A GUIDE TO INTEGRATED FISH HEALTH MANAGEMENT IN THE GREAT LAKES BASIN

edited by Fred P. Meyer
James W. Warren
Timothy G. Carey

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Great Lakes Fishery Commission

1451 Green Road
Ann Arbor, Michigan 48105

April 1983

The Great Lakes Fishery Commission, established by the 1955 Convention on Great Lakes Fisheries between Canada and the United States, was organized in April 1956 and assumed its duties as set forth in the Convention on July 1, 1956. The Commission has three major responsibilities: to develop and coordinate fishery research programs, to advise governments on measures to improve the fisheries; and to develop measures and implement programs to manage sea lamprey. The Commission is also required to publish or authorize the publication of scientific or other information obtained in the performance of its duties.

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A GUIDE TO INTEGRATED FISH HEALTH MANAGEMENT IN THE GREAT LAKES BASIN

edited by

FRED P. MEYER
U.S. Fish and Wildlife Service
National Fishery Research Laboratory
P.O. Box 818
La Crosse. Wisconsin 54601

JAMES W. WARREN
U.S. Fish and Wildlife Service
Fish Disease Control Center
P.O. Box 1595
La Crosse, Wisconsin 54601-0146

and

TIMOTHY G. CAREY
Department of Fisheries and Oceans
Aquaculture and Resource Development Branch
240 Sparks Street
Ottawa, Ontario, Canada K1A 0E6

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Ann Arbor, Michigan 48105

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CONTRIBUTORS

Timothy G. Carey, Aquaculture and Resource Development Branch, Department of Fisheries and Oceans, 240 Sparks Street, Ottawa, Ontario K1A 0E6

C. Young Cho, Fish Nutrition Laboratory, Fisheries Branch, Ontario Ministry of Natural Resources and Department of Fish Nutrition, University of Guelph, Guelph, Ontario N1G 2W1

John B. Daily, Section of Fisheries, Department of Natural Resources, Box 12, Centennial Office Building, St. Paul, MN 55155

Philip P. Economon, Department of Natural Resources, 390 Centennial Office Building, St. Paul, MN 55155

Hugh W. Ferguson, Department of Veterinary Pathology, Ontario Veterinary College, University of Guelph, Guelph, Ontario N1G 2W1

David B. Goldthwaite, Fishery Resources, U.S. Fish and Wildlife Service, One Gateway Center, Suite 700, Newton Comer, MA 02158

Robert H. Griffiths, Supervisor of Fish Culture, New York Department of Environmental Conservation, 50 Wolf Road, Albany, NY 12233

Jack R. Hammond, Michigan Department of Natural Resources, Fisheries Division, Box 30028, Lansing, MI 48909

John G. Hnath, Michigan Department of Natural Resources, Wolf Lake Fish Hatchery, 24270 C.R. 652, Mattawan, MI 49071

Rodney W. Homer, Division of Fish and Wildlife Resources, Department of Conservation, R.R. 3, Clear-view Estates, Manito, IL 61546

Vincent A. Mudrak, Pennsylvania Fish Commission, Benner Springs Fish Research Station, R.D. 1, Box 485, Bellefonte, PA 16823

Lyle L. Pettijohn, U.S. Fish and Wildlife Service, National Fish Health Research Laboratory, Leetown, Route 3, Box 700, Kearneysville, WV 25430

Richard Poynter, Oden State Fish Hatchery, Fisheries Division Michigan Department of Natural Resources, 3377 ½ Oden Road, Oden, MI 49764

Roger W. Ritzert, Corporate Technology, Owens-Illinois, One Seagate, Toledo, OH 43666

John H. Schachte, Jr., Fish Pathology Laboratory, New York Department of Environmental Conservation, 8314 Fish Hatchery Road, Rome, NY 13440

Al J. Sippel, Fisheries Branch, Ministry of Natural Resources, 99 Wellesley Street, Toronto, Ontario M7A 1W3

Brian Souter, Fisheries Research Branch, Department of Fisheries and Oceans, 501 University Crescent, Winnipeg, Manitoba R3T 2N6

James W. Warren, U.S. Fish and Wildlife Service, Fish Disease Control Center, P.O. Box 1595, La Crosse, WI 54601-0146

Harry Westers, Michigan Department of Natural Resources, Fisheries Division, Box 30028, Lansing, MI 48909

PREFACE

The Great Lakes Fishery Commission is pleased to publish A guide to integrated fish health management' and wishes to express its thanks to the member agencies of the Commission for their efforts. Credit for this valuable contribution to fish disease control belongs to the members of the Great Lakes Fish Disease Control Committee (GLFDCC) and, in particular, to Jim Warren, the committee chairman, Fred Meyer and Tim Carey who have skillfully guided this project to completion.

