardines below this threshold has
35 doubled since 2008 and is maximal in January and February (the peak of the reproductive season),
36 now reaching almost 10% of the population at that time. These results indicate that the demographic
37 changes observed in the wild may result in part from starvation-related adult mortality at the end of
38 the winter reproductive period, despite adaptive plastic responses.

39 Introduction

40 Survival is one of the main factors driving population dynamics but it is difficult to assess. This is
41 because it either requires longitudinal monitoring of individuals by capture, mark and recapture, or
42 the ability to find dead individuals if there are extreme events such as massive die-offs (Begon et al.,
43 1996). Aquatic ecosystems pose particular challenges because dead fishes are very rarely found,
44 either sinking to the bottom or being consumed by other animals (but see Griffiths and Kirkwood,
45 1995). The difficulty of observing the vast underwater realm generally precludes assessment of
46 patterns of mortality and what their main drivers might be. There are a few exceptions, such as when
47 dead fishes wash up along shorelines, enabling researchers to investigate the cause of deaths (e.g.
48 discover the occurrence of pathogens; Whittington et al., 2008)Whittington et al., 2008)Whittington
49 et al., 2008).

50 As in all animals, mortality in fishes has multiple sources, such as epidemics, predation, starvation or
51 aging. According to life-history strategy, life-history traits such as survival result from a trade-off in
52 energy allocation among maintenance, growth and reproduction (Stearns, 1976; Williams, 1966).
53 Species have evolved life-history strategies to maximise individual fitness, resulting in improved
54 population growth and stability across time (Stearns, 1992), but these may not always be optimal,
55 especially if there are sudden unexpected environmental changes. While massive die-offs often occur
56 in response to an abrupt change and extreme conditions, such as the effect of the so-called 'blob'
57 heat waves on seabird mortality in the Pacific (Jones et al., 2018), less extreme conditions may
58 modify the energy balance of individuals. This may involve an increase in energy demands and
59 expenditure (e.g. an increase in temperature and metabolism, maturation of gonads, etc.) or a
60 decrease in energy resources (e.g. change in food availability or quality). In both cases, energy
61 allocation could be modified, leading to declines in body condition that affect main vital rates and
62 potentially threaten survival. Indeed, starvation is known to disturb the demographics of animal
63 populations, for example penguins (Morgenthaler et al., 2018) or gannets (Grémillet et al., 2016);
64 and even cause mortalities in some cases (Sherman and Runge, 2002). For fishes, reduced food
65 resources are commonly assumed to threaten survival in winter (Hurst, 2007) but, although
66 starvation may be a major driver of mortalities in fish populations, there have been very few
67 attempts to investigate this directly (Dutil and Lambert, 2000). In particular, plankton production and
68 availability for recruitment dynamics of small pelagic fish has been well documented (e.g. in
69 Palomera et al. 2007), but less is known about adult mortality due to starvation.

70 Sardines are small pelagic planktivorous fish with a worldwide distribution that are known for the
71 profound fluctuations in their populations and their importance as fishery resources (Pikitch et al.,

72 2012). Alternation of boom and bust periods, with major changes in abundance, has been observed
73 in various upwelling ecosystems (in particular the Eastern-Boundary Upwelling Systems). This is
74 thought to be firstly the result of high and low recruitment rates due to variation in environmental
75 conditions (Checkley, 2009; Field et al., 2009; Gushing and Dickson, 1977; Schwartzlose et al., 1999),
76 although overfishing can also modify the dynamics and accelerate the decline of those populations
77 and generate their collapse (Essington et al., 2015; Lindegren et al., 2013; Toresen and Ostvedt,
78 2000).

79 In the North-Western Mediterranean Sea, sardines *Sardina pilchardus* had, for centuries, been a very
80 important fishery. Over the last decade, however, sardine biomass has decreased due to a sharp
81 decline in individual size and mass, although abundance has increased and recruitment is high (see
82 Saraux et al., 2019; Van Beveren et al., 2014 for more details). The decrease in size is primarily
83 related to higher natural mortality of older individuals but this cannot be accounted for by
84 overfishing, predation pressure or pathogens (Queiros et al., 2018; Van Beveren et al., 2016a; Van
85 Beveren et al., 2016b; Van Beveren et al., 2017). Therefore, a bottom-up control of the sardine
86 population, linked to a regime shift in their planktonic prey towards smaller less nutritious species,
87 has been proposed as a mechanism underlying lower growth and body condition (Brosset et al.,
88 2016a; Saraux et al., 2019). Experiments on captive adult sardines showed that body condition,
89 growth and energetic reserves were significantly impacted by both food size and food quantity.
90 Sardines feeding on small items needed to consume twice as much as those feeding on large items to
91 reach the same body condition and growth rate. They also had to feed on large items in large
92 quantity to accumulate lipid reserves (Queiros et al., 2019). Despite the low body condition under
93 some feeding treatments, mortality was too low to test hypotheses of natural mortality from
94 starvation (Queiros et al., 2019). Notably, in the wild, survival of low body condition adults may be
95 especially challenged by energy allocation towards reproduction, which occurs in winter (Brosset et
96 al., 2016b). This is when food availability is lowest, potentially leading to exhaustion of reserves and
97 starvation before spring blooms. Thus, we designed a fasting experiment to gain insight into whether
98 the disappearance of older individuals from sardine populations might be linked to mortality from
99 starvation post-breeding, in late winter.

100 Individuals with low body reserves before winter find themselves on the razor's edge if they have to
101 cope with prolonged food restrictions, endangering their survival or reproduction (see Boos et al.,
102 2005; Grémillet et al., 2005; Olsson and van der Jeugd, 2002). Thus, a further interrogation concerns
103 the capacity of this species to show adaptive responses to sustained periods of low food resources,
104 to decrease these risks. As previous experiments have suggested that the slower growth and lower
105 condition of sardines in the Gulf of Lions did not result from natural selection, we focussed upon a

106 role of adaptive phenotypic plasticity (Gienapp et al., 2008; Nussey et al., 2007; Visser, 2008). Such
107 plasticity can either be fixed by exposure to particular environmental conditions during development
108 or can be reset cyclically, for example labile traits linked to spawning periods (Nussey et al., 2007).
109 Phenotypic plasticity in maturation or growth has been documented as a response of marine fish to
110 exploitation (Jorgensen et al., 2007). While it is unlikely that adaptive phenotypic plasticity could
111 compensate for extreme climatic events and prevent massive die-offs (see Pershing et al., 2015), it
112 may play an important role in compensating slower or more predictable changes (Levins, 1968), such
113 as modifications in food resources. Plasticity in the short term would then provide the potential for
114 species to adapt in the medium to long term. Sardines are particularly interesting because they
115 exhibit a marked flexibility in feeding behaviour, using a direct prey capture when food items are
116 large but shifting to a filter-feeding strategy when prey are small (Garrido et al., 2007). Such
117 behavioural flexibility may be linked to other adaptive plastic responses that improve tolerance of
118 poor feeding conditions.

119 To better understand first the link between sardine adult condition and mortality and to estimate the
120 incidence of death by starvation in wild populations, we used a combination of lab experiments on
121 wild-caught fish maintained in captivity and in situ data. As for birds and mammals, fishes usually
122 undergo 3 different phases of fasting (Bar, 2014) where: phase I is characterized by a rapid decrease
123 in body mass, the use of glycogen reserves and the progressive use of lipids; phase II involves a
124 relatively extended period where body mass loss is relatively low and constant, and lipid reserves are
125 the main energy source, and phase III is when reserves are exhausted so the main energy substrate is
126 structural proteins and rates of body mass loss increase again (Bar, 2014; Cherel and Le Maho, 1985;
127 Cherel et al., 1987; Cherel et al., 1991; Le Maho et al., 1981). Using specific body mass loss (i.e. the
128 rate at which body mass decreases) and respiratory metabolism as indices, we investigated when
129 sardines entered the critical phase III of fasting. This enabled us to evaluate mortality probabilities
130 and sardine physiological states at different body conditions. We were then able to estimate risk of
131 adult mortality in wild populations, based upon their condition over recent years, and also as a
132 function of their annual breeding cycle.

