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Farmer monitoring reveals the effect of tidal height on mortality risk of oysters during a herpesvirus outbreak

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Abstract :

The intertidal zone is characterized by a sharp vertical gradient of environmental stress, which structures species distribution and their interactions. Few studies, however, have examined the influence of tidal height on host–pathogen interactions. Here, we investigated how the tidal height influence outbreak of the Ostreid herpesvirus type 1 (OsHV-1) affecting the Pacific oyster. A volunteer network composed of 20 oyster growers monitored the survival of 28 batches of oysters during an epizootic event in Southern Brittany, France. Oysters were spat from wild collection or hatchery production. The sampling sites were spread over a 150-km² area with a tidal height ranging from 0.98 to 2.90 m. Concomitantly, we followed survival of oyster spats in relation with OsHV-1 DNA detection at two sites and conducted risk analysis. We found that tidal height was associated with a lower risk of mortality. This effect was higher for hatchery than for wild oysters probably reflecting differences in health status. Our study opens perspectives for mitigation strategies based on tidal height and emphasizes the value of volunteer science in marine epidemiological studies.

Keywords : citizen science, marine epidemiology and health, OsHV-1, risk analysis.

32 **Introduction**

33 Since the mid-1970s, disease epidemics and mass mortalities have been occurring in marine
34 environments at a historically unprecedented rate (Harvell et al., 1999) with consequences for
35 fisheries and aquaculture (Lafferty et al., 2015). The risk of disease outbreak depends on
36 interactions between hosts, pathogens, and the environment, and any change in one or more
37 of these components may potentially increase or decrease this risk (Burge et al., 2014). The
38 intertidal zone is characterised by a vertical gradient of environmental stress with increasing
39 elevation which structure species distribution, zonation and community dynamics (Connell,
40 1972). This feature makes it an important model system for marine ecological studies.
41 Nevertheless, few studies have examined the influence of tidal height on host-pathogen
42 interaction (but see Burrell et al., 1984; Ben-Horin et al., 2013; Malek and Breitbart, 2016;
43 Malek and Byers, 2017). It is likely that host species that spend less time underwater are less
44 exposed to pathogens vectored by the seawater. Also, replication or proliferation of obligate
45 parasites that depend on the host cell machinery is probably altered because host metabolism
46 and growth are lowered (Widdows et al., 1979; Somero, 2002).

47 One of the most striking examples of recent disease emergence is the Pacific Oyster Mortality
48 Syndrome (POMS), killing up to 100% of the farmed oysters locally every year since 2008 in

49 France (AHAW, 2015; Barbosa Solomieu et al., 2015; Pernet et al., 2016; Alfaro et al., 2018).
50 Almost all French production areas were severely hit by POMS, thus resulting in a supply
51 shortage and a rise in prices (Le Bihan et al., in press). This syndrome is caused by the ostreid
52 herpesvirus 1 (OsHV-1) which alters the immune state of oysters and leads to fatal
53 bacteraemia and death (de Lorgeril et al., 2018). Epizootics caused by OsHV-1 occur every year
54 when seawater temperature is between 16°C and 24°C in France (Pernet et al., 2012; Renault
55 et al., 2014). Infection starts when viral particles come into contact with susceptible hosts via
56 suspension feeding. There is a threshold dose for infection and a dose-response effect of
57 OsHV-1 on mortality (Paul-Pont et al., 2015; Petton et al., in revision). Like other
58 herpesviruses, latent and asymptomatic OsHV-1 infections are able to persist in hosts (Segarra
59 et al., 2014). In this case, virus reactivation can occur several weeks to months after initial
60 exposure (Pernet et al., 2015; Petton et al., 2015), viral particles are shed into the water
61 column and dispersal to new hosts occurs via water currents (Pernet et al., 2012). The
62 economic costs associated with increased oyster mortalities have promoted investigation of
63 disease risk factors to improve management of shellfish farms, but the effect of tidal height
64 has rarely been investigated (Pernet et al., 2016).

65 A review of early experiments conducted in France during the first disease outbreaks suggest
66 that tidal height can be associated with lower mortality of oysters (Soletchnik et al., 2011).
67 This hypothesis has only been partially confirmed. High rearing height is related to reduced
68 mortality in adults but not in spats for which only a mortality delay occurs (Paul-Pont et al.,
69 2013; Whittington et al., 2015a). More recently, tidal height is associated with a lower risk of
70 mortality in only 9 out of 40 tested oyster spat families (Azema et al., 2017). Broad-scale
71 epidemiological studies reveal apparently conflicting results. A survey conducted in Irish

72 oyster farms suggests that emersion time is negatively associated with mortality (Peeler et al.,
73 2012) but another one carried out in Australia shows no relation (de Kantzow et al., 2017).
74 Mortality risk factors associated with OsHV-1 have recently been identified along an inshore-
75 offshore gradient in permanently immersed animals, but not in inshore farming areas where
76 animals are held at various tidal heights and are regularly emerged (Pernet et al., 2018). Here
77 we supplemented this epidemiological study focusing on the farming areas. The objective was
78 to investigate the effect of tidal height on mortality risk of oysters. An original aspect to the
79 present study is that survival of farmed oysters was monitored by a network of growers to
80 obtain simultaneous data from multiple remote sites. The results obtained by oyster growers
81 were strengthened by the Ifremer observatory network of oyster mortality. Although the roots
82 of citizen science go back to the very beginnings of modern science, projects in which
83 volunteers associate with scientists are new in the field of marine epidemiology (Foster-Smith
84 and Evans, 2003; Silvertown, 2009; Dickinson et al., 2010).

85

86 **Material and method**

87 *Study site*

88 The study sites covered a surface area of *ca.* 150 km² located in South Brittany, France, and
89 encompass six production areas located in inlets, rivers or gulfs where oysters were hit by
90 OsHV-1 every year since 2008 (Figure 1) (Fleury et al., 2018). Although the number of oyster
91 companies in this area has decreased by 25% since 2001 partly because of the virus, there
92 were 241 companies in 2012 that supported 522 full-time jobs (Agreste, 2014). The coastline
93 is heterogenous, alternating bedrock, sandy beaches and sandy-loamy to sandy-muddy tidal
94 flats. However, the large majority of oysters (70%) are cultivated off-bottom in mesh bags

95 attached to iron tables (Buestel et al., 2009). This area is influenced by semi-diurnal tides and
96 the tidal range was between 0.22 m and 5.88 m during the study period.