Because fish cultural activities are an important element in the rehabilitation of the Great Lakes fishery, the Commission established the GLFDCC in 1973 to recommend measures to protect the health of cultured and native fish populations. In 1975, the Commission adopted recommendations from the GLFDCC and issued a fish disease control policy and model program. The model program coordinates public and private efforts to reduce infectious disease losses in hatcheries, helps reduce the spread of fish diseases in the Great Lakes basin, and helps to prevent the introduction of new diseases into the basin. However, the model program was not designed to provide specific, detailed information on the methods to be used to prevent or control serious fish diseases. Additional information was needed on topics ranging from hatchery water supplies and facility design, to disease eradication procedures and cooperative fish health management programs to fill out the framework of the model fish disease control program. Such a compendium was needed to place information from many scattered sources within the reach of resource administrators, fish culturists, fish pathologists, students and interested lay people.

With confidence and enthusiasm, the GLFDCC set forth in April 1981 to develop the needed compendium. From the beginning it was agreed that the committee would not attempt to prepare a definitive text on fish health management. On the other hand, the compendium was to be an integrated structured guide to the implementation of fish health programs at all operational levels in the Great Lakes basin and elsewhere. The project has contributed significantly to the cooperative spirit and unity of the GLFDCC and its member agencies. The Commission is grateful to the contributors whose efforts made the completion of this compendium possible, and to the many experts in the USA and Canada who commented on individual contributions as part of the peer review process required for Commission publications.

K. H. Loftus,
Chairman,
Great Lakes Fishery Commission

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INTRODUCTION

T.G. CAREY
Department of Fisheries and Oceans
Ottawa, Ont.

and

D.B. GOLDTHWAITE
U.S. Department of the Interior
Fish and Wildlife Service
Newton Comer, MA

Fish culture has assumed increasing importance in North America as an alternative strategy for increasing production from the aquatic resource. Coincidental with the expansion of culture activities has been an increase in the incidence and spread of fish diseases which, in turn, stimulated extensive related research and surveys. Publications are currently available that describe the distribution of many fish disease agents, disease detection and identification procedures, fish pathology, and treatment methods (Amlacher 1970; Davis 1953; Mawdesley-Thomas 1972; Roberts 1978; Roberts and Shepherd 1974; Snieszko 1970; Van Duijn 1973; Warren 1981). However, the subject of planned programs for the reduction of risk exposure to diseases, as a specific approach for improving fish health, has not been adequately covered in the literature.

The purpose of this "Guide to Integrated Fish Health Management in the Great Lakes Basin" is to create an awareness on the part of fish culturists and administrators of the benefits of implementing continuous, integrated disease control procedures rather than reacting to disease problems as and when they arise. Information is presented in the Guide on important factors that contribute to the introduction and dissemination of fish diseases, and the measures available to reduce the risks of disease exposure.

The Guide is sub-divided into six parts, including sections on The Fish Health Management Problem (Part I), Disease Control Options in Hatchery Management (Part II), Administrative Options in Disease Control (Part III), and Integrating Fish Health Management Options (Part IV). For reference purposes, information on fish diseases of concern in the Great Lakes region is provided

(Part V), as well as scientific terms, common and scientific names of selected fish species, training needs (Part VI), and details of the Great Lakes Model Fish Disease Control Program.

In Part I, The Fish Health Management Problem, clarification is provided on the actions, reactions and interactions of the host/pathogen/ environment complex in fish culture, and the delicate balance that must be maintained between these factors if disease outbreaks are to be avoided. No single approach to providing good health can be effective on its own. Steps must be taken to prevent stress in the host, to provide an environment that is more favorable to the host than to the pathogen, and to prevent introduction or to reduce the density of pathogens. The importance of pre-planning to integrate these actions into a comprehensive program for individual fish culture operations is emphasized.

Communicable fish diseases are generally concentrated in or close to fish culture operations, thus, efforts to reduce or eliminate the incidence of diseases should focus first on these sites. Fish culture, in the context of this Guide, includes all strategies involving confinement of fish in aquatic ecosystems and ranges from extensive culture (close-to-natural environment) to intensive systems characterized by specialized rearing facilities, high stock densities, use of artificial feeds, and partially or wholly controlled environments.

