INTRODUCTION

It is well established that jellyfish (especially of the Phylum Cnidaria) can impact negatively on coastal industries and services such as tourism (stinging of bathers), fishing (clogging of nets, increased labour) and power generation (clogging of cooling intakes) (reviewed by Purcell et al. 2007). A less widely known problem is the negative impact of jellyfish on finfish, particularly Atlantic salmon *Salmo salar*, in aquaculture. The most well-publicised incident occurred in 2007, when the scyphozoan jellyfish *Pelagia noctiluca* devastated the entire stock of 250,000 harvest-sized Atlantic salmon in Northern Ireland (Doyle et al. 2008, Hay & Murray 2008). Other examples of acute mass mortality events include the mortality of >100,000 farmed salmon in Norway caused by the siphonophore *Muggiaea atlantica* (Fosså et al. 2003). This species was also a suspected causative agent of over 1,000,000 salmon killed off northwest Ireland in 2003 (Cronin et al. 2004). The siphonophore *Apolemia uvaria*, the oceanic hydromedusa *Solmaris corona* and the neritic hydromedusa *Phialella quadrata* have also been previously implicated in fish kill events (Bruno & Ellis 1985, Båmstedt et al. 1998).
Large mortalities do not occur at every site nor every year; however, recent investigations have documented that gill disorders (a more chronic problem in comparison to acute mass mortality events) in farmed salmon are a rising problem for the aquaculture industry (Rodger 2007). Clinical signs of gill disorders in marine-farmed salmon can present as a change in normal behaviour (such as a reduction in feeding, increased respiratory rate, frantic jumping and head shaking) and on examination of affected fish by bleeding gills, pale/thickened patches on the gills, focal lesions and necrosis of the gill lamellae and rakers. Gill disorders may be multi-factorial, with some cases considered to be caused by primary damage from jellyfish, with secondary bacterial infections and opportunistic parasites exacerbating issues (Ferguson et al. 2010, Rodger et al. 2011). However, jellyfish are not the only agents of gill disorders; other agents include phytoplankton, parasites, bacteria and viruses (Rodger 2007). In Ireland, between 2003 and 2005, gill disorders were considered to be one of the most serious causes of mortality in marine-farmed salmon. During this period, farms suffered an average of 12% mortality due to gill disorders (Rodger & Mitchell 2005). Whilst some marine farms suffer significant mortalities due to gill disease, many more suffer lower level mortalities that nonetheless result in considerable economic losses for the industry.

Many small jellyfish such as small hydromedusae and siphonophores are capable of forming high density blooms and are small enough to pass through the mesh of the aquaculture cages and to be inhaled by fish. On inhalation, they pass over the gills and inflict serious injuries when the nematocysts discharge (Fossà et al. 2003). In addition to physical damage caused to the gills, there may also be potentially toxic effects. When nematocysts discharge, they often release haemolytic, cytotoxic and/or neurotoxic chemicals to kill or paralyse their prey (Lotan et al. 1996). Recently, Helmholz et al. (2010) have demonstrated that the toxicity of whole venoms from Cyanea capillata and Aurelia aurita on gill cell cultures of rainbow trout Oncorhynchus mykiss caused a significant reduction in gill cell viability.

Whilst rigorous monitoring of harmful algal blooms around sites of shellfish and finfish aquaculture in Ireland is being conducted (Browne & Deegan 2006), little or no monitoring of zooplankton exists in these areas. Therefore, a year-long monitoring programme was initiated at 2 Atlantic salmon farms on the west coast of Ireland for: jellyfish (hydromedusae, siphonophores and ephyrae of scyphomedusae) previously implicated as agents of gill disorders (Fig. 1), other gelatinous zooplankton (cnenophores, urochordates and chaetognaths) that are useful indicators of water masses (Pierrot-Bults & Chidgey 1988, Edwards et al. 1999) (Fig. 1), phytoplankton and fish pathogens (ectoparasites and bacterial infections of the gills and viral disease). For the purpose of the present study, ‘gelatinous zooplankton’ is purely a descriptive term. Much like Haddock’s (2004) use of ‘gelata’; this term is not used in a taxonomic sense. ‘Jellyfish’ will be used hereafter to refer to the purely cnidarian component of the gelatinous zooplankton.