97

98 *Volunteer network*

99 The experimental design was set-up in collaboration with local oyster farmers. Oyster growers
100 were informed by the regional shellfish committee that participatory monitoring of large-scale
101 oyster mortalities was taking place and that volunteers were needed to survey their own
102 concessions. A meeting was held with all respondents in mid-April 2013 to outline the
103 monitoring protocol. Twenty farmers agreed to participate to the experiment. Farmers were
104 in charge of one (N=14 farmers), two (N=4) or three (N=2) batches of oysters for a total of 28
105 batches (#1-28). The sentinel oysters were chosen among the farmed stock by the farmers
106 themselves. To be included in the experimental design, the sentinel oysters needed be at the
107 spat stage and free of abnormal mortality since they arrived at the monitoring site. At the
108 onset of the monitoring, sentinel oysters were 10 to 25 mm shell length and they had spent
109 between 8 and 205 days on the farm (mean=70 ± 64 days). The sentinel oysters were deployed
110 after 15 October 2012 when seawater temperature was < 16°C, so that OsHV-1 infection at
111 the monitoring site was unlikely. They originated from hatcheries or from natural collection in
112 the wild. Oysters from hatcheries were not genetically selected for disease resistance. At the
113 time of this study, work on OsHV-1 oyster resistance in France was conducted on an
114 experimental scale only. In fact, some private hatcheries and the National Shellfish Committee
115 launched their own breeding programs in 2013-14 (Dégremont et al., 2015). Wild oysters were
116 settled on limed tiles or plastic tubes during the preceding summer and the spat were
117 removed during the fall or early winter according to standard practices (Heral, 1989). Wild
118 oysters generally spent more time on the farm before the start of monitoring than those from

119 hatcheries (116 vs. 40 d respectively). A subsample (n=100) of the farmed stock selected as
120 sentinels was transferred into small mesh bags (22×20×2 cm, Ø=6.0 mm) attached to iron
121 tables. Farmers provide exact positions of each oyster bag, the date of arrival on the rearing
122 site and the origin of the oysters (wild or hatchery). Bathymetric data were provided by the
123 Service Hydrographique et Océanographique de la Marine (SHOM). The survival monitoring
124 started on 8 May 2013, before the onset of the epizootics, and lasted for 133 d until 18
125 September 2013. Live and dead animals were counted twice a month by the farmer, generally
126 during spring tides (22 and 29 May, 12 and 26 June, 10 and 24 July, 8 and 22 August and 6 and
127 18 September).

128

129 *Observatory network*

130 The Ifremer observatory network followed the survival of two batches of oyster spats
131 originating from both wild or hatchery production at two sites located within the farming
132 areas (#29-32, Figure 1). Oysters consisted of (i) wild spat collected on limed tiles in Arcachon
133 Bay during summer 2012 or (ii) 3-month-old animals produced by a private hatchery. These
134 two batches of oysters were gathered at the Ifremer laboratory (La Trinité-sur-Mer, France)
135 between 10 and 26 March 2013. Before deployment in the field, a subsample of 50 oysters
136 from each batch were individually screened for OsHV-1 DNA by qPCR and exposed to a
137 thermal elevation at 21°C for one month in cohabitation with healthy spats to reveal both
138 disease expression and transmission (Petton et al., 2015). Wild oysters were asymptomatic
139 carriers of OsHV-1 as the virus was detected in 3 out of 50 individuals and mortalities
140 associated with OsHV-1 occurred in both the tested and the cohabited healthy oysters after
141 thermal elevation at 21°C (see table S1, batch n°42 in Petton et al., 2015). In contrast, oysters
142 from the hatchery were considered specific-pathogen-free because OsHV-1 DNA was not

143 detected and no mortality occurred after thermal elevation at 21°C (see table S1, batch n°40
144 in Petton et al., 2015).
145 These two batches of oysters were placed in three regular-sized mesh bags per site (350
146 individuals per bag). Live and dead animals were counted twice a month during spring tides
147 from April to December in one of the three bags. Only survival data collected at the same time
148 as the farmers were considered. Three pools of three live hatchery oysters were collected
149 from early May to mid-September in each bag and analyzed for the detection and
150 quantification of OsHV-1 DNA (one pool per bag) by LABOCEA (Quimper, France) using a
151 standard real-time PCR protocol (Pepin et al., 2008). Seawater temperature was measured
152 every 15 minutes using temperature probes placed in one oyster bag at each site.

153

154 *Statistics*

155 Nonparametric estimates of the survivor function were computed using the Kaplan–Meier
156 method (Kaplan and Meier, 1958). Survival time was measured as number of days after 8 May,
157 the onset of the monitoring period, until 18 September 2013. The data were read as the
158 number of dead animals within each bag at each time interval. Survival curves were presented
159 for each oyster bag as a function of rearing site and origin of oysters. A Frailty model
160 (Hougaard, 1995) was fitted to survival data to investigate the effect of tidal height, origin and
161 their interaction. The Frailty model is a proportional Cox regression model (Cox, 1972) with a
162 random effect (site). Then, survival probabilities predicted by the model were plotted for
163 every set of significant covariates or their interactions. These analyses were conducted with
164 the LIFETEST and PHREG procedures of the SAS software package (SAS 9.4, SAS Institute).

165

166 **Results**

167 Survival was measured on 32 wild (n=20 batches) or hatchery (n=12 batches) oyster batches,
168 spread over 22 sites within 6 production areas. Tidal height for these sites ranges from 0.98
169 to 2.90 m (mean \pm SD=1.94 \pm 0.67 m), corresponding to an immersion time varying between
170 59.3 to 96.9% (mean \pm SD=78.9 \pm 13.3%, Figure 2).

171 The oysters were hit by the mass mortality event at all sites (Figure 3). Mortality was first
172 observed 35 to 49 days after the onset of the monitoring (i.e. 12 to 26 June) for 20 batches
173 out of 32. Survival functions of oysters differed depending on their origin (Figure 2, log-rank
174 test, $\chi^2=20.0$, $p<0.001$). Indeed, mortality occurred earlier for wild than for hatchery oysters.
175 Final survival varied widely from 20 to 97 % (mean=51.0 \pm 20.4%) and it was similar between
176 origin (wild: 49.5% vs. hatchery: 53.5%, $\chi^2=1.8$, $p=0.187$). The observatory network revealed
177 that these mortalities occurred while seawater temperature exceeded 16°C and coincided
178 with the detection of high levels of OshV-1 DNA in oyster tissues ($>10^6$ cp mg g⁻¹ wet tissue,
179 Figure 4).