Part II of this Guide provides a range of options available to producers, individual hatchery managers, or program administrators for reducing the risk of exposure to diseases in fish culture facilities. For each option, information is provided on the purpose of the action, what goals can be achieved, the basic facilities and equipment required, and a general assessment of the effectiveness of options in different situations. Subject areas relate to disease prevention, disease control, and eradication of disease.

In Part III, Regional Control of Communicable Diseases of Fish presents broader strategies for reducing risk of introduction and spread of fish diseases, offering approaches that are regional, state/provincial, or national in scope. Control measures described take into account the threat of disease to natural as well as cultured populations, and serve to protect innocent parties and good operators who have developed high hygiene standards. Reference is made to accountability for fish health protection at different levels of administration, and regulatory mechanisms available for control of communicable diseases are described.

Part IV, Synthesis of a Fish Health Management Program, provides the basis for, and explains the importance of, integrating fish health management with all other fish culture activities to maximize health protection. It also identifies the parallel between integrated fish health management and integrated pest management in agricultural science and stresses the need to coordinate all control methods into a unified program directed at optimal control,

Finally, because the field of fish health encompasses a wide range of biological and technical disciplines, three Appendices and a Glossary have been included to identify sources of training and information, to describe the Great Lakes Fish Disease Control Program, to clarify scientific terms, and to list the scientific and common names of fish species referred to in the text. Names of fishes used in the text are the accepted common names in North America.

Mention of drugs or chemicals in this volume does not constitute recommended use. Readers should always follow recommendations on the product

label. If the desired use is not specified, the Food and Drug Administration should be contacted before a product is used. Likewise, the use of brand names or model numbers does not in any way constitute recommendation or endorsement of any products or equipment.

SOME IMPORTANT INFECTIOUS DISEASES OF SALMONIDS

Severe epizootics among salmonid fishes can be caused by a variety of physical, chemical, and biological disease agents. Although the importance of physical and chemical agents cannot be disregarded, these are seldom a problem in fish cultural facilities with adequate and reliable water supplies. Biological agents of infectious disease include viruses, bacteria and parasites, each of which presents complex and challenging control problems in the Great Lakes basin.

According to Wolf (1972), fish viruses fit the known patterns established for animal virology in general. They cannot be seen by the light microscope, require a living cell in which to replicate, and, for the most part, are unaffected by therapeutic drugs and chemicals used to control bacterial and parasitic diseases of fish. Infectious pancreatic necrosis (IPN) has been diagnosed in several locations in and around the Great Lakes basin and, at present, is the only important viral disease of salmonids known to be established in the basin. Infectious hematopoietic necrosis (IHN) has occurred at isolated locations in Minnesota and New York, but neither of these cases were located within the Great Lakes basin and both outbreaks were eradicated. IHN continues to be a persistent, serious problem on the West Coast of North America. Viral hemorrhagic septicemia (VHS) has not been reported in North America but causes serious economic impact on trout producers in Western Europe. IPN and IHN infections cause serious losses of fry and fingerlings, VHS can cause heavy mortalities among older fish. Both IPN and IHN can be spread through the shipment of contaminated eggs taken from virus-carrying adult spawners. Viral hemorrhagic septicemia may also be egg-transmissible, but it is spread primarily through the transfer of carrier fish and possibly through the shipment of frozen fish. Avoidance is the best procedure for minimizing the threat of viral diseases.

Bacterial diseases have long been a source of serious problems for cultured salmonids. External infections, like those caused by gill disease bacteria, are common and often costly. Columnaris disease, caused by *Flexibacter columnaris* can occur as a superficial external problem and as a systemic infection. Both forms of columnaris may require antibiotic therapy for control in certain cases. Furunculosis, a disease caused by *Aeromonas salmonicida,* is commonly associated with less than ideal environmental conditions for the cultured fish stock. Enteric redmouth (ERM), caused by *Yersinia* ruckeri, is a new problem in the Great Lakes basin. Its spread has been traced through the introduction of carrier fish from Idaho where the disease is well established. Bacterial kidney disease (BKD) remains a particularly insidious bacterial infection because it can be transmitted with eggs from infected adults, cannot be effectively controlled with antibiotics, and often does not become apparent until the affected fish are more than a year old. Each of these diseases presents significant control problems, some of which defy prompt solution.

Two parasitic diseases of salmonids are designated for special consideration in the Great Lakes basin. Whirling disease, caused by *Myxosoma cerebralis,* has

been a major concern since it was first detected in Michigan in 1968. It remains a potentially serious problem but, thus far, it has been effectively confined to certain watersheds of *Michigan's* lower peninsula. *Ceratomyxa shasta* is a serious problem at some trout and salmon facilities on the West Coast of North America. It has not been detected in the Great Lakes basin.

