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REVIEWS

Non-infectious gill disorders of marine salmonid fish

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Abstract Gill disorders present a significant challenge in salmon (*Salmo salar* and *Oncorhynchus* sp.) farming regions throughout the world. This review of gill disorders and diseases of marine fish is focused on the non-infectious causes of gill disease in marine stage salmonids and these are grouped into harmful algae, such as *Karenia mikimotoi*, harmful zooplankton, such as *Pelagia noctiluca*, other environmental challenges, such as pollutants, as well as nutritional and genetic or congenital causes. The present level of understanding of these gill disorders is reviewed with regard to risk factors, potential impacting factors, methods of best practice to mitigate non-infectious gill disease and disorders, as well as knowledge gaps and avenues for future research.

Keywords Gills · Disease · Non-infectious · Farmed · Salmon

Introduction

Gill disease, in particular that associated with harmful algal blooms and jellyfish swarms, is a significant challenge for global marine salmonid (*Salmo salar* and *Oncorhynchus* sp.) farming (Rutter 2010). There are numerous aetiological agents considered to be involved in, or responsible for gill disease, and there are a range of clinical and pathological presentations (Ferguson 2006; National Veterinary Institute 2009; Roberts and Rodger 2001; Rodger 2007). Gill disease has been responsible for large scale mass mortalities as well as poor growth and performance in farmed salmon and has been a serious financial burden for some sectors of the industry. The major non-infectious causes of gill disease in marine fish are reviewed as either specific agents or the gill disorders they have been associated with, with an emphasis on the marine stage salmonids in particular. The non-infectious aetiologies are grouped into the harmful algae, the harmful zooplankton, other environmental challenges as well as nutritional and genetic or congential disorders.

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Harmful algae

Aetiology

Reports of algal blooms affecting finfish or salmonid culture are numerous and details from some of these are presented in Table 1. Algal blooms can be termed 'harmful' when these occurrences impact on human public health, wildlife and fisheries or aquaculture. Approximately 4,000 marine microalgae have been described to date. Of these between 60 and 80 species

Table 1 Examples of marine phytoplankton species associated with fish mortalities

Phytoplankton species	Finfish affected	Country/region	Details	References
Karenia mikimotoi	*Gadus morhua, S. salar*	Ireland	Cod hatchery juveniles killed, 500 mortalities of farmed salmon	Silke et al. (2005), Mitchell and Rodger (2007)
	S. salar	Scotland	Gill damage in farmed salmon	Jones et al. (1982), Davidson et al. (2009)
	S. salar, O. mykiss	Norway	2.6–100% mortalities farmed fish	Dahl and Tangen (1993)
Karenia brevisulcata	*Sardinops sagax*	New Zealand	0.5 tonne pilchards killed	Jones and Rhodes (1994)
Cochlodinium polykrikoides	*Various farmed species*	Korea, Japan, China		Kim et al. (1997), Yuki and Yoshimatsu (1989), Qi et al. (1993)
		Philippines	Wild reef fish	Azanza et al. (2008)
Gonyaulax excavata	*S. salar*	Faroe Islands		Mortensen (1985)
Karlodinium micrum	*Mugil cephalus, Sciaenops ocellatus*	South Carolina	Retention pond fish kill	Kempton et al. (2002)
	Morone saxatilis x chrysops	Chesapeake Bay	15,000 2–2.75 kg fish in 1 day	Deeds et al. (2002)
Gymnodinium sp	*Liza macrolepis, Acanthropagrus cuvieri*	Araban Gulf	Farmed and wild fish	Heil et al. (2001)
Chaetoceros wighami	*S. salar*	Scotland	550,000 fish died or were culled, 44 tonnes mortality	Bruno et al. (1989), Treasurer et al. (2003)
Chaetoceros convolutus	*Oncorhynchus kisutch*	British Columbia	60,000 40 g fish within 1 week	Speare et al. (1989), Albright et al. (1993)
Ceratium furca	*S. aurata, Liza klunzingeri*	Arabian Gulf	Fish kill of wild mullet and farmed bream	Glibert et al. (2002)
Ceratium furca	*Salmo salar*	Scotland (Skye)	2.69T of 4 kg fish died (670 fish)	ICES report (2005)
Ceratium fusus	*Various species*	Japan	Farmed fish	Onoue (1990)
Skeletonema sp.	*S. salar*	British Columbia	Farmed fish (4% mortality, approx. 16,000 fish, mixed bloom)	Kent et al. (1995)
Chrysochromulina polylepis	*S. salar*	Scandinavia	Farmed and wild fish	Dahl et al. (1989)
Heterosigma akashiwo	*Oncorhynchus tshawytscha*	New Zealand, Japan	Farmed fish	Chang et al. (1990), Khan et al. (1997)
Alexandrium tamarense	*S. salar*	Nova Scotia	Farmed fish	Cembella et al. (2002)
Pseudonitschia sp.	*S. salar*	British Columbia	Farmed fish	Kent et al. (1995)

are identified as being toxic and 200 species as potential causes of harmful algal blooms (HABs; Sournia 1995; Smayda 1997). Algal blooms are naturally occurring phenomena, however, in recent decades there are suggestions that there has been an increase in the frequency and intensity of these blooms and that they are reported to be covering a wider geographical distribution (Daranas et al. 2001; Anderson 2009). Hallegraeff (1995) proposed four main explanations for this:

1. Eutrophication of coastal waters due to anthropogenic activities possibly in combination with unusual meteorological conditions
2. Increased practice of aquaculture in coastal waters

3. Increased awareness amongst scientists of toxic planktonic species
4. Movement of resting forms of toxic planktonic species between regions, for example, in ballast waters of boats (Bolch and de Salas 2007; Edwards et al. 2001).

Although questions remain with regard to the suggestions that harmful algae blooms are increasing in frequency and geographic extent, there is increased interest in such blooms in the scientific community, the general public and governments (Granéli and Turner 2006).