180 The relationship between the mortality risk and site, tidal height and origin of oysters was
181 investigated using the Cox regression model (Table 1). Oyster origin and tidal height exerted
182 a major influence on survival. Tidal height was associated with a lower mortality risk, with a
183 greater effect on oysters originating from hatcheries than for those from the wild (Table 1).
184 Any 1 m increase in tidal height led to a 48.8% reduction in mortality hazard for hatchery
185 oysters compared to only 15.7% for wild oysters. Hazard ratios were 0.512 (95% confidence
186 interval [CI]=0.452-0.580) and 0.843 (0.764-0.930) respectively. Survival probabilities
187 predicted by the model at the end of the monitoring of hatchery oysters held at 0, 0.98, 1.95
188 and 2.90 m were respectively 19%, 40%, 61% and 76%, compared to 42%, 44%, 47% and 50%
189 for wild oysters (Figure 5A). Therefore, tidal height had more influence on the survival of
190 hatchery oysters than on wild oysters. The mortality risk was the same for both wild and

191 hatchery oysters when tidal height was 1.2 m (hazard ratio=0.999, 95%CI [0.878-1.111]),
192 corresponding to an average immersion time of 94.6% (Figure 5B). For tidal heights above 1.2
193 m, any increase reduced the risk of wild oyster mortality to a lesser extent than that of
194 hatchery oysters, which meant that the survival of wild oysters was lower than that of
195 hatchery oysters. Conversely, at tidal height below 1.2 m, any decrease increased the risk of
196 hatchery oyster mortality to a greater extent than that of wild ones, conferring a higher
197 survival on hatchery compared to wild oysters.

198

199 **Discussion**

200 Here we validate the hypothesis that increasing tidal height decreases the risk of oyster
201 mortality during an OsHV-1 epizootic, as indicated for adult oysters in Australia and some spat
202 families in France (Paul-Pont et al., 2013; Whittington et al., 2015a; Azema et al., 2017).
203 Improved survival with increasing tidal height can be explained by a shorter immersion time,
204 leading to a lower exposure to viral particles in the water. Indeed, survival of healthy oysters
205 in laboratory conditions decreases with cohabitation time with infected oysters and reflects
206 the cumulative exposure to OsHV-1 (Petton et al., in revision). In line with this, survival of
207 oysters decreases with biomass of infected oysters and increases with seawater renewal, two
208 parameters that influence pathogen concentrations (Petton et al., 2015). Also, survival of
209 oysters injected with OsHV-1 decrease with increasing viral particle concentration (Paul-Pont
210 et al., 2015; Segarra et al., 2016). Therefore, oysters that spend less time immersed will be
211 less exposed to OsHV-1 particles vectored by seawater and show higher survival.

212 In addition, since herpesviruses replication is directly dependent on host cell activity, it may
213 be reduced during emersion as metabolism and growth of the host decline (Widdows et al.,
214 1979; Somero, 2002). For instance, virus replication in shrimp depends on cellular growth and

215 proliferation of the host (Su et al., 2014). Further to this, fast-growing oysters are more
216 susceptible to OsHV-1 is than slow-growers (Pernet et al. unpublished data).

217 Finally, differences in temperature regime between low and high tidal levels could also
218 contribute to differences in survival of oysters exposed to OsHV-1. Indeed, during daytime
219 exposure at low tide, the body temperature of a shelled mollusks can rapidly rise from that of
220 the ambient seawater to 15°C above air temperature (e. g. Helmuth, 1999). In parallel, high
221 temperature (>26°C) decreased the susceptibility of oysters to OsHV-1 (Petton et al., 2013; de
222 Kantzow et al., 2016; Delisle et al., 2018). It is possible that longer exposure to higher air
223 temperature at low tide makes oysters less susceptible to the virus at high tidal level. Lower
224 virus exposure, reduction of metabolic rate and exposure to high air temperature during
225 emersion are not mutually exclusive hypotheses, and further studies are necessary to identify
226 which mechanism prevails.

227 Interestingly, we found that the mortality risk of wild oysters was generally less influenced
228 by the tidal height than that of hatchery ones. A likely and logical hypothesis relate to
229 differences in their initial health status. Indeed, wild oysters used in the observatory network
230 were asymptomatic carriers of the virus whereas hatchery oysters were free of OsHV-1. This
231 observation is in line with a major study showing that 60% of the wild oyster batches tested
232 in France are asymptomatic carriers of OsHV-1 during their first winter, exhibiting mortality
233 after thermal elevation and infecting cohabiting healthy hatchery oysters, whereas those from
234 hatcheries and nurseries are free of OsHV-1 (Petton et al., 2015). Indeed, wild oysters are
235 unpredictably exposed to the pathogen during their early life history whereas those from the
236 hatcheries are generally protected from disease by means of prophylactic measures (Petton
237 et al., 2015; Whittington et al., 2015b). It is therefore highly likely that a majority of wild oyster

238 batches used in the volunteer network were initially asymptomatic carriers of the disease
239 while hatchery oysters were free of OsHV-1.

240 Lower influence of tidal height on the mortality risk in wild oysters compared to that of
241 hatchery oysters could also be explained by the fact that (i) tidal height decreased the risk of
242 oyster mortality by reducing viral exposure during emersion and (ii) the mortality risk of wild
243 animals, which were presumably asymptomatic carriers of the virus, was less dependent on
244 external sources of virus particles than that of hatchery oysters which were presumably free
245 of OsHV-1. Indeed, mortality risk of oysters is associated with the cumulative exposure to
246 OsHV-1 (Petton et al., in revision) and the reactivation of the virus in asymptomatic carriers
247 does not require additional exposure to the pathogen; a thermal elevation beyond the
248 permissivity threshold (16°C in Europe) is sufficient (Pernet et al., 2015; Petton et al., 2015).

249 More particularly, survival of hatchery oysters was higher than that of wild oysters at tidal
250 heights >1.2 m (immersion time <89.6%) but for tidal heights <1.2 m the opposite was true.
251 At tidal heights >1.2 m, regular emersion may have partially protected hatchery oysters from
252 pathogens, whereas wild oysters most likely died because the virus reactivated when
253 seawater temperature increased to 16°C. Conversely, at lower tidal levels, hatchery oysters
254 were at higher risk of mortality because the presumed protection from emersion was
255 decreased, and their susceptibility to disease was greater than that of wild oysters. As wild
256 oysters are more likely to be exposed to the pathogen than their hatchery counterparts, they
257 are also more likely to be selected for greater resistance to the disease (Dégremont, 2011;
258 Pernet et al., 2012). It is possible, however, that confounding effects other than health status
259 were associated with oyster origins and contributed to the interactive effect of tidal height
260 and the origin of oysters.

261 We also found that the onset of mortality in wild oysters occurred earlier than in hatchery
262 oysters. Similarly, OsHV-1 outbreaks are first manifest in wild oysters followed by those from
263 hatcheries and nurseries (Pernet et al., 2010; Degremont and Benabdelmouna, 2014). This
264 likely reflects the reactivation of the virus in wild oysters (asymptomatic carriers) as soon as
265 the seawater temperature reached 16°C, thus leading to an earlier death of the host. In
266 hatchery oysters (presumably free of OsHV-1), mortality would appear not only above a
267 certain temperature threshold, but after exposure to the virus and infection. Therefore, the
268 lag between mortality outbreaks of asymptomatic carriers and healthy oysters probably
269 corresponds to the time required for disease transmission, infection and expression once
270 environmental conditions have become permissive.