Other important viral, bacterial and parasitic diseases of cultured fish can be more troublesome than those listed here. The listed diseases, however, have been identified by the conservation agencies in the Great Lakes area for specific prevention and control efforts. Specific information is provided on these diseases later in this compendium and mention will be made of these diseases when describing various options for dealing with the particular disease.

REFERENCES

Amlacher, E. 1970. Textbook of fish diseases. D.A. Conroy and R.L. Herman (trans.). TFH Publications, Inc., Neptune City, NJ. 302 p.

Davis, H.S. 1953. Culture and diseases of game fishes. Univ. of Calif. Press, Berkley, CA. 332 p.

Mawdesley-Thomas, L.E. (ed.). 1972. Diseases of fish. Academic Press, London. 380 p.

Roberts, R. J. 1978. Fish Pathology. Balliere Tindall, London. 318 p.

Roberts, R. J., and C. J. Shepherd. 1974. Handbook of trout and salmon diseases. Fishing News Books Ltd., Surrey, England. 168 p.

Snieszko, S.E (ed.). 1970. A symposium on diseases of fishes and shellfishes. Am. Fish. Soc., Spec. Publ. No. 5. Bethesda, MD. 526 p.

Van Duijn, C. 1973. Diseases of fishes. Charles Thomas, Springfield, Il. 372 p.

Warren, J.W. 1981. Diseases of hatchery fish: a fish disease manual U.S. Fish and Wildlife Service, Twin Cities, MN. 91 p.

Wolf, K. 1972. Advances in fish virology: A review 1966-1971, p. 305-331. In L.E. Mawdesley-Thomas, (ed.). Diseases of fish. Symp. Zool. Soc. London, Symp. No. 30. Academic Press, New York, NY.

PART I

THE FISH HEALTH MANAGEMENT PROBLEM

1. THE NATURE OF FISH DISEASES

2. THE ROLE OF IMPROVED HUSBANDRY PRACTICES

3. PLANNING A FISH HEALTH PROGRAM FOR HATCHERY OPERATION

1

THE NATURE OF FISH DISEASES

J.W. WARREN
U.S. Department of the Interior
Fish and Wildlife Service
La Crosse, WI

Many bacteria and parasites that are capable of causing serious diseases of fish are normal inhabitants of the aquatic environment. In spite of their presence, disease may not occur. Are these potential pathogens and their fish hosts merely players in a larger environmental scenario that controls their interrelationship? Dr. S.E Snieszko (1972), in addressing this question, concluded a review article entitled "Progress in fish pathology in this century" with a quote from George Bernard Shaw's "Doctor's Dilemma" which says:

> "The popular theory of disease is that every disease had its microbe duly created in the Garden of Eden, and has been steadily propagating itself and producing widening circles of malignant disease ever since. It was plain from the first that if this had been approximately true, the whole human race would have been wiped out by the plague long ago, and that every epidemic, instead of fading out as mysteriously as it rushed in, would spread over the whole world. It was also evident that the characteristic microbe of a disease might be a symptom instead of a cause."

Snieszko ended his article with the rhetorical question: "Was he (Shaw) right?" It is the purpose of this paper to provide information that will give insight into this question.

Simply stated, disease is literally a lack of ease. It can be defined as a morbid process or condition in the body or its parts with characteristics which distinguish it from other morbid processes or conditions and from the normal state. Diseases can be infectious, that is, communicable from one host to another, or non-infectious. The course of a disease may range from short-term, lethal effects to chronic, inapparent conditions which are detectable only by necropsy or by specific tests conducted at appropriate times. Obviously these definitions are broad and encompass a wide variety of circumstances and results. Disease is

a complex interaction between the fish, disease agents and the environment. Understanding the processes involved is vital when considering diagnosis, prevention and cure.

In fish populations, one of the earliest signs that disease may be present is a nonspecific increase in the mortality rate. Fish that die at this stage may be those that are unusually susceptible to the pathogen present or may be those that are most susceptible to the adverse environmental conditions that may trigger epizootics. In any event, a fish culturist or biologist who is attempting to cope with a disease problem must first assemble some basic information.