Impact of HABs

It has been estimated by Hoagland and Scatasta (2006) that the total cost of HABs in the USA averaged $82 million per annum during the period 1987–2000 when impacts on public health, commercial fisheries, recreation, tourism and monitoring where considered.

The financial cost of algal blooms is a significant concern to the aquaculture industry. Taylor (1993) reported blooms of *Chaetoceros* sp., *Heterosigma* sp. and another undescribed chloromonad that cost the salmonid farming industry in British Columbia approximately $14 million between 1988 and 1993. In Korea approximately $1.31 million per year was estimated as the financial impact incurred by HABs to aquaculture (Kim 2006). Imai (2005) reported that the average economic loss due to HABs is over $10 million a year in Japan.

Clinical signs in salmonids associated with HABs

The clinical signs in farmed salmonids associated with HABs include fish swimming in an elevated position in the water column, lethargy, gasping at the surface, increased respiratory rate and mortalities. There may be excessive mucus production on the gills or gills may have small areas of thickening which appear as pale areas or spots (Rodger 2007). Small blood spots or petechiae are seen with some irritant phytoplankton, however, in some cases with toxic algae the gills may show no gross abnormalities. Bleeding, or blood leakage, from phytoplankton damaged gills may be seen during anaesthesia of fish or following transport. Sometimes there are associated mortalities in wild fish stocks and invertebrate benthic species associated with harmful blooms (Silke et al. 2005; Mitchell and Rodger 2007). Algal blooms can cause the affected waters to become intensely discoloured and while they have been frequently associated with 'red tides' the discolouration can in fact vary from red to white or from golden brown to emerald green (Zingone and Enevoldsen 2000).

Histopathology of fish associated with HABs

Histopathological observations of gill lesions caused by HAB events include acute necrosis, sloughing of epithelial cells, severe oedematous separation of the epithelium from the lamellar branchial vessels, necrosis of epithelial cells (Jones et al. 1982; Fernández-Tejedor et al. 2004; Mitchell and Rodger 2007; Fig. 1), swelling and pyknosis of primary lamellar epithelium and congestion of branchial vessels (Roberts et al. 1983). In addition to the gill pathologies there may also be liver and gastrointestinal histopathologies observed (Mitchell and Rodger 2007).

Mechanisms involved in HABs

Black et al. (1991) surmised that there are four main mechanisms by which fish mortalities can be caused by harmful algal blooms: (1) physical damage, (2) asphyxiation due to oxygen depletion, (3) gas-bubble

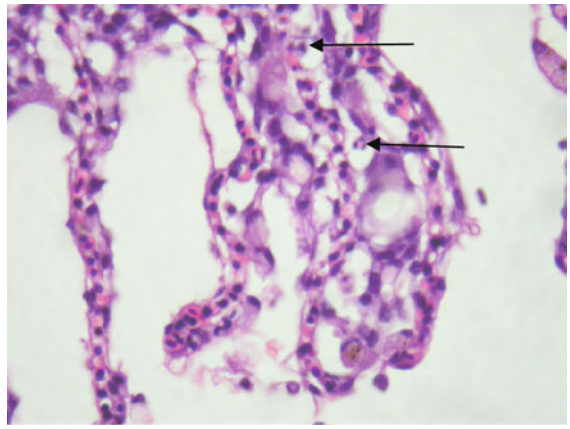

Fig. 1 Histopathological section of Atlantic salmon gills exposed to harmful phytoplankton (*K. mikimotoi*) exhibiting necrotic epithelial cells, irregular surface and epithelial lifting (H&E ×100)

trauma due to oxygen super-saturation caused by algal photosynthesis, and (4) ichthyotoxin damage.

Physical damage caused by HABs

Several species of marine diatom have been implicated, to a greater or lesser extent, in mortalities of farmed salmonids. These species include *Chaetoceros* sp. (Albright et al. 1993; Yang and Albright 1992), *Skeletonema* sp., *Thalassiosira* sp., *Thalassionema* sp., *Coscinodiscus* and *Pseudonitzschia* (Kent et al. 1995; National Veterinary Institute 2009). It has been demonstrated how phytoplankton such as *Chaetoceros* sp. possess harmful setae with spicules (Albright et al. 1993) which when filtered past the gill lamellae become retained due to the spines catching on the epithelial surfaces. This causes an irritation of the epithelia and overproduction of mucus by goblet cells. Physical damage to the epithelium then predisposes the fish to secondary infection with bacteria as demonstrated by Albright et al. (1993).

Factors that influence how damaging certain *Chaetoceros* species are to salmonids include the species of salmonid (Atlantic salmon and trout species are more susceptible to *Chaetoceros* sp. than Pacific salmon (*Oncorhynchus* sp.)), exposure time to the diatom and its concentration in the water column, size of fish (smaller fish are more susceptible), and presence of concurrent disease (sub-lethal concentrations of 0.4–5 cells of harmful *Chaetoceros* species/ml have been shown to cause mortalities in chinook (*O. tschawytscha*) and coho salmon (*O. kisutch*) due to secondary infection with *Vibrio* sp or bacterial kidney disease; Albright et al. 1993).

Asphyxiation by HABs

Despite the identification of potential ichthyotoxins associated with blooms of *Karenia mikimotoi* (Satake et al. 2002, 2005) mortalities due to this planktonic species are suggested to be due in part to decreased levels of oxygen saturation resulting from the decomposition of the algae in the later stages of the bloom (Silke et al. 2005). In addition to this, reduced oxygen conditions are enhanced by the respiration of the algae themselves and of the bacteria involved during the bloom breakdown. Consequently oxygen shortages primarily occur overnight although overcast weather in daylight may incur similar effects (Bruno and Poppe 1996).

Jones and Rhodes (1994) reported that the deaths of 500 kg of pilchards (*Sardinops sagax*) was attributed to reduced dissolved oxygen levels in combination with a bloom of the green microalgae, *Tetraselmus* sp. in a small lagoon in New Zealand. Mechanical clogging of the gill lamellae was also observed.

