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Injuries and deformities in fish: their potential impacts upon aquacultural production and welfare

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Abstract Fish can be the recipients of numerous injuries that are potentially deleterious to aquacultural production performance and welfare. This review will employ a systematic approach that classifies injuries in relation to specific anatomical areas of the fish and will evaluate the effects of injury upon production and welfare. The selected areas include the (1) mouth, (2) eye, (3) epidermis and (4) fins. These areas cover a large number of external anatomical features that can be injured during aquacultural procedures and husbandry practices. In particular, these injuries can be diagnosed on live fish, in a farm environment. For each anatomical feature, this review addresses (a) its structure and function and (b) defines key injuries that can affect the fish from a production and a welfare perspective. Particular attention is then given to (c) defining known and potential aquacultural risk factors before (d) identifying and outlining potential short- and long-term farming practices and mitigation strategies to reduce the incidence and prevalence of these injuries. The review then concludes with an analysis of potential synergies between risk factors the type of injury, in addition to identifying potential synergies in mitigation strategies. The paper covers both aquaculture and capture-based aquaculture.

Keywords Aquaculture · Deformities · Fish welfare · Injuries · Production

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Introduction

Injuries and deformities can occur in both wild (Slooff 1982) and farmed fish (Matsuoka 2003) and can occur at any time from the larval (López-Albors et al. 1995) to the adult stage (Korsøen et al. 2009) of the life cycle. In this review, the term injury is defined as 'physical damage', whilst the term deformity is defined as 'an acquired or congenital distortion of an organ or part'

(Collins English Dictionary 1998). Injuries are direct damage to live tissue (Ellis et al. 2008), and deformities are abnormalities that can lead to functional impairment. Both can be detrimental to fish welfare (Huntingford et al. 2006) and production performance. The subject of fish welfare is gaining prominence amongst researchers, aquaculturists, retailers, quality assurance schemes, NGOs and also consumers (e.g. Huntingford et al. 2006). Recent years have seen the aquaculture industry taking an active role in employing strategies that incorporate fish welfare considerations into daily production practices, and a number of national and international legislations and policies provide specific guidelines on preventing and dealing with injuries during common aquacultural production practices (e.g. the Council of Europe recommendations for farmed fish, 2005 http://www.coe.int/t/e/legal_affairs/legal_co-operation/biological_safety,_use_of_animals/Farming/Rec%20fish%20E.asp). A specific EU Council Directive (98/58/EC) also outlines minimum standards for protecting farm animals, including fish http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=CELEX:31998L0058:EN:HTML.

Any good aquaculturist will attempt to protect their fish from potentially injurious situations as part of their general moral and ethical responsibility towards their livestock. In addition, from a purely production perspective, injuries can lead to reduced growth (Miyashita et al. 2000), reduced feeding (Dyková et al. 1998), reduced feeding ability (Kurokawa et al. 2008), increased susceptibility to infection (Turnbull et al. 1996) and increased levels of mortality (Cobcroft and Battaglene 2009). Injuries can also reduce the market value of farmed fish (e.g. Michie 2001; the Norwegian Industry Standard for Fish http://fhl.nsp01cp.nhosp.no/files/Quality_grading_of_farmed_salmon.pdf). Farmers therefore have a vested interest in reducing the occurrence and incidence of injuries in farmed fish. An additional incentive for reducing the prevalence of injuries can be gaining added value, as fish produced to high welfare standards can command a price premium (Olesen et al. 2010).

An important step for improving the welfare of farmed fish is to reduce the occurrence of injuries and deformities. This can be accomplished through a risk factor/welfare intervention approach where one identifies potential aquacultural risk factors in addition to outlining potential short- and long-term farming practices and mitigation strategies. By utilising this approach, a fish culturist can identify procedures or husbandry practices that may be detrimental to fish welfare and production performance, and then devise and implement a number of mitigation strategies to reduce or eliminate these risks. To the authors' knowledge, there is no published review covering this burgeoning field of research, and none have utilised this risk factor/mitigation strategy approach.

This review will classify fish injuries and deformities in relation to specific parts of the anatomy and the selected areas include the (1) mouth, (2) eye, (3) epidermis and (4) fins. These areas cover a large number of external anatomical features that can be injured during aquacultural procedures and husbandry practices. Most importantly, each injury can be diagnosed on live fish and can be easily identified by eye, or by simple veterinary procedures. The review will then conclude with a summary of potential synergies between risk factors and injury type, before identifying potential synergies in operational farming practices and mitigation strategies. The paper will restrict its scope to the aquaculture and capture-based aquaculture of teleostean fish, and citations will not be exhaustive due to space limitations.

Mouth injuries

Mouth structure and function

In fish, the mouth is primarily used to take in water (and in some cases, air) and also to ingest food (Guillaume and Choubert 2001). Its location can be (1) superior, opening dorsally; (2) inferior, opening ventrally; or (3) terminal and located at the front of the head. The mouth consists of an upper and lower jaw, and the oral cavity contains a tongue. The upper jaw bones are classified as the premaxilla at the front, and the maxilla at the sides, and each premaxilla is joined anteriorly by a symphysis, whilst the lower jaw consists of a dentary bone (McEachron and Fechhelm 1998). If the fish possess teeth, they are primarily used for prey capture (Guillaume and Choubert 2001).

Types of mouth damage

Mouth damage and deformities occur in wild fish populations (Slooff 1982) and in aquaculture, but are more commonly seen in cultured fish (Sadler et al.

2001). Their occurrence has been documented throughout the lifecycle, from yolk sac larvae to adults (e.g. Lein et al. 1997; Miyashita et al. 2000).

Mouth deformities include (1) pug-headedness (Matsuoka 2003), (2) lower jaw deformity syndrome LJD (Sadler et al. 2001), (3) double mouth (Swan 1968), (4) cross bite (Barahona-Fernandes 1982) and (5) gaping jaws syndrome (Pittman et al. 1990) along with many documented incidences of deformity not given specific names. Mouth deformities and damage can also include one or more of the following: elongation or lengthening of either the upper or lower jaw (Matsuoka 2003), shortening of the entire snout (Yamauchi et al. 2006), the bending downwards or sideways of the lower jaw (Okamura et al. 2007), total absence of an upper jaw (Sumagaysay et al. 1999), the fusion of jaws in an open mouthed position (Roberts et al. 2001), pinching or twisting of the upper and lower jaws (Fraser and de Nys 2005), and the presence of extra bones or lack of bones (Martinez et al. 2007). Deformities caused by mechanical damage include broken bones (Miyashita et al. 2000), mouth lesions and haemorrhages, tissue erosion around the mouth and snout deformation (Shiau and Suen 1992; Cobcroft and Battaglene 2009). Mechanical erosion and haemorrhaging have been observed on the snout of rainbow trout *Oncorhynchus mykiss* and on the snout and underside of the lower jaw in tank held Atlantic cod *Gadus morhua* (observations of authors). In addition to the immediate detrimental effects of such mechanical damage, its occurrence can also make fish susceptible to infection by pathogens (Barthel et al. 2003). In most cases, mouth deformities occur in conjunction with deformation to the operculum and spine of fish (Sadler et al. 2001).

In Europe, Atlantic salmon is one of the most important aquacultural species by both volume and market value (FEAP statistics; Anon 2008). Two mouth deformities previously highlighted as serious welfare issues for this species are LJD and pug-headedness (or brachygnathia; Sadler et al. 2001; Branson and Turnbull 2008). LJD is characterised by downward curvature of the lower jaw and ankylosis of mandibular articulation which results in the jaw being locked in a permanently open position (Bruno 1990). This deformity can occur in conjunction with other skeletal deformities including short, folded and sometimes softened opercula (Roberts et al. 2001). LJD in Atlantic salmon has been recorded in Scotland (Bruno 1990), Ireland (Quigley 1995), Canada (McGeachy et al. 1996), Norway (Sadler et al. 2001), Australia (Jungawalla 1991) and Chile (Roberts et al. 2001) and affects salmon in both freshwater and seawater (Sadler et al. 2001; Roberts et al. 2001). It affects both males and females (Sadler et al. 2001), and its prevalence is also much higher in triploid than in diploid stocks (Jungawalla 1991).