The aims of the present study were to (1) identify the role of small hydrozoan jellyfish as agents of gill disorders, and (2) investigate inter-site variations in jellyfish and gill disorders at salmon farms. In order to fully investigate these aspects, samples of phytoplankton were taken and histological screening of fish tissues was conducted to assess the presence, or otherwise, of other potential causative agents of gill disorders and mortality.

**MATERIALS AND METHODS**

**Study sites.** Two marine Atlantic salmon farms, monitored for biotic and abiotic factors as well as fish health, were located 370 km (overland distance) apart on the west coast of Ireland (Fig. 2). Site choice was limited by sites that had previously been affected by gill disorders, geographical separation and a willingness of the farms to participate in the study. The southernmost site was located in Bantry Bay, a 40 km long river (drowned river valley) to the southwest of Ireland. Most of the bay averages 30 m in depth, increasing to 70 m near the mouth. Throughout the summer, cyclical coastal upwelling in the bay has been known to drive the development of toxic algal blooms, with shelf waters intruding into the bay on the relaxation of upwelling (Edwards et al. 1996). The northern site in Clifden Bay, Connemara (northwest Ireland), is in comparison, a relatively sheltered, shallow bay (maximum depth ~14 m) only 9 km in length. There are numerous, small freshwater inputs draining into the bay from the mountainous valley that surrounds the area.

**Sample collection and processing.** Gelatinous zooplankton: Gelatinous zooplankton samples were collected between April 2009 and March 2010 inclusive. Samples were taken during daylight hours at high tide (fortnightly April–October and monthly thereafter) during the neap phase of the tidal cycle. Samples were collected using a 0.4 m ring net with 200 µm mesh and a non-filtering cod-end. Five vertical net hauls were conducted at each site—inside and outside a ‘sentinel’ salmon cage (which was repeatedly monitored throughout the course of the study for gelatinous zooplankton and fish health) and at 3 other locations around the farm (Fig. 2). The cages reached a depth of 12 m in
Fig. 1. Representatives of the gelatinous zooplankton identified during the present study from 4 phyla. a: trachymedusan hydro-
medusa – *Aglantha digitale*; b: calycophoran siphonophore – *Muggiaea atlantica*; c: calycophoran eudoxid – *M. atlantica*; d:
larvacean – *Oikopleura* sp.; e: cydippid ctenophore – *Pleurobrachia pileus*; f: chaetognath – *Sagitta elegans*; g: leptomedusan
hydromedusa – *Phialella quadrata*; h: leptomedusan hydromedusa – *Obelia* spp.; i: agalmid ‘Athorybia’ larvae – *Agalma elegans*;
j: narcomedusan hydromedusa – *Solmaris corona*; k: doliolid – *Doliolum* sp. Scale bar = 1 mm for each organism
Bantry Bay and 10 m in Clifden Bay, with vertical hauls inside the cage (i.e. standing on the pontoon, lowering the plankton net into the cage and hauling the net in vertically keeping it away from the side of the cage) and outside the cage taken from the depth at which the cage starts to form a cone (i.e. 10 and 6 m in Bantry Bay and Clifden Bay, respectively). Samples taken from around the cages were conducted to a depth of 25 m in Bantry Bay and 12 m in Clifden Bay.