THE HOST, PATHOGEN, ENVIRONMENT RELATIONSHIP

Three factors must be considered in any fish disease investigation. These are the susceptible host, the virulent pathogen, and the environment in which they encounter one another. Even though all three may be present, a host and pathogen may interact without resultant disease. However, if a disturbance in any of the three factors disrupts the relationship, disease can appear and spread. One can think of this graphically as a weighted balance in which one of the pans represents the pathogen and the other pan represents the host. Environmental conditions would be represented as weights placed on either pan since these factors may affect the host in either positive or negative ways. If the pathogen and the host are in equilibrium, the balance remains at rest. If weights (environmental factors) are added to one side only, the balance will tilt. The causes of this imbalance must be determined if one is to develop an understanding of these three important factors and their interplay.

THE SUSCEPTIBLE HOST

If a plant or animal host is not susceptible to a disease, that disease cannot occur. This, of course, has been the basis for mass immunization programs in human medicine against poliomyelitis, measles, and other diseases. Susceptibility, however, is not governed only by immunity. Habits and customs affect the susceptibility of certain ethnic groups to food-borne infections. For example, those who do not eat pork cannot get trichinosis from that source. Similarly, the practices and techniques used by fish culturists also play an important role in the infectious agents to which their fish may be exposed. A high-quality diet and a clean water supply will eliminate a number of potential sources of disease just as would avoiding the introduction of new fish from another hatchery.

Within the fish itself, the defense mechanisms are varied and complex. Resistance to infection is arrayed like an army, in several echelons. The front line of defense is the skin, scales and mucous membranes which limit the entry of toxic, infectious, and parasitic agents. The next rank of defense is physiological. White blood cells that engulf pathogens, avoidance mechanisms, the ability of the liver to detoxify chemicals from the water or diet, storage of certain metals in the bones, local tissue reactions, and other reponses all help fish to keep noxious agents from overwhelming the body. The last line of defense is the immune system and its specific activity against biological agents such as viruses, bacteria and parasites.

Other, more general factors also join in defending the host against disease. Certain diseases affect fish of certain age groups or species more severely than others. A constitutional factor, or the will to live, may help fish resist the effects of disease or trauma. Evidence of this might be an adult salmons instinct to complete its spawning migration.

THE VIRULENT PATHOGEN

A virulent pathogen is usually thought of as a microbe, capable of causing infectious disease. Obviously, if such a pathogen is absent, there can be no infection or disease. However, one must not forget those factors whose absence may cause non-infectious diseases. Vitamin deficiencies, a deficiency of oxygen, and mineral deficiency in the water can all cause survival problems. Pathogenic agents can therefore be classified as physical, chemical, and biological.

Physical agents may be mechanical, thermal, or radiant in origin. One of the most common examples of mechanical agents in cultured fish is the "boot fever" suffered when fish are stepped on, injured, or stressed during handling, sorting, or moving. Simple traumatic injuries share many pathogenic features with some of the more complex disease problems. Temperature extremes are physical factors that threaten both cultured and wild fish. Unlike warm-blooded animals, the environment controls the body temperature of fish. In the wild, free-ranging fish sometimes have a greater opportunity to seek more desirable temperatures, but occasionally oxygen levels are too low in cooler water near the bottom of a pond or lake where fish seek relief from higher temperatures. Radiant agents include ultraviolet rays from the sun which can cause sunburn or possibly cataracts in sensitive fish. Physical agents are sometimes responsible for sudden, explosive mortalities in both cultured and wild fish populations.

Chemical agents can cause illness in a variety of ways. Environ- mental contaminants, taken in by adults, may be concentrated in the oils that are deposited in their eggs. Such contaminants may later cause the death of sac fry during early development. Fish feeds contaminated by aflatoxins, produced by molds during storage, can cause hepatomas in rainbow trout. Nutritional disturbances ranging from excess body fat and hypervitaminoses to malnutrition and vitamin deficiencies are caused by the overabundance or lack of dietary elements. Drug and chemical overdosages that cause mortalities would be classified with chemical agents.

Biologic agents have played a major role in the initiation of disease and are the primary focus of attention when infectious diseases are encountered. They include multicellular organisms like flukes and worms: single-celled organisms such as Ichthyophthirius, *Trichodina* and a myriad of other forms including microscopic pathogens like bacteria; and finally, viruses that are too small to be seen under the ordinary microscope. All are important disease agents which can be responsible for serious epizootics.

A complete understanding of all of the agents of disease is usually not possible for every worker but fishery personnel should have a working knowledge of the nature of pathogens, their manner of spread, routes of entry into fish cultural facilities, and methods for their possible avoidance or eradication.