271 Here we showed a beneficial effect of tidal height on oyster survival during an OsHV-1
272 epizootic. However, increasing tidal height is not always associated with higher oyster survival
273 (Paul-Pont et al., 2013; Whittington et al., 2015a). Indeed, tidal height delays the onset of spat
274 mortality without increasing final survival (Whittington et al., 2015a). Although there is no
275 apparent reason for delaying mortality if there is no survival gain at the end, this is a first step
276 towards increasing survival and restoring the health of ecosystems. Therefore, studies to
277 define the risk factors for disease management should consider the survival dynamics rather
278 than the end-point.

279 We therefore propose that farmers may mitigate oyster mortality by temporarily raising
280 the culture height during the OsHV-1 mortality period, when seawater temperature is above
281 15°C. Oyster mortality mitigation strategies based on tidal height have already been suggested
282 (Peeler et al., 2012; Paul-Pont et al., 2013; Whittington et al., 2015a). Such a practice would
283 be particularly relevant for oysters under one year old which are the most susceptible to the
284 virus, and for oysters that are initially free of OsHV-1. However, to limit stunting due to the

285 reduced feeding time that comes with increased emersion, farmers may consider lowering
286 the culture height during the second summer, once oysters have become more resistant to
287 the virus.

288 In line with this, rearing height of oyster spat in Australia was changed in response to OsHV-
289 1 on 47% of leases, and 26% of these observed higher mortality when oysters were held low
290 in the intertidal zone (Ugalde et al., 2018). Yet, farmers consider this factor to be moderately
291 important in limiting the losses caused by the virus. Also, a survey of 93 growers spread over
292 two French farming areas (including the region covered by this study) conducted in 2014
293 reveals that 75% of them think that changing the rearing height is not an effective measure
294 for controlling oyster mortalities caused by the virus (Le Bihan, Lupo and Pernet, unpublished
295 data). This probably reflects that the effect of tidal height on oyster mortality is not
296 straightforward as it interacts with the origin of oysters, and more particularly with their initial
297 health status (OsHV-1-free or asymptomatic carrier, this study) or their genetic make-up
298 (Azema et al., 2017). These interactions could skew the point of view of the growers.

299 Finally, we must evaluate the economic efficiency of such a measure. Does the survival gain
300 compensate for reduced growth and increased handling? Then, we must assess acceptability,
301 that is the willingness of farmers to put into action disease control measures. In France, oyster
302 leases at high tide are usually dedicated to storing commercial oysters to limit their growth
303 until they are shipped to markets. As a result, increasing rearing height of oyster spat could
304 create an unwanted competition for space on the upper foreshore.

305 This study is based on the observation of a large number of oyster batches spread over the
306 entire farming area at different tidal heights and over several sampling periods. This effort
307 was made possible by the establishment of a volunteer network, thus highlighting its
308 usefulness and power in marine epidemiology studies. In comparison, conventional

309 experimental approaches are generally conducted on a more local scale and involve
310 comparing final survival at two or three tidal heights in a restricted number of batches.
311 However, associating observed mortalities with OsHV-1 was possible owing to the
312 simultaneous presence of a long-term oyster mortality monitoring network with a scientific
313 experiment carried out in the same location. Further hypothesis-driven controlled
314 experiments are needed to elucidate the mechanism of increased disease resistance with tidal
315 height. Although citizen science has proven useful for marine epidemiology, more local
316 hypothesis-driven research remains essential to uncover mechanisms underlying ecological
317 patterns (Dickinson et al., 2010).

318

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325

326 **References**

327 Agreste 2014. Chute de l'huître en Bretagne- Sud, progression de la moule en Bretagne-
328 Nord. *In* Recensement de la conchyliculture 2012. Ed. by Direction Régionale de
329 l'Alimentation, de l'Agriculture et de la Forêt – Service régional de l'information
330 statistique et économique.

331 AHAW, E. P. A. H. W. 2015. Oyster mortality EFSA Panel on Animal Health and Welfare
332 (AHAW). EFSA Journal, 13.

333 Alfaro, A. C., Nguyen, T. V., and Merien, F. 2018. The complex interactions of Ostreid
334 herpesvirus 1, Vibrio bacteria, environment and host factors in mass mortality
335 outbreaks of *Crassostrea gigas*. *Reviews in Aquaculture*.

336 Azema, P., Maurouard, E., Lamy, J. B., and Degremont, L. 2017. The use of size and growing
337 height to improve *Crassostrea gigas* farming and breeding techniques against OsHV-
338 1. *Aquaculture*, 471: 121-129.

339 Barbosa Solomieu, V., Renault, T., and Travers, M.-A. 2015. Mass mortality in bivalves and
340 the intricate case of the Pacific oyster, *Crassostrea gigas*. *Journal of Invertebrate*
341 *Pathology*, 131: 2-10.

342 Ben-Horin, T., Lenihan, H. S., and Lafferty, K. D. 2013. Variable intertidal temperature
343 explains why disease endangers black abalone. *Ecology*, 94: 161-168.

344 Buestel, D., Ropert, M., Prou, J., and Gouilletquer, P. 2009. History, status, and future of
345 oyster culture in France. *Journal of Shellfish Research*, 28: 813-820.

346 Burge, C. A., Mark Eakin, C., Friedman, C. S., Froelich, B., Hershberger, P. K., Hofmann, E. E.,
347 Petes, L. E., et al. 2014. Climate Change Influences on Marine Infectious Diseases:
348 Implications for Management and Society. *Annual Review of Marine Science*, 6: 249-
349 277.

350 Burrell, V. G., Bobo, M. Y., and Manzi, J. J. 1984. A comparison of seasonal incidence and
351 intensity of *perkinsus marinus* between subtidal and intertidal oyster populations in
352 South Carolina. *Journal of the World Mariculture Society*, 15: 301-309.

353 Connell, J. H. 1972. Community Interactions on Marine Rocky Intertidal Shores. *Annual*
354 *Review of Ecology and Systematics*, 3: 169-192.

355 Cox, D. R. 1972. Regression Models and Life Tables. *Journal of the Royal Statistical Society:*
356 *Series B (Statistical Methodology)*, 20: 187-220.

357 de Kantzow, M., Hick, P., Becker, J. A., and Whittington, R. J. 2016. Effect of water
358 temperature on mortality of Pacific oysters *Crassostrea gigas* associated with
359 microvariant ostreid herpesvirus 1 (OsHV-1 μ Var). *Aquaculture Environment*
360 *Interactions*, 8: 419-428.

361 de Kantzow, M. C., Hick, P. M., Dhand, N. K., and Whittington, R. J. 2017. Risk factors for
362 mortality during the first occurrence of Pacific Oyster Mortality Syndrome due to
363 Ostreid herpesvirus–1 in Tasmania, 2016. *Aquaculture*, 468: 328-336.