Gas-bubble trauma

Gill pathology and disease has been associated with gas bubble disease (Roberts 2001; Speare 1998) in freshwater species, however, reports in marine fish are less frequent. The main gill pathologies include oedema of the lamellae with concomitant degeneration of the overlying epithelium. Renfro (1963) indicated that an excess of oxygen was associated with fish mortalities at Galveston Bay, Texas. The levels of supersaturation implicated in these mortalities were as high as 250–327%. There are reports into the effects of lower levels of supersaturation in marine fish with Gray et al. (1985) investigating gas supersaturation in sea bass (*Dicentrarchus labrax*) and mullet (*Mugil cephalus*) and it was demonstrated that the 96 h LC_{50} for post larvae at 20°C were similar at 127.2% total gas pressure (125.2–129.4% TGP) and 129.4% TGP (124.7–135.0% TGP) respectively. Cod (*Gadus morhua*) larvae (32–64 days post hatch) exposed to total dissolved gas of 103 and 106% did not exhibit increased mortality but a chronic effect was observed as a reduction in growth when compared to controls (Gunnarsli et al. 2009).

Icthyotoxins produced by HABs

There are a variety of phytoplankton species that are of concern as toxin producers and some of these are summarised in Table 1.

Karenia species, previously known as *Gyrodinium* (Silke et al. 2005 records that *K. mikimotoi* is synonymous with G. *aureolum*, *G.* cf *aureolum*, *G. nagsakiense* and *G. mikimotoi*) has been associated with fish kills for many years. Roberts et al. (1983) demonstrated that *K mikimotoi* (*Gyrodinium aureolum*) did not cause mechanical occlusion of the gills but did induce severe histopathological damage, including necrotic degeneration and disintegration of

lamellar epithelia. Two cytotoxic polyethers, Gymnocin A (Satake et al. 2002) and Gymnocin B (Satake et al. 2005), have been isolated from *K. mikimoto*, and these may explain the mechanism by which this dinoflagellate damages fish species (Mitchell and Rodger 2007).

The rate of fish mortalities of chinook salmon smolts due to *Heterosigma akashiwo* was found by Black et al. (1991) to be dependant on the concentration of planktonic cells and water temperature. Normal amounts of mucus and no pathological gill or organ damage was associated with the fish deaths, and it was surmised that a rapidly acting neurological agent was involved. Carrasquero-Verde (1999) conducted experiments to determine the role of heterotrophic bacteria in the toxicity of *H. carterae* to salmonids. He found that under the given experimental conditions the presence of bacteria was necessary for the phytoplankton species to be toxic to chinook and coho salmon. It was also suggested that the bacterium indirectly caused toxin production in the phytoplankton by acting as a stressor to the phytoplankton or by producing a precursor chemical which would instigate the synthesis of toxin. The main clinical sign observed during exposure was an increased respiratory rate. Just prior to death the fish behaved as if they were anaesthetised and lost their equilibrium. Finally they rested on the bottom of the experimental tanks, respiratory rate decreased and muscular spasms were reported immediately prior to death. There was no apparent increase in mucus on the gills of either control or treated fish.

Mitigation and control of HABs

Mitigation and control strategies for HABs in finfish aquaculture have been reviewed by Kim (2006) and include those precautionary measures such as monitoring and prediction with actions such as early harvesting, reducing feed to fish, and use of clay, water pumps or aerators as well as indirect controls such as reducing nutrient inputs, modifying water circulation and bio-remediation. Following a HAB the actions that could be undertaken include moving the pens to another unaffected area, enclosing the nets pens, changing the water circulation in the farms, using aeration or oxygenation and direct actions on the bloom by physical (removal with skimmer or screen filter), biological (using algicidal microorganisms) or chemical means (clays and long chain polymers or surfactants such as sophorolipid; Kim 2006). At present the predictive models and mitigation techniques employed to minimise the harmful effects of algal blooms are insufficiently advanced or developed to ensure the complete safety of commercial fisheries or aquaculture although examples and recent advances such as the underwater sensor developed by a team at NOAA's National Centres for Coastal Ocean Science and the Monterey Bay Aquarium Research Institute (Scholin et al. 2009), the physical-biological model developed by a team at the Woods Hole Oceanographic Institution (McGillicuddy et al. 2003) and detection of *Karenia brevis* blooms in the Gulf of Mexico using backscattering and fluorescence data (Cannizzaro et al. 2009) hold much promise. Mitigation measures on the impact of algal blooms on aquaculture cages include the upwelling of deep water and increased oxygenation of cage water. Both strategies may, however, be counterproductive in the instance of a bloom of icthyotoxin producing species of algae. Riley et al. (1989) demonstrated that physical agitation of the water column actually increased the lethal effect of toxic blooms. Short term submergence of Atlantic salmon (*Salmo salar*) pens has been experimentally shown as a potential method to avoid negative surface algal blooms (Dempster et al. 2009). Rigby et al. (1993) eschewed the strict adherence to correct procedure in the physical and chemical treatment of transport ships and vehicles to reduce the possibility of transfer of harmful algae into new geographical regions. Various measures have been attempted and/or employed to monitor the occurrence of harmful algal blooms in the marine environment. This has included the use of electronic microarray platforms (Barlann et al. 2007) and the development of 3D fluorometric method to discriminate between causative species of HABs (Zhang et al. 2009a). McManus et al. (2008) suggested that the use of autonomous high-resolution vertical profilers coupled with targeted sampling could allow for earlier detection of HABs in the coastal environment. Zhang et al. (2009b) developed a loop-mediated isothermal amplification (LAMP) assay for the sensitive and rapid detection of the genomic DNA of *Karenia mikimotoi* and suggested that this technique has the potential to be a highly sensitive, specific and rapid predictor of HABs. In Korea, the dispersion of yellow clay is considered to be the best practical method to mitigate

HABs (Seo et al. 2008), as it is considered to be inexpensive, not difficult to apply in the field and to have little significant impact on water quality. Biosurfactants have been investigated in different regions as a method to control HABs and warrant further and more in-depth investigation (Gustafsson et al. 2009). Other mitigation methods examined in Korea include the use of marine bacteria to kill red tide microalgae, microscreen filtration and ozone, ultraviolet radiation to destroy HAB species and sodium hypochlorite (NaOCl) as a by-product of the electrolysis of natural seawater (Lee et al. 2008), however these techniques were expensive, environmentally detrimental and difficult to employ. Satellite imagery can also be used to monitor the physical factors that lead to the development of blooms both temporally and spatially (Ahn et al. 2006; Vanhouette-Brunier et al. 2008). Advances are also being made in the development of simple screening techniques for the detection of cytotoxic substances by algal blooms (Katsuo et al. 2007) and the utilization of geographic information systems to analyse primary data on HABs and develop risk assessments (Wang and Wu 2009).