THE ENVIRONMENT

The causative agents of disease and their fish hosts do not carry on their struggle in a vacuum. The environment in which the encounter takes place may favor one or the other. Changes in the environment, whether natural or fish cultural, may shift the balance from one side to the other and will often determine whether the host will survive or succumb. Understanding and managing the environment is the key to successful fish culture, and a knowledge of the role of the environment in the nature and occurrence of disease is essential to fish disease control.

The physical environment provided for fish culture is affected by the location (geography) of the facility. Latitude governs the ambient temperature of ground water and temperature and altitude both determine oxygen saturation levels. Season and climate are also tied closely to geography. Climatic conditions have a great influence on the sources, quantity and quality of water available and consequently, in the determination of the species and numbers of fish that can be safely reared at a given facility. The physical conditions set by the environment also affect which fish pathogens might flourish and which ones might be of little significance. For example, columnaris disease is seldom a severe problem at water temperatures below 55°C (12.7°C) (Holt et al. 1975) so it is less of a problem in northern regions than in the south. Oxygen depletions due to snow-covered ponds are a problem in the winter in the north, while oxygen depletions due to decomposition following heavy algae blooms, are more common in southern pond culture during the summer.

Facility design and construction can have a profound bearing on the occurrence of disease. Earthern facilities provide environmental conditions that favor certain disease agents that need not be considered when concrete facilities are used. Sanitation, a clean water supply, proper collection and disposal of moribund and dead fish, and proper management of population densities are among the measures that contribute to the production of healthy fish (Wedemeyer et al. 1976).

Biological factors in the environment are of particular importance in the evaluation of fish cultural conditions and in the occurrence of infectious diseases. It is important to know whether fish reside in the water supply. If they do, the culturist must be alert to the possibility that such fish may be the reservoir for infections that occur in cultured fish. Infectious disease cannot occur in the absence of a virulent pathogen and fish in the water supply system are a common source. Hatcheries with springs and wells for water supplies have greater control over the entry of disease agents than facilities that are stream-fed.

Variations in water quality are a major source of environmental stress encountered by fish. Human beings maintain a nearly constant body temperature of 98.6°F (37°C) and breathe atmospheric air which, at sea level, normally contains 21% oxygen, 78 % nitrogen, 1% argon and 0.04 % carbon dioxide. On the other hand, in the aquatic environment, temperatures and concentrations of dissolved gases are highly variable. Supersaturation of water by air and other gasses can be a source of environmental stress. Although fish are well adapted to underwater life, they are at the mercy of the environment.

THE OCCURRENCE OF DISEASE

Wedemeyer, et al (1976) devoted an entire book to the role of environmental stress in fish diseases. In explaining the role of stress as a predisposing factor, they quote Dubos (1955):

> "There are many situations in which the microbe is a constant and ubiquitous component of the environment but causes disease only when some weakening of the patient by another factor allow infection to proceed unrestrained, at least for a while. Theories of disease must account for the surprising fact that, in any community, a large percentage of healthy and normal individuals continually harbor potentially pathogenic microbes without suffering any symptoms or lesions."

The pertinence of this quotation to the occurrence of disease in cultured or wild fish populations is obvious. Snieszko (1973) illustrates the situation with three overlapping circles to graphically represent the interrelationship between the susceptible host, the virulent pathogen and adverse environmental conditions (Fig. 1). As the significance of each of the contributing factors increases, the circles can be visualized to increasingly overlap, thus enlarging the common central area that represents circumstances under which coinciding elements will result in the occurrence of disease. By decreasing the impact of any of the three elements, there is a corresponding reduction in the magnitude of the disease threat (Fig. 2 and 3). This interrelationship is another way to express the example given earlier regarding the weighted balance concept.

When a disease outbreak is encountered, the pattern of losses, the species and sizes of fish involved, and the duration of the epizootic can provide a great deal of useful information (Wedemeyer et al. 1976). Sudden, explosive die-offs, involving all fish present (and sometimes tadpoles and other aquatic animals) usually indicate the occurrence of an acute environmental problem, such as a lack of oxygen, a lethal chemical toxicant, or lethal temperatures (Fig. 4). Mortalities that begin with the appearance of a few sick fish, unusual behavior, or a loss of appetite can signal the onset of infectious disease. These diseases have incubation periods ranging from a day or two for virulent pathogens like some outbreaks of columnaris (Becker and Fujihara 1978) to prolonged periods of several months in cases of bacterial kidney disease (Fryer and Sanders 1981). Infectious disease outbreaks in wild fish populations often affect only a single species (Wedemeyer et al. 1976; Unpublished case history records, U.S. F. W. S., Fish Disease Control Center, La Crosse, WI).