364 de Lorgeril, J., Lucasson, A., Petton, B., Toulza, E., Montagnani, C., Clerissi, C., Vidal-Dupiol, J.,
365 et al. 2018. Immune-suppression by OsHV-1 viral infection causes fatal bacteraemia
366 in Pacific oysters. *Nature Communications*, 9: 4215.

367 Dégremont, L. 2011. Evidence of herpesvirus (OsHV-1) resistance in juvenile *Crassostrea*
368 *gigas* selected for high resistance to the summer mortality phenomenon.
369 *Aquaculture*, 317: 94-98.

370 Degremont, L., and Benabdelmouna, A. 2014. Mortality associated with OsHV-1 in spat
371 *Crassostrea gigas*: role of wild-caught spat in the horizontal transmission of the
372 disease. *Aquaculture International*: 1-15.

373 Dégremont, L., Garcia, C., and Allen Jr, S. K. 2015. Genetic improvement for disease
374 resistance in oysters: A review. *Journal of Invertebrate Pathology*, 131: 226-241.

375 Delisle, L., Petton, B., Burguin, J. F., Morga, B., Corporeau, C., and Pernet, F. 2018.
376 Temperature modulate disease susceptibility of the Pacific oyster *Crassostrea gigas*
377 and virulence of the Ostreid herpesvirus type 1. *Fish & Shellfish Immunology*, 80: 71-
378 79.

379 Dickinson, J. L., Zuckerberg, B., and Bonter, D. N. 2010. Citizen Science as an Ecological
380 Research Tool: Challenges and Benefits. *Annual Review of Ecology, Evolution, and*
381 *Systematics*, 41: 149-172.

382 Fleury, E., Normand, J., Lamoureux, A., Bouget, J.-F., Lupo, C., Cochennec-Laureau, N.,
383 Petton, S., et al. 2018. RESCO REMORA Database : National monitoring network of
384 mortality and growth rates of the sentinel oyster *Crassostrea gigas*. SEANOE.

385 Foster-Smith, J., and Evans, S. M. 2003. The value of marine ecological data collected by
386 volunteers. *Biological Conservation*, 113: 199-213.

387 Harvell, C. D., Kim, K., Burkholder, J. M., Colwell, R. R., Epstein, P. R., Grimes, D. J., Hofmann,
388 E. E., et al. 1999. Review: Marine ecology - Emerging marine diseases - Climate links
389 and anthropogenic factors. *Science*, 285: 1505-1510.

390 Helmuth, B. 1999. Thermal biology of rocky intertidal mussels: Quantifying body
391 temperatures using climatological data. *Ecology*, 80: 15-34.

392 Heral, M. 1989. Traditional oyster culture in France. Barnabe Aquaculture, Ellis Horwood.

393 Hougaard, P. 1995. Frailty models for survival data. *Lifetime Data Analysis*, 1: 255-273.

394 Kaplan, E. L., and Meier, P. 1958. Nonparametric Estimation from Incomplete Observations.
395 *Journal of the American Statistical Association*, 53: 457-481.

396 Lafferty, K. D., Harvell, C. D., Conrad, J. M., Friedman, C. S., Kent, M. L., Kuris, A. M., Powell,
397 E. N., et al. 2015. Infectious diseases affect marine fisheries and aquaculture
398 economics. *Annual Review of Marine Science*, 7: 471-496.

399 Le Bihan, V., Catalo, M., and Le Bihan, J. in press. Reorganization of the value chain activities
400 of oyster companies on the Atlantic coast following health crises in France (2006–
401 2013). *Marine Policy*.

402 Malek, J. C., and Breitburg, D. L. 2016. Effects of air-exposure gradients on spatial infection
403 patterns of *Perkinsus marinus* in the eastern oyster *Crassostrea virginica*. *Diseases of*
404 *Aquatic Organisms*, 118: 139-151.

405 Malek, J. C., and Byers, J. E. 2017. The effects of tidal elevation on parasite heterogeneity
406 and co-infection in the eastern oyster, *Crassostrea virginica*. *Journal of Experimental*
407 *Marine Biology and Ecology*, 494: 32-37.

408 Paul-Pont, I., Dhand, N. K., and Whittington, R. J. 2013. Influence of husbandry practices on
409 OsHV-1 associated mortality of Pacific oysters *Crassostrea gigas*. *Aquaculture*, 412–
410 413: 202-214.

411 Paul-Pont, I., Evans, O., Dhand, N. K., and Whittington, R. J. 2015. Experimental infections of
412 Pacific oyster *Crassostrea gigas* using the Australian ostreid herpesvirus-1 (OsHV-1)
413 μ Var strain. *Diseases of Aquatic Organisms*, 113: 137-147.

414 Peeler, E. J., Reese, R. A., Cheslett, D. L., Geoghegan, F., Power, A., and Thrush, M. A. 2012.
415 Investigation of mortality in Pacific oysters associated with Ostreid herpesvirus-1
416 μ Var in the Republic of Ireland in 2009. *Preventive Veterinary Medicine*, 105: 136-
417 143.

418 Pepin, J. F., Riou, A., and Renault, T. 2008. Rapid and sensitive detection of ostreid
419 herpesvirus 1 in oyster samples by real-time PCR. *Journal of Virological Methods*,
420 149: 269-276.

421 Pernet, F., Barret, J., Gall, P. L., Corporeau, C., Dégremont, L., Lagarde, F., Pépin, J.-F., et al.
422 2012. Mass mortalities of Pacific oysters *Crassostrea gigas* reflect infectious diseases
423 and vary with farming practises in the Thau lagoon. *Aquaculture Environment*
424 *Interactions*, 2: 215–237.

425 Pernet, F., Barret, J., Marty, C., Moal, J., Le Gall, P., and Boudry, P. 2010. Environmental
426 anomalies, energetic reserves and fatty acid modifications in oysters coincide with an
427 exceptional mortality event. *Marine Ecology-Progress Series*, 401: 129-146.

428 Pernet, F., Fuhrmann, M., Petton, B., Mazurié, J., Bouget, J.-F., Fleury, E., Daigle, G., et al.
429 2018. Determination of risk factors for herpesvirus outbreak in oysters using a broad-
430 scale spatial epidemiology framework. *Scientific Reports*, 8.

431 Pernet, F., Lupo, C., Bacher, C., and Whittington, R. J. 2016. Infectious diseases in oyster
432 aquaculture require a new integrated approach. *Philosophical Transactions of the*
433 *Royal Society of London. Series B: Biological Sciences*, 371: 20150213.

434 Pernet, F., Tamayo, D., and Petton, B. 2015. Influence of low temperatures on the survival of
435 the Pacific oyster (*Crassostrea gigas*) infected with ostreid herpes virus type 1.
436 *Aquaculture*, 445: 57-62.