All samples were immediately preserved in 4% seawater formalin. All gelatinous zooplankton were identified and enumerated to the lowest possible taxonomic level within a day of collection, and the abundance of gelatinous zooplankton was converted to the number of individuals m\(^{-3}\) (see Fig. 4), assuming 100% filtration efficiency of the net due to vertical haul over a short distance. The distance travelled was assumed to be equivalent to the depth sampled; calibrated with individual temperature–depth profiles obtained from a G6 temperature–depth data storage tag (Centre for Environment, Fisheries & Aquaculture Science Technologies Limited, CTL; www.cefastechnology.co.uk/g5/default.htm) attached to the plankton net.

**Phytoplankton:** Phytoplankton samples were taken weekly or fortnightly in one location at each site from April 2009 and March 2010 inclusive as part of the Marine Institute Phytoplankton Monitoring Programme. Integrated water column samples were taken by the salmon farmers using a Lund tube (a weighted polyethylene hose 2 cm in diameter) (Lund & Talling 1957). A standard 15 m long Lund tube was used in Bantry Bay and a 10 m long tube in the shallower Clifden Bay — taking an integrated sample from the surface to the previously stated depths. On each phytoplankton sampling occasion, the entire sample was mixed in a bucket, and a 25 ml subsample (preserved with 4 to 7 drops of neutral Lugol’s Iodine) was sent to the Marine Institute Phytoplankton Monitoring Group for analysis. Each sample was poured into an Utermöhl chamber and left to settle for a minimum of 12 h before analysis. The Phytoplankton Monitoring Group provided data on the number of algal cells l\(^{-1}\) for each sample. The data were then screened for species which have been previously noted to adversely affect finfish. It was these species that were used in further analyses.
Atlantic salmon: Five fish were randomly sampled from the sentinel cage at each site, directly after zooplankton sampling. Fish were caught with feed using a hand net to catch fish from the healthy, feeding population (to avoid catching sick fish that may be near the surface). Fish were put into a large bin containing a lethal dose of the anaesthetic tricaine methanesulfonate (MS-222: 100 mg l⁻¹). Blood samples were taken from the caudal vein for the detection of salmonid alpha virus (SAV), the causal agent of pancreas disease (PD). The second gill arch on the left hand side of each fish was excised, along with small portions of heart, brain, liver, kidney, pyloric caeca, spleen and muscle/skin. All tissues were immediately fixed in 10% neutral-buffered formalin. For histopathological examination, 5 µm sections were cut from paraffin embedded tissues and stained with haematoxylin and eosin. Slides were scanned microscopically at 40×, 100× and 400× magnifications. Gills were inspected for signs of gross pathology (excess mucus, pale gill filaments, swelling, haemorrhage or discolouration), and visceral organs were examined for gross- and histopathology.

Temperature data: Temperature data was obtained via temperature sensors at each site that form part of a network maintained by the Marine Institute. StowAway® TidbiT™ temperature sensors were attached to a rig hanging from one cage pontoon at each site. The sensors logged temperature hourly, providing continuous data measurements from 9 April 2009 until 31 March 2010. The rigs were composed of 3 temperature sensors located at different depths; 1, 8 and 12 m in Bantry Bay and 1, 5 and 10 m in the shallower Clifden Bay. A daily average for the temperature at each depth was calculated and used to plot temperature–depth profiles (Fig. 3).

Data analysis. Gelatinous zooplankton: One-way analysis of variance (ANOVA) was used to compare the abundances of total gelatinous zooplankton and jellyfish between samples taken inside and outside the sentinel salmon cage at each location. In addition, comparisons were made between the abundances from samples taken at the 3 locations around the cages. Normality and homoscedasticity were tested for prior to conducting ANOVA using box-plot visualisation and Levene’s test respectively.

Gill damage: On histopathological examination of the gill tissues, the presence/absence of gill damage was noted for each fish sampled. Gill damage was assessed with a semi-quantitative system designed and under development by Mitchell et al. (S. O. Mitchell pers. comm.). Any gill damage was assessed for the severity and extent of epithelia hyperplasia, lamellar fusion and necrosis. If inflammation, circulatory damage, parasites and/or pathogens were present, these factors were then combined with the above parameters to give an overall categorisation of the gill damage as mild, moderate or severe (with corresponding levels of clinical significance).

