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

Pernet Fabrice <sup>1,\*</sup>, Gachelin Sonia <sup>2</sup>, Stanisière Jean-Yves <sup>3</sup>, Petton Bruno <sup>1</sup>, Fleury Elodie <sup>1</sup>, Mazurié Joseph <sup>3</sup>

<sup>1</sup> Laboratoire des Sciences de l'Environnement Marin (LEMAR), UMR 6539 UBO/CNRS/IRD/Ifremer, CS 10070, Ifremer, Plouzané, France

<sup>2</sup> Comité régional de la conchyliculture de Bretagne Sud, ZA Porte Océane, 7 rue du Danemark, Auray, France

<sup>3</sup> Unité Littorale, Laboratoire Environnement Ressource du Morbihan Pays-de-la-Loire, Ifremer, 12 Rue des Résistants, La Trinité-sur-Mer, France

\* Corresponding author : Fabrice Pernet, email address : [Fabrice.Pernet@ifremer.fr](mailto:Fabrice.Pernet@ifremer.fr)

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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-km<sup>2</sup> 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 km<sup>2</sup> 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<sup>-1</sup> 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

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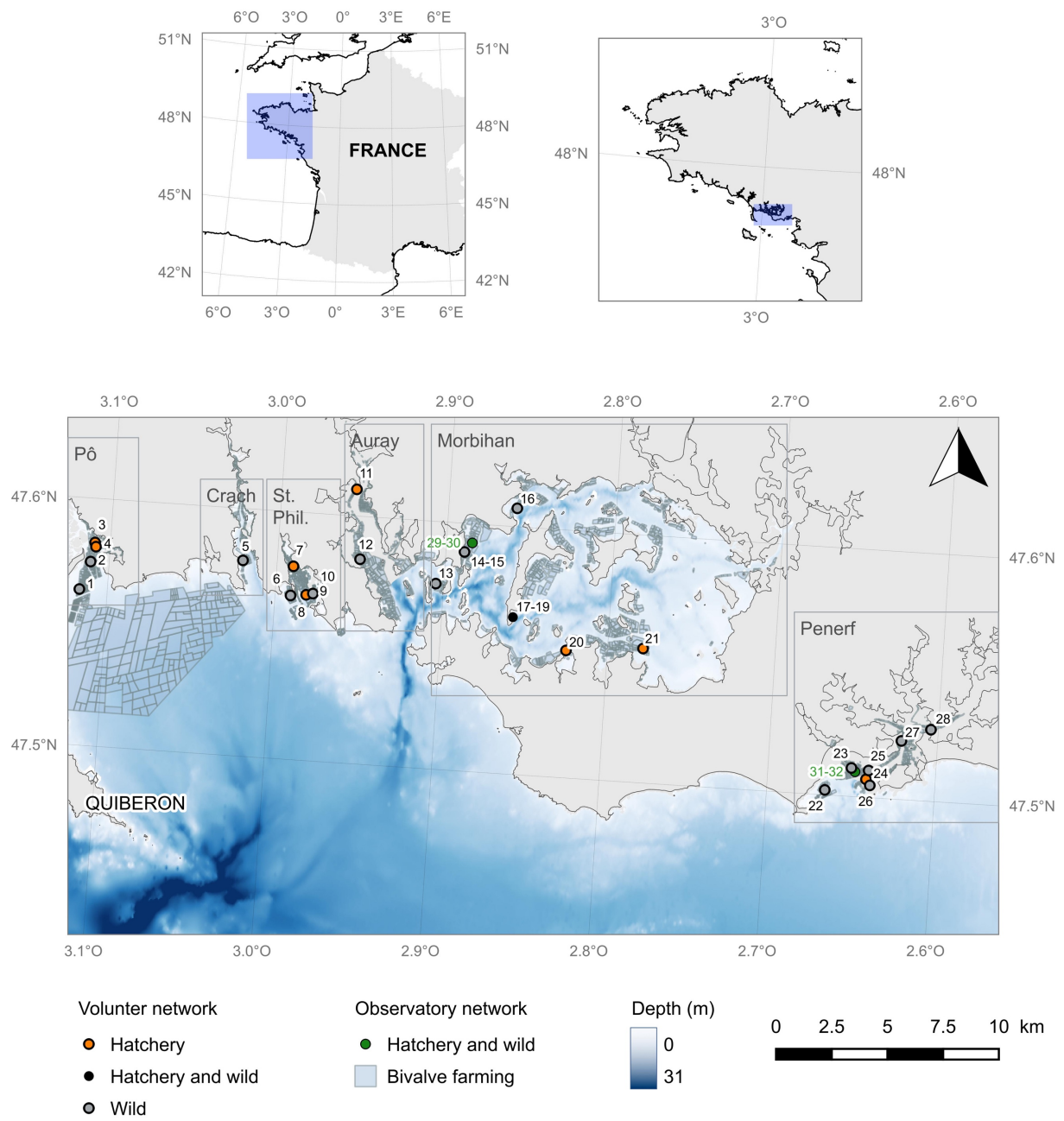
483