437 Petton, B., Boudry, P., Alunno-Bruscia, M., and Pernet, F. 2015. Factors influencing disease-
438 induced mortality of Pacific oysters *Crassostrea gigas*. *Aquaculture Environment*
439 *Interactions*, 6: 205-222.

440 Petton, B., de Lorgeril, J., Mitta, G., Daigle, G., Pernet, F., and Alunno Bruscia, M. in revision.
441 Fine-scale temporal dynamics of a polymicrobial infection in the Pacific oyster
442 (*Crassostrea gigas*) using an ecologically-realistic experimental pathosystem. *Disease*
443 *of Aquatic Organism*.

444 Petton, B., Pernet, F., Robert, R., and Boudry, P. 2013. Temperature influence on pathogen
445 transmission and subsequent mortalities in juvenile Pacific oysters *Crassostrea gigas*.
446 *Aquaculture Environment Interactions*, 3: 257-273.

447 Renault, T., Bouquet, A. L., Maurice, J.-T., Lupo, C., and Blachier, P. 2014. Ostreid herpesvirus
448 1 infection among Pacific oyster (*Crassostrea gigas*) spat: relevance of water
449 temperature to virus replication and circulation prior to the onset of mortality.
450 *Applied and Environmental Microbiology*, 80: 5419-5426.

451 Segarra, A., Baillon, L., Faury, N., Tourbiez, D., and Renault, T. 2016. Detection and
452 distribution of ostreid herpesvirus 1 in experimentally infected Pacific oyster spat.
453 *Journal of Invertebrate Pathology*, 133: 59-65.

454 Segarra, A., Baillon, L., Tourbiez, D., Benabdelmouna, A., Faury, N., Bourgougnon, N., and
455 Renault, T. 2014. Ostreid herpesvirus type 1 replication and host response in adult
456 Pacific oysters, *Crassostrea gigas*. *Veterinary Research*, 45: 1-10.

457 Silvertown, J. 2009. A new dawn for citizen science. *Trends in Ecology & Evolution*, 24: 467-
458 471.

459 Soletchnik, P., Mazurie, J., Pernet, F., Benabdelmouna, A., Bouquet, A. L., Petton, B., Blin, J.
460 L., et al. 2011. Les pratiques culturales peuvent-elles permettre de réduire la
461 surmortalité du naissain d'huîtres creuses? Récapitulatif des essais d'élevage et
462 expérimentations zootechniques menés sur le territoire français entre 2008 et 2010.

463 Somero, G. N. 2002. Thermal Physiology and Vertical Zonation of Intertidal Animals: Optima,
464 Limits, and Costs of Living. *Integrative and Comparative Biology*, 42: 780-789.

465 Su, M.-A., Huang, Y.-T., Chen, I. T., Lee, D.-Y., Hsieh, Y.-C., Li, C.-Y., Ng, T. H., et al. 2014. An
466 Invertebrate Warburg Effect: A Shrimp Virus Achieves Successful Replication by
467 Altering the Host Metabolome via the PI3K-Akt-mTOR Pathway. *PLOS Pathogens*, 10:
468 e1004196.

469 Ugalde, S. C., Preston, J., Ogier, E., and Crawford, C. 2018. Analysis of farm management
470 strategies following herpesvirus (OsHV-1) disease outbreaks in Pacific oysters in
471 Tasmania, Australia. *Aquaculture*, 495: 179-186.

472 Whittington, R. J., Dhand, N. K., Evans, O., and Paul-Pont, I. 2015a. Further observations on
473 the influence of husbandry practices on OsHV-1 μ Var mortality in Pacific oysters
474 *Crassostrea gigas*: Age, cultivation structures and growing height. *Aquaculture*, 438:
475 82-97.

476 Whittington, R. J., Hick, P. M., Evans, O., Rubio, A., Alford, B., Dhand, N., and Paul-Pont, I.
477 2015b. Protection of Pacific oyster (*Crassostrea gigas*) spat from mortality due to
478 ostreid herpesvirus 1 (OsHV-1 μ Var) using simple treatments of incoming seawater in
479 land-based upwellers. *Aquaculture*, 437: 10-20.

480 Widdows, J., Bayne, B., Livingstone, D., Newell, R., and Donkin, P. 1979. Physiological and
481 biochemical responses of bivalve mollusks to exposure to air. *Comparative*
482 *Biochemistry and Physiology a-Physiology*, 62: 301-308.