133 Finally, to investigate whether phenotypic plasticity played a role in affecting the relationship
134 between adult body condition and mortality, we used sardines that had been maintained on four
135 different feeding treatments for seven months prior to the fasting experiment, one mimicking
136 conditions in the wild before 2008, one mimicking the current period after 2008, and two
137 intermediate conditions (see Queiros et al., 2019 and below for more details). If sardines are able to
138 display phenotypic plasticity, individuals that were subjected to caloric restriction during the initial
139 period might cope better with fasting. Alternatively, sardines under caloric restriction in the initial

140 phase might have suffered physiological costs, which would make them less able to cope with
141 fasting.

142

143 **Methods**

144 All procedures were in accordance with the French and the EU legislation regarding animal
145 experimentation (APAFIS, Permissions N° 7097-2016093008412692 and N° 10622-
146 2017071711101242).

147 ***Sardine provenance***

148 Adult sardines had initially been captured in October 2016 by a dedicated commercial purse seiner
149 off Frontignan (Hérault, South of France) and transported to the IFREMER research station at Palavas-
150 les-Flots for a first feeding experiment (Queiros et al., 2019). To run this previous experiment,
151 sardines had been weaned from live food (*Artemia nauplii*) onto commercial aquaculture pellets and
152 individually marked under anaesthesia (benzocaine at 140 ppm), using a tiny RFID (Radio Frequency
153 Identification) tag (Biolog-id, Bernay, France, 0.03g, i.e. <0.2% of sardine lowest body mass)
154 implanted in the dorsal muscle with a specific injector. 449 sardines had been attributed to one of
155 four feeding treatments (2 tanks per treatment), so that both the mean and coefficient of variance in
156 length and mass were similar between tanks.

157 The four feeding treatments comprised food particles (commercial pellets) of two different sizes in
158 sardine natural feeding range (0.1mm and 1.2mm, eliciting filtration versus particulate-feeding,
159 respectively) at two different ration levels (0.3% and 0.6% of the biomass in tanks, based on
160 preliminary tests). That is, i) treatment LP-LQ: large food particles in large quantities, ii) LP-SQ: large
161 particles in small quantities, iii) SP-LQ: small food particles in large quantities and iv) SP-SQ: small
162 particles in small quantities. Results of this experiment are presented in Queiros et al., (2019) and
163 showed an effect of both food item size and quantity on sardine growth, condition and physiological
164 state.

165 ***Experimental design***

166 After 7 months of experiment (June 15th, 2017), sardines were sampled randomly from the four
167 feeding treatments and assigned to 8 smaller tanks (50L), to start the fasting respirometry
168 experiment. About 150g of sardines were placed per tank but, because of differences in mean body
169 mass of sardines from the four treatments, the number of individuals varied from 8 to 16 among

170 tanks. Sardines were left to acclimate for 12 days before the experiment started, i.e. time to develop
171 natural swimming and schooling behaviours in the new tanks.

172 One day prior to the experiment, sardines were measured again (length and mass) and body
173 condition estimated as the Le Cren index (see Brosset et al., 2015):

$$174 \quad K_n = W / W_{th} \quad [1]$$

175 where W is the wet mass of the fish in g and $W_{th} = 0.00607 \times TL^{3.057}$ the wet theoretical mass of a fish
176 of a given total length TL in cm.

177 Similarly to previous results on growth, condition and physiology (Queiros et al., 2019), body
178 condition of the randomly sampled sardines from treatments SP-LQ and LP-SQ were similar but
179 differed from the other two treatments (Fig. S1). Therefore, we had three initial treatments in this
180 experiment: (i) good initial feeding condition (sardines fed on LP-LQ), (ii) intermediate initial feeding
181 condition (sardines fed on SP-LQ and LP-SQ) and (iii) poor initial feeding condition (sardines fed on
182 SP-SQ). Unfortunately, due to a problem in the air system in two tanks, sardines died during one
183 night (1 tank from the LP-LQ and 1 tank from the LP-SQ), the experiment was finally run in 6 tanks
184 (see table 1).

185 Biometries were performed once a week with all sardines measured individually (tag read, length
186 and body mass recorded) under anaesthesia (benzocaine at 140 ppm). Both body mass and total
187 length were assumed to change linearly in between two biometries such that, when needed, daily
188 values were estimated through interpolation. Tanks were checked at least three times a day for
189 mortality and dead or moribund fish (unable to maintain its balance) were removed on these
190 occasions and immediately measured and weighed. At each biometry, the number and biomass of
191 fish per tank were checked. Whenever the number of fish decreased below 5 in one of the tanks, fish
192 of this tank were transferred to the other tank of the same treatment.

193 The specific body mass loss, namely the rate at which body mass decreases was calculated as;

$$194 \quad dm/mdt = (m_{t_1} - m_{t_0}) / (m_{t_0} \times (t_1 - t_0)) \quad [2]$$

195 where t_i is the time at i and m_{t_i} is the body mass at time t_i

196 Tanks were supplied with the same water pumped directly from the sea and filtered through sand
197 (30–40 μm). The photoperiod was adjusted each week to follow the prevailing natural cycle. Sea
198 water temperature was not controlled during the experiment to mimic natural conditions (varied
199 between 19°C and 24°C during the fasting experiment).

200

201 **Respirometry**

202 The rearing tanks were custom-designed to measure metabolic rate as O₂ uptake by automated stop-
203 flow respirometry (Steffensen, 1989), as previously described in McKenzie et al. (2007), see
204 supplementary material for detailed methods. Measurements were taken continuously throughout
205 the fasting period with a 1h cycle (45min flow/ 15min stop). Oxygen uptake by the fish (MO₂) was
206 then calculated on the stored files using R software and a custom script. The MO₂ was calculated in
207 mg kg⁻¹ h⁻¹, from the decline in water O₂ concentration and considering the total volume of water
208 and the total biomass of the fish (McKenzie et al., 2007; Steffensen, 1989). The hourly measures of
209 MO₂ were averaged to provide a measure of metabolic rate for the entire day. Standard metabolic
210 rates represent metabolic costs of maintenance and were estimated as the 10%-quantile of daily
211 measurements per tank for days in which more than 10 measurements were available (Chabot et al.,
212 2016).

213 **Sardines in the wild**

214 To compare our results obtained in tanks with fish in natural conditions, we used a long-term dataset
215 of sardine size (to the nearest mm) and body mass (to the nearest 0.1g), as well as maturity stage (by
216 visual assessment according to (ICES, 2008)). Maturity stages were described on a scale from 1 to 6,
217 with increasing development of gonads in stages 2 to 4, spawning period during stage 5 and post-
218 spawning period during stage 6 and a resting period during stage 1. These sardines had been
219 sampled from PELMED scientific surveys (PELagiques en MEDiterranée, DOI: 10.18142/19) and
220 commercial fisheries in the Gulf of Lions (NW Mediterranean Sea) from 1971 to 1978 and 1993 to
221 2018. Samples consisted of one crate of fish taken randomly from a pelagic trawl or a purse seine net
222 before any sorting had occurred. Body condition was estimated for all sardines with the Le Cren
223 index ((Brosset et al., 2015), see Equation [1] above). According to previous studies, sardine body
224 condition decreased profoundly in 2008, to remain low since then (Saraux et al., 2019; Van Beveren
225 et al., 2014). As a consequence, data were categorised into two periods: i) the past, being all data
226 collected before 2008, i.e. 6764 sardines, and ii) the present, being all data collected since 2008, i.e.
227 14,668 sardines.