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	$\chi^2$	P
Origin	Hatchery	1	0.597	0.127	21.9	<b>&lt;0.0001</b>
Tidal height	–	1	-0.171	0.050	11.5	<b>0.0007</b>
Tidal height × Origin	Hatchery	1	-0.498	0.075	44.0	<b>&lt;0.0001</b>
Site	–	–	–	–	58.5	<b>&lt;0.0001</b>

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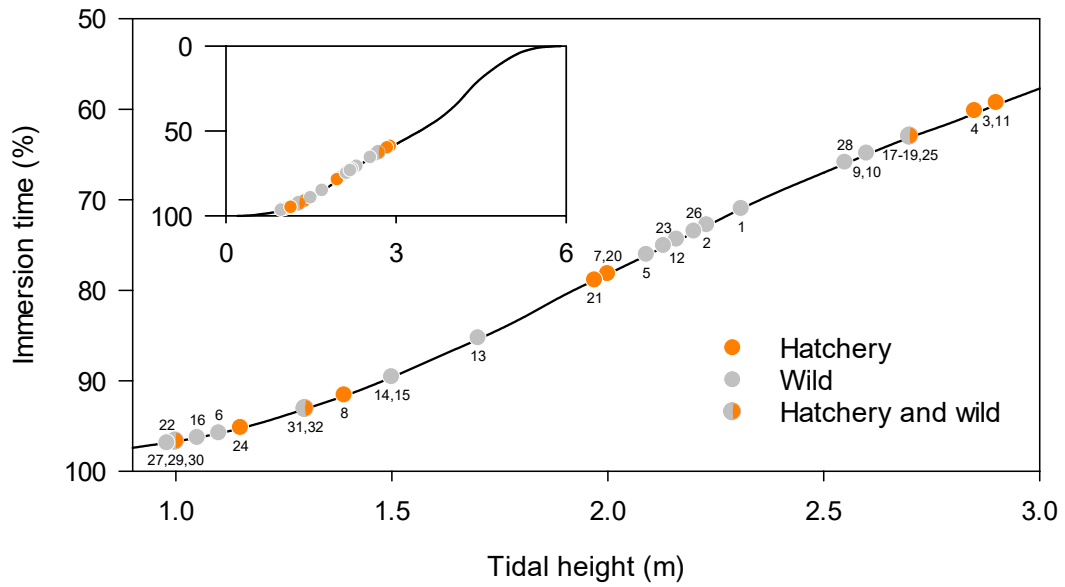
489 **Figure caption**