The second mouth deformity previously highlighted as a welfare issue for Atlantic salmon is pug-headedness and is characterised by a severe underdevelopment of the upper jaw, resulting in the appearance of a much protruded lower jaw (Bæverfjord et al. 1998a). Whilst the number of fish that suffer from this is normally low, there can be serious welfare implications for individual fish with the deformity (Branson and Turnbull 2008).

As stated earlier, mouth deformities can develop throughout the lifecycle, but most literature sources relate to the early life stages. Much of this research specifically focuses on the difficulties of developing suitable husbandry practices for larval and juvenile fish to allow aquaculture production of these species. The production of high value species such as bluefin tuna (Miyashita et al. 2000), striped trumpeter *Latris lineata* (Cobcroft and Battaglene 2009) and Japanese eel *Anguilla japonica* (Okamura et al. 2007) are hindered by the occurrence of mouth damage and deformation during the early life stages.

Effect of mouth injuries on production and welfare

Mouth damage and deformity can affect fish from both a production and a welfare perspective. In terms of production, reduced market prices due to the unappealing appearance of fish with mouth deformities present a major problem for industry (Sadler et al. 2001). Given that mouth deformities often inhibit a fish's ability to ingest food (Branson and Turnbull 2008), growth rates can also be adversely affected and survival rates reduced (Miyashita et al. 2000; Cobcroft and Battaglene 2009).

From a functional perspective, fish with mouth damage and deformations face two main issues resulting from the inability to properly open and close their mouths. The first is an impaired ability to feed such as that seen in fish suffering from LJD and pug-headedness (Branson and Turnbull 2008). In extreme cases, mouth deformities and damage can stop fish

from moving their mouths, leading to a total inability to eat, which can in turn leading to starvation and eventual death (Pittman et al. 1990). The second main problem facing these fish is breathing impairment resulting from a reduced ability to use buccal-opercular pumping to properly ventilate their gills. This again has been noted as an issue for fish with LJD and pug-headedness (Lijalad and Powell 2009). It is thought that fish with these mouth deformities may have to swim more than other fish in order to achieve sufficient ram irrigation to ensure adequate water flow over their gills. As a result, high stocking densities that can potentially inhibit swimming may further compromise the welfare of these fish.

It is still under debate as to whether fish can experience pain and suffering, and this is quite a controversial issue (Huntingford et al. 2006). Irrespective of this, previous research has shown that rainbow trout injected with a weak acetic acid solution or bee venom demonstrate behaviour and physiological changes suggesting discomfort (Sneddon 2003). These animals temporarily cease feeding, rest on the substratum and rub their snouts on the walls and base of their tanks. The use of morphine has been shown to reduce these adverse behaviours (Sneddon 2003). Whilst these findings may indicate simply a reflex response to potentially painful stimulus, i.e. nociception (Broom 1998) the possibility it indicates the presence of pain cannot be ruled out. With this in mind, damage to the mouth and jaws of fish should be regarded as a potential concern.

Further, it is likely that an inability to express normal behaviours may help explain the occurrence of mechanical damage to the mouths of cultured fish. In bluefin tuna culture, for example, night-time collisions with tank walls and cage netting are common during the juvenile and early adult stages (Ishibashi et al. 2009). In the wild, these fish would rarely encounter such obstacles as they are generally found in open pelagic waters.

Risk factors for mouth injuries

In the case of LJD in Atlantic salmon, two important risk factors have been identified relating to nutrition and genetics. Investigations into LJD in Chile indicated that its occurrence was associated with feed lacking sufficient phosphorus and vitamin C around the time of introduction to the sea (Roberts et al. 2001). In Australia, LJD has been linked with genetics, specifically ploidy and occurs more frequently in triploid than diploid fish. During the seawater phase, LJD has been shown to affect <30% of commercially produced all-female triploid Atlantic salmon (Jungawalla 1991). In addition, dietary deficiencies in phosphorus and vitamins A, C, D and K have also been suggested as causative factors for triploid fish (King and Lee 1993). In both Chile and Australia, it has been suggested that nutritional deficiencies may arise from the use of feeds designed for slower growing fish. This concept is supported in Chile where problems occur at a time of elevated temperatures with subsequently faster growth (Roberts et al. 2001).

Definitive risk factors for pug-headedness in Atlantic salmon have not been determined, but it has been suggested that the cause may be genetic or epigenetic (Slooff 1982). Two potential risk factors include egg incubation at inappropriate temperatures (Bæverfjord et al. 1998a) and exposure to heavy metals such as zinc (Slooff 1982). In addition to those already listed for LJD in Atlantic salmon, nutritional risk factors demonstrated in other species include diets deficient in niacin (Shiau and Suen 1992) and highly unsaturated fatty acids (HUFA; Kestemont et al. 2007). However, it is not only deficiencies that can cause problems. Excess vitamin A has also been shown to be a risk factor. In hatchery-reared flatfish, supplementation of diets with vitamin A is routinely used to stimulate pigmentation development. The problem is that excess dietary vitamin A can lead to high levels of its metabolite retinoic acid, which has been shown to dramatically increase the occurrence of mouth deformities (Suzuki et al. 2000).

The use of inappropriate rearing temperatures during the early development of fish is also a well-documented risk factor in the development of mouth deformities. During egg incubation and larval development, many fish are particularly susceptible to inappropriate culturing temperatures. Temperatures that are too high, too low or that fluctuate too much can potentially increase the incidence of jaw deformation in these early stages (Lein et al. 1997; Okamura et al. 2007). In addition to this, inappropriate salinities (Okamoto et al. 2009) and light intensities (Bolla and Holmefjord 1988) can also strongly increase the incidence of mouth deformities in these early life stages.

The presence of pollutants presents another risk. It has been reported that exposure to dioxins during the embryonic development of red sea bream *Pagrus major* can result in a shortened snout and deformity of the lower jaw (Yamauchi et al. 2006). Similarly, if the common bream *Abramis brama* is grown in polluted waters, they develop higher levels of mouth deformity, in most cases pug-headedness (Slooff 1982).

There are also many risk factors that specifically relate to the behaviour of fish and how they interact with the rearing environment. Self-inflicted mechanical damage such as that caused by physical contact with tank walls or cage nets is a major cause of mouth damage. In bluefin, tuna darkness is considered a major risk factor given that most collisions occur at night when these structures are not clearly visible (Miyashita et al. 2000), especially given the poor visual function of these fish in dim lighting conditions (Matsumoto et al. 2009). Flashing lights, loud noises and vibrations through the tank walls also cause fish to panic and increase collisions (Miyashita et al. 2000). Another risk factor relating to lighting is the length of dawn and dusk. It has been found that changing too quickly from night-time to daytime light intensities causes visual disorientation, thus increasing the risk of collision (Masuma et al. 2001). In the case of walling behaviour, it has been shown that for larval striped trumpeter, inappropriate tank coloration is a risk factor. The occurrence of walling behaviour and subsequent jaw deformations in this species changed depending on the colour of housing tank used (Cobcroft and Battaglene 2009). Collisions that do not result in instant death often cause injury to the snout and lower jaw (Miyashita et al. 2000).

Netting materials used to make sea cages and trawl nets may also represent a risk of mouth injury in both aquaculture and capture-based aquaculture via mechanical damage. The nets used to contain and capture fish present surfaces against which mechanical damage can occur. In bluefin, tuna fractures to the jaw bones often result from fish catching their mouths on nets (Miyashita et al. 2000). Nets may also cause abrasive damage in other species. This is possibly the case for Atlantic salmon, especially during the time immediately following transfer to sea. If the intensity of lighting in the freshwater hatcheries is markedly lower than that of the sea cages, these fish can exhibit light avoidance behaviours and push into the cage walls and base (Halls 1994). In this case, the cage netting and the increased light intensity may form a combined risk for mouth damage. In both sea cages and tanks, any overly abrasive structures or materials that fish may run into or rub up against also represent a risk for mouth damage. With respect to capture-based aquaculture, previous research has shown that Baltic cod *Gadus morhua* caught in trawl nets sustain damage such as lesions on their snouts and dark net marks behind their heads (Suuronen et al. 1996).