228 **Statistical analyses**

229 All statistical analyses were performed with R v.3.5.0 (R Core Team, 2018). Values are given in the
230 text as mean ± SE, and statistical tests were considered significant at p < 0.05. When data were not

231 independent from each other due to repetitions within individuals (e.g. body condition over time), a
232 mixed model was used (either linear mixed model (LMM) or generalized linear mixed model (GLMM),
233 depending on the distribution of the data) with the individual effect set as a random intercept.
234 Number of observations (n) and number of individuals (N) are reported. Model selection was
235 performed by Akaike's information Criterion (AIC) and the most parsimonious model was retained
236 when a difference in AIC was less than 2 (Burnham and Anderson, 1998). Survival analyses were
237 performed based on the distribution of survival times using the Cox proportional-hazards regression
238 model (Cox, 1972). Treatments or maturity stages were compared using Wilcoxon tests, as normality
239 was violated. When multiple testing was performed (comparison between treatments, etc.), a
240 Bonferroni correction was used (Legendre and Legendre, 2012). Finally, whenever appropriate,
241 breakpoints in the data were identified using the "segmented" package in R (Mugeo, 2008). Residuals
242 of all models (LM, LMM and segmented regressions) were checked and both ANOVA tables and
243 diagnostic plots are presented in supplementary material. Whenever, the distribution of the residuals
244 was not normal and/or included too many outliers, additional analyses were performed to test for
245 the robustness of the results. First, data were transformed using a monotonous positive function
246 through the BoxCox transformation, i.e. . Normality of the residuals was checked again and if
247 validated the results of both models (on raw and transformed data) were compared. If the normality
248 of the residuals was still violated, we performed the model removing outliers, so as to ensure a
249 better residual distribution. Again, the results of both models (all raw data and raw data without
250 outliers) were compared. If results were similar, we present results and figures with raw data in the
251 main text, as we had no biological reason to remove outliers and raw data are easier to read than
252 transformed ones. However, results of the second more statistically appropriate models are
253 mentioned in the main text and fully detailed in the supplementary material. In one analysis, the
254 normality of the residuals seemed violated due to the individual random effect (close to normal
255 residuals when we used a linear model, but non-normal residuals when adding the individual random
256 effect). In that case, a second analysis was performed by running separate linear models, one per
257 individual, and the distributions of the estimated slopes were then used to test for the robustness of
258 the results of the linear mixed model.

259

260 **Results**

261 ***Changes in body condition***

262 Body condition of sardines decreased through time (number of fasting days) for the three treatments
263 (Fig. 2A). When modeling body condition through time using only individuals measured at least three

264 times, the number of fasting days, the treatment and their interaction were all retained in the best
265 model (as selected by AIC, LMM, $n = 289$, $N = 78$). So, the decrease in body condition throughout the
266 fasting period varied among treatments and the rate of decrease was also treatment dependent, as
267 indicated by the significant interaction between treatment and fasting days (Fig. 1 ; Table S1). The
268 rate of decrease in body condition was higher in sardines in good initial feeding condition ($-0.008 \pm$
269 0.000 per day) than in sardines in intermediate or poor condition (-0.006 ± 0.000 per day for both
270 cases). Because the normality of model residuals was not verified (Fig. S3), we also ran a second set
271 of analyses to check the robustness of these results. Estimating the decrease in condition for each
272 individual through a linear model (for individuals with at least 3 measurements), we found very
273 similar results, with a higher decrease in body condition for sardines initially in good body condition
274 (-0.009 ± 0.000) compared to sardines initially in intermediate or poor condition (-0.006 ± 0.000 , Fig.
275 S4; note that the distributions of the residuals of these linear models were in general satisfactory).

276 ***Survival analyses***

277 During the experiment, sardines died between day 1 and day 57 (Fig. S5). The cumulative death rate
278 shows that a quarter of the sardines died after 2 weeks, half after 3 weeks and 90% after 50 days of
279 fasting. The first mortality event occurred at a body condition of 0.84 for sardines in good initial
280 feeding condition and slightly later for sardines in intermediate or poor initial condition (0.77; Fig.
281 S6).

282 The patterns of survival against body condition were similar among the three treatments, but the
283 good initial condition was clearly shifted (to the right) along the x-axis (Fig. 2A). The survival analysis
284 indicated that the one-week survival probability of further fasting was affected by body condition
285 and the treatment the sardine originated from (GLMM binomial, $n=313$, $N=78$, Fig. 2A, Table S2).
286 Survival decreased with decreasing body condition, and was lower in sardines in good initial feeding
287 condition than in sardines in intermediate or poor initial condition (Fig. 2A). The probability of
288 surviving one week of further fasting was steady while body condition remained higher than 0.90 for
289 fish in good initial condition and higher than 0.75 for fish in intermediate or in poor initial condition.
290 Past these thresholds, this probability decreased rapidly reaching 50% at 0.80 for fish in good initial
291 condition and at around 0.65 for both fish in intermediate and poor initial condition (Fig. 2B).

292 The mean duration for which sardines were able to sustain fasting was higher for animals in good or
293 intermediate initial condition (31.5 ± 4.3 d and 32.8 ± 2.5 d) compared to those in poor initial
294 condition (16.3 ± 1.8 d, see Fig. 3A). The odds of dying were higher for sardines in poor initial
295 condition (Cox survival regression: $n = 78$, odds-ratio = 2.88 [1.30-6.41], $P = 0.009$) compared to
296 sardines in good initial condition, while they did not differ between sardines in intermediate and

297 good initial condition (odds-ratio = 0.71 [0.33-1.54], $P = 0.384$). When initial body condition was
298 included in this model, we found a negative effect of initial condition on the odds of dying (odds-
299 ratio of scaled condition = 0.14 [0.07-0.27], $P < 0.001$), i.e. sardines with a higher initial body
300 condition (e.g. an increase of 1SD, i.e. + 0.11 here) had a decreased daily survival (by 86% in this
301 example, Fig. 3B). Interestingly, the difference between initial treatments differed once initial body
302 condition was added in the model. The odds of dying were then lower in sardines of poor and
303 intermediate initial feeding conditions compared to those of good initial feeding conditions (odds
304 ratio = 0.02 [0.00-0.12] and 0.04 [0.01-0.13], respectively, both $P < 0.001$).

305 ***Changes in body mass loss***

306 The specific body mass loss displayed an abrupt increase a few days before death for all the sardines
307 (Fig. 4). To detect inflexion point(s), we applied a segmented regression model, using body mass loss
308 as the dependent variable. The best model selected by AIC comprised two breakpoints at 10 and 2
309 days before death ($\Delta AIC = -670$ with simple regression with no breakpoint and $\Delta AIC = -87$ with
310 segmented regression model including 1 breakpoint, see Table S3 for an ANOVA table of the selected
311 model). More precisely, during their first period of fasting, body mass loss was fairly low around 0.80
312 ± 0.00 % of body mass per day (Fig. 4). Ten days before death, this rate started increasing by 0.1%
313 point per day until two days before death, when specific body mass loss increased very sharply to
314 reach a mean body mass loss of $3.59\% \pm 0.36\%$ the day before death (Fig. 4). When looking
315 separately at each treatment, two breakpoints were also always found: two days before death for all
316 treatments and 16 days before death for the intermediate treatment, 9 and 8 days before death for
317 the poor and good treatment respectively (Fig. S7).

318 The specific body mass loss was relatively low and stable (0.84 ± 0.02 % of body mass per day) for
319 sardine body conditions greater than 0.75, but it rapidly increased at lower body conditions (Fig. 5).
320 When applying a segmented regression between body condition and specific body mass loss, the
321 best model selected by AIC retained one breakpoint at around 0.72 ($\Delta AIC = -157$ with simple
322 regression with no breakpoint and $\Delta AIC = -2.5$ with segmented regression model including 2
323 breakpoints, see Table S4 for an ANOVA table of the selected model and Fig. S8). Yet, the residuals of
324 this model presented a very skewed distribution (Fig. S9 – *All data*), which was barely improved by
325 data transformation. Rather, this seemed due to a distribution of specific body mass loss with a lot of
326 high values (Fig. S10). To assess the robustness of the breakpoints, we ran 2 additional analyses
327 based on the distribution of specific body mass loss: (a) using only data lower than 4 (thus discarding
328 extreme values) and (b) using only data lower than 2 (keeping data within the first and main mode of

329 distribution from Fig. S10). The normality of the residuals clearly improved in these two analyses (Fig.
330 S9) and the values of the breakpoint remained quite stable: (a) 0.72 ± 0.01 and (b) 0.74 ± 0.01 .