490

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

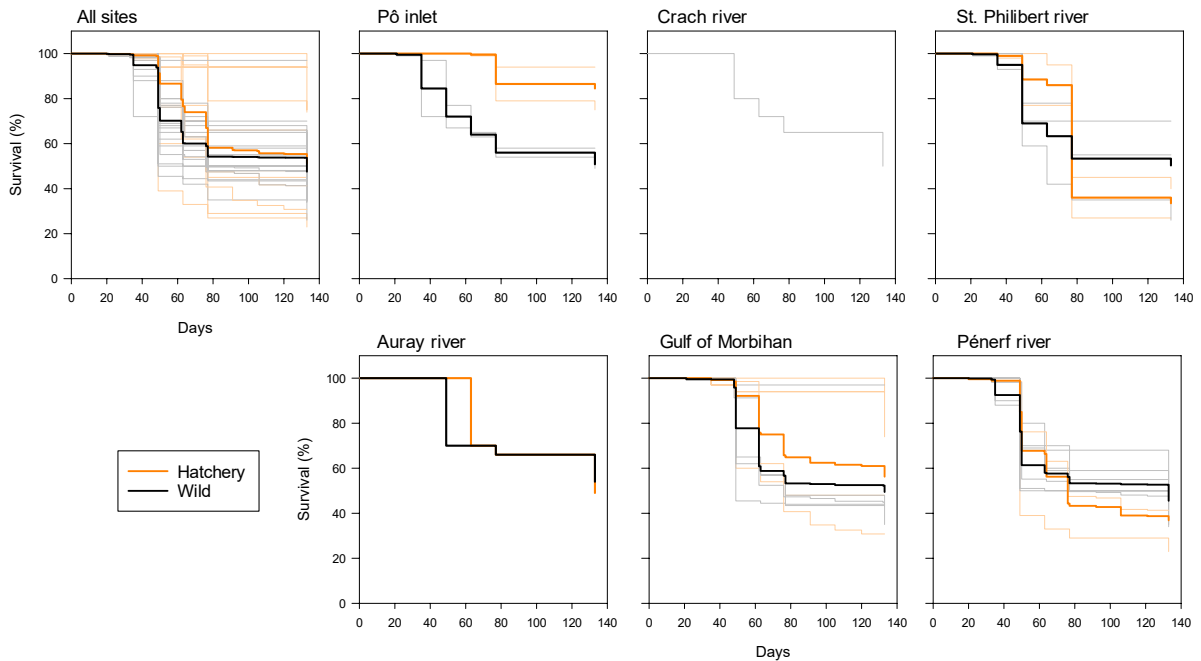
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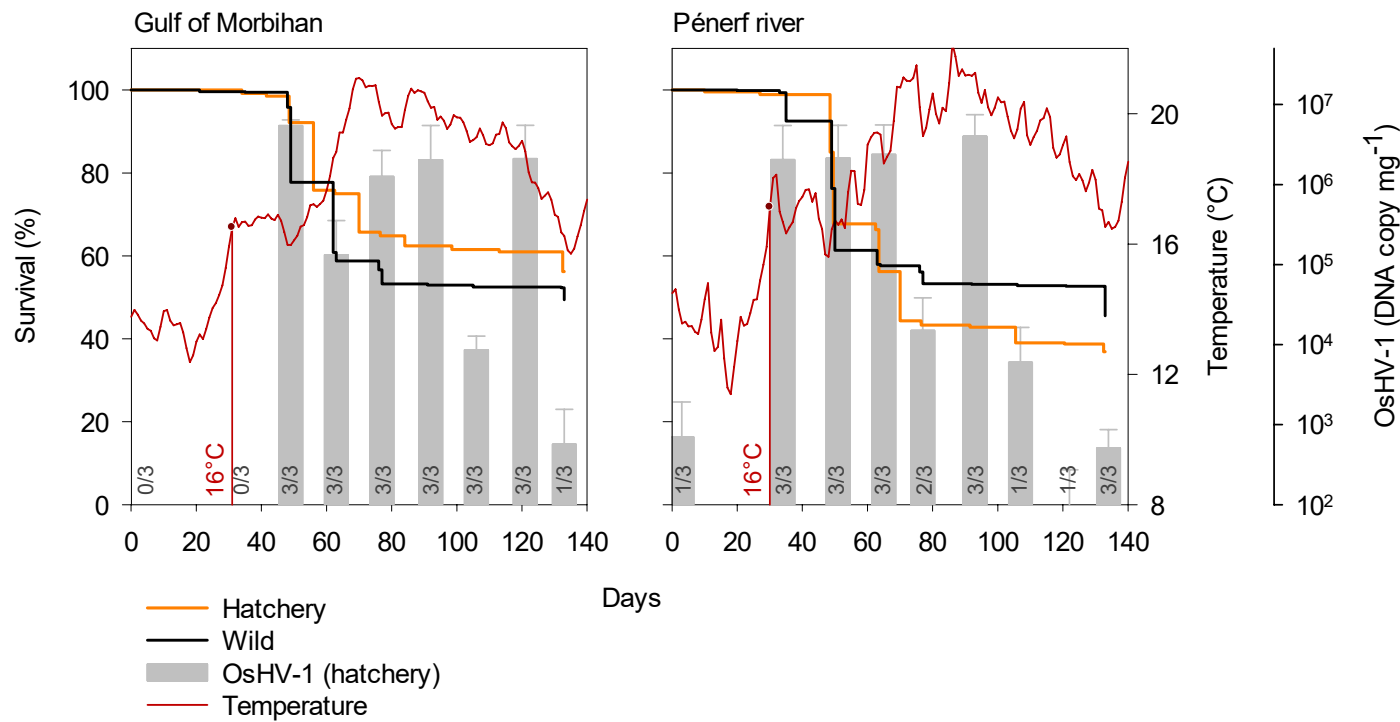
494 **Figure 2.** Relationship between immersion time and bathymetry. Circles represent the  
 495 position of the oyster survival monitoring sites. Inset shows the entire bathymetric range  
 496 during the period of study.

