

# **Fin Erosion in Aquaculture and Natural Environments**

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**ABSTRACT:** Fin erosion occurs when the fins of afflicted fish become degraded from a variety of sources, which can include abrasion with rough surfaces, fin damage from aggressive encounters between fish, nutritional deficiencies, and bacterial infection. Fin erosion has become a concern in fisheries management because of aesthetic and fish survival issues. Preventative measures for controlling fin erosion in hatcheries include: (i) feeding fish to satiation, (ii) increasing water velocities such that the energetic costs of fighting outweigh the gains, (iii) duoculture to reduce intraspecific aggression, (iv) use of a sand or cobble substrate on the bottom of rearing raceways and tanks to reduce abrasion, and (v) tank design. Various effective chemical treatments of fin necrosis have been developed which will be discussed. Fish diet supplementation, particularly with amino acids such as lysine, is a promising area of research to reduce fin erosion. Additionally, fish diets devoid of micronutrients such as copper have been found to lead to substantial fraying of dorsal and caudal fins in salmonids. Fin necrosis is related to fin erosion and is reviewed as well, especially bacteriology and stress-related aspects. For instance, stress is a factor that has been found to exacerbate both conditions. This review examines all the potential causes and effects of this serious condition, which occurs in propagated and wild fish populations.

**KEY WORDS:** fin erosion, fin necrosis, fin nipping, fin rot.

## **I. INTRODUCTION**

Fin erosion in fish is a multi-faceted issue that has become of concern in recent years due to increased production of fish in hatcheries and supplementation of wild stocks with hatchery-reared fish. The high incidence of fin erosion in the hatchery environment has resulted in extensive research on its causes. In aquaculture operations, the occurrence of fin erosion is substantially greater than in the natural environment, possibly due to the unnatural densities, feeds provided, and rearing environments (Mork et al., 1989). Fin condition assessment has been used as a definitive indicator of hatchery origin (Bosakowski and Wagner, 1994a).

Fin erosion is defined as the condition where fins are degraded, frayed, or reduced in size from abrasion, bacterial infections, or fin nipping. Fin necrosis is the

process of fins becoming abraded and frayed via infection from a variety of bacteria. Fin rot is the resultant condition from fin necrosis (Bodammer, 2000).

This review focuses on methods of measuring fin condition, behavioral aspects, and impacts on fin erosion, bacteriological and hematological aspects of fin erosion, the relation of nutrition to the incidence of fin erosion, and fin erosion in hatcheries. The main concerns regarding fin erosion are compromised swimming ability reducing survival in the wild (Barker et al., 1994) and aesthetics. Some anglers feel that their fishing experience has been compromised when they catch a hatchery-raised fish with eroded fins. A considerable amount of research has been conducted to ascertain causes of fin erosion and also to determine ways to reduce it in aquaculture operations. Reasons for reducing the incidence of fin erosion are twofold: (i) to increase the survivability of hatchery-raised fish to be stocked in the wild, and (ii) increase the aesthetic value of stocked fish to the angler and public fish consumer alike (Bosakowski and Wagner, 1995). Principal factors leading to fin erosion in hatcheries include: overcrowding, malnutrition, poor water quality, abrasive rearing surfaces, and bacterial infections (Bosakowski and Wagner, 1995).

Although the practice of fin clipping and the prevalence of fin erosion have been linked (Gjerde and Refstie, 1988), this topic is not discussed herein due to the breadth of this area of knowledge being sufficient to comprise a separate review paper. For further information on fin clipping, see Gjerde and Refstie (1988), Heimer et al. (1985), and Vincent-Lang (1993).

## II. METHODS OF ASSESSING FIN CONDITION

The three most popular fin condition indices developed to quantify fin condition include: (i) the Health Condition Profile fin condition index (Goede and Barton, 1990), (ii) the Kindschi Fin Condition Index (Kindschi, 1987), and (iii) the Frantsi Fin Condition Index (Frantsi et al., 1972). Other methods include those by Brown and Norden (1970), Piper et al. (1982), and Speare and MacNair (1996).

Goede and Barton's (1990) method of fin condition assessment is a part of a larger organosomatic index of fish health. Goede-Barton's index scores fin condition as follows: 0-all fins or other extremities intact; 1-previous fin damage that has healed over; 2-current fin damage but relatively mild degeneration with possible slight hemorrhaging; and 3-extensive active tissue degradation, possibly accompanied by hemorrhaging and secondary infection. Open wounds from damage to extremities can be entry sites for pathogens, and can increase the probability of fungal infections or ion loss (Goede and Barton, 1990).

Kindschi (1987) developed the fin condition factor as a way to quantify the degree of fin erosion in a given population of fish. The equation for the fin factor in percentage form is:

$$\text{Fin factor(\%)} = (\text{Fin length} \times 100) / (\text{total fish length}).$$

The fin factor equation given above is based on the concept that appendages of vertebrates grow proportionally to the overall body length. The relationship was

corroborated for wild salmonids by Bosakowski and Wagner (1994b). Fin length is measured from the median point of attachment to a fish's body to the most distal median point on the fin.