Mitigation strategies to reduce or eliminate risk factors for injury

In order to mitigate against mouth and jaw deformities and their resulting impacts upon welfare, it is important to ensure that where possible, the growing conditions are optimised in relation to the ecology of the fish. Mitigating measures against mouth deformities include ensuring the diets of fish contain appropriate levels of phosphorus, niacin, HUFA, vitamins A, C, D, K (Shiau and Suen 1992; King and Lee 1993; Roberts et al. 2001; Kestemont et al. 2007; Martinez et al. 2007). Rearing temperatures should be optimised to avoid the ranges where each species has proven susceptibility to increased mouth deformities (e.g. Lein et al. 1997). Rearing salinities (Okamoto et al. 2009) and light regimes (Bolla and Holmefjord 1988) that are known to reduce the occurrence of these deformities should be utilised. Fish should also be grown in water free from pollutants such as dioxins and heavy metals (Yamauchi et al. 2006), and where acute exposure to these pollutants occurs, rearing systems should be rapidly flushed with contaminant free water.

In the case of LJD in Atlantic salmon, mitigation measures include the adequate formulation of diets to ensure that nutritional requirements are met, especially when growth rates are higher than normal (Roberts et al. 2001). In the case of triploid fish, it has been suggested that these fish have different dietary utilisation and uptake abilities than diploid fish (Sadler et al. 2001). Mitigation measures may therefore require identifying any such differences and altering diet formulation for triploid fish accordingly.

In order to mitigate against mechanical damage resulting from physical contact with tank walls or cage nets, a number of measures may be appropriate. In bluefin tuna, the use of all-night lighting has been shown to reduce collisions. The removal of excess stimulation such as flashing lights, loud noises and

vibrations also helps reduce panic and subsequent collisions (Miyashita et al. 2000). The use of lighting strategies in Atlantic salmon hatcheries that minimise the difference between hatchery intensities and the ambient levels in sea cages may help prevent fish exhibiting light avoidance behaviours when first introduced to cages (Halls 1994). Walling behaviour can be reduced by choosing appropriate tank colours (Cobcroft and Battaglene 2009) or using turbid culturing conditions (Bristow and Summerfelt 1994). As mentioned earlier, appropriate choices of materials used for cages and trawl nets will also help mitigate against mouth damage through abrasion.

Eye injuries

Eye structure and function

The basic eye anatomy of fish is very similar to that of other vertebrates. The globe is maintained in position by three pairs of oculomotor muscles, which are attached to the sclera and are innervated by the third cranial nerve. The sclera is made of laminated fibrous layers reinforced with hyaline cartilage. Eyelids and lacrimal apparatus are not present as they are not needed in the aquatic environment (Branson 1993). The cornea is the anterior transparent window of the eye which permits a sharp image of the external environment to be displayed onto the retina. The cornea consists of the outermost epithelium, stroma and innermost endothelium layers. The corneal epithelium acts as a barrier to the fish's external aqueous environment (Ubels and Edelhauser 1987), and the integrity of the corneal epithelium is essential for normal vision. The cornea contributes to the structural integrity of the globe and protects the inner eye from invasion of unwanted organisms and environmental changes, whilst balancing the intra-and extra-ocular pressure (Collin and Collin 2001). The lens is generally spherical and protrudes through the iris, generating a very wide angle of view. The iris is fixed and has weakly developed sphincter muscles, possibly because fish rarely need to rapidly adapt to changes in light levels (Branson 1993).

Types of eye damage

Probably the most frequently described eye condition found in almost any fish health textbook is the condition of 'pop eye' or exophthalmia. Exophthalmia can be caused by many factors (Bouck 1980), but sometimes it can be caused by gas bubble disease. Characteristic signs of gas bubble trauma are gas bubbles in the blood, lateral line, gills filaments, fins, under the skin and of course, the eyes. Emboli and gas bubbles can only form when the sum of the dissolved gas pressures exceeds the sum of the hydrostatic pressure—simply put super-saturation of gas in the water (Bouck 1980). As a general rule, anything more than 110% air saturation will eventually cause gas bubble disease. If gas bubbles occur between the cornea and the lens of the eye, the eye may swell and protrude or 'pop' out (van Duijn 1973).

Other injuries can be a product of eye snapping. Eye snapping is an odd problem that happens when rays of light enter a tank and reflect off the eyes of fish. The reflected flash attacks from conspecifics and can result in one-sided periocular dermatitis, ulceration of the cornea and even loss of the eye. The angle at which light enters the tank in combination with fish distribution can favour damage to the right or left eye (Speare 2008).

A common form of eye damage is cataracts, which are opaqueness or clouding of the eye lens. Several reported factors can cause cataracts including nutritional deficiencies, toxic agents, parasites, exposure to ultraviolet light, hereditary factors, variation in water temperature and rapid growth (reviewed by Björnsson 2004).

Effect of eye injuries on production and welfare

There is concern that eye wounds can lead to secondary bacterial infections (Greaves and Tuene 2001) and that fish with eye injuries may also be subject to parasite infections. Eye flukes generally decrease the size of the eye lens, which can lead to deleterious effects like cataract formation; the amount of lens area covered by cataracts increases with infection intensity, and in some cases, eye fluke infection can cause blindness (Karvonen and Seppälä 2008). Other changes may also be noticeable with parasite infection, including increased ventilation rate, heart rate and swimming activity (Laitinen et al. 1996).

Regardless of the type or severity, eye damage has detrimental effects upon fish welfare as it can increase behavioural and physiological stress (Thatcher 1979).

At worst, most consider injuries to the eyes as potentially lethal (Ostrand et al. 2006). At best, defective eyes or visual impairment can decrease feeding ability and reduce avoidance behaviour as fish are not able to discern the exact location of their target, whether it be a pellet of food or avoiding a dip-net (Thatcher 1979; Mesa et al. 1994).

Risk factors for eye injuries

Common aquaculture husbandry and management practices such as handling and netting the fish during for example vaccination, grading and bathing procedures can cause eye injuries. For example, if many fish are netted or lifted at once, the weight of the fish bearing down can injure those on the bottom. Lift nets and different pumping technologies can cause injury (Grizzle et al. 1992), and even turbulent shear flow of water directed at fish from certain angles in turbines can cause damage to their eyes (Deng et al. 2005). Grizzle et al. (1992) found higher incidence of injuries such as abrasion and lacerations using a turbine pump and netting accompanied by an increase in injury related enzymatic activity (LDH and AST) in channel catfish. Interestingly, Grizzle and Lovshin (1994) later found that extreme turbine pump speeds (390 rpm vs. 330 rpm) resulted in a higher incidence of injuries in the same species.

Behavioural interactions can lead to eye damage, and eye injuries are a significant concern for halibut producers, given that aggressive behaviours including 'eye snapping' has been known to happen especially amongst juvenile halibut (Ottesen and Strand 1996). In a study of aggressive behaviour in farmed Atlantic halibut, Greaves and Tuene (2001) found that accidental or deliberate aggressive contact between fish primarily occurred during feeding periods, resulting in an estimated 3–5% of young halibut have at least one damaged or absent eye.

UV light exposure is a documented risk factor for cataract formation in a number of farmed species. For example, in a study where juvenile Atlantic cod were reared in recirculating aquaculture facilities, the frequency of cataracts significantly increased after UV water disinfection was utilised (Björnsson 2004). The increase was greatest in the tank closest to the UV light. Interestingly, UV light does not have to shine on fish directly so long as the water is treated with UV. UV-induced cataracts can also be a problem in farmed halibut (Treasurer et al. 2007). Cataracts are also a problem in farmed Atlantic salmon. Histidine deficiency has been suspected of being a contributing factor. It has been shown that increasing dietary histidine and adding 5% NaCl had decreased cataract frequency and severity (Rhodes et al. 2010). Fluctuating water salinity and increases in water temperature can also be factors that contribute to cataract development (Bjerkås and Sveier 2004). Brandt et al. (1986) noted corneal cloudiness and cataract formation following transport in largemouth bass *Micropterus salmoides*. Ubels and Edelhauser (1987) hypothesised that corneal opacity was due to the repeated contact of fish eyes with spines and scales of tank-mates, nets and tank walls during the high-density confinement typical of transport. Besides abrasions caused by collisions with other fish, abrasions can be caused by fish brushing against netting. Fish reared in net pens in marine or lake environments, especially, are contained within netted walls. Sometimes nets harbour hard-shelled organisms and fish become injured after swimming against them, losing scales and increasing their risk of developing bacterial and viral infections (Braithwaite and McEvoy 2005).