Research undertaken in Canada on the use of in-feed mucolytic agents for salmon at risk of exposure to harmful algae has been undertaken; Yang and Albright (1994) fed L-cysteine ethyl ester to coho salmon and then exposed them to lethal levels of *Chaetocerus concavicornis*, an algae which causes physical gill damage and dramatically increases mucus levels on the gills. The experimental work showed greatly reduced mucus production in treated fish when exposed to the harmful algae.

Harmful zooplankton

Aetiology of zooplankton swarms

Gelatinous zooplankton swarms are a significant cause of production loss to the aquaculture industry. The increase of reports in the literature on harmful algal and zooplankton blooms may be related to increased scientific awareness and interest, but concerns are mounting that the marine ecosystem food chain is rapidly changing with a trend towards dominance of jellyfish (Hay 2006; Lynam et al. 2006). The species which have been associated with fish kills are listed in Table 2. Mills (2001) also documented how non-indigenous jellyfish species have begun to invade new ecosystems, for example, *Rhopilema*, originally from the Indo-Pacific region, was introduced into the Mediteranean.

The abundance of gelatinous zooplankton appears to be influenced by numerous climatic variables including temperature, salinity, North Atlantic Oscillation (NAO), North Pacific Decadel Oscillation (NPDO) and El Nino Southern Oscillation (ENSO; Purcell 2005; Lynam et al. 2006). Purcell (2005) discussed how increasing temperatures and shifting isotherms promote reproduction of jellyfish and allow the expansion of species ranges northwards. Coastal development, pollution and overfishing are impacting on the populations of natural predators of jellyfish, such as loggerhead turtles and also competitors such as tuna, sardines and anchovies (Purcell 2005). Purcell et al. (2007) and Lo et al. (2008) when discussing shellfish culture and jellyfish swarms proposed that the floating aquaculture structures provided a favourable habitat for increased polyp reproduction, especially in situations with restricted water flow. Zooplankton swarms are synchronous with natural climate changes, however, global warming resulting in milder winters may allow for greater survival of individuals between seasons and leads to increases in bloom strength earlier in the following calendar year.

Impact of gelatinous zooplankton swarms

Hay and Murray (2008) reported that, in Scotland between the period of 1999 and 2005, approximately 60% of 4.7 million fish mortalities (9,500 tonnes) were caused by jellyfish or harmful zooplankton. The authors also observed that in Skye and the Outer Hebrides in 2002 fish losses caused by a combination of zoo and phytoplankton accounted for greater than 10% of mortalities in numbers and approximately 17% of the biomass lost. Rodger (2007) observed that first sea year salmon in farms are especially vulnerable to gill pathology and gave an estimate of approximately 12% for annual mortalities due to gill disease in Ireland, however the gill disease had various aetiologies and included harmful zooplankton. Hellberg et al. (2003) reported sudden acute losses of farmed salmon in Western Norway in 2002 associated with swarms of *Muggiaea atlantica*.

Besides the obvious direct loss of mortalities in aquaculture, the implications of zooplankton swarms

Table 2 Examples of gelatinous zooplankton associated with mortalities in farmed salmonids

Zooplankton species	Fish affected	Country/region	Details	References
Aurelia aurita (Scyphozoan)	*S. salar*	Norway	Farmed salmon	Bamstedt et al. (1998)
		Shetland	Farmed salmon	Bruno and Poppe 1996
		Ireland	Farmed salmon	Mitchell (personal observation)
Pelagia noctiluca (Scyphozoan)	*S. salar*	Northern Ireland	Approx. 250,000 fish killed	Doyle et al. (2008)
	S. salar	Ireland, Scotland	O'Connor (2002), Hay and Murray (2008)	
	S. salar	France	Significant mortalities in farmed fish	Merceron et al. (1995)
Muggiaea atlantica (Siphonphore)	*S. salar*	West coast of Norway	>100,000 farmed salmon, 2000 siphonophores/m^3	Fossa et al. (2003), Hellberg et al. (2003)
	S. salar	Scotland	Mortalities in farmed fish	Sourd (pers. comm)
Phialella quadrata (Leptomedusa)	*S. salar*	Shetland	1,500 fish died	Bruno and Ellis (1985)
		Norway		Bamstedt et al. (1998)
Cyanea capillata (Scyphozoan)	*S. salar*	Scotland, Ireland	Farmed salmon (90,000 mortalities in Ireland in 2004)	Bruno and Ellis (1985),
				Rodger (personal observation)
Solmaris corona (Narcomedusa)	*S. salar*	Scotland	Farmed salmon (650,000 mortalities in 2 days in 2002)	Sourd (pers. comm.)
				Bamstedt et al. (1998)
				Hay and Murray (2008)
Apolemia uvaria (Siphonophore)	*S. salar*	Sweden, Norway	Farmed salmon	Bamstedt et al. (1998)
				Rodger (personal observation)
Bolinopsis infundibulum (Ctenophore)	*S. salar*	Norway	Farmed salmon	Bamstedt et al. (1998)
Vellella vellella (Athecata)	*S. salar*	Ireland	Skin and gill pathology observed	Rodger (personal observation)

are far reaching. A considerable cost is incurred during retrieval and disposal of these dead fish. Zooplankton swarms increase stress in affected fish populations and the incidence of disease and possible secondary bacterial (*Tenacibaculum* sp.) infections. Reduced growth during and/or after exposure to swarms is possible. Costly mitigation actions may also be required. Loss of production potential may occur if emergency slaughtering is deemed necessary. Insurance premiums may increase following claims for damage due to plankton blooms and as there may be a preset limit for claims many smaller losses due to zooplankton go unreported.