Frantsi et al. (1972) developed an ordinal index for grading degrees of fin erosion: 0-no erosion; 1-less than 1/3 of fin eroded; 2-more than 1/3 but less than 2/3 eroded; and 3-more than 2/3 of fin eroded. Generally, the Frantsi Index assesses caudal fins and the other fins separately. All fins are examined for signs of fraying and hemorrhaging within the fin, and at the base of the fin as well. The Frantsi Noncaudal Fin Condition Index is calculated with the following formula:

Fin Condition Index

$$= \frac{\text{total sum of noncaudal fin erosion observed in the sample of fish}}{(\text{Maximum total of fin erosion possible for a single fish}) \times (\text{Number of fish in sample})} \times 100$$

There are seven noncaudal fins and under the Frantsi system, each is assigned a potential value of 3; thus, the value of the maximum total of fin erosion for a single fish is 21 (e.g.,  $3 \times 7 = 21$ ). Under the Frantsi condition index, the upper and lower caudal fin lobes are evaluated as two separate fins. Again, the potential value is 3, and the maximum total of fin erosion is 6 (e.g.,  $3 \times 2 = 6$ ).

Brown and Norden's (1970) fin erosion measurement method estimated the percentage of caudal fin erosion by comparing eroded caudal fins to normal caudal fins as the "healthy standard." The Piper et al. (1982) method involved estimating the degree of fin erosion from length-weight tables for a particular species or population of fish. Generally, the higher the length-weight ratio, the better condition a fish and fins were deemed to be in. This method was only found to be applicable to the caudal fin only.

The Speare and MacNair (1996) fin erosion scale is a numerical system along the same lines as the Frantsi Index and the Goede-Barton index. The Speare and MacNair fin condition scale is comprised of the following increments: 0 = normal; 1 = up to 25% of the fin missing or damaged; 2 = from 25% to 75% of the fin missing or damaged; 3 = from 75% to 100% of the fin missing; and 4 = 100% of the fin missing or damaged and adjacent skin also affected.

All of the above fin erosion measurement systems have advantages and disadvantages. Each quantifies the degree of fin erosion numerically, but an inherent flaw with the numerical quantification of fin erosion is that there needs to be an index fish with perfectly healthy fins for comparison. Additionally, fin erosion measurement methods that are categorical such as those that employ percentages or fractions as the basis of definition for the degree of fin erosion, could introduce error since the degree of fin erosion seen may be subjective, especially if a sample with perfect fins is not present. Steelhead trout have been reared individually (Winfrey et al., 1998; Kindschi et al., 1991b) which provide fish with perfect fins for treatment comparisons. In the case of the Goede and Barton health condition profile, categories of fin erosion are used as a barometer of fish health. Of all the techniques reviewed above, the Kindschi (1987) fin factor method appears to be the most accurate method, and is the least affected by subjective bias, but it requires more time to conduct than

other categorical methods. The Kindschi method is the only method reviewed that quantifies fin erosion for all fins.

### **III. BEHAVIORAL CAUSES OF FIN EROSION AND FISH CULTURE CONSIDERATIONS**

#### **A. FIN NIPPING AND REARING DENSITY**

Aggressive behavior among fish has been strongly correlated with the incidence of fin erosion. Fin erosion is often caused by fin nipping, with the dorsal fin appearing to be the most popular fin to be nipped in aggressive confrontations between salmonids (Abbott and Dill, 1985). The pectoral and caudal fins are also points of attack in aggressive encounters, but not with the frequency that the dorsal fin tends to be attacked (Abbott and Dill, 1985). In Atlantic salmon (*Salmo salar*) parr, dorsal and caudal fins are the targets attacked most often in intraspecific aggressive encounters (Turnbull et al., 1998). Pectoral fins were found to be susceptible to fin damage as well, but were generally not favored parts to attack. As a consequence of this, Turnbull et al. (1998) hypothesized that most pectoral fin damage was sustained from abrasion with concrete raceway walls. In the culture of banded pearl spot (*Etroplus suratensis*), the caudal fin is particularly susceptible to fin rot, primarily because of territoriality and the consequent fin nipping that tends to accompany this behavior (Rattan and Parulekar, 1998). *Aeromonas* and *Pseudomonas* bacteria are considered to be the principal causative organisms of fin rot in banded pearl spot; the antibiotic furacin was the most effective treatment (Rattan and Parulekar, 1998).

Fin nipping is the primary reason for dorsal fin damage in hatchery-reared salmonids (Arndt et al., 2001). Bacteria that cause fin necrosis generally cannot maintain the infection without there being consistent physical damage to the host's tissues in the afflicted area (Turnbull et al., 1998).

Fin damage by nipping appears to be affected by age, but not gender (Mork et al., 1989). Mork et al. (1989) found that the incidence of dorsal fin erosion was lower in sexually mature males than immature Atlantic salmon males, with no noticeable difference between immature males and females. In most aquaculture operations, fish of roughly the same size and species are cultured separately (Adams et al., 2000). Fin nipping and fin erosion were significantly reduced when two size classes of Atlantic salmon were reared together versus one size class by itself (Adams et al., 2000). Although the larger fish attacked the smaller fish, the frequency was reduced compared to that at which fish of the same size class attacked each other (Adams et al., 2000).