Eye flukes of the genus *Diplostomum* possess a stage in their life cycle where they infect the eyes of fish. *Diplostomum* penetrate the lens capsule and access the eye lens; this may cause lens material to leak into the surrounding tissues and cause a subsequent reduction in lens size. These parasites can also establish themselves in the lens, growing and developing for several weeks, during which time, they may damage the physiology of the lens and exploit energy resources that would otherwise be used by the host for normal eye functioning (Karvonen and Seppälä 2008).

Mitigation strategies to reduce or eliminate risk factors for eye injury

As the majority of the injuries described revolve around wounding, damaging or causing abrasions to the eye, it is logical that any husbandry or management practice involving the handling of these animals be carefully considered as to its design; the same applies to equipment. For instance, fish pumps, nets, sorting and grading apparatus come in a variety of materials and designs. Hence, there has been much commercial interest in identifying the methods that reduce the possibilities of damage when moving fish. Moving and

transferring fish by pumping them in pipes where they are not removed from water has been considered a less damaging option than netting (Conte 2004) as the pump can move large quantities of fish quickly and without injury. One such pump is a hidrostal pump—a screw-type centrifugal impeller. Helfrich et al. (2004) looked at the effects of live transport on mortality, descaling and injury rate in striped bass *Morone Saxatilis* and rainbow trout using this pump and found the frequency of eye injuries was 2.8% for bass and 0% for trout. Similar studies of large hidrostal pumps on other species of fish, such as splittail *Pogonichthys macrolepidotus* and Chinook salmon *Oncorhynchus tshawytscha,* have yielded comparable results (Helfrich et al. 2001). Helfrich et al. (2004) have therefore suggested that large hidrostal pumps be used to transport live fish at high densities.

Greaves and Tuene (2001) have suggested that injuries can be minimised by reducing aggression around meal times. This can be achieved by dispersing a satiation ration over a wide surface area for example by using appetite-based demand feeding systems. This should diffuse the sudden burst of fish activity and reduce the occurrence of competition, aggression and accidental fish collisions. In addition, manipulating how light or shade hits the top of a tank may reduce or prevent eye-snapping behaviour. With regard to controlling eye fluke infections, it has been suggested to eliminate water snails (the intermediate host in *Diplostomum*'s 3-host life cycle). Molluscicides, filtration and removal of vegetation can help (Southgate 1993). The risk of inducing corneal opacity during live transport can be minimised by regularly monitoring for corneal abrasions in fish operations as abrasions can easily be detected by touching a fish's eye with a sodium fluorescein ophthalmic strip (Ubels and Edelhauser 1987).

Finally, although cataracts can cause serious problems in an array of farmed species, studies have shown that there are different strategies to combat this condition. Nutritional, toxic, parasite, UV light, hereditary and environmental factors have all been associated with cataract formation (reviewed in Björnsson 2004); therefore, it is important to monitor these aspects. Limiting the amount of exposure to direct and indirect sources of UV light has been shown to reduce cataract development (Björnsson 2004), and these authors suggested that systems are designed so that they allow the UV-treated water to first flow though a large enough reservoir tank before flowing into rearing tanks. Another way to help mitigate nutritionally related cataract formation is to balance the diet (Waagbø et al. 2003). Parasite-induced cataracts (Karvonen et al. 2004) may be alleviated by rapidly treating parasite infections. In addition, maintaining an optimal water temperature (e.g. avoiding warm water) and preventing temperature fluctuations can also help to prevent the development and severity of cataracts (Bjerkås et al. 2001).

Epidermal injuries

Epidermal structure and function

The epidermis of fish mainly consists of two types of cells: filamentous cells and mucous production cells. They are arranged according to three distinct but continuous zones (basal, medial and peripheral) of the fish epidermis (Harris and Hunt 1975). The epidermis represents a biological barrier between the fish and the aquatic environment, conferring protection against infectious diseases (Ellis 2001), friction (Daniel 1981) and pollution, and it is actively involved in ion regulation (Hoar and Randall 1969).

Types of epidermal damage

Skins injures and lesions have been defined as a visible loss of epidermis accompanied by changes in skin colour, haemorrhages or ulcers involving dermal, subdermal and/or muscle tissues occurring in any part of the body (Vågsholm and Djupvik 1998). Skin lesion also includes papillomatous lesions characterised as uni- or multi-focal mass of hypertrophied skin, absence of hyperplasia and ulceration (Ottesen et al. 2007).

Effects of epidermal injuries on production and welfare

From a production perspective, a direct decrease in the average price of 2% occurs when injuries are present in 10% or more of the fish (Vågsholm and Djupvik 1998). Also, consumers are currently demanding high-quality seafood products, and gross appearance, including skin, appears to be a quality selection parameter (Huidobro et al. 2000). There is no direct

association between skin injuries and fish mortalities, but several bacterial infections such as winter ulcers (Løvoll et al. 2009), piscirickettsiosis (Smith et al. 1999) or infectious salmon anaemia (Totland et al. 1996) can colonise the initial lesion and lead to mortalities. For example, mortalities due to winter ulcers are usually <10%, but can also lead to decreased fillet quality at slaughter, having a major dual impact on the productivity of a farm (Løvoll et al. 2009). Also, infectious salmon anaemia outbreaks have been associated with skin lesions (Nylund et al. 1994) and injuries which occur previous to or concurrently with sea lice infestations of *L. salmonis* and *C. elongatus* have been implicated with high mortalities (Boxshall and Defaye 1993).

Bacterial skin infections and parasitic infestations can be treated with antimicrobials or antiparasitic drugs whose purchase directly increases production costs or can increase production losses through reduced appetite and increased mortalities. This reduced appetite or anorexia can lead to reduced growth (Vågsholm and Djupvik 1998).

Skin injuries and lesions have a direct effect upon nociception, as fish possess free nerve cells close to and throughout the skin surface (Kotrschal et al. 1993). In addition, skin injuries have a direct effect on disease susceptibility and are a fertile environment for the proliferation of bacterial and viral diseases and perpetuation of the skin damage (Nylund et al. 1994).

Risk factors for epidermal injuries

Several aetiological agents for epidermal injuries have been identified, and these can be divided into abiotic and biotic factors (Tørud and Håstein 2008). Causes of skin injuries related to common husbandry practices such as sorting, pumping and netting have been investigated in several farmed fish (see earlier section on eye injuries). The main skin lesions associated with these practices were skin abrasions characterised by a discontinuity of the epidermis and accompanying subcutaneous haemorrhaging. Mechanically induced skin injuries have also been described in the abocular side of flat fish such as Atlantic halibut and southern flounder *Paralichthys lethostigma* under commercial conditions (Ottesen and Strand 1996). In Atlantic halibut, smooth tank surfaces have been associated with the occurrence of papillomas, characterised as masses with multiples nodules and skin erosion consisting mainly of eroded epithelium and hyperplasic areas (Ottesen et al. 2007). Further, skin lesions in Atlantic salmon have been associated with vaccination against *Vibrio anguillarum*, *Vibrio salmonicida* and *Aeromonas salmonicida* sp *salmonicida* using plant oils as vaccine adjuvants instead of mineral oils (Vågsholm and Djupvik 1998). The same authors investigated other risk factors in Atlantic salmon at the slaughter line and found a higher risk of skin injuries in smaller fish, in fish slaughtered in summer or fish that have been held under an extended seawater growth phase.

Other management practices such as preventive or therapeutic formalin baths can cause thinning of the epidermal strata (Sanchez et al. 1998), which leads to mucous cell opening (Buchmann et al. 2004) that can be associated with bacterial infections such as *Flavobacterium psychrophilum* (Madsen and Dalsgaard 1999) that perpetuate the initial skin lesions. High mucosal cell differentiation and openings are found using high doses (200–300 ppm for 1 h) or low doses for longer time periods (50 ppm for 24 h; Buchmann et al. 2004). Ultraviolet radiation has been identified as a risk factor for the development of skin lesions in broodstock rainbow trout in Bolivia (Bullock and Coutts 1985) and during the summer season in Atlantic salmon in Ireland (McArdle and Bullock 1987). Presumably, latitude (associated with season) could also be considered as a risk factor in aquaculture farms located in the southern hemisphere where the protective atmospheric ozone layer is thinning as this has been associated with detrimental effects on other aquatic organisms (Villafane et al. 2001).