Ferguson et al. (2010) have recently reported the association of the bacterial fish pathogen *Tenacibaculum maritimum* with the jellyfish *Philalella quadrata* and proposed that jellyfish could act as vectors of bacterial disease for farmed salmon. Certainly *T. maritimum* is commonly observed in, and on, fish gill (and skin) following some episodes of zooplankton damage (Rodger 2007). Other workers have also reported how jellyfish aggregations affect the bacterial populations in the marine environment (Riemann et al. 2006), however, it is not established what impact, if any, such would have on farmed fish in the vicinity of such a change.

Clinical signs associated with harmful zooplankton swarms

Clinical signs seen during a harmful zooplankton bloom are similar to those during a phytoplankton

bloom. Fish may be lethargic, swim close to the surface or exhibit bursts of jumping behaviour. They may also stop feeding (Bruno and Ellis 1985). There may be increased numbers of moribund fish and/or mortalities. Respiratory stress may be obvious and shaking of heads has also been observed (Rodger 2007). On examination of the gill filaments there may be mottling and discolouration and localised, punctate erosion of the gills, gill rakers and operculum (Figs. 2, 3). Other damage observed can include lesions on the skin and eyes (Fig. 4).

Mechanisms involved in harmful zooplankton swarms and fish interactions

Many gelatinous zooplankton possess epidermal cells known as cnidocytes which contain stinging structures (nematocysts) which can damage fish gills and skin (Barnes 1987). These nematocysts discharge when triggered and a filament penetrates the prey delivering protein toxins which paralyse and damage cells. Much interspecies variation exists in the type and number of stinging cells possessed. Also the chemical composition, potency and effects of the toxins released vary greatly between species. Cell death can then occur in contact areas and adjacent tissues are prone to secondary bacterial infection.

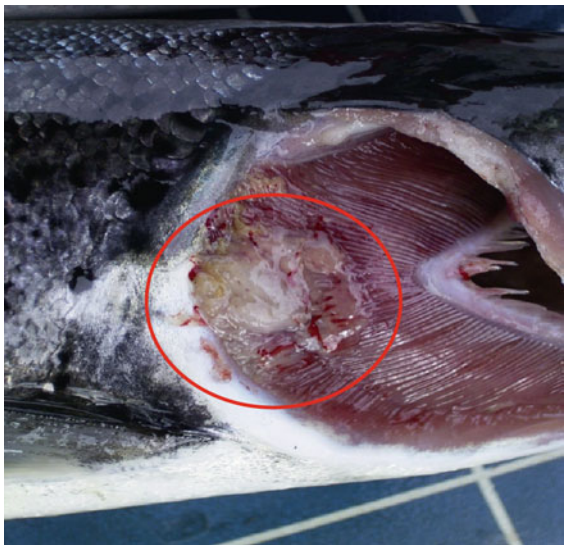

Fig. 2 Gills of farmed Atlantic salmon exhibiting patch of necrotic tissue (*ringed*) as caused by harmful gelatinous zooplankton

Fig. 3 Gills rakers of marine farmed rainbow (*O. mykiss*) trout exhibiting haemorrhagic necrotic lesions (*ringed*) due to harmful gelatinous zooplankton

Fig. 4 Nose, eye and skin ulceration and erosion of farmed Atlantic salmon due to exposure to swarms of *Pelagia noctiluca*

Experimental work undertaken by Helmholz et al. (2010) has elegantly demonstrated the gill cell toxicity of the cnidocyte extracts from the scyphomedusae *Cyanea capillata* and *Aurelia aurita* using rainbow trout gill cell line (RTgill-W1). The physical presence of high numbers of gelatinous zooplankton may in some cases give rise to physical clogging of gills and lack of availability of oxygen. This has been recorded with some species which do not have

nematocysts, such as *Bolinopsis infundibulum* (Båmstedt et al. 1998).

Histopathology associated with harmful gelatinous zooplankton

Focal necrosis of the gills, with haemorrhage, epithelial sloughing, discharge of eosinophilic granular cells and oedema have been associated with exposure of fish to harmful gelatinous zooplankton (Roberts and Rodger 2001). Occasionally the culprits may be observed in histological sections as in Fig. 5.

Mitigation of zooplankton swarms

The goal of mitigation strategies in the treatment or control of a zooplankton swarm is to protect public health, fisheries resources, aquaculture initiatives and marine ecosystems in general. Potential long-term strategies to minimise the impact of zoo-plankton swarms should include the reduction of eutrophication, reduction of over-fishing and minimisation of global warming (Richardson et al. 2009).

An early warning system of impending swarms is a desired aim of many aquaculture producers and good communication on the occurrence and intensity of blooms between farms is necessary. The pilot studies of early warning systems in Scotland (JEWWI and CAPTOR) as outlined by the Marine Scotland Marine Laboratory (www.frs-scotland.gov.uk) held promise but further work has not continued. Oxygenation and recirculation of deep 'bloom free' water around caged fish may help as would reduction in, or complete cessation of, feeding activities (Hay and Murray 2008). Movement of cages away from the swarm area could be considered if feasible. Prophylactic antibiotic cover for possible secondary infections may also be beneficial. Employment of protective nets, booms or bubble curtains may be considered worthwhile at high risk sites (Rodger 2007). Bubble curtain trials in a salmon farm in Co. Donegal, Ireland where monitoring of zooplankton was undertaken, revealed that a significantly greater density (20%) of plankton was recorded on the outside of the curtain as compared to the inside (Ratcliff 2004). Although this difference in density was considered positive, from a fish health perspective, it was not considered great enough to remove the threat of problematic species whilst blooming. Further, the energy costs of operating the bubble curtain were very high but nevertheless further trials with the bubble curtain were recommended (Ratcliff 2004). Hull-cleaning and ballast water cleaning protocols must be strictly enforced to prevent the spread of alien species into new ecological systems (Richardson et al. 2009). In marine farms the cleaning of pontoons and nets should be undertaken on a regular basis before heavy fouling with organisms such as hydroids occurs.