The effects of fish rearing density on fin erosion varies with different species. Bonneville cutthroat trout, *Oncorhynchus clarki utah*, experienced a significantly higher level of fin erosion at increased rearing densities (Wagner et al., 1997), although this result was inconsistent. Degraded fin conditions at higher rearing densities were attributed to a higher level of agonistic interactions. For rainbow trout supplied with supplemental oxygen, higher rearing densities ranging from 9.83–9.94 kg/m<sup>3</sup> led to greater amounts of fin erosion (Miller et al., 1995).

For Arctic char (*Salvelinus alpinus*), higher levels of aggression did not occur at higher rearing densities including 8.7 kg/m<sup>3</sup>, 20.0 kg/m<sup>3</sup>, and 44.0 kg/m<sup>3</sup> (Brown

et al., 1992). Jobling (1995) also found that greater fish densities, as well as higher current velocities, reduced the number of agonistic interactions among Arctic char. Caudal fin erosion was found to be more severe in walleyes than in their hybrids under intensive aquaculture conditions (Clayton et al., 1998).

The link between higher levels of aggression and fin nipping and erosion under both natural and unnatural fish population conditions were confirmed by Stringer and Hoar (1955). Stringer and Hoar (1955) sought to document aggressive behavior of Kamloops trout (*Oncorhynchus mykiss*) in a simulated natural habitat. Fin nipping was found to be an integral component for territorial defense, with the caudal fin being the preferred region to attack (Stringer and Hoar, 1955). Thus, territoriality results in a higher degree of fin erosion because of a higher incidence of fin nipping in both the wild and hatchery situation (Stringer and Hoar, 1955). Generally, dominant mature fish tend to experience less fin erosion than subdominant less mature fish (Mork et al., 1989).

In some studies, fish rearing density had no effect on fin erosion. In rearing densities of steelhead (*Oncorhynchus mykiss*) reared at 56 kg/m<sup>3</sup>, 171 kg/m<sup>3</sup>, 250 kg/m<sup>3</sup>, or 252 kg/m<sup>3</sup>, dorsal and pectoral fin erosion was seen equally at all densities, and the degree and severity of fin erosion seen was unaffected by higher rearing densities (Kindschi et al., 1991b). In Atlantic salmon, pectoral and dorsal fin conditions were unaffected by rearing densities ranging from 80–229 kg/m<sup>3</sup> (Soderberg and Meade, 1987). No differences in fin erosion were found among several different salmonid species at densities ranging from 361 fish/m<sup>3</sup> to 556 fish/m<sup>3</sup> (Wagner and Bosakowski, 1994). When fry were reared at densities up to 43,926 fish/m<sup>3</sup>, the high density rearing experience did not significantly influence fin condition in fingerlings (Wagner et al., 1996b).

## B. DUOCULTURE

Fin erosion has been found to be more prevalent where only one species of fish was raised in confinement (Tinus and Reeves, 2001). Tinus and Reeves (2001) found that fin damage via nipping was significantly reduced in small juvenile steelhead when shoals of reidside shiner (*Richardsonius balteatus*) were present. Dorsal fin rot was substantially reduced in Atlantic salmon raised with Arctic char (Holm, 1989). However, Atlantic salmon raised with brown trout were found to have a much higher incidence of fin erosion than when raised in monoculture (Jobling et al., 1998). Mixed-species fish rearing was considered a more economical approach to producing quality fish with aesthetically appealing fins (Holm, 1989; Baker and Ayles, 1990). Sympatric associations of fish exist in nature, where different species of fish have been shown to school together. Atlantic salmon, brown trout, and Arctic char have been shown to have a sympatric distribution in many river and lake systems (Holm, 1989).

## C. WATER FLOW AND TANK DESIGN

One strategy to reduce aggression and fin nipping between fish is to limit the benefit that fish gain from fighting. Perhaps aggression between fish could be

reduced by making conditions so that the energetic costs of fighting are not worth the gains (Adams et al., 2000). With Arctic char, agonistic behavior (directly linked to fin nipping and fin erosion) was substantially reduced when fish were forced to swim harder to overcome faster water velocities (Christiansen and Jobling, 1989). However, other evidence indicates that water exchange rate has no bearing on the frequency of aggressive interactions between salmonids under aquaculture conditions (Ross et al., 1995).

Hatchery tank and water flow designs have been correlated with the prevalence of fin erosion. Raceways implementing a cross-flow water current (Watten and Johnson, 1990) reduced fin erosion, possibly because fish avoided each other more as a consequence of working against the current and hence the number of aggressive encounters was significantly reduced (Arndt et al., 2001). Steelhead reared in tanks with baffles experienced greater fin erosion compared to fish reared in tanks without baffles (Kindschi et al., 1991a). Fish raised in net pen culture situations tend to be more susceptible to fin erosion than fish raised in tanks (Mormg, 1982).

Abrasion from rearing surfaces can contribute to fin erosion (Bosakowski and Wagner, 1995). Eroded fins are more characteristic of concrete raceways and small ponds as opposed to large ponds and earthen raceways (Larmoyeux and Piper, 1971; Bosakowski and Wagner, 1994a). However, Arndt et al. (2001) found that coating the surface of raceways with a polymer to smooth the raceway surface actually increased the incidence of fin erosion. Factors correlated with fin erosion severity included lower alkalinities, unnatural bottom substrates (e.g., concrete or steel), higher unionized ammonia levels, and higher fish densities (Kindschi et al., 1991b; Bosakowski and Wagner, 1994a).