CONCLUSION

At the outset, a rhetorical question was asked regarding whether or not the presence of "the characteristic microbe of a disease might be a symptom instead of the cause." In many situations, cultured fish live healthy, normal lives in the continuous presence of pathogens. However, when environmental stresses occur and the balance tips in favor of disease, the characteristic microbes flourish. If the fish cannot adequately adjust or, if fish cultural corrections are not made, disease may occur. If losses mount in typical patterns, the fish culturist must act. By resolving environmental problems and applying effective therapeutants, a balance between the host and the pathogen can be restored. The

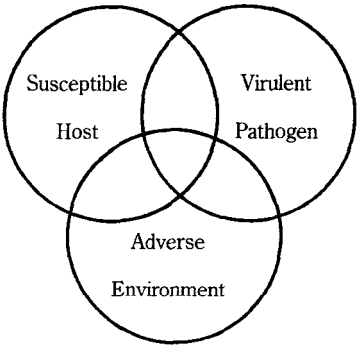


FIG. 1 Host-Pathogen-Environment interrelationship with disease

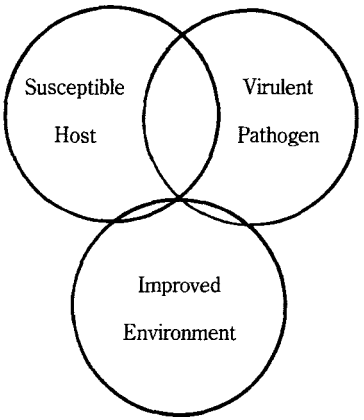


FIG. 2. Host-Pathogen-Environment interrelationship: No disease, improved environmental conditions

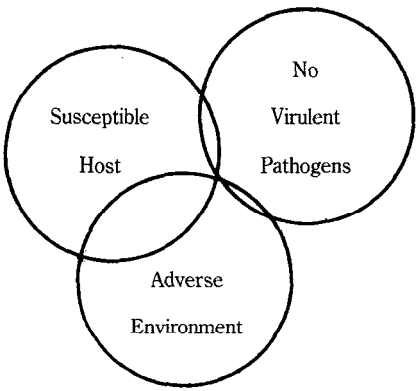


FIG. 3 Host-Pathogen-Environment interrelationship: No disease no virulent pathogens

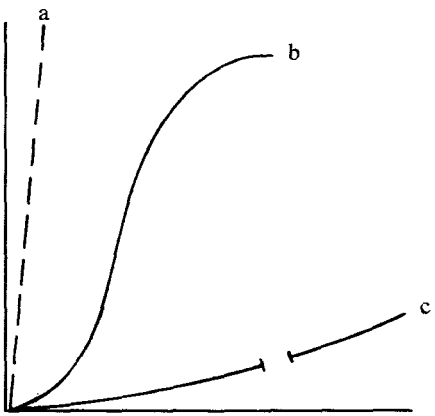


FIG. 4 Mortality curves: (a) acute environmental failure, (b) acute & (c) chronic infectious diseases

question still remains; Was the disease caused by the microbe or were the microbes and the fish merely players in a larger environmental scenario? A microbial infection can often be the symptom of environmental failure and an urgent signal that conditions must be changed. Successful fish culture often hinges on whether correction of adverse environmental conditions can be achieved in time to prevent losses (Snieszko and Bullock 1975). The skills related to fish culture which are required to maintain the balance between the host and the pathogen in the face of changing environmental conditions indicate that there is still a great deal of "art" in the "science" of fish culture.

REFERENCES

Becker, C.D., and poM.F! Fujihara. 1978. The bacterial pathogen *Flexibacter columnaris* and its epizootiology among Columbia River fish. Am. Fish. Soc. Monogr. 2. Bethesda, MD. 92 p.

Dubos, R.J. 1955. Second thoughts on the germ theory. Sci. Am. 192: 31-35.

Fryer, J. and J. E. Sanders. 1981. Bacterial kidney disease of salmonid fish. Annu. Rev. Microbiol. 35: 273-298.

Holt, R.A., J.E. Sanders, J.L. Zinn, J.L. Fryer, and K.S. Pilcher. 1975. Relation of water temperature to *Flexibacter columnaris* infection in steelhead trout *(Salmo gairdneri),* coho *(Oncorhynchus kisutch)* and chinook (0. *tshawytscha)* salmon. J. Fish Res. Board Can. 32: 1553-1559.