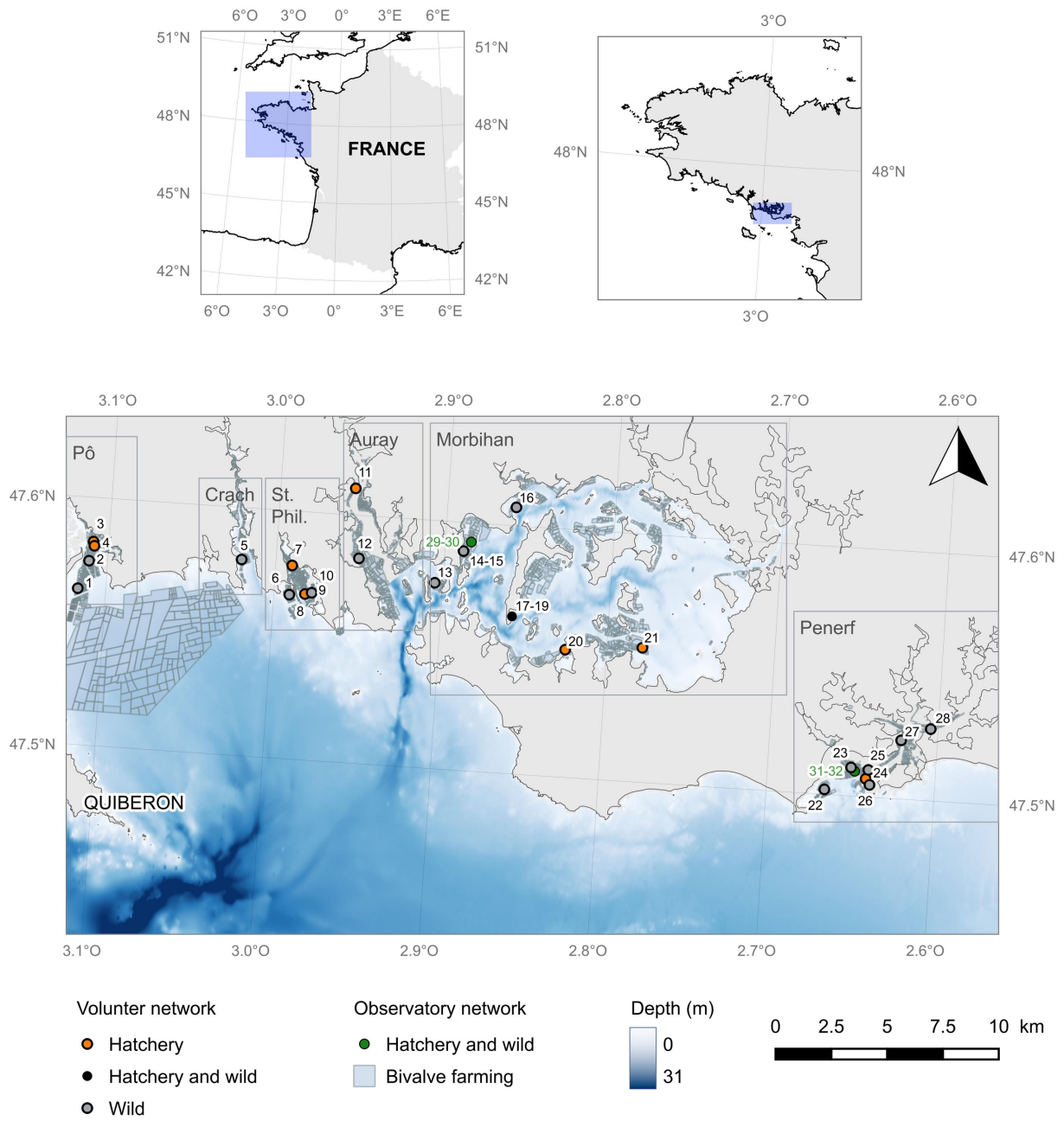
483

484 **Table 1.** Model parameter estimates from the Cox regression model. The columns display
 485 the parameter name, the degrees of freedom that are associated with the parameter (df),
 486 the estimated parameter value, the standard error of the parameter estimate (SE), the Wald
 487 chi-square statistic, and the associated p-value for testing the significance of the parameter.

Parameter	Level	df	Estimate	SE	χ^2	P
Origin	Hatchery	1	0.597	0.127	21.9	<0.0001
Tidal height	–	1	-0.171	0.050	11.5	0.0007
Tidal height × Origin	Hatchery	1	-0.498	0.075	44.0	<0.0001
Site	–	–	–	–	58.5	<0.0001

488

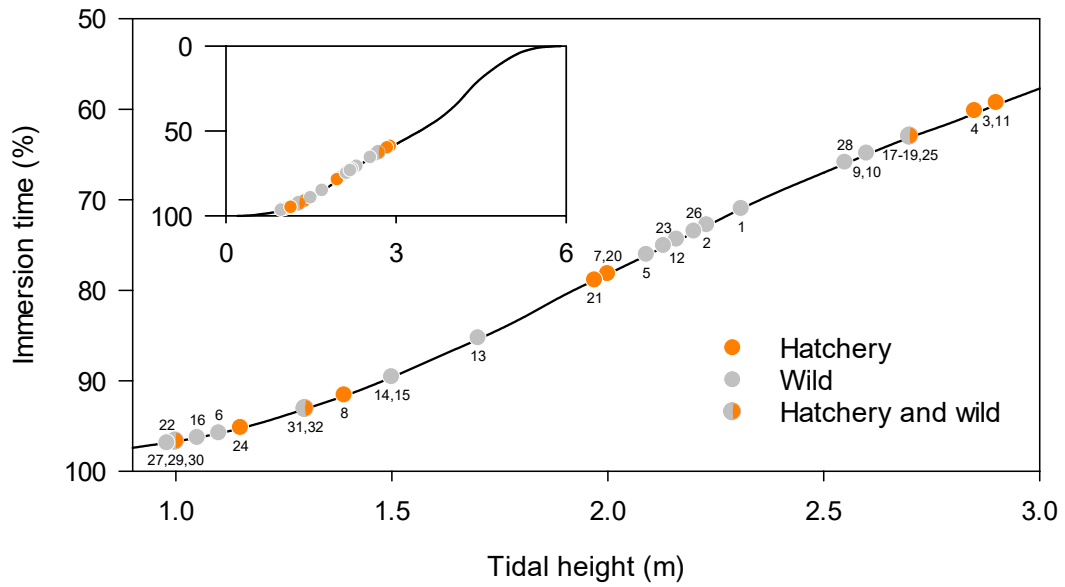
489 **Figure caption**



490

491 **Figure 1.** Spatial distribution of oyster survival monitoring sites.