Bottom substrate also affects the occurrence of fin erosion. With Bear Lake cutthroat trout and rainbow trout fingerlings, fin erosion was substantially reduced when fish were raised in cobble-bottomed raceways (Bosakowski and Wagner, 1995; Arndt et al., 2001). Fin condition in albino rainbow trout was also found to be markedly improved with cobble substrates (Wagner et al., 1996c).

#### **D. FEEDING STRATEGIES**

Feeding to satiation can help to reduce the incidence of fin erosion, since fish fed to satiation are less likely to nip each other (Larmoyeux and Piper, 1971). In lower ration groups, subordinate fish were found to suffer the most dorsal fin damage (Moutou et al., 1998). Feeding fish to satiation reduced the incidence of fin nipping and fin erosion, but did not eliminate it (Wagner et al., 1996a). Gregory and Wood (1999) found that rainbow trout fed a half-satiation diet had reduced swimming ability, attributed to a higher degree of fin damage, compared to fish fed to satiation. The addition of 30% nondigestible bulk to the diet has been shown to help satiate hunger while substantially reducing fin erosion at minimal cost (Larmoyeux and Piper, 1971). However, this practice would lead to increased solid wastes. In the case of feeding intervals, Klontz et al. (1991) found that neither daily nor intermittent feeding had any impact on the incidence of fin erosion in steelhead.

## **IV. BACTERIOLOGICAL AND HEMATOLOGICAL ASPECTS OF FIN EROSION**

### **A. DISEASES SIMILAR TO FIN EROSION**

Many of the symptoms of fin erosion such as hemorrhagic fins, lesions, and dark coloration of the skin, overlap those of other fish diseases commonly encountered by fisheries professionals (Piper et al., 1982). Symptoms of fin rot can be very similar to those of ulcer disease, peduncle disease, and furunculosis (Mahoney et al., 1973). Additionally, fin necrosis is one of the signs and symptoms of salmonid herpesvirus type-2 infections (Kumagai et al., 1994).

### **B. BACTERIAL AND PROTOZOAN AGENTS OF FIN EROSION**

Fin erosion can be exacerbated by a variety of bacteria. The main bacterial fish pathogens known to cause fin rot are *Aeromonas salmonicida*, *Aeromonas liquefaciens*, and common water bacteria (Loganathan et al., 1989; Okaeme, 1989; McVicar et al., 1993; Hettiarachchi and Cheong, 1994; Hossain et al., 1994; Chowdhury, 1998). Bacteria from the genera *Pseudomonas* and *Vibrio* have been implicated as causative agents of fin rot as well (Giles et al., 1978; Barker et al., 1994; LeaMaster and Ostrowski, 1988; Angelini and Seigneur, 1988; Chowdhury, 1998; Hossain et al., 1994; Loganathan et al., 1989). The *Dermocystidium* parasite has also been found to cause fin erosion in farmed Atlantic salmon (Hoglund et al., 1997). *Flexibacter columnaris* bacteria was implicated as a causative agent of fin rot in guppies *Poecilia reticulata* (Yamazaki et al., 1990) and tilapia (Chun and Sohn, 1985). In addition to attacking the fins themselves, the bacteria responsible for fin erosion can also attack the skin and dermal musculature (Khan et al., 1981; Bodammer, 2000). Fish with substantial surface abrasions generally prove to be the fish most susceptible to fin erosion, particularly because bacteria that invade are opportunistic (Khan et al., 1981). Virulent strains of *Flexibacter* and *Aeromonas salmonicida* have been implicated as causes of fin rot, possibly why high mortality is seen in afflicted fish (Bullock, 1968; Pickering and Pottinger, 1988). Flavobacterium-Cytophaga group bacteria have been implicated as a causative agent of fin erosion in cultured marine fish as well (Bernardet, 1998).

*Vibrio anguillarum* bacteria were repeatedly found to be present in the skin and muscle lesions of winter flounder (*Pleuronectes americanus*) afflicted with fin necrosis and related conditions (Levin et al., 1972). In mullet (*Mugil cephalus*), normal skin microflora is composed of 5–6 colony types. However, when an environmental stressor is applied, such as pollution in the form of crude oil, the microflora can be reduced to 1–2 colony types, one of which is the *Vibrio* bacterium (Giles et al., 1978). In pike perch (*Stizostedion lucioperca*), the principal ciliate protozoan parasite found responsible for fin erosion was *Ichthyobodo necator* (Rahkonen, 1994). In a study on the effects of *Trypanosoma murmanensis* infection on *Gadus morhua*, *Pseudopleuronectes americanus*, *Myoxocephalus scorpius*, and *Myoxocephalus octodecemspinosus*, Khan (1985) found that when fin rot occurred simultaneously with *T. murmanensis* infection, death would occur. When *T. murmanensis* and fin rot

infections occurred separately, no mortality would result; reasons as to why *T. murmanensis* infections coupled with fin rot caused mortality were unknown (Khan, 1985).