Fish mortality: Data on the fish mortalities at the salmon farms were provided by the farm management on request. Data from the Bantry Bay farm were available on a cage-by-cage basis for almost every day. However, the mortality data from Clifden Bay were only available at a monthly resolution for the farm as a whole.
whole, which impeded further in-depth analyses. The monthly mortalities at each farm site were converted to a percentage of the remaining stock to give the overall trend in mortalities for the year and an indication of the onset of any diseases suffered was given.

Repeated Pearson’s Product Moment correlation analyses were performed between the abundance of a range of possible planktonic agents and the average daily fish mortality across the Bantry Bay site resampled as a function of time lag. The mortality data were not always collected daily (often 2 d were combined); therefore, the data were smoothed with a 3 d moving average algorithm to remove erroneous stationary periods of mortality whilst attaining the major trends. The 3 d moving-averaged fish mortality data were tested for Pearson's correlation with (1) the abundance of potentially harmful jellyfish species (*Muggiaea atlantica, Phialella quadrata* and *Solmaris corona*); (2) the abundance of all other jellyfish species; (3) the abundance of gelatinous, non-cnidarian zooplankton; and (4) the abundance of all phytoplankton considered harmful to finfish, to determine the level of co-variation between the different subsets of the zooplankton and phytoplankton time series as a function of time lag applied to the fish mortality (Yaffee & McGee 2000).

When performing this analysis, it is preferable to use the same sampling frequency for each series to avoid spurious results; therefore, the fish mortality data (adjusted for each time lag applied) and the phytoplankton data were re-sampled at the same frequency as the gelatinous zooplankton series. Although this may decrease the robustness of the analysis, it allows comparisons of the series on the same scale (Guadayol et al. 2009) and the inclusion of specified time lags. Correlation coefficients were tested for significance at $\alpha = 0.05$ through the process of randomisation of the original data (2000 times). Correlation coefficients were computed and ranked between the randomised and original data (2-tailed approach) and p-values calculated (Manly 2007).

**RESULTS**

**Hydrography**

Seasonal differences in temperature stratification around the cages were present in Bantry Bay (Fig. 3a) over the course of the year. Initially, a thermocline developed in the surface waters in June, deepening throughout the water column (with complete mixing at times) in July and August. In early September, the water became stratified again with the surface waters much warmer than deeper in the water column. This indicated an influx of cold North Atlantic water from outside the bay, which slowly mixed through the water column by the end of September. The bay slowly cooled and remained completely mixed over the winter and spring. In the shallower Clifden Bay, the water column remained mixed almost the entire year. In early June there was a slight inverse stratification recorded when the waters at 10 m were warmer than the surface waters (Fig. 3b).

**Gelatinous zooplankton abundance and occurrence**

From a total of 200 zooplankton samples collected at both sites throughout the year, 31 species/genera representing 6 different taxa were identified (Fig. 4). Overall, hydromedusae were the most species-rich taxon with 21 species/genera recorded in Bantry Bay and 11 in Clifden Bay. Three species of siphonophore were recorded (Fig. 5), including the adult (polygastric) and ‘Athorybia’ larval stage of *Agalma elegans* and polygastric and reproductive (eudoxid) stages of *Muggiaea atlantica* (Fig. 1). Two species of chaetognath were identified (*Sagitta setosa* and *S. elegans*) as well as scyphozoan ephyrae; however, ephyrae were only recorded on 3 occasions (Fig. 4).