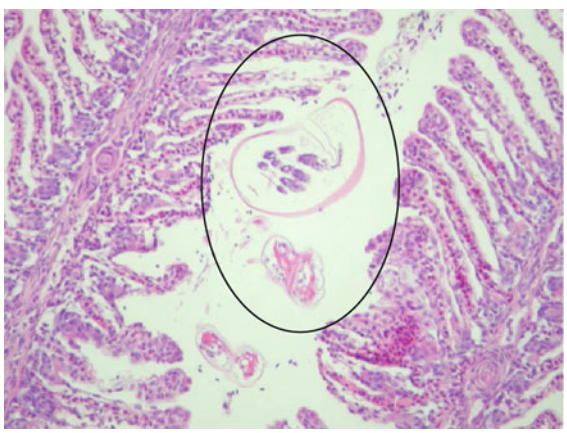

Fig. 5 Histopathological section of Atlantic salmon gills exhibiting hyperplasia, haemorrhage, lamellar fusion and necrosis of the epithelium with an unidentified zooplankton in the interlamellar space (*encircled*)

Improved methods for monitoring gelatinous zooplankton species are required to enable early warning of harmful species to all water users and stakeholders who may be affected. Although early warning systems are desired, they will only become really useful if there is an effective system already in place that can realistically protect the farmed fish from the harmful zooplankton blooms (or HABs). More research is required in the area of mitigation, especially in the design of bubble nets that have indications of benefits but have proven expensive to operate. There are significant knowledge gaps in area of the pathogenic zooplankton species for finfish, especially among the siphonophores and there is little or no work on the pathogenesis of the nematocysts and toxins on fish. There is a requirement for research to be undertaken into methods and means to reduce the impact of harmful zooplankton on farmed fish through dietary, physical or genetic means.

Other environmental aetiologies of gill disease

Medicines and remedies

Remedies used for the control of infectious disease have, in some cases, been associated with damage to the gills of the fish treated, for example, formalin (Speare et al. 1997), hydrogen peroxide (Kiemer and Black 1997) and chloramine-T (Sanchez et al. 1997, Powell et al. 1998).

Formalin treatments are used commonly on freshwater aquaculture farms, both prophylactically and against external diseases caused by parasites, bacteria and fungi. Formalin is rarely used in marine fish farms but is occasionally used for the treatment of external parasites. The morphometric and histological results presented by Speare et al. (1997) on gills of Atlantic salmon and rainbow trout did not represent significant gill pathology, despite testing repeated treatments with doses comparable with routine regimes used on farms. Histological changes observed in Atlantic salmon included a slight dose-related increase in lamellar fusion. Rainbow trout gills showed slightly more changes: fusion, inflammation and hyperplasia of filament epithelia. While both species showed an increase in mucus production there was no evidence of oedema or necrosis. Rucker et al. (1963) observed that rainbow trout were more susceptible to formalin than Pacific salmon.

Hydrogen peroxide (H_2O_2) may be used as a bath treatment for external parasites of salmon, such as sea lice, *Lepeophthirius salmonis* and *Caligus elongatus*. Kiemer and Black (1997) showed that there is a very narrow margin between the effective treatment range and concentrations which will induce gill damage and possibly death. Increasing temperatures reduce this error margin further and this is a disadvantage of the treatment given that sea lice reproduction and multiplication also increase with temperature. Gill histopathology shown experimentally in the same paper showed that the damage was not uniform. Areas of severe damage were side by side with healthy tissue. Secondary lamellae and the outer surfaces of gill arches were most severely affected. Pathology included hypertrophy, clubbing of secondary lamellae, mucous cell hyperplasia, hyperaemia, irregular surface of epithelial cells, haemorrhage, necrosis, fusion and lifting of the epithelial surface. The severity of pathology was consistent with length of exposure time and concentration of H_2O_2. As a result it is generally accepted that hydrogen peroxide use is safest when water temperatures are lower (<13°C). Speare et al. (1999) also demonstrated gill lesions in rainbow trout with exposure to hydrogen peroxide at 1,000–1,500 mg/l for 20 min and also at 750 mg/l. The pathology was characterised by foci of epithelial hyperplasia with lamellar fusion, pillar cell necrosis and pillar channel aneurysms. A significant decline in the number of lesions occurred over the 3 week sampling period following treatment. Hydrogen peroxide is used by land based industries as a disinfectant and as a bleaching agent and inadvertent discharge to waterways can result in significant fish mortalities and pathology (Fig. 6).

Chloramine T (N-sodium-Nchloro-para-toluene-sulphonamide) is commonly used prophylactically and as a treatment against bacterial gill disease and ectoparasitic disease in salmonid hatcheries in freshwater. It has also been investigated as a treatment for amoebic gill disease in marine salmonids (Harris et al. 2004). Several studies have been performed to determine the effects of this potentially toxic chemical on the gills of salmonids (Sanchez et al. 1997, 1998). Gill response seen by Sanchez et al. (1997) was limited to a change in the type of mucous cells predominating in the epithelia and hyperplasia of mucous cells. Dose levels and treatment durations

