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GILL DISEASES OF FISH

I. INTRODUCTION

The gill diseases of fish under confined; i.e., farm ponds or raceways, and free-living conditions are very complex and, in many cases, not well understood from epidemiological and etiological standpoints. This group of diseases, in the opinion of many, is one of the major production-limiting factors in farmed fishes (Klontz, 1973, 1974; Snieszko, 1974). Subclinical episodes are often quite difficult to detect due to their insidious onset. Clinical episodes are frequently dramatic in terms of the mortality involved, which has a rapid onset and often an exponential daily increase.

One of the major cost-incurring factors in this group of diseases is the chemotherapeutic regimens, which could best be described as "categorical." That is, fish with the clinical signs of rapid, shallow respiratory movements, grossly enlarged gill tissues, and incomplete opercular closure are "treated" with one of the many medicaments added to the pond water. The results have ranged from "rewarding" to "well, we guessed wrong." the latter cases could have been prevented, perhaps, by elucidating the nature of the problem prior to treatment.

The purpose of this presentation is to provide information about the broad nature of gill diseases in fish. It is not intended as a review of each and every described gill disease. Such can be obtained from the texts written or edited by Plehn (1924), Halver (1972), Davis (1956), Sindermann (1969), Hoar and Randall (1984), Mawdesley-Thomas (1972), Reichenbach-Klinke and Elkan (1965), Snieszko and Axelrod (1971), Snieszko (1970), Roberts (1978), Ribelin and Migaki (1975) and Ellis (1985).

II. NATURE OF GILL DISEASES OF FISH

The known causal factors in gill diseases of confined and free-living fishes are myriad. For exemplification, these factors can be grouped into those which are (1) infectious or are (2) noninfectious, with each group being further subdivided into those factors which are primary and those which are secondary.

The primary causal factors are those which have a direct effect on the respiratory tissues and/or processes. Included in this group are the nonsystemic myxobacteria, eubacteria and fungi, the external protozoans and monogenetic trematodes, the water-borne chemical and physical agents, and the genetic and nutritional factors.

The secondary causal factors are those which affect the respiratory tissues and processes indirectly. Included in this group are the systemic bacterial and viral pathogens, the systemic protozoans and digenetic trematodes, the environmental chemical and physical stressors and certain metastatic neoplastic processes. These factors, in most cases, exert their pathogenic capabilities throughout the body with the gill involvement being just a part of the somatic process.

The major factors predisposing fish to subclinical and/or clinical episodes of gill diseases are (1) the stress response; (2) age; (3) environmental conditions favoring the proliferation of infectious agents; i.e., myxobacteria, eubacteria, protozoa and fungi. The three factors are virtually always involved in clinical episodes.

The stress response, particularly to chronic stressors such as high population densities, has been shown to create pathological changes in the gill tissues which are conducive to the involvement of secondary infectious and/or noninfectious factors.

The susceptibility to noninfectious and infectious respiratory disease-inducing factors decreases with age. The highest incidence of respiratory diseases occurs in the sac fry stage during which the most common malady is sestonosis with subsequent myxobacterial and protozoal involvements. This condition is typified by the accumulation of particulate materials and fungal elements on the buccal aspect of the gill rakers. Death is usually by suffocation. Because of its nature, sestonosis is virtually untreatable. Strict cleanliness is the only sure method of prophylaxis.

The bacterial gill diseases are quite common in populations of fishes from the fingerling stage through the mid-juvenile stage. This group of maladies is most frequently a sequel to a noninfectious process, environmental gill disease (EGD). The EGD syndrome is considered to be, first, stress-mediated and, second, environmentally-mediated. By itself; i.e., uncomplicated by pathogens or noninfectious factors, it is more a debilitating process than it is lethal. This aspect is, perhaps, what makes it such an economically significant disease process. There is no specific recommended treatment regimen largely because the causal factors are often quite obscure, if evident at all.

III. PATHOLOGICAL CHANGES IN RESPIRATORY DISEASES OF FISH

A. Noninfectious causal factors: The gross and microscopic pathological changes in gill tissues, especially the lamellae, are dose:response dependent. The inflammatory process ensuring from the initial exposure begins with lamellar epithelial hypertrophy in which there is focal-to-generalized involvement of the squamous epithelial cells. If the irritant exposure is short-lived, the hypertrophic condition subsides within a few days, usually without notice.

If the irritant exposure continues, the hypertrophic condition is "joined" by lamellar epithelial-capillary endothelial separation (ECS). In this lesion the squamous epithelium becomes separated from the underlying capillary endothelium and the resultant space is filled with a serous exudate. At this point in the process, especially if there is generalized involvement, the fish could be exhibiting clinical respiratory disease, particularly during or immediately following physical handling. This condition can be readily seen in wet mounts of gill tissues following a 1-hour exposure to certain water-administered chemotherapeutants such as formalin.