All species/taxa were more frequently recorded and abundant in Bantry Bay compared to Clifden Bay (with the exception of *Oikopleura spp.* and *Obelia spp.*) (Fig. 4). Jellyfish were highly abundant in Bantry Bay, peaking from August to November, and 3 species previously implicated in mass mortality events of farmed salmonids were identified at this site (*Muggiaea atlantica, Phialella quadrata* and *Solmaris corona*). The largest peak in hydromedusae occurred in September, with a mean abundance of ~230 ind. m$^{-3}$; this was representative of an influx of large numbers of the oceanic narcomedusa *S. corona* (Fig. 4). Doliolids, of the genus *Doliolum*, indicators of oceanic water masses, also occurred in the samples over this period. The hydromedusa *P. quadrata*, previously implicated in the mortality of farmed Atlantic salmon in Scotland, was relatively common throughout the year, though not abundant; the maximum abundance of *P. quadrata* (17.9 ind. m$^{-3}$) occurred in June (Fig. 4). The calycophoran siphonophore *M. atlantica*, first appeared in late-July and became a dominant member of the community from the end of August until November (Fig. 4). A highly reproductive population was apparent over this period with high abundances of both polygastric colonies and eudoxid stages recorded (maximum abundance: 227.6 ind. m$^{-3}$). Whilst the oceanic species *S. corona* was entirely absent at the Clifden Bay site, both *M. atlantica* and *P. quadrata* did occur; however, they were scarcely abundant when present. In general, the number and abundance of gelatinous zooplankton
in Bantry Bay were much higher than in Clifden Bay (especially in terms of jellyfish and those species previously implicated in fish kill events) (Fig. 4).

There was no significant difference (p > 0.05) in total gelatinous zooplankton or jellyfish abundance between samples taken inside and outside the cages at Bantry Bay (total: $F_{1,36} = 0.48$; jellyfish: $F_{1,36} = 0.51$) and Clifden Bay (total: $F_{1,40} = 0.11$; jellyfish: $F_{1,40} = 0.01$). There was also no significant difference (p > 0.05) in zooplankton or jellyfish abundance between the samples taken around the cages at the Bantry (total: $F_{1,60} = 0.08$; jellyfish: $F_{1,60} = 0.02$) and Clifden Bay (total: $F_{1,60} = 0.78$; jellyfish: $F_{1,60} = 0.84$) salmon farms.

**Phytoplankton abundance and occurrence**

All phytoplankton species previously identified as harmful to finfish were quantified from the 136 samples taken across both sites. In Bantry Bay, there...
were 3 peaks in phytoplankton abundance over the course of the study, one in the early spring of each year and one in early autumn (Fig. 4). These blooms were predominated by Chaetoceros socialis and Phaeocystis globosa (April 2009), Pseudo-nitzschia seriata, Thalassionema nitzschoide and Thalassiosira spp. (maximum abundance of $7.2 \times 10^5$ algal cells l$^{-1}$ in September 2009), and C. socialis, P. seriata and Thalassiosira rotula (March 2010). The September bloom in Bantry Bay was also concurrent with the influx of oceanic water into the bay and the occurrence of Solmaris corona and doliolids in the zooplankton samples. In comparison, there was an increase in phytoplankton abundance from spring into summer in Clifden Bay, peaking in June and July before decreasing thereafter (Fig. 4). Chaetoceros spp. dominated the phytoplankton community over the summer period in Clifden Bay.