Biotic risk factors for skin injuries are varied and can include bacterial, parasitic and fungal fish pathogens, and predators (Vågsholm and Djupvik 1998). *Moritella viscosa* has been described as one of the major bacterial fish pathogens that can cause winter ulcer syndrome, characterised by skin injuries and lesions in Atlantic salmon, Atlantic cod, turbot *Psetta maxima* and Atlantic halibut (Lunder et al. 1995; Björnsdóttir et al. 2004; Løvoll et al. 2009). The clinical signs of the disease appear during the cold season when water temperature drops below 10°C. Associated skin injuries can cover up to one-third of fish body and are characterised by severe ulcerations exposing subcutaneous and muscle tissue accompanied with liquefactive necrosis and haemorrhaging of

the surrounding tissues. Parasitic infestation such as sea lice (*Lepeophteirus salmonis*, *Caligus elongatus* and *Caligus rogercresseyi*) can cause severe itching, skin irritation and ulcerations in farmed Atlantic salmon and trout (Boxshall and Defaye 1993). Risk factors include high stocking densities, hydrological factors (tides and water current) and delays to implement biosecurity measures such as cage and farm disinfections (Hardy-Smith 2006). Further, skins injuries and ulcers can be caused by *Saprolegnia* spp. colonisation of the epidermis (Dykstra et al. 1989). Other biological causes of skin lesions in farmed fish include stings from jellyfish blooms leading to ulceration and subsequent secondary bacterial infections (Tørud and Håstein 2008). In addition, in farmed olive flounder, the parasite *Philasterides dicentrarachi* infiltrates the epidermis, producing skin injuries and lesions histologically characterised by the presence of necrosis, red blood cells and a low inflammatory responses with few monocytes, macrophages and thrombocytes (Jin et al. 2009).

Mitigation strategies to reduce or eliminate risk factors for epidermal injury

Potential operational interventions to decrease skin injuries and lesion include mitigation of contributing risk factors and causes. As such, the use of vacuum pumps to transport or sort fish into well boats should be encouraged, as well as the use of intermediate pump velocities whenever turbine pumps are employed. Similarly, tanks with rigged floors should be used in flatfish aquacultural activities. Chemical baths used for the treatment or prevention of diseases should be utilised in low quantities and for the shortest time possible (Buchmann et al. 2004). Whilst prevention of infectious disease is important, farmers should use vaccines based on mineral oil coadjuvant instead of plant oil in order to decrease the risk of skin injuries (Vågsholm and Djupvik 1998). Vaccine development companies should also be encouraged to change their coadjuvant and demonstrate no skin injuries after inoculation. A well-implemented biosecurity plan should be set-up to decrease the spread of fish infectious diseases associated with skin injuries. Finally, although environmental factors such as water temperature, water currents and tides and ultraviolet exposure are difficult or impossible to control in cage aquaculture, these factors should be taken into account when setting up new farm sites or expanding existing farm production facilities.

Fin injuries

Fin structure and function

The teleost fin consists of an epithelial layer or fold of skin supported by fin rays, which form part of a larger skeletal structure (see Videler 1993). The fin rays are controlled by a series of depressor, erector and inclinator muscles (see Winterbottom 1974) and are connected to each other and the endoskeleton by collagenous fibres. The base of the fin rays can be individually rotated (Westneat et al. 2004), and fish can also adjust the curvature of a fin ray (Standen and Lauder 2005), allowing a fish to rotate, spread, undulate, elevate and depress a fin (Videler 1993) in relation to manoeuvring requirements or environmental disturbances such as variable flows or currents.

It is widely accepted that the primary functions of fins are to help a fish to control its posture, and to generate and control propulsion during locomotion (e.g. Standen and Lauder 2005). For example, the caudal fin of teleosts is primarily used to generate forward locomotion, but may also have a role in generating lift (Lauder 2000). The dorsal fin can enhance stability and can also generate thrust in salmonids (Drucker and Lauder 2005), and its elevation or extension may also be used to communicate status during aggressive contests (e.g. Abbott and Dill 1985). The role of the adipose fin in salmonids is somewhat uncertain, but it has a hypothesised role in locomotion (Drucker and Lauder 2005). With regard to the paired fins, the pectoral fins can be utilised in braking manoeuvres, turns and can also aid stability in salmonids (Drucker and Lauder 2003). Both the pectoral and pelvic fins can be used to augment station holding in juvenile salmonids (Arnold et al. 1991), whereas the pelvic fins can be used by males to grasp females during reproductive rituals and also during intra-specific aggressive encounters between males in Atlantic cod (e.g. Brawn 1961).

Types of fin damage

Fin damage has been recorded in wild fish, especially in degraded habitats (see Latremouille 2003 for a

review) but is more common under aquaculture conditions (Bosakowski and Wagner 1994). Fin damage can be described in numerous ways, but the basic forms of damage can be divided into three categories: (1) splitting, (2) erosion and (3) thickening (e.g. MacLean et al. 2000). An additional form of fin damage can also be malformed fins (Turnbull et al. 1996). Splitting refers to splits or clefts in epithelial tissue between fin rays. It appears to be a relatively mild form of fin damage that leaves the skeletal fin rays intact and undamaged. Erosion refers to damage that results in loss of both the epithelial fin tissue and also the whole or part of the fin ray (Turnbull et al. 1996). Thickening refers to nodular, opaque thickening along the distal edge of an affected fin. This thickening appears to be the result of a hyperplasia in the fin epithelia and may contain some necrotic cells. Deformed or malformed fins contain twisted or abnormally branched fin rays that can lead to folding or twisting of the entire fin structure (see Turnbull et al. 1996). All forms of fin damage may result in diffuse haemorrhaging throughout the fin tissue or focal haemorrhaging in proximity to the fin base.

Fins have a capacity for healing and regeneration, and the regeneration process has been extensively reviewed by Akimenko et al. (2003). In brief, epithelial healing begins with the rapid migration of epithelial cells to form a wound epidermis. The formation of a blastema then allows the re-growth of fin rays and connective tissue, permitting fin regeneration (Akimenko et al. 2003). Regeneration may not be a straight forward process, as regenerated fins may also contain twisted fin rays, or may exhibit abnormal branching patterns (Turnbull et al. 1996). Previous research has reported that fins may not regenerate if there is complete erosion of fin ray and epithelial tissue (Goss and Stagg 1957). However, a recent study (Shao et al. 2009) reported that four species of fish including zebrafish *Danio rerio* and koi carp *Cyprinus carpio* can regenerate an amputated caudal fin, even when the amputation is at the level of the endoskeleton.

Effect of fin damage on production and welfare

Fin damage can affect fish from both a production and a welfare perspective. Fin damage may have a detrimental effect upon two common production performance parameters, growth and survival and may also affect product quality. It may also impact upon welfare due to being a direct injury to living tissue (Ellis et al. 2008) and may increase susceptibility to infection and potentially reduce swimming ability.

From a production perspective, there is no published, quantifiable data on the effect of different types of fin damage upon product quality. However, some authors have implied from personal experience or unpublished data that fin damage reduces the market value of an affected fish from both table fish processors and live fish buyers for re-stocking purposes (e.g. Hoyle et al. 2007). Further, fin damage can be used as a potential quality indicator (Winfree et al. 1998). These statements are supported by literature from industrial sources and quality assurance schemes. For example, the processing and quality grading scheme of the Alaska Quality Seafood Program http://www.alaskaqualityseafood.com/pdf/h_g_specifications.pdf, incorporates both fin loss and fin erosion into its quality classifications. In addition, the Norwegian Industry Standard for Fish http://fhl.nsp01cp.nhosp.no/files/Quality_grading_of_farmed_salmon.pdf also incorporates the presence/absence of fin damage into their quality scheme. Further, the farmer's standard quality assessment procedure, the Quality Index Method (QIM), incorporates fin haemorrhaging into its assessment criteria (Knowles et al. 2007).