331 ***Changes in metabolic rates***

332 We then looked at the metabolic rates (per unit mass), as quantified through O₂ uptake during the
333 first 10 days of the experiment. These metabolic rates varied depending on the initial feeding
334 condition of the sardines (Fig. 6). Notably, sardines in poor condition had a lower metabolic rate than
335 sardines in good or intermediate condition ($P = 0.019$ and $P = 0.022$ respectively, Bonferroni-
336 corrected Wilcoxon tests). Standard metabolic rates (estimated as the lowest daily 10% quantile and
337 representing mostly maintenance metabolism) did not differ among treatments ($P > 0.126$,
338 Bonferroni-corrected Wilcoxon tests; Fig. 6). However, the difference between mean daily respiration
339 rate and daily standard respiration rate was significantly lower in sardines in intermediate or poor
340 initial feeding condition than in those in good initial condition ($P < 0.001$ for both, Bonferroni-
341 corrected Wilcoxon tests; Fig. 6).

342 According to segmented linear regression on all raw data, metabolic rate increased strongly when
343 sardine mean body condition decreased below 0.64 ± 0.01 , while it was constant above this body
344 condition ($305.5 \pm 4.7 \text{ mg O}_2 \cdot \text{h}^{-1} \cdot \text{kg}^{-1}$; Fig. 7, Table S5). However, the diagnostic plots of this model
345 revealed some patterns in the residuals (Fig. S11). Running the segmented model on transformed
346 respiration data (using a BoxCox transformation of -0.71) allowed removal of most of the patterns in
347 the residuals (Fig. S11), while leading to the same result with a breakpoint at 0.64 ± 0.01 (Table S6),
348 below which the transformed respiration increased strongly. Interestingly, the breakpoint obtained
349 on raw data was similar in sardines in poor or intermediate initial condition (0.63 ± 0.02 and $0.65 \pm$
350 0.01 respectively), but was much higher in sardines in good initial condition (0.78 ± 0.04) (Table S5).
351 Similar results were obtained when using BoxCox transformed data to improve the normality of the
352 residuals (Fig. S11), with a critical body condition detected at 0.64 ± 0.03 , 0.65 ± 0.01 and 0.79 ± 0.04
353 for sardines in poor, intermediate and good initial conditions respectively (see Table S6).

354 ***Body condition in the wild***

355 Sardine body condition in the wild was significantly higher in the past (i.e. before 2008) than in the
356 present (1.12 ± 0.00 vs. 0.98 ± 0.00 , Wilcoxon test, $P < 0.001$, $n = 6764/14668$ respectively, Fig. S12).
357 Further, body condition varied among months, peaking in spring/summer (as well as in early autumn
358 for the past period only) and reaching its lowest level in December, January and February for both
359 periods (Fig. S12). Finally, body condition decreased with maturity stages in both periods (Fig. S13,
360 Bonferroni-corrected Wilcoxon tests). While it decreased almost linearly with maturity stages in the

361 present, the contrast in body condition appeared mainly to be between maturity stages 5 and 6 (i.e.
362 during or post-spawning) and among the first four maturity stages in the past (despite no significant
363 differences in some cases due to very low sample size in some maturity stages).

364 When comparing against the critical body condition defined in our fasting experiments, only 0.1% of
365 the sardines sampled before 2008 were below the 0.65 threshold that appeared critical for 1-week
366 survival, and only 0.2% of the sardines sampled since 2008. The occurrence of sardines below the
367 second body condition threshold (i.e. 0.72 which corresponds to the entry into phase 3 fasting
368 according to body mass loss, Fig 5) also appeared rather low, although it almost doubled when
369 comparing the present to the past (2.3% vs. 1.2%; Fig. 8). The occurrences were not, however, evenly
370 distributed among months, being more probable during winter, especially in the present period,
371 where they reached 6 and 9 % of the population in January and February, respectively (Fig. 8).

372

373 **Discussion**

374 Although there has been quite significant research focus on starvation as a cause of mortality in
375 larval and juvenile fishes (Hurst, 2007), studies on adult fishes are much rarer (Dutil and Lambert,
376 2000; Lambert and Dutil, 1997). As mortality is complicated to observe in marine fish populations, we
377 used experiments in tanks to investigate the extent to which wild adult sardines might be at risk of
378 death from starvation. We used measurements on individuals and treatment groups to study the
379 physiological and behavioural consequences of caloric restriction. Most of our conclusions on the
380 effects of fasting on sardines were based on measurements of individual sardines within the
381 treatment groups. The exception was oxygen consumption, which was measured on entire tanks
382 because individual measurements are not feasible in this species. Due to the unfortunate failure of
383 the oxygen supply prior to the fasting experiment, we lost one tank of sardines in initial good
384 condition, so could only collect oxygen consumption throughout fasting on a single replicate for this
385 treatment. There were, however, no tank effects on fish condition or respirometry in the other two
386 treatments, and we also found no tanks effects on growth, condition, body composition and
387 oxidative stress in a previous long-term study on sardines (Queiros et al., 2019). The conclusions
388 regarding how starvation affected metabolic rate were also based upon common patterns in six
389 tanks, irrespective of their initial treatment. Further, we performed additional analyses when some
390 statements about model assumption were not verified (e.g. normality of residuals), which confirmed
391 and ensured the robustness of our results.

392 The results revealed that sardines were able to survive fasting for extended periods (up to 57 days
393 under our conditions) and to reach very low body condition before they risked mortality from
394 starvation depending on their initial condition. It is well known that fishes can survive extended
395 periods of food deprivation, although the actual duration varies among species, life stage and water
396 temperature (McCue, 2010; Navarro and Gutiérrez, 1995; Wang et al., 2006). In our study, survival in
397 sardines initially in intermediate and poor conditions remained high until a Le Cren's body condition
398 of 0.75 (i.e. 25% lower than the global average from long-term measurements on wild populations).
399 When fish condition decreased below this, survival dropped rapidly, to only 50% at a body condition
400 of 0.65. For fish in good initial condition, however, 50% survival was reached when body condition
401 decreased below a Le Cren's factor of 0.8.

402 The experimental approach was able to reveal when the sardines started relying on energetic protein
403 substrates as the main fuel for metabolism (contrary to high energetic lipid substrate). That is,
404 specific body mass loss and mass-specific metabolic rates increased markedly below a certain body
405 condition, enabling us to define a critical threshold that indicated when sardines entered into phase
406 III of fasting. Despite clear thresholds for mean population responses (see for instance Fig. 5), there
407 was significant variation among individuals, especially when they were at very low condition. Such
408 inter-individual differences might reveal the importance of individual quality in sardine physiological
409 responses to starvation and survival, despite individual quality being debated as a concept and
410 terminology (Bergeron et al., 2011; Wilson and Nussey, 2010). Individuals within fish species are
411 known to exhibit wide variation in their tolerance of feed deprivation, and this can have both a
412 physiological and behavioural basis (Auer et al., 2016; McKenzie et al., 2014; Norin and Metcalfe,
413 2019; O'Connor et al., 2000). Thus, further studies are required to elucidate the physiological or
414 behavioural correlates of the individual responses to a fasting challenge in sardines. In the current
415 study, the critical threshold for entry into phase III of fasting was much more accurately defined by
416 the specific body mass loss (estimation at the individual level) than by the metabolic rate (estimation
417 at the tank level). That is, although individual fish in a given tank derived from the same initial
418 feeding condition, their body condition varied at the beginning of the experiment (Table 1). Further,
419 rates of mass loss and metabolism both sped up during phase III of fasting, leading to death in about
420 8 days. Fish transitioned from phase II to phase III over a short period, such that a mixture of fish in
421 the different fasting phases were present in a given tank at a given time, contributing to the group's
422 overall metabolic rate. Nonetheless, the tank respirometry clearly indicates that entry into phase III
423 of starvation was associated with a marked increase in metabolic rate by the sardines, in all
424 treatment groups. It is not known why this occurred; it may have reflected the lower energetic
425 efficiency of using proteins as a main fuel compared to lipids (Schmidt-Nielsen, 1997) or a desperate

426 increase in activity in search of food (the so-called refeeding signal described in birds and mammals;
427 (Groscolas et al., 2000; Koubi et al., 1991; Robin et al., 1998; Spée et al., 2010), which would
428 obviously have exacerbated the rate of mass loss.