**Histological screening and gill damage**

From April until the end of August, the gills of the fish sampled from the sentinel cage in Bantry Bay had only minor pathological damage (mild gill damage), such as a low level of epithelial hyperplasia and the occasional area of lamellar fusion, as observed on histology sections (Fig. 5a,b). This level of background pathology was considered to be typical of gills regularly observed in marine-farmed fish.
(H. D. Rodger pers. obs.). Throughout September and October some of the salmon sampled showed signs of gill disorders on gross examination (small grey-white areas 2 to 5 mm in diameter) obvious on the otherwise healthy-looking red gill filaments. High levels of gill mucus were also obvious on clinical examination in some fish. The peak in the severity of the gill damage, observed via histopathology, occurred in October and November. The gills presented with a more extensive epithelial hyperplasia, multi-local lamellae fusion, numerous necrotic epithelial cells, and in some cases focal inflammation as well as an increase in eosinophilic granular cells in the filaments (Fig. 5c). Focal telangiectasis, haemorrhage, areas of focal sloughing and loss of the epithelium was also apparent. This degree of gill damage (moderate or severe for 50% of the fish sampled in October) was considered to have severe clinical significance for the fish. There were no signs of bleeding gills, widespread damage (i.e. no multi-local lesions), bacterial infection, amoebic gill disease or other ectoparasites on inspection or histology. The number of fish with such gill damage increased to 80% of the fish sampled at the beginning of November (Fig. 6a). Though persistent gill damage was present for 3 mo, by December, fish sampled from the Bantry Bay farm showed a reduction in the severity of the gill disorders which by then had reverted back to minor gill damage (low level of hyperplasia and focal lamellar fusion) and then a gradual return to healthy gills.

Clinical PD, as caused by the SAV, was first diagnosed on site on 20 August 2009 from the histopathology and sera samples collected (Fig. 6a), although fish in 4 out of the 6 cages (including the sentinel cage) had been previously vaccinated. PD was confirmed as present in fish from all cages; however, the mortality of fish from vaccinated cages was 7.2 percentage points lower than the unvaccinated cages over the course of the year. Histological examination of the fish tissues sampled over the year showed an absence of parasites and damage other than that caused by the gelatinous agents of gill disorders or PD.

Throughout most of the year, the salmon in the Clifden Bay farm had only minor histopathological damage to the gills. Mild focal hyperplasia and fusion of gill lamellae was observed though not considered of clinical significance. On 6 October 2009, the protozoan parasite *Trichodina* sp. was observed on the gills as well as the bacterial pathogen *Tenacibaculum* sp. The presence of these pathogens was concurrent with an increase in epithelial hyperplasia, fusion and necrosis (Fig. 5a,d). Of the fish sampled, 80% had moderate or severe gill damage on 6 October. Damage of the same nature was also observed in early November (Fig. 6a). However, on both occasions, a return to healthy gills was apparent by the next sampling occasion, unlike the persistent damage observed in Bantry Bay.

Clinical PD was diagnosed at the Clifden Bay site on 15 June 2009 (none of the fish had been vaccinated) and bacterial disease was also present at the end of September (Fig. 6b).
Potential causes of fish mortality

The correlation analyses revealed that fish mortality across the fish in all cages at the Bantry Bay site was as follows: significantly and positively correlated with the abundance of harmful jellyfish, with a lag of 1 to 7 d (p < 0.05), though not a lag of 0 d (p > 0.05); significantly and negatively correlated with both the abundance of all other jellyfish (p < 0.001) and the abundance of gelatinous but non-cnidarian zooplankton (p < 0.001) at all lags; and not significantly correlated with the abundance of harmful phytoplankton (p > 0.05) (Figs. 6a & 7). The highest correlation with harmful jellyfish occurred 3 d post sampling (Fig. 7).

DISCUSSION

Jellyfish have been identified as the causative agents in a number of fish kill events of marine-farmed salmonids throughout northern Europe in recent decades (Purcell et al. 2007). However, due to a lack of long-term monitoring, there are often insufficient data on jellyfish abundances before, during and after a fish kill event, which prevents the ultimate cause of mortality from being identified. Furthermore, instances of jellyfish being linked to the underlying problem of gill disorders (chronic exposure to lower abundances than those that cause acute mass mortality event) are especially scarce (Rodger et al. 2011). Thus, the data presented here are unique in this regard with 2 sites monitored for phytoplankton, gelatinous zooplankton and fish health over the course of a year. From a total of 31 gelatinous zooplankton species/genera identified throughout the study, only 3 of these have been previously implicated in fish kill events (Muggiaea atlantica, Phialella quadrata and Solmaris corona). All 3 species occurred in Bantry Bay, with M. atlantica and S. corona in high densities (Fig. 4), whereas only M. atlantica and P. quadrata occurred in the northern Clifden Bay site at relatively low or negligible densities. Numerous species of phytoplankton identified as harmful from previous fish kill events were also recorded (e.g. Chaetoceros spp., Pseudo-nitzschia seriata, Skeletonema spp., Thalassionema spp. and Thalassiosira spp.).