A concise and well-researched review of the potential effect of fin damage upon fish welfare was carried out by Ellis and his co-authors in 2008, and they highlighted the lack of quantified studies that have directly investigated the effects of fin damage upon fish welfare. In the absence of this direct evidence, a number of associative inferences must be drawn from other studies.

To the authors' knowledge, the effect of fin damage upon on-farm growth performance and survival has not been documented. However, the potential effects of fin damage can be inferred from tagging studies investigating the effect of fin clipping upon growth and survival. The effects upon growth and survival are dependent upon the type and number of fins removed. For example, the removal of a single fin does not reduce growth in rainbow trout, but does reduce survival, and the level of mortality depends upon the type of fin removed (Nicola and Cordone 1973). Further, the survival of fish with multiple fin excisions is lower than those which have a single fin removed (Mears and Hatch 1976). In a study by Coble (1967),

the growth performance of yellow perch *Perca flavescens* was only affected when the pectoral fin was clipped. The causes and mechanisms responsible for reduced growth and survival have not been elucidated in the above studies.

Fin damage is a direct injury to living tissue (Ellis et al. 2008), and this tissue contains nerve cells both within and between fin rays (Roques et al. 2010). These nerve cells contain C-fibres and A-δ fibres that are involved in pain perception, and Roques et al. (2010) have validated an acute response to a painful stimulus, caudal fin clipping, in Nile tilapia *Oreochromis niloticus*. This response was quantified via increased swimming activity up to 6 h after the damage occurred and also transient increases in both gill Na+/K+-ATPase activity and branchial mucus cell activity 1 h after clipping. Irrespective of the debate on whether fish feel pain or not (see Yue Cottee in press), the fins are nociceptive (Chervova 1996) and are capable of detecting noxious stimuli via the nervous system. Fin damage may also be detrimental to fish health. For example, fin damage may increase a fish's susceptibility to infection via opportunistic pathogens such as *Aeromonas salmonicida* which causes furunculosis (Turnbull et al. 1996). The base of the fin is also a documented entry point for infectious haematopoietic necrosis virus, IHNV (Harmache et al. 2006).

One could surmise that fin damage could reduce fin function and have a detrimental effect upon a fish's capacity to control its posture or reduce its swimming ability (as noted in Barthel et al. 2003) during routine swimming or feeding. However, this would depend upon the severity and type of fin damage (erosion, splitting) and also the frequency and type of fins affected. For example, recent research on the effect of experimentally induced pectoral fin erosion upon the prey-capturing ability of bluegill sunfish found that a reduction in 35% of pectoral fin area did not have an effect upon maximal swimming velocity or braking capacity during prey capture (Higham et al. 2005). The authors suggested bluegill sunfish make behavioural compensations to adjust for reduced pectoral fin size. This same result was also noted in muskellunge *Esox masquinongy* that were subject to clipping of one or both pectoral and pelvic fins or all paired fins (Wagner et al. 2009). However, complete loss of pectoral fins due to experimental amputation can affect fish in other ways for example by reducing the station-holding capacity of Atlantic salmon parr (Arnold et al. 1991).

Risk factors for fin damage

It should first be noted that there are a multitude of factors that may cause or aggravate fin damage amongst farmed fish, and these factors can interact with others to exacerbate this damage. In addition, one risk factor may mask or be related to another for example ration size can affect fin damage (Moutou et al. 1998), but this may be due to competition and increased levels of aggression. For additional summaries of the numerous non-aquacultural factors implicated with fin damage, a reader can refer to two previous reviews (Latremouille 2003; Ellis et al. 2008).

Both the choice of rearing system and the choice of its construction material can affect fin damage. For example, when rainbow trout are reared in re-circulating aquaculture systems versus flow-through tanks, fish in the re-circulation systems had greater pectoral and dorsal fin erosion than their counterparts. The authors suggested this was due to changes in tank flow dynamics (Roque d'Orbcastel et al. 2009). Concrete raceways also lead to greater ventral fin erosion in rainbow trout than raceways that have cobbles incorporated into their substrate (Wagner et al. 1996). Baffles in raceways also lead to greater pectoral erosion (Barnes et al. 1996). Recent research on the effects of submersible cage culture upon fish performance and welfare reported that short-term submersion (ca 3 weeks) has no effect upon fin damage (Dempster et al. 2009), whilst longer-term submersion (>6 weeks) is detrimental to fin condition in Atlantic salmon (Korsøen et al. 2009). The choice of light regime can also influence the incidence of fin damage. When silver catfish *Rhamdia quelen* are held under 24 h darkness, they exhibit no fin damage in comparison with those held under 24 h light or a 10:14 LD cycle (Piaia et al. 1999).

There are numerous husbandry practices that can be detrimental to fin damage such as transferring fish using a turbine pump rather than a vacuum pump (Grizzle et al. 1992) and handling fish with knotted instead of knotless nets (Barthel et al. 2003). Feeding practices and the nutritional content of feed can also influence the incidence of fin injuries. Numerous studies have shown that underfeeding can increase the prevalence of fin damage (e.g. Moutou et al. 1998), whereas others have reported no effect (e.g. Klontz et al. 1991). Feeding frequency can also be a risk factor

for fin damage. When rainbow trout receive a daily satiation ration from self-feeders, reducing feeding frequency from 3 meals to 1 meal a day significantly hinders recovery from historical dorsal fin erosion (Noble et al. 2007a). However, when fish receive a fixed satiation ration, an increase in the feeding frequency from 1 meal to 3 meals a day increases pectoral fin damage in rainbow trout (Rasmussen et al. 2007). The authors' attributed this increase to escalated competition around repeated feed events. Numerous studies have also reported that diet formulation, such as the choice of fishmeal versus krill as a protein or lipid source, can increase fin damage (Lellis and Barrows 1997).

The relationship between stocking density and the incidence of fin damage is somewhat unclear and may be related to other factors such as poor water quality or feed access. Some studies have suggested fin damage escalates with increasing stocking density in rainbow trout (North et al. 2006) and sea bass (Person-Le Ruyet and Le Bayon 2009), but others have reported that stocking density has no effect in Atlantic salmon (Hosfeld et al. 2009). The potential detrimental effects of stocking density can also depend upon the species under investigation, as previous work by Siikavuopio and Jobling (1995) has shown that fin damage decreases at increasing stocking densities in Arctic charr *Salvelinus alpinus*. Further work by Rasmussen et al. (2007) found that the effect of stocking density upon fin damage differs depending on which fin is examined. At low densities, the anal fin of rainbow trout was in better condition than the anal fins of fish under higher densities, whereas the dorsal fin was in better condition at high compared to low densities. However, group living does appear to be detrimental to fin damage, as individually held fish can have better fins than those held in groups (Winfree et al. 1998).

Other abiotic factors such as temperature can affect the prevalence of fin damage amongst farmed fish, but the effects again differ with species. Fin damage increases in rainbow trout (Winfree et al. 1998) and sea bass (Person-Le Ruyet and Le Bayon 2009) when temperatures are high, whereas it increases in Atlantic salmon when temperatures are low (Schneider and Nicholson 1980). Wagner et al. (1998) found no clear effects of temperature upon fin damage in Bonneville cutthroat trout, *Oncorhynchus clarki*. Oxygen saturations greater than 100% have also emerged as a risk factor for fin damage in sea bass (Person-Le Ruyet and Le Bayon 2009). Exposure to high light intensity can lead to sunburn and increased damage to the upper caudal and dorsal fins (Bullock 1988). Bosakowski and Wagner (1994) reported increased fin erosion in trout exposed to low alkalinity and high ammonia levels.

Triploid rainbow trout exhibit less fin erosion than diploids (Wagner et al. 2006), and genetic strain can also affect the incidence of fin damage, with albino fish exhibiting less erosion than their pigmented counterparts (Wagner et al. 1996). Sexual maturity can also play a role in the degree of fin damage exhibited by fish. Mature Atlantic salmon male parr (precocious parr) exhibit less fin erosion than immature parr (Mork et al. 1989), but this may be related to behavioural adaptations on the part of mature precocious males to avoid conflict with conspecifics. Intraspecific competition in the form of overt biting has also been identified as a risk factor for dorsal fin damage (Cañon Jones et al. 2010). Physiological stressors may also play a role as cortisol-treated fish exhibited greater fin damage than their counterparts in experimental studies on rainbow trout (Gregory and Wood 1999). Further, parasites such as sea lice, *Caligurus* spp., have also been reported as risk factors for fin damage (Dawson 1998).