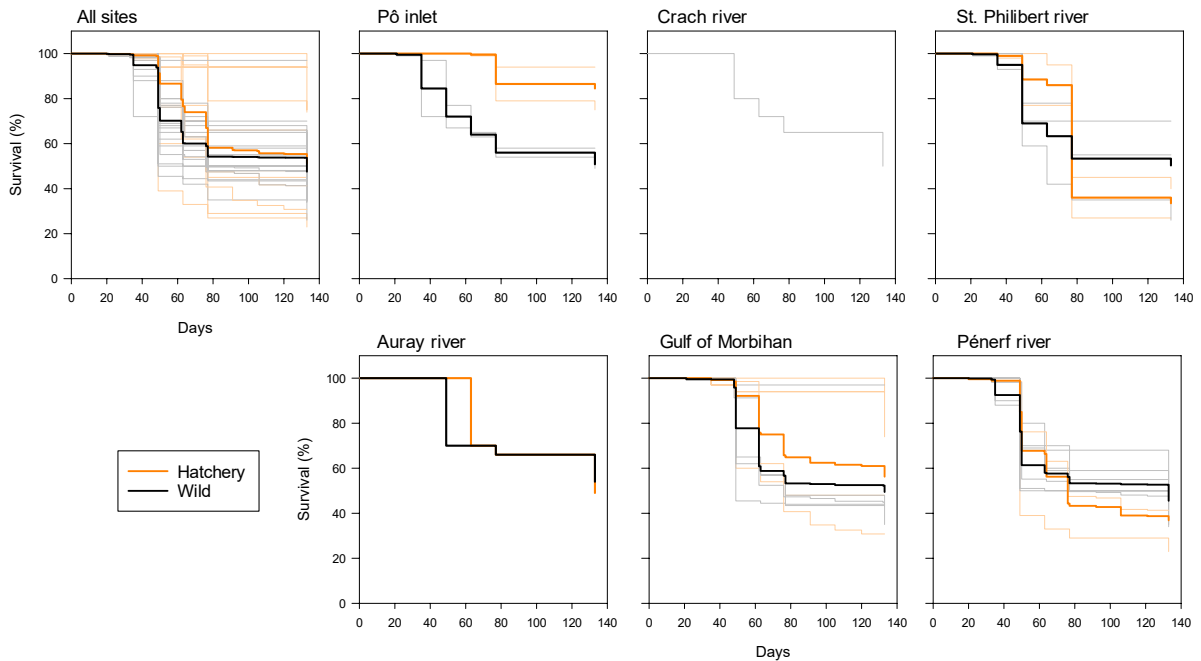
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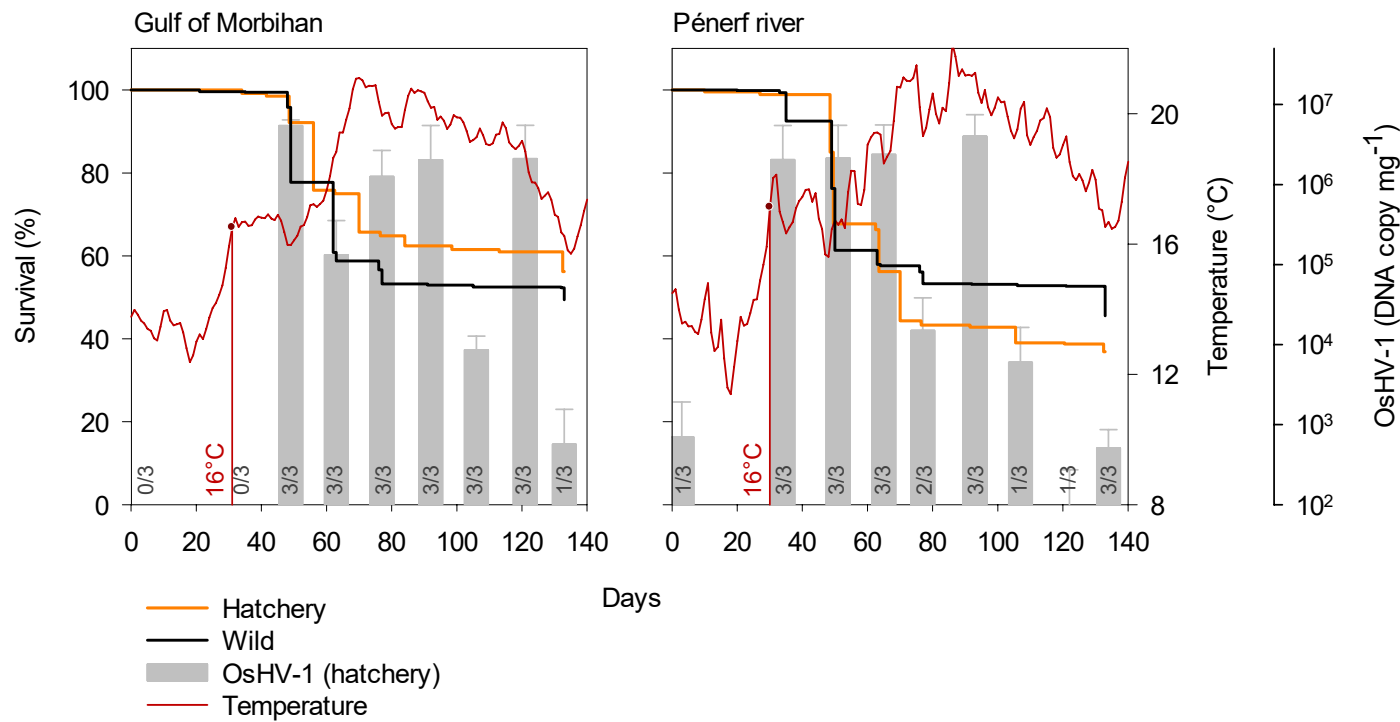
494 **Figure 2.** Relationship between immersion time and bathymetry. Circles represent the
 495 position of the oyster survival monitoring sites. Inset shows the entire bathymetric range
 496 during the period of study.

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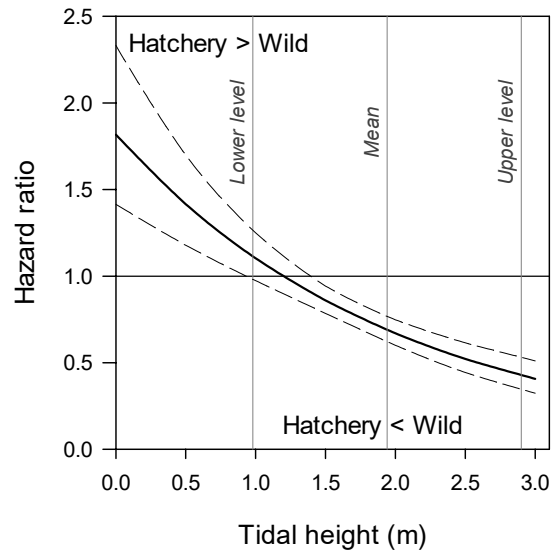
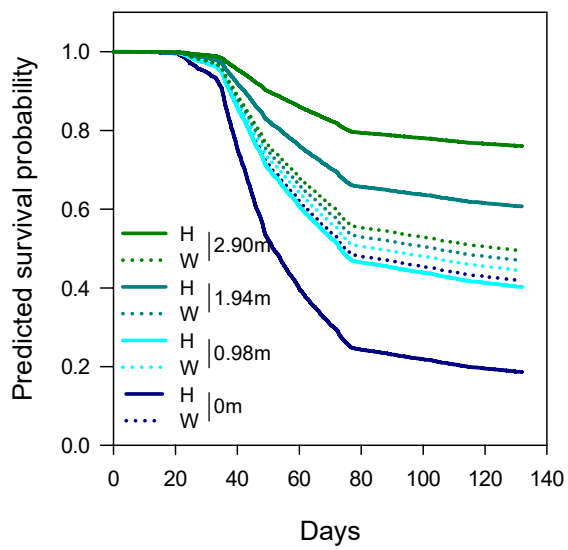
499 **Figure 3.** Survival of oysters. Thick lines represent the average survival per origin. Day 0
 500 corresponds to 8 May 2013.



501

502 **Figure 4.** Oyster survival in two sites from the Ifremer observatory network in relation with
 503 seawater temperature and OsHV-1 DNA detection. Left axis: survival functions of wild and
 504 hatchery oysters. Right axis: evolution of seawater temperature and levels of OsHV-1 DNA in
 505 oyster tissues collected in hatchery oysters (mean \pm SD, n=3 pools of 3 individuals). Ratios
 506 indicate the number of positive samples out of the total number analysed. Day 0 correspond
 507 to 8 May 2013.

508



509

510 **Figure 5.** Predicted survival probability curves of oysters as a function of tidal height and origin
 511 of oysters (A). Hazard ratio of hatchery vs. wild oysters as a function of tidal height (B). Day 0
 512 corresponds to 8 May 2013. Tidal height of 0 m represents permanent immersion whereas
 513 0.98, 1.94 m and 2.9 m were the lowest, the average and the highest level recorded in our
 514 study. Values predicted at tidal height < 0.98 m are extrapolations.