429 When comparing our overall critical condition threshold to in situ values observed over the years, the
430 proportion of the population sampled in nature that was below the critical condition of entry in
431 phase III of fasting was minimal ($\leq 2\%$). Moreover, almost none of the fish sampled in-situ exhibited
432 the low body condition with a 50% probability of surviving a week (i.e. 0.65). Such a result is very
433 similar to that obtained in Atlantic cod (Dutil and Lambert, 2000) but needs to be taken cautiously, as
434 sardines of low body condition might be excluded from schools or might already have died for other
435 reasons linked to a weakened physiological state. Indeed, low body condition can seriously impair
436 fish swimming activity (Faria et al., 2011; Martinez, 2003). As a consequence, starving sardines might
437 not be able to sustain the continuous aerobic swimming required to follow the school and might get
438 isolated. Poor swimming performance, especially burst swimming capacity (Martínez et al., 2004)
439 would render them prone to predation, also due to the absence of the dilution effect that is gained
440 by being in a school (Lehtonen and Jaatinen, 2016; Rieucan et al., 2014). This suggests that our
441 estimates of survival in tanks might represent a maximum potential of sardines to resist fasting and
442 that it is likely that mortality would occur earlier in the wild. Indeed, the fasted sardines were very
443 weak during the experiment, as attested by the increase in death rate during and after biometries
444 (Fig. S5), which had never occurred in other experiments where sardines were fed (QQ, CS, EG, GD
445 pers. comm.). Apart from handling once a week, our fish were subjected to no other stress, with no
446 predation, pathogens, etc. Nonetheless, such results need to be taken cautiously as mortality might
447 result from a combination of weakness of sardines and handling. Further, we examined the absolute
448 physiological thresholds of starvation; sardines most likely would not have to cope with such severe
449 food deprivation for such a long period in the wild. Thus, further studies may examine variations in
450 physiological thresholds as a function of different levels of food restriction (e.g. Hill et al., 1984; Hill
451 et al., 1985). Nevertheless, the proportion of Gulf of Lions sardines that were below the critical
452 condition of entry in phase III of fasting (i.e. 0.72) was about twice as high in the present period
453 (2.3%), after the changes in population condition and age structure were observed (Saraux et al.,
454 2019; Van Beveren et al., 2014), than in the past (1.2%). Additionally, sardines were fed with the
455 same food levels during 7 months prior to the fasting experiment and may have developed plastic
456 responses to their feeding conditions. However, resources and food quantity in particular is known to
457 fluctuate over time making adaptation more complicated in the wild. Further, sardines are known to
458 be capital breeders, i.e. they have to increase their energy reserves during summer to sustain
459 reproduction during winter, when food resources decline (Ganias, 2009; McBride et al., 2015). This

460 reproductive strategy leads to an increase in body conditions before winter (Fig. 8), which might raise
461 the critical body condition of entry into phase III and also decrease the probability of surviving a one
462 week of fasting (see Fig. S6 and Fig. 2 for 'good conditions', respectively). Interestingly, characteristics
463 of sardine populations have changed over the last decade (e.g. decrease of length at first maturity)
464 whereas the gonadosomatic index has increased (see Fig. 4 in Brosset et al. 2016b) suggesting higher
465 reproductive investment despite the decrease in body condition. While potential down regulation of
466 the reproduction through skipped spawning remains unknown for this species (but see Kennedy et
467 al., 2010 on herring), such investment toward reproduction despite low energy reserves was also
468 supported by a rather low atresia prevalence and intensity (Brosset et al. 2016b). This could result
469 from a change in energy allocation between the main functions, as a cause or consequence of higher
470 adult mortality (Brosset et al 2016b). Further, when looking at the monthly values, the critical
471 condition of entry into phase III occurred mostly in January/February (reaching 9%), which
472 corresponds to the end of the spawning season for sardines and also to the coldest period. The fact
473 that, among maturity stages, the proportion of body condition below the critical threshold was
474 highest in stage 6, post-spawning, suggests that a depletion of reserves partly due to reproduction
475 remains a valid hypothesis (Brosset et al., 2016b). The plasticity of the digestive tract in the context
476 of caloric restriction (e.g. atrophy or downregulation of function (Secor and Carey, 2016; Zaldúa and
477 Naya, 2014)) might not allow fish to cope with energy reserve depletion despite eruption of
478 planktonic blooms in spring. While we cannot derive actual mortality estimates directly from this
479 study, the results confirm that mortality might have increased after 2008 and be primarily in winter
480 at the end of reproduction.

481 Additionally, sardines that were in intermediate or poor feeding condition at the outset displayed a
482 much stronger resistance to fasting than fish that were in good feeding condition. This not only
483 shows that extended caloric restriction promotes subsequent tolerance of fasting (McCue et al.,
484 2017) but, most of all, it clearly demonstrates a plastic response by the fish to their environmental
485 conditions. All sardines were collected from the wild and our assignment to the different treatments
486 ensured that they were in similar body condition before starting the feeding trials described in
487 experiment (Queiros et al., 2019). Thus, it seems extremely unlikely that the differences in fasting
488 tolerance among the treatments in the current study could derive from genetic selection or early
489 environment exposure. They were more likely a plastic response at the adult stage (McCue et al.,
490 2017). The fact that body condition did not drop as fast in sardines that had been maintained for an
491 extended period in a state of caloric restriction presumably reflects differences in energy
492 expenditure. This is borne out by the tank respirometry. At the start of the experiment (1st 10 days),
493 daily metabolic rates were lower in sardines in poor initial feeding condition than in the other two

494 treatments (Fig. 6). It is well established that fish reduce their metabolic expenditure when exposed
495 to caloric restriction or extended fasting and that the two main strategies are a reduction in
496 spontaneous activity and a reduction in basal metabolism (Auer et al., 2016; McKenzie et al., 2014;
497 Norin and Metcalfe, 2019). These two processes are not mutually exclusive (Auer et al., 2016). While
498 further work is required to establish the relative contributions of behaviour and physiology, in
499 particular video tracking to quantify activity and studies of metabolism from the whole animal down
500 to the cell and mitochondrion (Auer et al., 2016), the tank respirometry provides some preliminary
501 insights. If we assume that the lowest 10%-quantile rate of O₂ uptake each day was an estimate of
502 standard metabolic rate (Chabot et al., 2016), this indicates that costs of maintenance were similar
503 among treatments. When these daily values were, however, subtracted from daily mean metabolic
504 rate, the difference was significantly higher in the fish in good initial condition, compared to the
505 other two treatments. This difference in metabolic rate, of the fasting fish, might represent in large
506 part costs of spontaneous swimming activity, perhaps exploratory foraging (Auer et al., 2016). This
507 indicates that the fish that had been exposed to caloric restriction had developed an adaptive
508 behavioural plasticity to reduce their energy expenditure. This energy conservation strategy is widely
509 known to be adopted by different taxonomic groups to cope with food restrictions (see Hill et al.,
510 1984; Lees et al., 2010; Secor and Carey, 2016; Stokkan, 1992; Zaldúa and Naya, 2014). The fact that
511 fish in good initial feeding condition, with no history of caloric restriction, were not able to engage
512 this adaptive response indicates that such behavioural plasticity is not instantaneous. One potential
513 explanation for this is that the responses to caloric conditions are under neuro-hormonal or
514 endocrine control, and require some weeks or months to be expressed (Secor and Carey, 2016).