### C. DISEASE PROGRESSION OF FIN EROSION

The steps of infection for fin necrosis proceeds as follows: (1) necrotic disintegration of fins, (2) necrotic ulcer formation, and (3) secondary infection of disintegrated fin by viruses, bacteria, and fungi (Tret'yakov et al., 1988; Sharples and Evans, 1996). Fin necrosis diseases in turbot progressed from the formation of white, discolored foci on skin, operculum, and base of dorsal and caudal fins to sloughing of the epithelial surface of the center of lesions, followed by production of shallow and hemorrhagic ulcers (Devesa et al., 1989). Ulcerative lesions had three different areas characterized by different types of invading microorganisms: (1) a peripheric pale zone with a ciliate protozoan (*Cryptocaryon sp.*), (2) an intermediate grayish dark zone with *Mycobacteria*, and (3) a central hemorrhagic zone with halophilic *Vibrio* strains (Devesa et al., 1989; Turnbull et al., 1996). The problems of fin erosion can be further compounded by malnutrition and decreased feeding (Schneider and Nicholson, 1980).

Generally, the biochemical progression of fin erosion as a disease is poorly understood (Tret'yakov et al., 1988). The physical-chemical characteristics of hemoglobin becomes altered in fish afflicted with fin necrosis. For example, membrane-structural enzymes such as adenilate cyclase change under fin necrosis disease conditions leading to compromised physiological function. However, the exact mechanisms of how the fin necrosis condition alters hemoglobin have yet to be elucidated (Tret'yakov et al., 1988).

### D. DISEASE SYMPTOMS OF FIN EROSION

Generally, the physical manifestation of fin rot appears as a mucoid, whitish, opaque, degenerative mass of tissue on the periphery of the skin (Schneider and Nicholson, 1980). In reared turbot (*Scophthalmus maximus*), fin necrosis symptoms include erratic swimming, respiratory distress, and inappetence (Devesa et al., 1989). Infection associated with fin erosion can lead to depressed hematocrit levels, depressed hemoglobin levels, decreased total plasma protein, increased numbers of immature erythrocytes, and increased numbers of immature neutrophils (Khan et al., 1981; Mahoney and McNulty, 1992). The aforementioned complications arising from fin erosion are believed to be the cause of death in cases of fin erosion, not the condition itself. The bacterial causative agents of fin erosion can also affect the heart, liver, spleen, kidney, intestine, gills, and skin (Khan et al., 1981). In fins, where erosion and rotting were the most pronounced, melanophores (essentially color pigment capsules) aggregate (Mearns and Sherwood, 1974). Histologically, fin erosion as a disease results in the disintegration of melanophores and destruction of pigmentary bodies (Wells and ZoBell, 1934). Additionally, lymphocytic infiltration in the afflicted fin is relatively minimal or absent (Mearns and Sherwood, 1974).



Four types of pathological conditions were found characteristic of tissues adjacent to areas of fin erosion in winter flounder: (i) epithelial cell hyperplasia, (ii) mucus cell hyperplasia and hypertrophy, (iii) spongiosis, and (iv) focal necrosis (Bodammer, 2000). Spongiosis and focal necrosis had not been seen in winter flounder living in polluted environments prior to this research (Bodammer, 2000).

Chemical treatments that have been used to eliminate fin rot include 10 to 50 ppm oxytetracycline and 1 to 2 ppm benzalkonium chloride (McVicar et al., 1993). Chloramine-T at concentrations less than 10 mg/L was a good prophylactic treatment for the prevention of various topical fish diseases such as fin erosion (Powell et al., 1994). Conroy (1963) found that antibiotic treatment cleared up fin rot symptoms effectively in afflicted goldfish. Baths of formalin, malachite green, and nitrofurazone as separate treatments were found to be effective treatments for fin erosion in reared turbot (Devesa et al., 1989). For Dover sole (*Microstomus pacificus*), a sand substrate in tanks prevented the occurrence of the disease and cured an established disease situation (McVicar and White, 1982). Antibiotics administered orally and through the liquid medium to Dover sole had little effect. Formalin treatment held the disease at bay, but with deleterious toxic effects to the fish (McVicar and White, 1982). In farmed catfish (*Clarias gariepinus*) and carp (*Puntius gonionotus*) in Bangladesh, *Aeromonas* and *Pseudomonas* bacteria were found to be 62–67% resistant to typical antibiotics used to treat fin rot, such as chloramphenicol and oxytetracycline (Chowdhury, 1998). In another study of carp and catfish species of Bangladesh, Hossain et al. (1994) found that the antibiotic Streptomycin was most effective against *Aeromonas* bacteria, and that Tetracycline was the most effective antibiotic against *Pseudomonas* bacteria. Amoxicillin was found to be the least effective antibiotic against *Aeromonas* bacteria, and nalidixic acid was the least effective treatment against *Pseudomonas* bacteria (Hossain et al., 1994).

### E. EFFECTS OF TEMPERATURE, LIGHT, AND OTHER FACTORS ON FIN EROSION

Temperature and light can also impact the prevalence of fin erosion. Colder water temperatures reduced fin erosion in steelhead: however, the growth rate was also reduced (Winfree et al., 1998). The extent and severity of fin rot in salmonids increased as water temperature decreased (Schneider and Nicholson, 1980). In alewives (*Alosa pseudoharengus*), on the other hand, rising water temperatures can suppress antibody production, possibly leading to an increase in susceptibility to bacterial infection (Brown and Norden, 1970). Temperature extremes can compromise immune system responses in fish (Tret'yakov et al., 1988). Whether it is heat or cold extremes that cause immunosuppression is species-dependent (Iwama and Nakanishi, 1996).