Mitigation strategies to reduce or eliminate risk factors for fin damage

In order to mitigate against fin splitting, thickening and erosion, it is important to identify and define straightforward operational strategies that can be carried out either during the setting up of a farm or during its daily operations. The correct choice of tank, net or raceway construction materials can reduce the prevalence and severity of fin erosion. Incorporating cobbles into the substrate of concrete raceways reduces erosion of the ventral fins (Wagner et al. 1996). Introducing some form of environmental enrichment, such as branch structures, overhead cover may also improve fin condition in steel head *Oncorhynchus mykiss* (Berejikian and Tezak 2005). With regard to submersible cage culture, Atlantic salmon cages should only be fully submerged for less than 3 weeks without surface exposure (Dempster et al. 2009). Where possible, water current should be increased in the rearing unit as this reduces fin damage in Arctic charr and Atlantic salmon (Jobling et al.

1993). This has the added benefit of exercising the fish, which can also improve fin condition (Jørgensen and Jobling 1993). In net culture, cages could be located in high flow areas to achieve the same effect, or a farmer could deploy recently developed technologies that induce fish swimming and exercise by utilising their optometric response.

To reduce the amount of fin damage that daily husbandry practices can inflict upon their fish, farmers should use knotless nets when handling their fish (Barthel et al. 2003), and limit the frequency of handling wherever possible. Further, during fish transfers, fish should be pumped rather than netted and fish should be pumped using vacuum pumps rather than turbine pumps (Grizzle et al. 1992). Feeding regimes that incorporate responsive rations via demand feeding technology can reduce fin damage in both cage and tank culture (Noble et al. 2007a, b; Suzuki et al. 2008), and this can in turn improve welfare. These demand feeding systems allow fish to dictate the size, frequency and timing of their daily ration and can be tailored for each rearing system (e.g. Blyth et al. 1993; Alanärä 1992). Formulating diets that match the nutritional requirements of the fish will also reduce the capacity for fin damage within a rearing system, such as replacing fish meal with a krill based diet in rainbow trout (Lellis and Barrows 1997).

Growing the fish within suitable ranges of (1) temperature (e.g. Person-Le Ruyet and Le Bayon 2009), (2) oxygen saturation (Person-Le Ruyet and Le Bayon 2009) and (3) light intensity (Bullock 1988) can decrease the risks for fin damage. This can be achieved through improved oxygen monitoring to maintain levels at an optimum. Raceways can be covered or shaded to reduce light intensity levels or reduce the risk of sunburn if this emerges as a risk factor. Increased water exchange rates will also reduce the risk of fin damage (Good et al. 2009) and will also reduce the risk of a build up of ammonia levels or reduce the risk of low alkalinity (Bosakowski and Wagner 1994).

Triploid rainbow trout also exhibit less fin erosion than diploids (Wagner et al. 2006) and can also regenerate their fins faster than diploids (Alonso et al. 2000). Hybrid fish also exhibit less caudal erosion than their pure strain counterparts (Clayton et al. 1998 in Latre 2003), so this may be an option to some farmers. Finally, research investigating fin damage in both duo- and mono-culture reported a reduced incidence of damage under duo-culture conditions for Atlantic salmon and Arctic charr (Holm 1989; Nortvedt and Holm 1991). However, this is dependent upon the species selected as fin damage can be unaffected (e.g. rainbow trout and masou salmon, Flood et al. 2010) or increase when fish are held in duo-culture (e.g. Baltic salmon and brown trout, Jobling et al. 1998).

Concluding summary: synergies between risk factors and mitigation strategies

As stated at the start of this review, injuries are deleterious to both production and welfare (e.g. Cobcroft and Battaglene 2009). Farmers have an ethical responsibility to reduce the frequency and severity of injuries and by doing so can increase the market value of their fish by direct gains in fillet quality (Vågsholm and Djupvik 1998) and added value for incorporating welfare considerations into their production strategies (Olesen et al. 2010).

The primary aim of this review was to identify risk factors for injuries and a number of potential or existing mitigation strategies. It is one of the first to adopt a mitigation approach, identifying husbandry practices and operational strategies for improving the welfare of farmed fish. This review covers a wide range of existing or emerging aquaculture/capture-based aquaculture species. It also identifies risk factors from numerous types of production and rearing systems and has shown that there are many synergies between risk factors and numerous types of injury (for a summary see Table 1). If a farmer acts upon these, they can actively reduce the frequency and severity of these injuries. For example, as a number of injuries involve abrasion during handling, it is therefore logical that any husbandry or management practice involving the handling of these animals be carefully considered as to its design; the same applies to equipment. For instance, fish pumps, nets, sorting and grading apparatus come in a variety of materials and designs. Giving careful thought to designs less likely to cause injury would be beneficial such as using knotless nets or vacuum pumps instead of turbine pumps when transferring fish. Further, manipulating diet quality and feed management practices can also reduce the prevalence of injuries. For example, providing fish with a balanced diet, containing sufficient mineral and protein requirements can reduce the

Table 1 Showing examples of key risk factors for injuries to a number of different species

Risk factor	Injury	Species	Mitigation strategies
Diet formulation	Lower jaw deformity syndrome (LJD)	Dietary deficiencies in phosphorus and vitamins A, C, D and K in Atlantic salmon (King and Lee 1993; Roberts et al. 2001)	Ensure diets contain appropriate levels of phosphorus and vitamins A, C, D and K
	Mouth lesions, haemorrhaging and erosion of tissue around the mouth	Deficiency of niacin in tilapia (Shiau and Suen 1992)	Ensure diets contain appropriate levels of niacin
	Early life stage jaw deformities	Excess vitamin A in Japanese flounder (Suzuki et al. 2000) and Summer flounder (Martinez et al. 2007) or deficiencies in HUFA in pikeperch (Kestemont et al. 2007)	Ensure diets contain appropriate levels of vitamin A
	Cataracts	Cod, salmon, lake trout, wolf-fish, sea bass and sea bream (see Björnsson 2004)	Balance diet for each specific species
	Fin damage	E.g. steelhead (Lellis and Barrows 1997)	Appropriate selection of protein and lipid sources
Underfeeding	Fin damage	Rainbow trout (Moutou et al. 1998), Atlantic salmon (Ytrestøyl et al. 2005), Atlantic cod (Hatlen et al. 2006)	Feed to satiation
Choice of feed regime	Fin damage	Atlantic salmon (Noble et al. 2007a, b) rainbow trout (Suzuki et al. 2008)	Use demand feeding systems
Parasitic infections	Cataracts	Cod, salmon, lake trout, wolf-fish, sea bass, sea bream (see Björnsson 2004)	Biosecurity measures to prevent pathogen entry
	Epidermal ulcers (winter ulcer)	Prevalence of *Moritella viscosa* on fish farms in Atlantic salmon, cod, turbot and halibut (Lunder et al. 1995; Björnsdóttir et al. 2004; Gudmundsdóttir et al. 2006; Løvoll et al. 2009)	Biosecurity measures to prevent pathogen entry (disinfection, transport restrictions). High salinity (>12–15 ppt). Low water temperature (>10°C) Use of antimicrobials at the correct dose
	Epidermal ulcers	Prevalence of sea lice (*L. salmonis, C. elongatus, C. rogercresseyi*) in Atlantic salmon and rainbow trout (Boxshall and Defaye 1993)	Reduce stocking densities, disinfection of cages and utensils, restrict fish transport and control of natural host Use of antiparasitic drugs, only if infestation is massive
	Epidermal ulcers and necrosis	Prevalence of *Philasterides dicentrarachi* in olive flounder (Jin et al. 2009)	Biosecurity measures to prevent pathogen entry (disinfection, transport restrictions) Use of antimicrobials at the correct dose
	Epidermal ulcers	Prevalence of *Saprolegnia* sp. in Rainbow trout (Dykstra et al. 1989)	Biosecurity measures to prevent pathogen entry (disinfection, transport restrictions)
Choice of rearing system	Fin damage	Re-circulating versus flow-through tanks in rainbow trout (Roque d'Orbcastel et al. 2009)	Farm fish in flow-through tanks or optimise flow dynamics in RAS tanks
		Concrete versus cobbled raceways in rainbow trout (Bosakowski and Wagner 1994; Wagner et al. 1996)	Incorporate cobble substrates into salmonid raceways during construction
		Baffles in raceways in rainbow trout (Barnes et al. 1996)	Omit baffles during raceway construction