In the subacute inflammatory process,the lamellar hypertrophy is replaced with epithelial hyperplasia of, first, the lamella epithelium and then the filamental epithelium. The hyperplastic response can be terminal; i.e., involving only the distal portions of the lamellae, or it can involve the entire lamellae. In the case of nutritional gill disease (NGD) the hyperplastic response typically begins at the interlamellar filamental space and progresses distal to - but not beyond - the lamellar tips. This condition, over a period of 1-2 weeks, gradually worsens to become interlamellar hyperplastic occlusion, in which the interlamellar spaces are completely obliterated. At this point the fish is often visibly distressed. Gill tissues frequently protrude from the opercular cavity and there is incomplete closure of the opercula. Grossly, the excised gill is quite characteristic with filamental separation due to the increased mucus production and the entrapped aquatic particulates. Many fish pathologists regard this stage in the process as the point where secondary involvement by bacterial and protozoan pathogens occurs. In contrast, many reports of myxobacterial gills disease include the conclusion that the myxobacterium was the exciting agent in this condition.

With proper chemotherapy and management practices the foregoing responses can be healed. The repair process in the more severe cases requires 2-3 weeks, provided there are no further insults to the physiological respiratory process.

In recent years there has been considerable interest in a pathological process called "gill necrosis" (GN). The syndrome occurs predominately in farm-raised carp and is thought to be the result of chronic exposure to "autointoxication by ammonia under the influence of pH"; however, by most it is considered to be an idiopathic (unknown)process.

Another idiopathic process affecting the gill lamellae both anatomically and physiologically is intralamellar engorgement. This condition has been reported as lamellar telangiectasis and as lamellar aneurysm. The lesion is quite characteristic and involves only isolated lamellae. The process apparently begins with a lemellum becoming engorged with blood to several times its usual size. At this point the lesion is quite susceptible to being physically ruptured. Without rupture there is, within a few days, thrombus formation followed by typical clot organization, fibrosis and resorption. Under optimum environmental conditions, the entire process from onset to complete resorption can require 30-50 days.

B. Infectious causal factors: The pathological changes associated with direct and indirect infectious causal factors are often quite confusing for the diagnostician. In the directly associated causal factors the pathogen usually occupied an opportunistic role. That is, it "set up housekeeping" on gill tissues already affected by some moderately long-standing noninfectious process.

Following the establishment of the host-parasite relationship a series of degenerative changes ensure within a matter of days. Among the degenerative changes are (1) hemorrhage; (2) necrosis; and (3) death due to anoxia or to critical loss of osmoregulatory function. This series of changes is most commonly seen in the bacterial gill disease (BGD) complex in which both ubiquitous aquatic myxobacteria and eubacteria are involved.

The changes associated with the parasitic protozoa and monogenetic trematodes are frequently less dramatic unless there are other noninfectious environmental factors operating. In the case of chronic Trichodina infection, during the winter months there is often a characteristic host response to the organism "hibernating" on the gill lamellae and/or filaments.

In systemic bacterial diseases such as furunculosis, enteric redmouth disease, vibriosis, bacterial hemorrhagic septicemia and edwardsiellosis there is often secondary, nonspecific involvement of the respiratory tissues. The main lesion in these cases is frank hemorrhage and necrosis with secondary fungal involvement.

Nonspecific necrotic changes in the gill tissues are often attributable to secondary involvement with Saprolegnia, a ubiquitous aquatic phycomycete. On the other hand, in the case of European gill rot or fungal gill rot, the causative organisms, Branchiomyces sanguinis and B. demigrans are intravascularly invasive, which causes an infarctive necrosis of gill lamellae. The terminal lesion has been termed "gangrenous branchitis," for which there is no therapeutic regimen.

One of the more infrequently observed, but nonetheless puzzling, lesions is the granuloma. This lesion appears sporadically and may be a benign neoplasm or of bacterial (Renibacterium salmoninarum or Mycobacterium sp..) origin. Similar lesions are protozoan sporocysts; e.g., Plistophora salmonis or developing metacercaria; e.g., Sanguinicola klamathensis. When these organisms emerge from the lamellae there can be a transient blood-loss anemia, which under certain circumstance can reduce the resistance of the host to secondary bacterial infections.

IV. PREVENTION AND CONTROL OF RESPIRATORY DISEASES OF FISH

There are several approaches to preventing and controlling respiratory disease episodes in confined fish populations. The first is avoidance of the conditions which are conducive to the occurrence of subclinical and clinical episodes. This is best accomplished by (1) maintaining the fish within the environmental "no-effect" limits with respect to settleable and suspended solids, ammonia-nitrogen, dissolved oxygen and population density (Klontz eta l, 1985a) and by (2) routine examination of gill wet mounts, production data and feed conversion ratios.