Fig. 6 Wild brown trout (*Salmo trutta*) exhibiting gill necrosis and corneal oedema after being exposed to hydrogen peroxide effluent from a paper mill in Scotland

used were similar to those used in the farm situation. The authors found that the type of mucus produced showed a change in composition from neutral to acid mucin. Anatomical changes monitored during this 11 week study included lamellar oedema, lamellar hyperplasia, lamellar inflammation, clavate lamellae, lamellar fusion, interlamellar inflammation and thrombus formation. It was found that control tanks had greater gill pathology than treated tanks, albeit minimal, and it was inferred that treatment may even improve the condition of gills in intensive rearing systems. Based on their results, Sanchez et al. (1997) also refuted the hypothesis that growth and feed conversion is inhibited by gill damage due to repeated chloramine-T exposure. However chloramine-T has been shown to have acute (within 12 h) respiratory and acid–base disturbances in fish (Powell et al. 1998). Sensitivity to the agent is also reported to be species dependant, with freshwater Atlantic salmon and rainbow trout *Oncorhynchus mykiss* having greater susceptibility than channel catfish *Ictalurus punctatus* (Powell and Harris 2004). Chloramine-T has also been shown to be more toxic to Atlantic salmon in seawater than in freshwater, although the main deleterious effect was seen to be the same for both environments, that is, extensive oxidative necrosis of the epithelium lining gill filaments and lamellae leading to acute disruption to osmoregulation (Powell and Harris 2004). Given this sensitivity to the treatment it is advisable to perform a bioassay prior to the administration of chloramine-T.

Eutrophication and pollution

Coastal eutrophication encourages the production of phyto-plankton and ultimately leads to phyto and zooplankton blooms (Glibert et al. 2002; Anderson et al. 2008; Richardson et al. 2009). Factors involved in this enrichment of nutrients include sewage, agriculture runoff and discharge as well as industrial effluent and discharge. In addition to their role in plankton enrichment of the marine ecosystem these factors also play a direct role in gill disease and mortalities in farmed finfish species (Liber et al. 2005). Damage to gill filaments or the salt secreting cells on the filaments decreases the ability of the fish to respire. This is important both in adult marine stage salmon and in smolts adapting to the marine environment (Clarke 1992).

Hydrogen sulphide (H_2S) may be produced naturally and under normal conditions in coastal waters but can also result from self-pollution under farm pens. Kiemer et al. (1995) describe the effects of this substance produced experimentally on Atlantic salmon smolts. Fish were exposed to sub-lethal levels of H_2S in either one of two regimes: chronic exposure (18 weeks) or acute exposure (14 days). In the chronic exposure cases an adaptive response was observed. Histological evidence of damage consisted of clubbing and thickening of the gill lamellae and increased numbers of mucous cells. Gill damage peaked after 6 weeks of exposure to the H_2S and gradual recovery of gills ensued after 8 weeks of exposure. On termination of the experiment at 18 weeks both exposed and control fish were healthy and normal, although exposed fish had slight thickening of the secondary gill lamellae. Acute exposure cases had major irreversible damage to the gill tissues. Extensive fusion of secondary lamellae was reported. In addition primary lamellae were thickened, the epithelial surface was rough, haemorrhages were apparent and there was separation of the epithelium from the underlying branchial vessels. Mucous cell numbers increased at the beginning and towards termination of the experiment but were almost absent in between.

The majority of references regarding toxicity of metals and pollutants to salmonid and non-salmonid fish and including gill pathologies relate to freshwater although Kroglund et al. (2007) assessed the effects of acidification and aluminium (Al) on survival of post-smolt Atlantic salmon in Norway. Exposures were of varying intensities and duration. It was found that the capacity for Atlantic salmon smolts to osmoregulate is extremely sensitive to Al concentration and that return rates of Al-exposed fish were reduced by between 20 and 50% when compared with control fish.

Nutritional aetiologies for gill disease

It has been shown that a number of nutritional deficiencies may lead to gill damage in farmed fish. This includes deficiencies in vitamin C, vitamin K, biotin and pantothenic acid (Waagbø 2008).

Histopathology associated with such nutritional changes includes lamellar degeneration or fusion. Haemorrhage in gills was one symptom reported in salmonids fed diets low in vitamin K (Taveekijakarn et al. 1996). Mæland and Waagbø (1998) demonstrated that several cold water marine teleost fish including Atlantic salmon, lack the ability to produce ascorbic acid, indicating the necessity for its inclusion in commercially formulated feeds.

The increasing cost of producing feed for the aquaculture industry based on fish meal has led to the use of alternative ingredients; however care should be taken to avoid the introduction of deficiencies into the diet. Maeland and Waagbø (1998) reported that the replacement of fish meal with spray dried hen egg white caused severe gill pathology due to a deficiency of biotin. This included fusion and thickening of the gill lamellae. It was shown that a protein contained in egg white, avidin, competes strongly with vitamins for binding on biotin and biocytin, thereby making the compounds unavailable for absorption.

Epithelial hyperplasia of gill lamellae has been shown to be characteristic of pantothenic acid (PA) deficiency (Poston and Page 1982; Karges and Woodward 1984). Wood and Yasutake (1957) described how the PA deficiency progressed with time as the hyperplasia extended from lamellar base to the distal filament tips. This was confirmed by work undertaken by Karges and Woodward (1984). Woodward (1994) suggested that the minimum dietary requirement of young salmonids for PA was 19.1 mg/kg of feed. Barrows et al. (2008) demonstrated pantothenic acid deficiency in rainbow trout fed a plant-based diet without added vitamins showed characteristic gill histopathology as described by Wood and Yasutake (1957) and Woodward (1994).

Genetic and congenital causes for gill disease

Shortening of the opercular cover of gills may be uni or bilateral. It results in the exposure of the gill filaments to the external environment and potential damage (Fig. 7). This can result in the shortening and thickening of filaments. On histological analysis such gills display varying pathologies including epithelial hyperplasia and lamellar fusion and are vulnerable to secondary bacterial infection. Fish with such a defect are unable to pump water across the gill effectively and are obliged to swim continuously in order to maintain a continuous flow of water past the gills (Branson and Turnbull 2008). In Atlantic salmon farms the cause for the deformity was considered to have a possible heritable factor as the defect appeared associated with specific strains of fish from certain regions however, the environmental conditions during egg incubation and first feeding may also have been a factor. The aetiology of this condition now appears, in many cases, to be related to elevated egg incubation temperature (>8°C; Baeverfjord et al. 1997; Ørnsrud et al. 2004).