Table 1 continued

Risk factor	Injury	Species	Mitigation strategies
		Long-term cage submergence in Atlantic salmon (Korsøen et al. 2009)	Limit cage submergence to periods <6 weeks (Dempster et al. 2009; Korsøen et al. 2009)
	Epidermal injuries	Smooth tank surfaces in Halibut and southern flounder (Ottesen et al. 2007; Ottesen and Strand 1996)	Use of rigged tanks floor
	Broken jaw bones through collisions with tanks walls and catching jaws on cage netting	Inappropriate cage netting material in bluefin tuna (Miyashita et al. 2000). Inappropriate tank coloration in striped trumpeter (Cobcroft and Battaglene 2009)	Use of smaller mesh sizes and rubber or knotless mesh to reduce jaw catching, or use of appropriate tank colours
	Mouth lesions, haemorrhaging and erosion of tissue around the mouth	Rubbing against cage netting, tanks walls or other structures in holding facilities in Atlantic salmon (Halls 1994); rainbow trout (authors observations); Atlantic cod (authors observations; Halls 1994; authors observations)	Ensure appropriate materials are used for cages netting and tanks and make efforts to minimise the presence of abrasive surfaces that may cause damage to the mouths of fish if they rub up against them
Pumping	Fin damage. Epidermal abrasion and laceration	Channel catfish, Ictalurus punctatus (Grizzle et al. 1992)	Use a vacuum pump to transfer large numbers of fish (Grizzle et al. 1992)
	Eye injuries	Striped bass, rainbow trout (Helfrich et al. 2004); splittail and salmon (Helfrich et al. 2001)	Use a vacuum pump to transfer large numbers of fish (Grizzle et al. 1992)
	Epidermal injuries	Rainbow trout an striped bass (Helfrich et al. 2004)	Use of vacuum pumps or turbine pumps at no more than 330 rpm
Handling—net type	Fin damage	Bluegill (Barthel et al. 2003)	Use knot-less nets (Barthel et al. 2003)
	Mouth damage	Baltic cod (Suuronen et al. 1996)	Use of smaller mesh sizes and rubber or knotless mesh to reduce damage
Inappropriate egg incubation temperatures	Pug-headedness (brachygnathia)	Atlantic salmon (Bæverfjord et al. 1998a, b; Branson and Turnbull 2008)	Utilise optimal egg incubation temperatures
Inappropriate rearing temperatures	Early life stage jaw deformities	Atlantic halibut (Lein et al. 1997; Bolla and Holmefjord 1988); Atlantic salmon (Ornsrud et al. 2004); Japanese eel (Okamura et al. 2007)	Utilise appropriate rearing temperatures
	Fin damage	High temperatures in rainbow trout (Winfree et al. 1998) and sea bass (Person-Le Ruyet and Le Bayon 2009); low rearing temperatures in Atlantic salmon (Schneider and Nicholson 1980)	Utilise appropriate rearing temperatures
Inappropriate lighting	Eye injury and snapping (Speare 2008)	Halibut (Greaves and Tuene 2001)	Avoid creating shadows and uneven lighting over tanks.
	Early life stage jaw deformities	Atlantic halibut (Bolla and Holmefjord 1988)	Utilise appropriate light regimes
	Broken jaw bones through collisions with tanks walls and catching jaws on cage netting	Bluefin tuna (Miyashita et al. 2000)	Use of all-night lighting to improve vision. Removal of flashing lights loud noises and vibrations

Table 1 continued

Risk factor	Injury	Species	Mitigation strategies
High UV	Epidermal abrasion	Rainbow trout in Bolivia (Bullock and Coutts 1985)	Avoid using farms located in zones with high ultraviolet incidence
		Atlantic salmon in Ireland during summer (McArdle and Bullock 1987)	
	Cataracts	Cod, salmon, lake trout, wolf-fish, sea bass, sea bream (see Björnsson 2004)	
Gas super-saturation	Exophthalmia	Most fish species, but mainly salmonids (Bouck 1980)	Aeration of water, monitor gas levels in water
High stocking density	Fin damage	Rainbow trout (North et al. 2006); sea bass (Person-Le Ruyet and Le Bayon 2009)	Reduce stocking densities
Low stocking density	Fin damage	Arctic charr (Siikavuopio and Jobling 1995)	Increase stocking densities
Low water current	Fin damage	A. charr and A. salmon (Jobling et al. 1993)	Optimise water current
Direct aggression	Eye injuries	Halibut (Greaves and Tuene 2001); flounder (Carter et al. 1996); sole (Howell 1997)	Reduce feeding aggression and competition by delivering multiple feedings and dispersing feed over wide surface area
	Fin damage	Atlantic salmon (Cañon Jones et al. 2010); rainbow trout (Abbott and Dill 1985)	Reduce feeding aggression and competition by delivering multiple feedings and dispersing feed over wide surface area
Triploidy	Lower Jaw deformity syndrome (LJD)	Atlantic salmon (Jungawalla 1991)	Farm diploids
Vaccination	Epidermal injuries and intra-peritoneal adherences	Atlantic salmon (Vågsholm and Djupvik 1998)	Use of mineral oil–based adjuvant
Therapeutic medicinal treatment	Epidermis thinning and mucous cell opening	Rainbow trout (Buchmann et al. 2004; Madsen and Dalsgaard 1999)	Use of bath with low concentration and for the shortest time
Pollutants	Pug-headedness (brachygnathia)	Common bream (Slooff 1982); Red seabream (Matsuoka 2003)	Use clean water free of pollutants to culture fish. Early detection
	Early life stage jaw deformities	Common bream (Slooff 1982); Red seabream (Yamauchi et al. 2006)	Grow fish in clean waters free of pollutants
	Cataracts	Cod, salmon, lake trout, wolf-fish, sea bass, sea bream (see Björnsson 2004)	Use clean water free of pollutants to culture fish. Monitor water quality
Jellyfish blooms	Epidermal damage	Atlantic salmon (Tørud and Håstein 2008)	Jellyfish traps

Possible operational mitigation strategies for each risk factor are also included

severity of both mouth and fin damage. In addition, providing fish with a responsive ration to satiation can reduce both eye injuries (Greaves and Tuene 2001) and fin damage (Noble et al. 2007a) and provides further evidence that a single mitigation strategy can reduce the prevalence of a number of injuries.

Mitigation strategies can be both acute and long term. For instance, egg incubation temperature is a risk factor for pug-headedness, in addition to gill deformities and spinal injuries (Bæverfjord et al. 1998b; Ornsrud et al. 2004), and this can be remedied by having knowledge of the optimal egg incubating temperatures for a given species. With regard to long-term mitigation strategies, diet formulation, which is a risk factor for lower jaw deformity syndrome (Roberts et al. 2001), mouth lesions, haemorrhaging and erosion of tissue around the mouth (Shiau and Suen 1992), early life stage jaw deformities (Suzuki et al.

2000) and fin damage (Lellis and Barrows 1997), can be mitigated by formulating diets with appropriate levels of vitamins C and K, and utilising optimal sources of protein and lipid.

The literature on mitigating the injuries of farmed fish is scant, but the subject matter is important. Some of the current knowledge about risk factors for injury and their associated mitigation strategies can be applied to existing or emerging aquacultural or capture-based aquacultural species. However, further research must identify species-specific risk factors and potential mitigation schemes. Many more areas need to be addressed than the ones commonly reported in this paper, and controlled quantitative studies in a diverse number of cultured fish species are needed. This requires an integrated approach, utilising inputs from aquaculturists, researchers, veterinarians and aquacultural technology manufacturers.

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