497



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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.

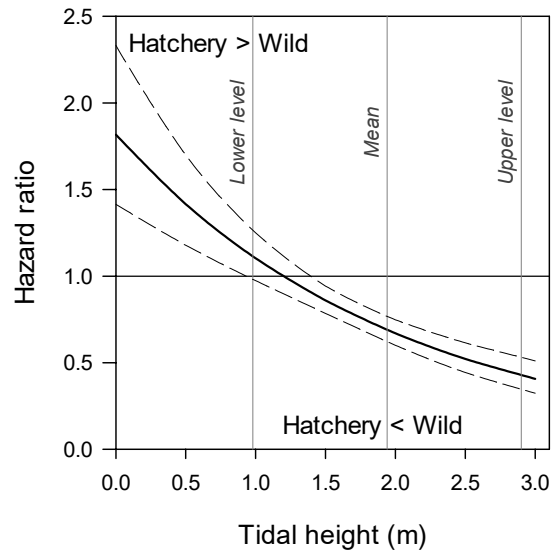
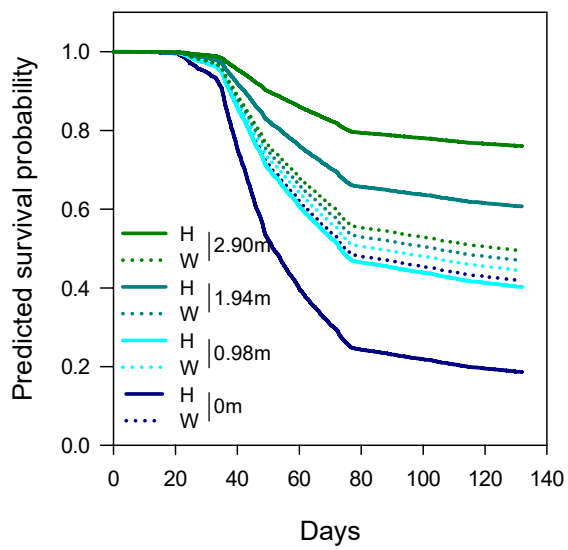


501

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.