Vincent-Lang (1993) hypothesized that because coho salmon *Oncorhynchus kisutch* experienced significant mortality in the wild from ventral fin clips, fin erosion would lead to a similar result. A radio tracking study of bluegills (*Lepomis macrochirus*) found that radio tags cause significant erosion of the pelvic fins (Knights and Lasse, 1996). The mechanical stress of handling with hand jigging and traps of sablefish (*Anoplopoma fimbria*) was found to cause significant caudal fin erosion (Rutecki and Meyers, 1992). Addition of cortisol to the bloodstream of

brown trout *Salmo trutta* at levels as low as 1 ng/ml caused increased susceptibility to fin rot (Pickering and Pottinger, 1985).

The use of covers on raceways did not have any effect on the incidence of fin erosion (Wagner and Bosakowski, 1994). The use of baffles along with raceway covers, however, was found to lead to a higher incidence of fin erosion on the pelvic and pectoral fins of rainbow trout (Barnes et al., 1996). Stringer and Hoar (1955) found that bright illumination increased the incidence of fin nipping. However, Wagner and Bosakowski (1995) found that shading of raceways induced only a transitory reduction of fin erosion.

Oxygen levels and whether fish are diploid or triploid can have an impact on the incidence of fin erosion. Larmoyeux and Piper (1973) showed that fish reared in low oxygen environments tended to have less dorsal fin erosion than fish reared under higher oxygen conditions. No difference in caudal fin damage was seen between diploid and triploid Atlantic salmon parr (Carter et al., 1994). For the dorsal fin, however, in mixed diploid-triploid parr groups, triploid parr experienced substantially more fin erosion than diploid parr. Triploid parr were hypothesized to be more likely targets of aggressive behavior than diploid parr (Carter et al., 1994).

## **F. SADDLEBACK DISEASE**

“Saddleback” disease in Atlantic salmon has been shown to have an etiology very similar to that of fin erosion (Morrison et al., 1981). *Flexibacter columnaris* is the principal causative bacterium of “saddleback” disease. To achieve infection of fish, the bacterium requires a break in the epidermis to infect the dermis; otherwise, infection will not take place (Morrison et al., 1981). The symptoms of saddleback disease are very similar to those of fin necrosis. Generally, the disease starts at base of most fins (rarely the caudal fin, however) and works outward, essentially the reverse of the fin rot process (Morrison et al., 1981).

## **G. BLACK PATCH NECROSIS**

In Dover sole, fin necrosis is termed Black Patch Necrosis (BPN) (Campbell and Buswell, 1982). Black Patch Necrosis is believed to be caused by a long, thin, filamentous gram-negative organism that strongly resembles *Flexibacter columnaris* (Campbell and Buswell, 1982). Black Patch Necrosis proved to be highly contagious between healthy and diseased stocks; however, the use of sand on tank floors brought BPN disease under control (Campbell and Bushnell, 1982).

## **V. NUTRITIONAL ASPECTS OF FIN EROSION**

Generally, for most species of fish reared in hatchery situations, good growth and feed conversions correlate directly with good fin condition (Lemm et al., 1988). Studies indicate salmonids thrive best on a diet with 45–50% usable protein and 23–27% unsaturated lipids from marine fishes, at temperatures between 10–20°C (Lemm et al., 1988). Fin condition was best in the fastest growing fish (Lemm et al., 1988).

For rainbow trout, the feeding of semi-purified diets containing no polyunsaturated fatty acids, particularly linolenic acid, led to poor growth and feed conversion and a higher incidence of fin erosion (Castell et al., 1971). The amino acid lysine was critical for the prevention of fin erosion in rainbow trout, with optimum levels of lysine being around 6.1% of dietary protein (Ketola, 1982). The addition of lysine, arginine, histidine, isoleucine, threonine, valine, and tryptophan to the corn gluten meal diet of hatchery-reared rainbow trout improved weight gain and nearly eliminated all fin erosion (Ketola, 1983).

Vitamin C is essential for good growth rates and a lower incidence of fin erosion (Mazik et al., 1987; Soliman et al., 1986). Collagen formation in fish requires an adequate supply of vitamin C (Mazik et al., 1987). Collagen is a fibrillar structural protein that provides flexible support to various fish tissues, muscles, and fins (Ostrander, 2000). In Nile tilapia *Oreochromis niloticus*, vitamin C deficiency was found to lead to caudal fin erosion, while an excess of vitamin C was found to be detrimental to growth but had no bearing on fin erosion (Soliman et al., 1994). Larval common carp (*Cyprinus carpio*) and larval channel catfish (*Ictalurus punctatus*) experienced significant fin erosion when fed a diet lacking vitamin C (Dabrowski et al., 1988; Dabrowski et al., 1996). Lochmann and Phillips (2001) found that vitamin C-free diets led to a higher incidence of fin erosion in juvenile golden shiners (*Notemigonus crysoleucas*). Sea bass (*Lates calcarifer*) (Phromkunthong et al., 1997) and grouper (*Epinephelus malabaricus*) (Phromkunthong et al., 1993) experienced fin erosion when fed a diet lacking vitamin C.