The "no-effect" limits of environmental factors have been established for some species of salmonids under certain, albeit limited, conditions. The best suggestion is for the individual aquaculturist to establish the unique limits for his/her facility. To accomplish this one must establish a protocol for measuring the environmental parameters and their effects on fish growth and gill tissues. This process should begin with sac fry and continue throughout the production cycle. One caveat is that the process is time-consuming and often frustrating, but always rewarding in the long term.

During the process of establishing the specific "no-effect" limits of environmental parameters, wet mounts of gill tissues must be examined on a regular basis. Fish should be taken from the "healthy" and the "unhealthy" or "sickly" portions of the populations. The fish are prepared for examination by killing them with a sharp blow to the dorsum of the head and exanguination by severing either the causal peduncle or the spinal cord immediately posterior to the base of the skill. An entire gill arch is removed and placed into 10% neutral buffered formalin for no longer than 1-2 minutes. It is either examined in toto (small fish only) or a few of the filaments are removed with scissors, mounted in pH 7.2 phosphate-buffered normal saline and examined using the 10X and 100X objectives. Paraffin-imbedded sections of the lamellae and filaments may be prepared but there are very time-consuming and expensive and not any more illustrative than are wet mount preparation, which can be done on site very rapidly.

If and when a clinical episode of respiratory disease occurs, then an accurate diagnosis must be made prior to initiating any therapeutic regimen. The sequence of changes occurring in the gill tissues is the best indicator of the nature of the causal factors involved. Lesions such as hypertrophy, ECS, and occlusive hyperplasia all suggest basic physiological upsets which may be reflected by alterations in other systems. The presence of bacteria and the so-called gill parasites often is a reflection of an underlying environmental problem, most common of which is "poor housekeeping." At this juncture, it might be apropos to present an oft-quoted saying by Frederick Fish, a fish pathologist of the 1930's, to wit: "In fish culture, cleanliness is not next to godliness - it supersedes it." (Fish, 1939).

Once the problem is defined; i.e., the major causal factors identified, the next step is to "re-balance" the system. This is best accomplished by, first, withholding feed for 3-4 days, if the fish are of sufficient size to permit this. This will (1) reduce the oxygen demand of the fish; (2) reduce the ammonia-nitrogen generation by the fish and (3) reduce the fecal and uneaten solids in the system. Second, administer sufficient salt (as granulated NaCl) to the system to obtain a 1-2% solution. This will (1) reduce the blood ammonia-nitrogen levels; (2) stimulate mucus secretion; and (3) have an astringent effect on the gill tissues. Third, reduce the population density to approximately one-half the oxygen-related carrying capacity of the system. This should be accomplished without unduly stressing the fish.

If, in addition to the environmental factors being corrected, there are infectious agents involved, the following treatment regimen has been effective. First, select a candidate chemotherapeutant which can be administered by water exposure. Second, conduct a bioassay using at least two and preferably three levels of the candidate chemotherapeutant administered in the fashion to be used for the affected population. The fish to be tested for efficacy and tolerance to the chemotherapeutant should be obtained from the clinically affected and "healthy" portions of the affected population. At the end of the bioassay period - usually an hour for pond conditions, the treated fish should be examined for efficacy. If the target organisms were killed or removed from the gills, the treatment was effective - and safe, if the test fish survived. These fish should remain under separate conditions for 12-18 hours following the bioassay to detect any delayed effect.

V. SUMMARY

As was implied at the outset of this presentation, respiratory diseases of fish in aquaculture systems have been dealt with in a somewhat haphazard fashion. Many aquaculturists regard them as a nuisance and apply all manners of medicaments to prevent their occurrence. This approach, although sound in some respects, has had its shortcomings; namely, the problem was identified but inadequately defined. As a result, the factors - intrinsic and extrinsic - involved have not been corrected. For some strange reason the term "disease" in the vocabulary of the majority of fish farmers and, to some extent, the practicing fish pathologists, denotes only those conditions involving a microbial pathogen. The noninfectious elements of a clinical episode are virtually ignored, thus being left to exert their respective influences another day. To illustrate this point, all one must do is to examine the textbooks of fish diseases and see that the causative agents of infectious diseases are described in what could best be defined as "loving terms." This quite understandable since the authors are, for the most part, well-known and highly respected microbiologists. Perhaps a case should be made to increase our understanding of the epidemiological facets of disease episodes. This aspect of medicine has a proven track record in human and veterinary medicine.

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