The development of significant gill disorders in the salmon from the Bantry Bay farm was evident during late summer/autumn. Histological examination of the gill tissues showed a clear increase in gill damage from

![Fig. 7. Correlation between daily fish mortalities and the abundance of different gelatinous zooplankton groups and phytoplankton (*) in Bantry Bay, Ireland. Harmful jellyfish group includes Muggiaea atlantica, Phialella quadrata and Solmaris corona. Fish mortality data were smoothed with a 3 d moving average algorithm. Mean (dashed line) and confidence intervals (95%) for randomised data are shown. * p < 0.05, ** p < 0.001](image-url)
a state of low level damage to gross gill damage (i.e. represented by lamellar fusion and necrosis of the epithelium, Fig. 5a,c). This coincided with the peak in harmful jellyfish abundance (September to November, abundance: ~450 ind. m⁻³) (Fig. 6a). This level of gill damage would have had a significant clinical impact of the fish’s health and ability to survive. Importantly, a highly significant, positive correlation between the abundance of harmful jellyfish (Muggiaea atlantica, Phialella quadrata and Solmaris corona) and the average daily fish mortalities was identified (Fig. 7). The results suggest that M. atlantica and S. corona were the causative agents of the gill disorders identified and may also have had an impact on the observed mortalities. The occurrence of P. quadrata at much lower densities than the other 2 species (maximum abundance: 17.9 compared to ~200 ind. m⁻³) and earlier peak in abundance (June compared to October) suggests that it is unlikely that P. quadrata played a large part in the positive correlation observed. There was a highly significant negative correlation between all other jellyfish and fish mortality, as well as non-cnidarian gelatinous zooplankton and fish mortality. This confirms that the seasonal peaks of these species do not coincide with the peak period in mortality or risk period for the fish and may therefore not be as much of a cause for concern as the aforementioned species.

Although high abundances of some harmful phytoplankton species were observed at times throughout the study, no gill damage thought to be attributable to phytoplankton was observed at either site. Phytoplankton damage often presents clinically as more widespread damage across the entire gill surface (compared to localised patches of damage with zooplankton), and in some cases blood can be observed in holding bins when affected fish are caught/anaesthetised (Rodger 2007), neither of which were observed here. Furthermore, debate still remains as to whether species like Thalassiosira spp. and Thalassionema spp. are capable of causing gill damage. Whilst Thalassiosira spp. and Skeletonema spp. have been associated with gill disease in Atlantic salmon in a single study, the abundances of each species were not given, and dense blooms of such species have often occurred without deleterious effects (previous having been designated as non-pathogenic) (Kent et al. 1995). In addition, it is unknown at what density these potentially harmful species may be lethal. A study using the controlled exposure of harmful algae species to Atlantic salmon found that concentrations of up to 4.0 × 10⁻⁶ cells l⁻¹ caused no deleterious effects (Burridge et al. 2010), concentrations that were higher than experienced when the peak in fish mortality and gill damage occurred in this field study (7.2 × 10⁵ algal cells l⁻¹ in September).