In Japanese flounder (*Paralichthys olivaceus*), retinoic acid (vitamin A) controls gene expression required for pharyngeal and pectoral fin development (Suzuki et al., 2000). Excess vitamin A fed to brook trout can cause reduced growth and severe caudal lesions (Brown and Norden, 1970). Inhibiting retinoic acid synthesis as well as collagen synthesis resulted in both fin and skeletal deformations in flounder (Suzuki et al., 2000). Vitamin A deficiency in Nile tilapia causes hemorrhages in the fins, whereas excess vitamin A at around 10,000 IUs and above causes caudal fin erosion (Saleh et al., 1995). Additionally, a diet lacking in pantothenic acid was found to cause caudal fin erosion in tilapia (Roem et al., 1991). Folic acid deficiency may lead to greater susceptibility to bacterial infections, particularly in salmonids, and also causes fragility in the caudal fins of salmon and trout (Brown and Norden, 1970). Copper has been found to be an element that when absent from the diet of rainbow trout can cause severe fraying of the dorsal and caudal fins (Campbell et al., 2002). A level of 730 mg per kg dry weight feed of copper was best for eliminating fin erosion in rainbow trout (Campbell et al., 2002). Copper has been found to be involved in the formation of connective tissue in fish (Lellis and Barrows, 1999).

Chitin is a polysaccharide found in the exoskeletons of various invertebrates. When used as a dietary supplement for juvenile steelhead, chitin reduced dorsal fin erosion (Lellis and Barrows, 2000). The authors hypothesized that chitinase (the enzyme in fish used to break down the chitin exoskeleton of invertebrates) plays a role against invasive pathogens and may reduce fin erosion (Lellis and Barrows, 2000). Additionally, adding krill ash to a fish-based diet was found to help prevent fin erosion (Hardy, 1997). In the case of steelhead, Lellis and Barrows (1997) found that a diet using marine invertebrates in the form of krill as the primary protein source led to the lowest incidence of dorsal fin erosion, versus a diet using marine teleosts as the primary protein source. Conversely, a commercial diet supplemented

with herring oil led to a greater degree of fin erosion in steelhead (Kindschi et al., 1991c). The protein/mineral content of fish diet has been found to be the dietary factor with the most relevance to the incidence of fin erosion (Lellis and Barrows, 1999). Overall, fish diet composition can have a significant impact on fin erosion.

## **VI. ENVIRONMENTAL CONSIDERATIONS RELATING TO FIN EROSION**

Although the prevalence of fin erosion is much greater under artificial rearing conditions than in the wild, there is still a fair degree of fin erosion seen in the wild, especially in cases of degraded habitat conditions (Wiklund and Bylund, 1996; Reash and Berra, 1989; Padmalatha and Singh, 1996). For example, a chemical environmental stressor, such as, crude oil interacting with surrounding natural habitat, led to a higher incidence of fin erosion in mullet (Minchew and Yarbrough, 1977; Hinkle-Conn et al., 1998). Pollock (*Theragra chalcogramma*) experienced substantial fin erosion when egg stages were exposed to water-soluble fractions of oil (Carls and Rice, 1989). Yellowfin sole (*Pleuronectes asper*), rock sole (*Pleuronectes bilineatus*), and Pacific halibut (*Hippoglossus stenolepis*) all experienced a high degree of fin erosion when exposed to oil-laden sediments (Moles and Norcross, 1998). Dover sole in habitats that were once degraded and then rehabilitated with flourishing kelp beds had the lowest incidence of fin erosion compared to fish reared in constantly degraded habitats (Stull, 1995). Crude oil contamination caused fin erosion when administered to mullet in an estuarine pond ecosystem, but not in a controlled laboratory test (Minchew and Yarbrough, 1977). In a follow-up study on mullet (Giles et al., 1978), *Vibrio* were the only bacteria present in or on the fish that were capable of utilizing crude oil in metabolic and respiratory pathways. Petroleum pollution favors certain groups of bacteria within the sediments in estuarine conditions (Giles et al., 1978). The authors concluded that because oil substantially reduced the exterior microflora of the mullet, pathogenic *Vibrio* bacteria were given a chance to proliferate and cause excessive fin damage (Giles et al., 1978). High, heavy metal concentrations such as increased levels of zinc, lead, and cadmium cause increased fin erosion in mullet as well (Bangaramma and Lakshmi, 1999). Degraded waters and habitat were found to be substantial contributors to incidences of fin erosion in winter flounder as well (Ziskowski and Murchelano, 1975). In a 1994 study by Barker et al., winter flounder had more external lesions (e.g., fin necrosis, ulcers) at sites closer to the effluent of a pulp and paper mill than farther away from it. The incidence of fin erosion in flounder increases with increased salinity (Lang et al., 1999). Flounder inhabiting sites closest to pulp- and paper-effluent discharge sites were the most stressed, with low condition (K) factors, low hepatosomatic indices, low blood hemoglobin, and low lymphocyte levels (Barker et al., 1994). Similar findings were found by Lindesjö, and Thulin (1990) for perch *Perca fluviatilis* and ruffe *Gymnocephalus cernua* living in habitats adjacent to a pulp and paper effluent. In another study with flounder, handling of fish coupled with environmental stressors was found to leave fish more susceptible to bacterial infections that cause fin necrosis (Vethaak et al., 1996). Retene (7-isopropyl-1-methylphenanthrene) is a compound often affiliated with pulp and paper effluents, which has been shown to