515 Understanding fluctuations in mortality is essential for the management of exploited species, but the
516 estimation of natural mortality in stock assessment models often remains restricted to predation
517 mortality and modelled either as a constant or as a function of size (Gislason et al., 2010), despite
518 evidence that natural mortality can significantly vary according to size and growth (Gislason et al.,
519 2010; Lorenzen, 1996) or density dependent processes (Fromentin et al., 2001). Here, our
520 experimental results showed that mortality increases in fish with low body condition, suggesting that
521 adult natural mortality from starvation may occur in the wild mainly in winter of recent years (as the
522 proportion of the natural population below critical body condition threshold is much higher in
523 January/February and doubled in recent years). Such a result is also of interest for fisheries science,
524 as misspecification of M in stock assessment models is known to lead to biased estimates of fisheries
525 reference points (Gislason et al., 2010; Johnson et al., 2014; Punt et al. 2021). A recent study on
526 Atlantic cod *Gadus morhua* found, for instance, that using a variable body-condition dependent M
527 instead of a constant one can result in drastic changes in stock diagnosis (up to 40% differences in

528 spawning stock biomass (SSB), F and recruitment; (Casini et al., 2016). In the case of sardines in the
529 Gulf of Lions, the present situation of limited age classes 0-2 prevents us for using an analytical
530 model and assess the importance of a variable M. Still, our study also suggests that body condition, a
531 fairly easy variable to monitor (Brosset et al., 2015), could provide a reliable indicator of a stock
532 health status, that could be widely applied. This could support management of data limited stocks,
533 where complete stock assessments are not possible, but also provide a basis for dynamic adaptive
534 management. Management is often conducted with a delay compared to the current state of a stock,
535 due to the time needed to collect the data, perform stock assessments, decide upon management
536 measures and, finally, implement them (Cochrane and Garcia, 2009). By explicitly showing when the
537 population is the most sensitive and the most at risk within the year, body condition could thus
538 provide rapid updated information on the stock to managers for them to engage in adaptive
539 measures, such as individual quotas combined with differential temporal allocation of fisheries effort
540 within the year or temporal closures (see e.g. (Hobday et al., 2010; Melnychuk et al., 2013).

541 In conclusion, we showed here that adult sardines were highly resistant to fasting when maintained
542 in tanks free of predation or pathogens. We showed that, when previously maintained under caloric
543 restriction, sardines could display significant behavioural plasticity that improved their ability to
544 tolerate fasting, by reducing rates of body mass loss and so increasing their survival. Experimental
545 measurements of specific body mass loss and metabolic rates enabled us to define a critical
546 threshold of body condition, which we could then relate to in situ measurements on wild
547 populations. The combination of laboratory and field data therefore reveals that death from
548 starvation is probably not a major factor in determining the mortality of sardines in the
549 Mediterranean Sea during most of the year, but mortality after spawning has increased in the last
550 decade becoming as high as 9%. Overall, death from starvation may be a significant driver of marine
551 fish population dynamics, especially if energy availability drops in marine ecosystems as a result of
552 ongoing global change.

553

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561

562 **Authors contribution**

563 CS, PB, DM conceived the idea; CS, QQ, EG, GD, DM, JMF designed the study; CS, QQ, GD, EG, AM,
564 DM collected the data; CS, QQ, JMF analysed the data; CS, QQ, DM wrote the manuscript; all authors
565 contributed critically to the drafts and gave final approval for publication.

566

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576

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823 **Figures**

824

825 Fig. 1: Mean \pm SE body condition per week along the fasting experiment for each of the three initial feeding
826 condition treatments (absence of standard errors is due to a single survivor in the given treatment).

827

828 Fig. 2: Probability of surviving one week of fasting according to body condition. A) Empirical data are shown as
829 0 and 1 survival points, while lines represent the probability to survive predicted by the model according to
830 their initial feeding conditions. B) Mean empirical probability of surviving one week of fasting according to bins
831 of body condition. The lines represent the 95% confidence interval associated with this empirical probability
832 (according to a Bernouilli distribution). Colour indicates the three initial feeding condition treatments.

833

834 Fig. 3: Survival probability of sardines across the fasting experiment according to their initial feeding treatment:
835 A. uncorrected, as predicted by the cox survival model and B. corrected by the initial fish body condition, as
836 predicted by the cox survival model including initial fish body condition as a covariate. Colour indicates the
837 feeding treatment sardines originated from and dotted vertical lines indicate the number of fasting days at
838 which survival equalled 50%.

839

840 Fig. 4: Mean \pm SE specific body mass loss (, i.e. the rate at which body mass decreases) per week along fasting
841 experiment. As individuals died at different time in the experiment, the number of days has been estimated
842 relative to death. The specific body mass loss is expressed as %. The vertical dashed line shows a rupture in the
843 slope.

844

845 Fig. 5: Mean \pm SE specific body mass loss () according to bins of body condition. The specific body mass loss is
846 expressed as %.

847

848 Fig. 6: Respiration rates (in $\text{mg O}_2 \cdot \text{h}^{-1} \cdot \text{kg}^{-1}$) during the first 10 days of the experiment according to the feeding
849 conditions sardines encountered before the start of the experiment. A) mean daily respiration rate, B) standard
850 respiration rate (as estimated by the lowest 10% quantile and representing mostly maintenance metabolism),
851 C) 'Activity' related respiration rate (as estimated by the difference between the daily mean and minimum
852 values). Boxes sharing common letters are not significantly different from each other according to Bonferroni-
853 corrected Wilcoxon tests.

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856 Fig. 7: Mean daily metabolic rate (expressed in $\text{mg O}_2 \cdot \text{h}^{-1} \cdot \text{kg}^{-1}$) of sardines in a given tank as a function of mean
857 body condition of sardines in that tank that day. Colour indicates the treatment sardines originated from. The
858 segmented regressions are indicated by the black line and the 95% confidence intervals with dashed lines. The

859 breakpoint along with its 95% CI is also indicated at the bottom of the figure in black. Breakpoints and their
860 95% CI estimated for each treatment are indicated at the bottom of the figure in colour.

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863 Fig. 8: Distribution of body condition of sardines sampled in the wild before (in blue, upper panel) or after 2008
864 (in red, lower panel) for each month of the year. Horizontal dashed lines indicate the threshold of body
865 condition corresponding to an entry in phase 3 of fasting. The percentage of the population below this critical
866 threshold of body condition each month is indicated at the bottom of each panel.

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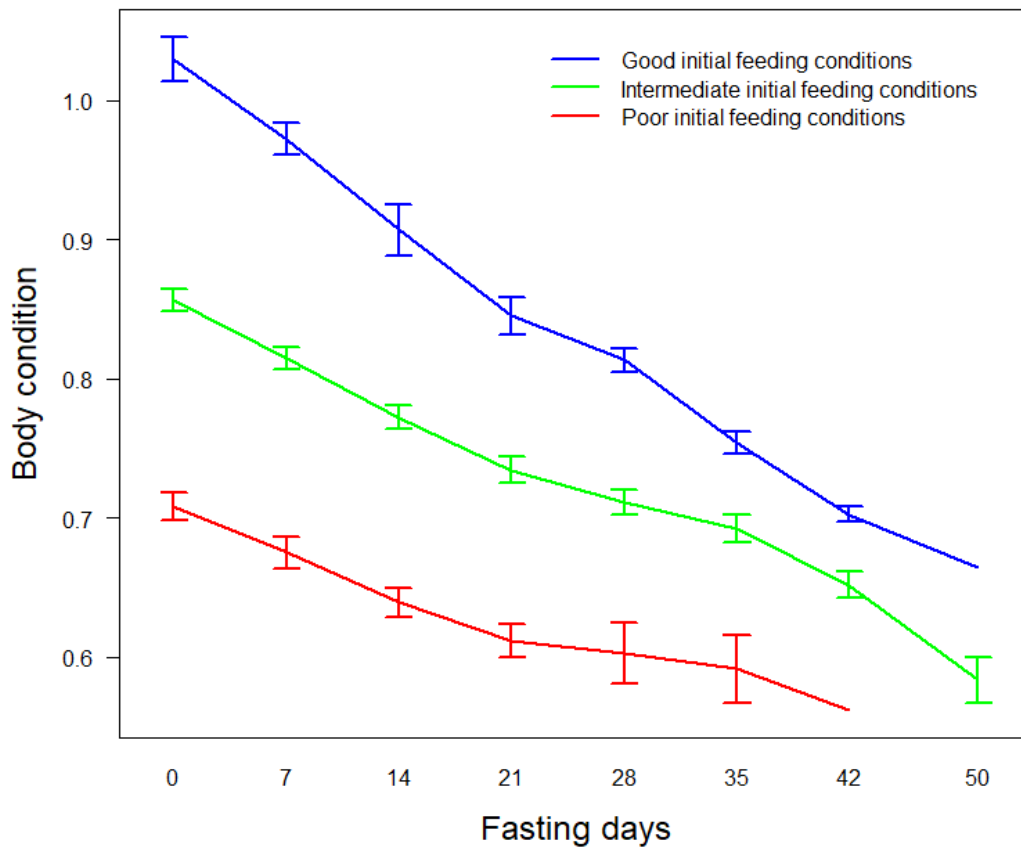
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869 Table 1 - Mean \pm SE body mass, length and body condition of sardines, as well as the number of fish and tanks
870 used per initial feeding conditions at the start of the experiment.