At the end of August the fish in the Bantry Bay farm were diagnosed with clinical PD, a disease which has also had a severe impact on the aquaculture industry in Ireland (Rodger & Mitchell 2007). Due to the occurrence of both PD and gill disorders, it is difficult to apportion a percentage of mortalities attributable to each condition (total mortalities for the sentinel cage: 4.3%). However, the presence of one condition may also have increased the impact or clinical significance of the other condition due to a reduction in immunity. Regardless of the presence of PD in the fish population, it is clear that there is a strong correlation between the abundance of harmful jellyfish and fish mortality (Fig. 7). In Clifden Bay, jellyfish were relatively scarce overall and harmful species were near absent. Nevertheless, the fish at this site suffered discrete periods of gill damage associated with the pathogens Trichodina sp. and Tenacibaculum sp. Although the gill analysis over the period when the pathogens were present assigned a higher severity to the damage than the gills of the fish in Bantry Bay, this may be due to the additive effect of the presence of multiple pathogens (Fig. 5a). It was considered that the gill damage suffered by fish in the Bantry Bay site was not only more clinically significant but also more long standing, potentially affecting the survival of the fish. In Clifden Bay, the mortalities were considered attributable to a combination of PD and bacterial infection (Fig. 6b).

Indeed, in the larger, deeper and more southerly Bantry Bay, there was evidence of localised population blooms of some species of gelatinous zooplankton and phytoplankton (Fig. 4a), perhaps indicative of more resource abundant and warmer waters that have been long known to promote harmful algal blooms in this region (Raine et al. 1993). Furthermore, influxes of oceanic species such as Solmaris corona and Doliolum sp. (Edwards et al. 1999) are indicators of an intrusion of an oceanic water mass into the bay, with such events previously resulting in the advection and stimulation of harmful algal blooms in the bay after the relaxation of upwelling events when currents are reversed (Raine et al. 1993, Edwards et al. 1996). The implications of such oceanic intrusion should be considered with the proposed movement of aquaculture to more offshore locations (Watson & Drumm 2007). Little is known about whether fish in offshore sites will be more or less vulnerable to damage by jellyfish blooms due to a lack of knowledge on the abundance and distribution of oceanic species in the relevant areas.

At present, marine-farmed finfish are not only vulnerable to diseases like PD and parasitic infections such as sea lice (Rodger & Mitchell 2007, Costello 2009), but also to gill damage caused by small jellyfish species, a threat which has received limited attention.
to date. The present study demonstrates that the abundance of detrimental species, local hydrography and environmental interactions may affect the likelihood of gill disorders (attributable to jellyfish) arising. In addition, the detrimental abundances of the siphonophore Muggiaea atlantica in Bantry Bay were at least an order of magnitude lower than the bloom which occurred in Norway (maximum recorded abundance of *M. atlantica*: 13,000 ind. m⁻³) killing hundreds of thousands of farmed salmon (Fossà et al. 2003). Yet, the abundances observed here were sufficiently high to cause significant gill damage and potentially low level mortalities (i.e. not rapid mass mortalities). An average mortality of 7.1% of the stock was suffered in total at the Bantry Bay site over the monitoring period, some of it potentially attributable to gill disorders. Hence, lower level abundances of small jellyfish may be more of a cause for concern than previously considered. Whilst the monitoring of harmful phytoplankton, PD and sea lice occurs regularly at finfish aquaculture sites around Ireland and elsewhere (Copley et al. 2001, Browne & Deegan 2006), there has been no routine monitoring of harmful gelatinous zooplankton until the present study.

Logistically, it was only possible to conduct this study over a year at 2 sites; however, it is becoming increasingly apparent that widespread routine monitoring of jellyfish around aquaculture sites is necessary and will be fundamental if the links between their blooms and detrimental effects on the fish are to be fully understood. Widespread monitoring will be vital to obtain site-specific information jellyfish populations, including their seasonal occurrence and abundance. As yet, no reliable and cost effective mitigation methods exist to prevent small hydrozoan jellyfish from entering the cages (Rodger 2007, Hay & Murray 2008) and this should also be the focus of future studies. Furthermore, controlled experimental studies on the specific pathologies of gill disorders could further assist the diagnosis of such diseases *in situ* and could prove vital to the future of the industry.

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**LITERATURE CITED**


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