Fig. 7 Lethargic pre-harvest farmed Atlantic salmon exhibiting significant opercular shortening

Sadler et al. (2001) investigated the prevalence of gill filament deformity syndrome (GFDS) of diploid and triploid Tasmanian Atlantic salmon. GFDS describes the absence of the primary gill filaments. These authors found that the skeletal deformity was an autosomal condition (non-sex-linked genotype). The deformity seen in larval fish was lethal. Triploid fish had markedly reduced gill surface area for gas exchange and acid–base balance compared with their diploid counterparts. This occurred whether gills were affected by GFDS or were completely normal. They concluded that the aetiology of the skeletal deformity was unclear, but that the reduced gill surface area would negatively impact on triploid fish under stressful or sub-optimal environmental conditions.

Summary and conclusions

In recent years greater interest in plankton blooms has led to the establishment of research projects that aim to increase scientific knowledge and be of benefit to science and industry alike. Examples of such initiatives include the GILPAT project in Ireland and others such as ECOHAB, a USA national research plan, GEOHAB, an international project concerned with Global Ecology and Oceanography of Harmful Algal Blooms, EUROHAB a European Commission funded initiative, and EUROGEL (European Gelatinous zooplankton: mechanisms behind jellyfish blooms and their ecological and socio-economic effects).

Development of early warning systems and the employment of oceanographic tracking models would be advantageous to predict deleterious coastal blooms. Such advance warning would allow for a wider choice of protective strategies and for greater time to put the chosen strategy in place. Devices such as LOKI (Lightframe Onsight Key species Investigation) have been developed with the aim of enabling the study of zoo-plankton distribution in direct relation to environmental parameters (Mengedoht et al. 2007). The predictive models and mitigation techniques employed to minimise the harmful effects of algal blooms are insufficiently advanced or developed to ensure the complete safety of commercial fisheries or aquaculture although examples such as the recent advances such as the underwater sensor developed by a team at NOAA's National Centres for Coastal Ocean Science and the Monterey Bay Aquarium Research Institute (Scholin et al. 2009) and the physical-biological model developed by a team at the Woods Hole Oceanographic Institution (McGillicuddy et al. 2003) hold much promise.

An example of potential biological control of jellyfish blooms is exemplified in the case of *Mnemiopsis* sp. blooms in the Black Sea in the early 1980's (Richardson et al. 2009). The ctenophore was accidentally introduced to the Black Sea probably through ballast waters from the USA. A second alien introduction of the ctenophore *Beroe* sp., again by ballast water, was fortuitous as the latter species is a voracious predator on *Mnemiopsis*. Combined with this event was a significant reduction in the use of agricultural fertiliser during the 1990's which reduced the effects of eutrophication and led to a significant reduction of biomass of *Mnemiopsis* sp. Zhou et al. (2009) comprehensively discuss the potential benefits and problems associated with using microorganisms in aquaculture, including their use in adjusting algal populations in a water body. Flocculant clays have been demonstrated to provide promising algal bloom control in Japan, Korea, China and the USA (Kim 2006) but environmental risk assessments should be carefully implemented to clarify the chronic impacts of clays on the marine ecosystem.

This review records the areas where work has already been undertaken into non-infectious gill disorders in the marine environment and also highlights the gaps in our knowledge. Areas that should be considered for further research and investigation as well as questions that arise include:

1. What is the pathophysiology and pathogenesis when gills are exposed to harmful zooplankton and phytoplankton?
2. Methods for the rapid identification of harmful zooplankton at farm level, especially the small species and hydroids (training of farm staff and vets will be an important aspect of this work)
3. Epidemiology of gill disease in finfish aquaculture
4. Screening of harmful phytoplankton and zooplankton species for specific pathogens i.e. *Tenacibaculum* and other bacteria and viruses
5. Is aeration or oxygenation post gill insult/challenge of benefit or otherwise?
6. Does dietary modification alter gill condition and can it help or hinder gill repair?
7. How do normal and abnormal bacterial gill floras compare and how does that change following an insult/challenge by harmful zoo or phytoplankton?
8. What genetic selection or markers can be identified to improve resistance to gill disease?
9. Do fouling organisms (hydroids) have any impact on farmed finfish gills?
10. What are the synergies between pathogens and non-infectious agents in the development of gill disease?
11. Although early warning systems are desired, they will only become useful if there is an effective system already in place that can realistically protect the farmed fish from the harmful zooplankton swarms (or HABs). More

Table 3 Selected non-infectious gill disease conditions with risk factors, methods of control and research priorities

Disease or syndrome	Critical parameters	Risk factors	Potential impacting factors	Methods of control, best practice to mitigate disease	Knowledge gaps and future research needs
HABs	Harmful algae	Geographical location	Genetics	Routine sampling for phytoplankton	Benefit or otherwise of aeration
		Environmental conditions	Bacteria on gills	Oxygenation/aeration?	Is gas supersaturation triggered in some blooms?
			Movement of fish, grading, etc.	Use of rapidly installed protective pen enclosures	Dietary modifications and benefits?
				Submersion of pens?	Use of biosurfactants?
					Use of clays?
HZSs	Harmful gelatinous zooplankton	Geographical location	Bacterial gill disease	Routine sampling for zooplankton	Predictive models needed
		Environmental conditions	Genetics?	Oxygenation/aeration?	Early warning systems (at mouths of bays)
			Size of fish?	Use of rapidly installed protective pen enclosures	Relationship of plankton with bacterial/viral pathogens
				Submersion of pens?	Significance of fouling organisms?
				Use of bubble curtains?	Challenge trials with suspect organisms.
				Towing of pens out of harmful bloom.	Improved mitigation of impact of swarms.

HABs harmful algal blooms, *HZBs* harmful zooplankton swarms

research is required in the area of mitigation, especially in the design of bubble nets that have indications of benefits but have proven expensive to operate.

Selected non-infectious gill disease conditions with their risk factors, potential impacting factors, methods of best practice to mitigate the disease, knowledge gaps and future research needs are shown in Table 3.

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