Treatment	Body mass (g)	Length (mm)	Body condition	Nb of fish	Nb of tanks
Initial good feeding condition (LP-LQ)	19.31 \pm 1.30	138.0 \pm 2.9	1.03 \pm 0.02	8	1
Initial intermediate feeding condition (LP-SQ & SP-LQ)	12.46 \pm 0.38	127.0 \pm 1.2	0.85 \pm 0.01	39	3
Initial poor feeding condition (SP-SQ)	9.84 \pm 0.37	125. 1 \pm 1.5	0.71 \pm 0.01	31	2

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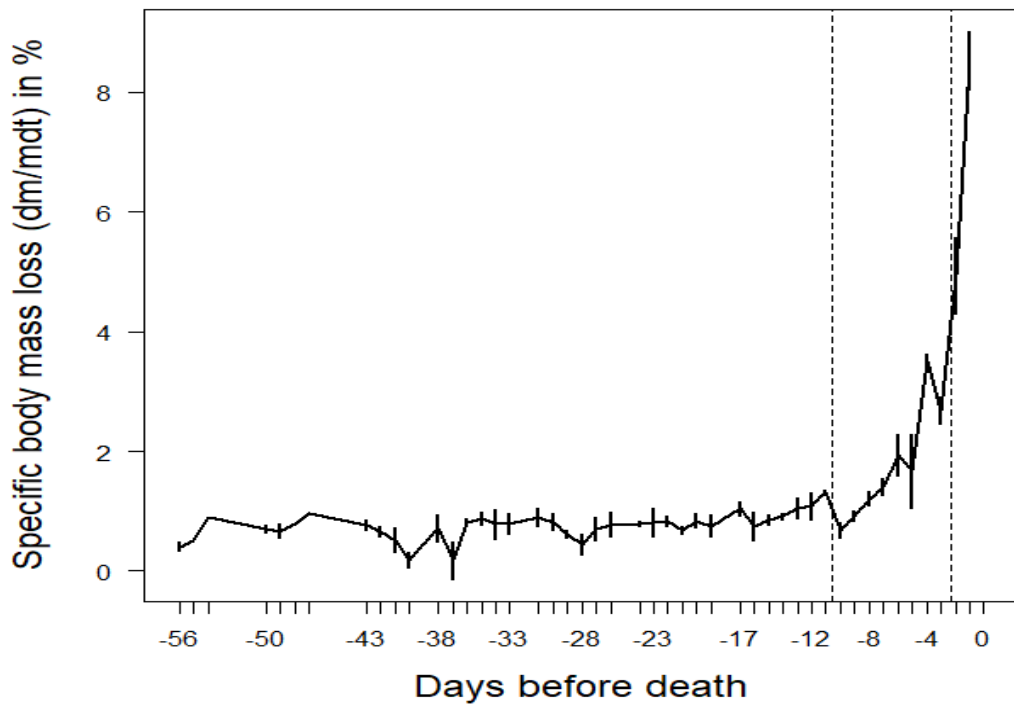
893 Figure 1.