cause fin erosion in larval zebrafish (*Danio rerio*) (Billiard et al., 1999). Rainbow trout exposed to untreated bleached kraft mill effluent have a higher incidence of fin necrosis than fish which have not been exposed (Couillard et al., 1988). In a feral population of goldfish (*Carassius auratus*) exposed to bleached kraft mill effluent, fin erosion peaked at sites closest to the effluent source, and progressively decreased at sites more distant from the effluent source (Sharples et al., 1994; Lindesjö and Thulin, 1994). Degraded habitat conditions coupled with the presence of *Aeromonas* and *Pseudomonas* bacteria gave rise to fin erosion in South American catfish, *Rhamdia sapo* (Angelini and Seigneur, 1988). In a study of the incidence of fin erosion in and near San Diego Bay, California, the incidence was found to be highest in the bay itself, which was the site most proximate to industrial and population centers and, hence, pollution (McCain et al., 1992). Sewage discharges were found to cause a higher incidence of fin erosion in the flatfishes of Chile (Leonardi and Tarifeno, 1996).

Polychlorinated biphenyls (PCBs) used in transformer oils cause substantial fin erosion when exposed to rainbow trout (Mayer et al., 1985). Polychlorinated biphenyl exposure also causes higher incidences of fin erosion in winter flounder (Khan, 1999). Food deprivation coupled with PCB exposure caused a higher incidence of fin erosion in Arctic charr than in starved fish not exposed to PCBs (Jorgensen et al., 1999). Similar findings were discovered with lake chubsuckers (*Erimyzon sucetta*); food deprivation in combination with exposure to coal combustion wastes caused significant fin erosion (Hopkins et al., 2002). Conversely, sunfish *Lepomis sp.* exposed to selenium-laden coal fly ash deposits experienced less fin erosion than fish not exposed (Lohner et al., 2001). Rainbow trout exposed to 10  $\mu\text{g}/\text{kg}$  of 2,3,7,8-tetrachlorodibenzo-p-dioxin experienced significant fin erosion (Spitsbergen et al., 1988). In another study with 2,3,7,8-tetrachlorodibenzo-p-dioxin, rainbow trout, yellow perch, carp, bluegill, largemouth bass, and bullhead were found to experience drastic fin erosion at an initial concentration of 25  $\mu\text{g}/\text{kg}$  (Kleeman et al., 1988). Conversely, another study with yellow perch found that 2,3,7,8-tetrachlorodibenzo-p-dioxin at a concentration of 494 parts per thousand had no bearing on the incidence of fin necrosis (Kleeman et al., 1986). Polycyclic aromatic hydrocarbons and polycyclic aromatic compounds are carcinogens that have been found to cause fin erosion in medaka (*Oryzias latipes*) (Fabacher et al., 1991). Pollution in the form of polyaromatic hydrocarbon compounds caused a higher incidence of fin erosion in Atlantic cod, (*Gadus morhua*) (Aas et al., 2001). Chlorinated hydrocarbon residues such as DDT cause a greater amount of fin erosion in winter flounder as well (Moore et al., 1996).

In sea trout (*Salmo trutta*), a higher prevalence of fin erosion was correlated with the chalimus stages of salmon lice, which tend to prefer fins as an infection site (Dawson, 1998). Additionally, in Baltic salmon (*Salmo salar*), the frequency of fin erosion was found to be higher in winter than other seasons (Vehanen et al., 1993).

Pollution that causes habitat degradation leads to fin erosion and other conditions deleterious to fish health (Noaksson et al., 2001; Burke et al., 1993). Fin erosion in the wild is considered one bioindicator of degraded habitat conditions (Sharples and Evans, 1996; Jacques et al., 1994; Ziskowski et al., 1987). Habitat degradation can include wide temperature changes, extreme salinity fluctuations, decreased dissolved oxygen concentrations, and greater susceptibility to mechanical injuries and parasitic

infections (Barker et al., 1994). The relationship between pollution and fish health is complex.

## VII. SUMMARY

Fin erosion is a condition that can be caused by a variety of sources. The main causes of fin erosion include: (1) abrasion with rough surfaces, (2) fin damage from aggressive confrontations, usually from fin nipping, (3) nutritional deficiencies, and (4) bacterial infection. Usually, several of the above causes of fin erosion exist simultaneously. For instance, fin damage from abrasion and nipping leaves afflicted fins susceptible to bacterial infection.

As a condition, a fair amount is known about fin erosion; however, there are still further aspects of the condition that need to be addressed. Further topics to pursue include: (1) micronutrient effects, (2) mineral deficiency effects, and (3) effects of the genetics or strain of fish being propagated.

For the aquaculture situation, more research is needed on how water velocities and different current flows affect the incidence of fin erosion. Specifically, more raceway designs need to be tested to determine their efficacy at reducing fin erosion. More research is also required on understanding which duoculture practices work best in an aquaculture situation to eliminate fin erosion. Further research needs to be done as to whether baffle usage by itself in raceway culture causes fin erosion. Additionally, more research is required on how to modify fish behaviors in aquaculture situations so that fin erosion is reduced. Despite the amount of research that has been dedicated to fin erosion, the problem still remains; thus further research is required to reduce the incidence of fin erosion.

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