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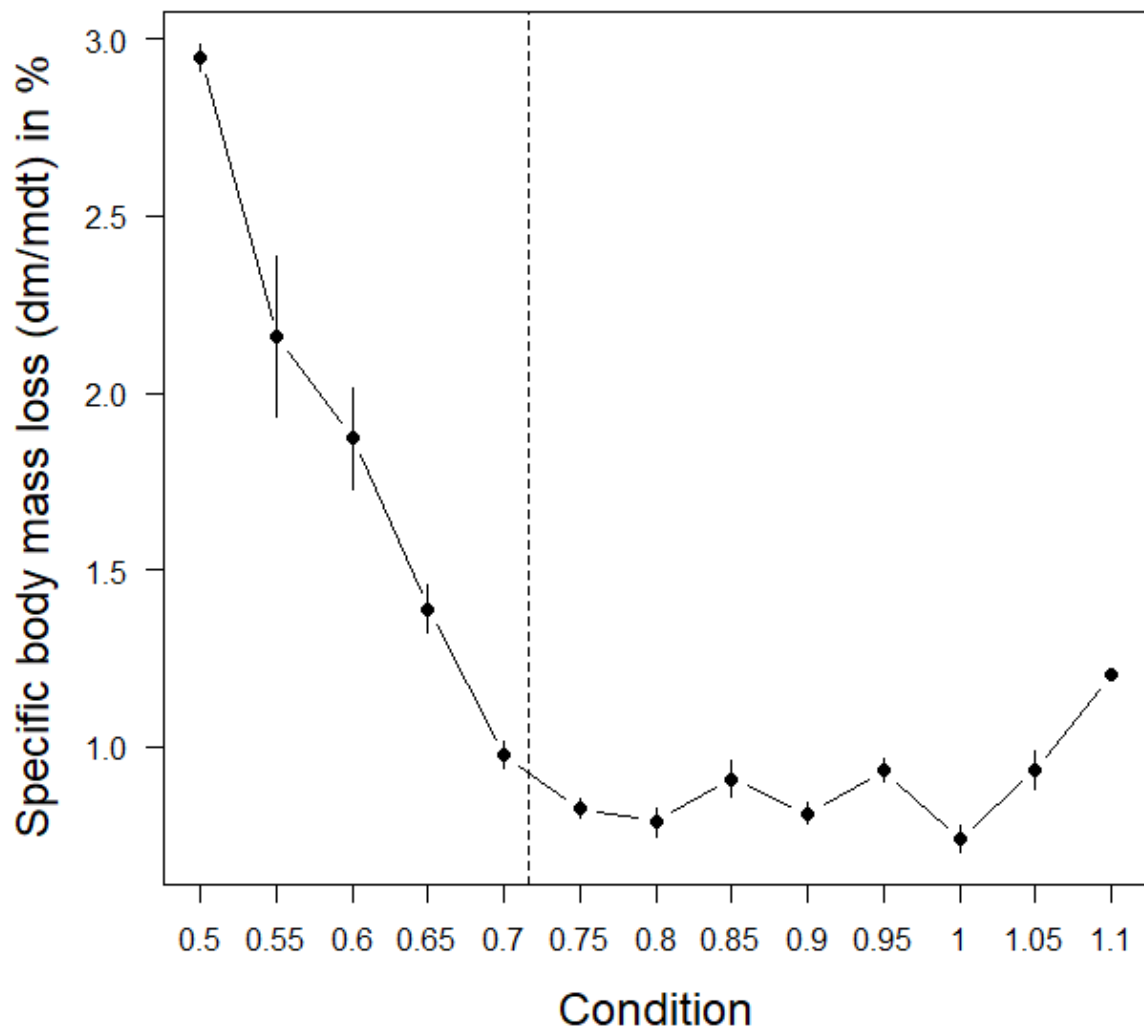
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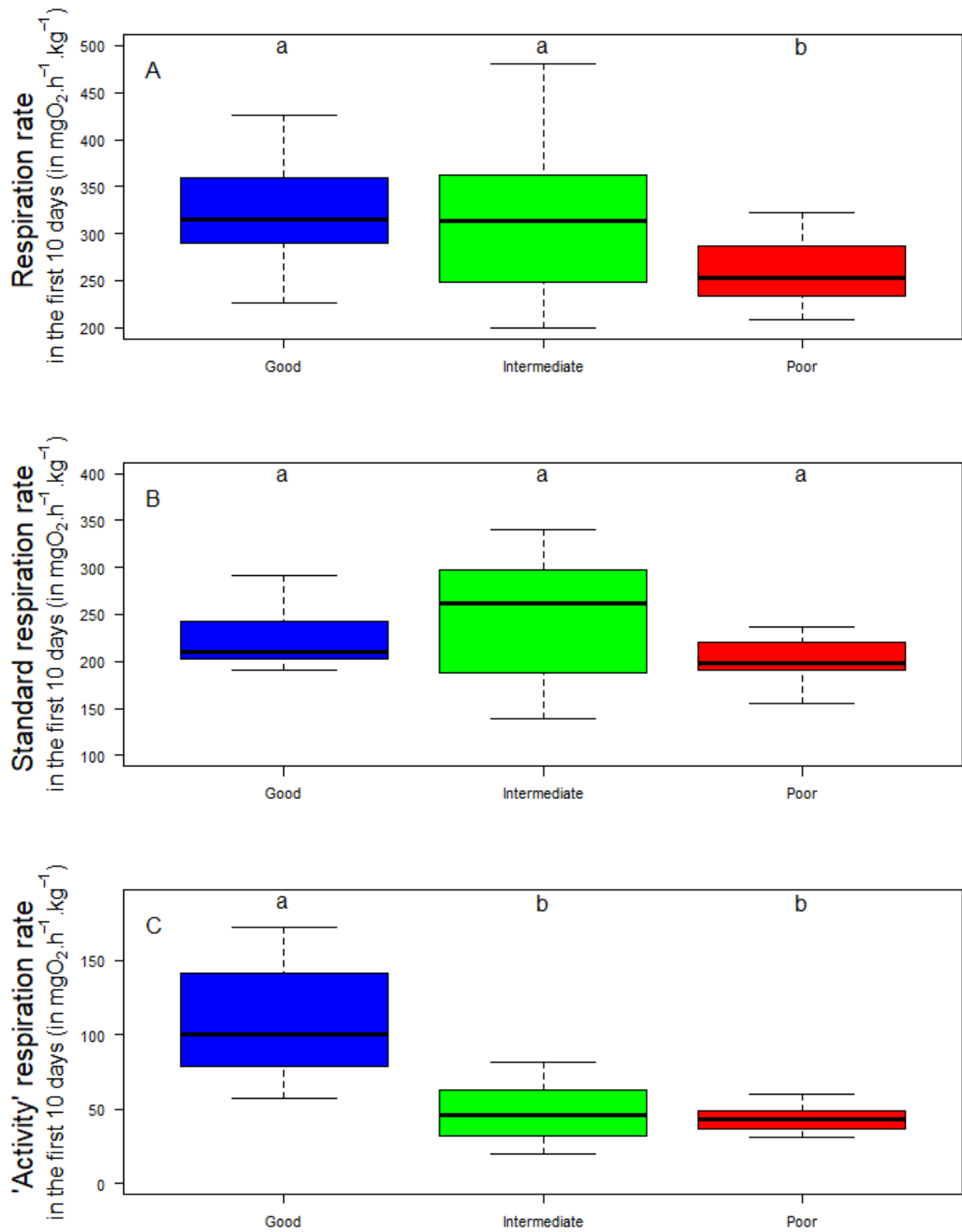


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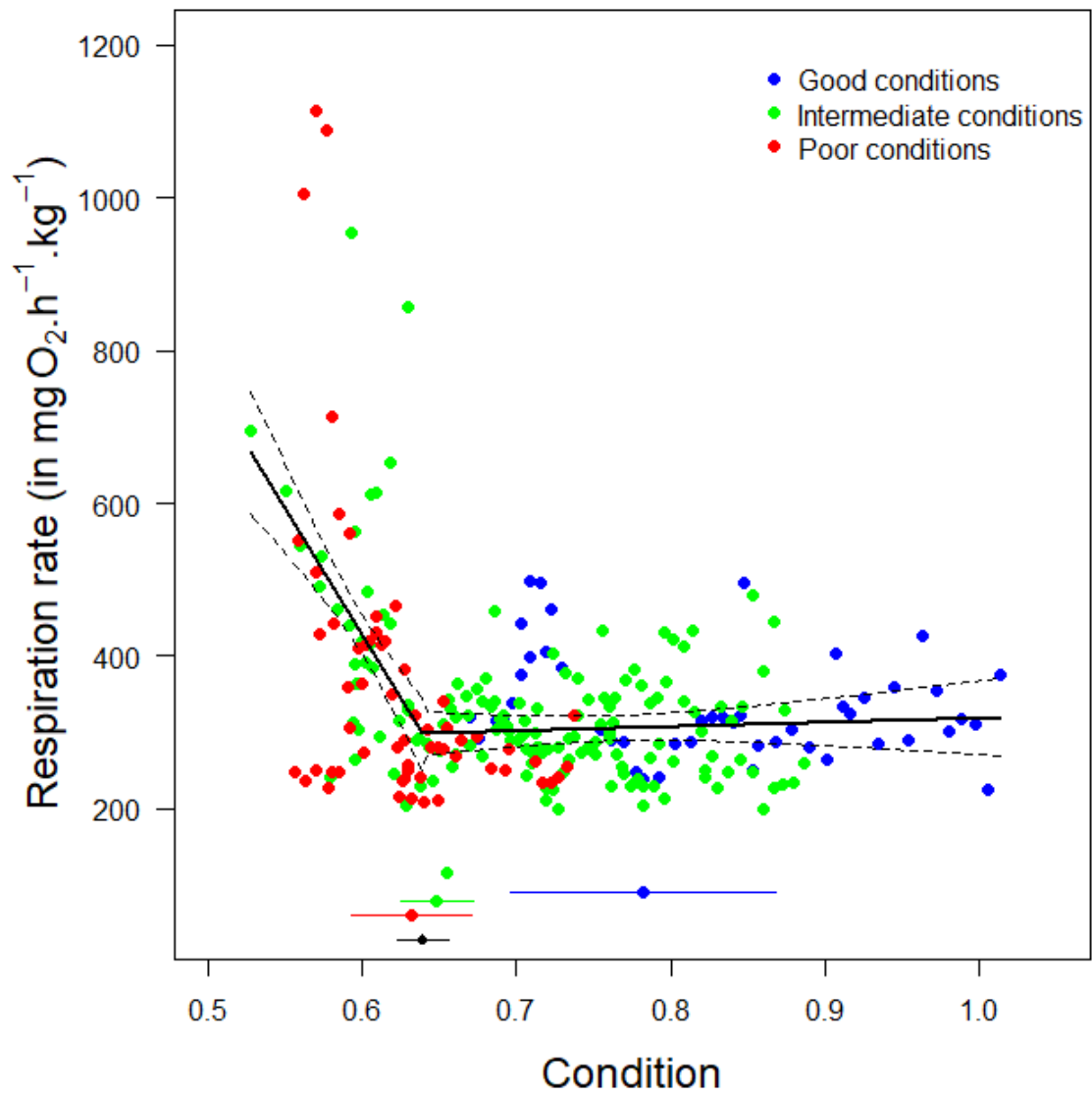
907 Figure 5

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