Snieszko, S.E 1972. Progress in fish pathology in this century, p. 1-15. In Diseases of fish. L.E. Mawdesley-Thomas, (ed.). Symp. Zool. Soc. London, No. 39. Academic Press, New York, NY. Symp. No. 39: 380 p.

Snieszko, S.E 1973. Recent advances in scientific knowledge and developments pertaining to diseases of fishes. Adv, Vet. Sci. Comp. Med. 17: 291-314.

Snieszko S.E, and G.L. Bullock. 1975. Fish furunculosis. U.S. Fish and Wildl. Serv., Fish Dis. Leafl. 43. Washington, DC. 10 p.

Wedemeyer, G.A., EI? Meyer, and L. Smith. 1976. Book 5 : Environmental stress and fish diseases. TFH Publications Inc., Neptune City, NJ. 192 p.

2

THE ROLE OF IMPROVED HUSBANDRY PRACTICES

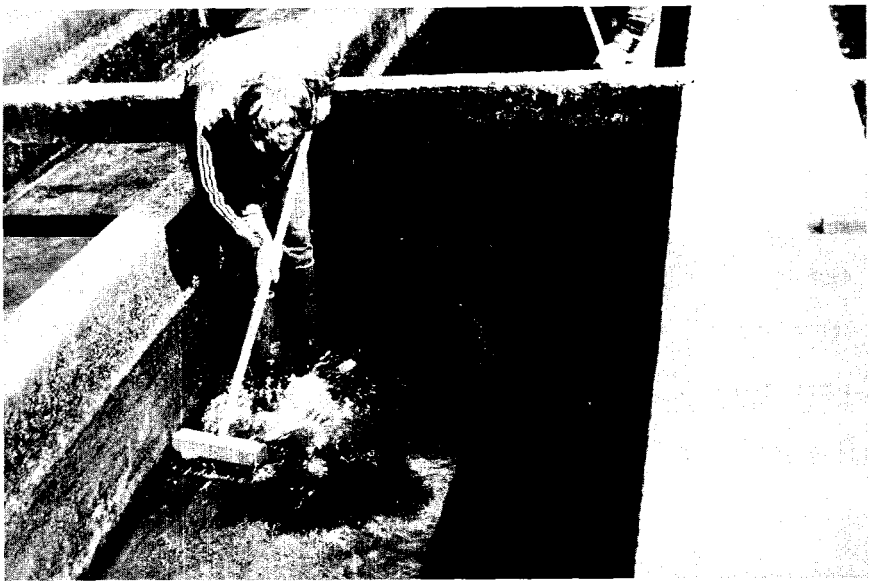
R.H. GRIFFITHS
New York Department of
Environmental Conservation
Albany, NY

and

J.W. WARREN
U.S. Department of the Interior
Fish and Wildlife Service
La Crosse, WI

One of the primary objectives of fish culture is the production of quality fish that are vigorous and healthy. Procedures for rearing salmonids have been described in detail by Davis (1953) and updated by Leitritz and Lewis (1980). Successful fish husbandry is an indication that good environmental conditions have been provided by the rearing facility. Improvements in fish husbandry practices can be used to reduce the effects of adverse environmental conditions, to overcome deficiencies in facility design, and to reduce the frequency and severity of stresses which contribute to the occurrence of disease.

Fish cultural practices must be coordinated with the construction and layout of the facility, the kinds of fish being reared, and the quantity and quality of the available water supply. Problems that lead to the occurrence of disease have been built into some facilities (Needham 1977). Water supplies may be too limited for the number of rearing units, may carry disease agents, or may be supersaturated with nitrogen gas. In recent years, water recirculating and reconditioning systems have been developed to squeeze the maximum fish production out of available water supplies. The frequency of mechanical and environmental breakdowns increases with the complexity of the facility (Buss



Regular cleaning of rearing ponds eliminates accumulated organic wastes, and removes a potential reservoir for fish pathogens (Ontario Min. of Nat. Res.)

1981). Westers and Pratt (1977) compared single-use, straight raceways with recirculating water systems in Burrows ponds and found that the single-use raceways produced more fish of better quality. The apparent reason for this was related to environmental conditions. In single-use raceways, fish spend much of their time at the head end of the raceway near the water source. Poorer water quality at the lower end of the raceway was avoided. In recirculating raceways, a less than optimal environment was present throughout the system from which the fish could find no respite.

Problems associated with fish stocks themselves can have far-reaching effects. Inbreeding can reduce the genetic diversity needed for survival and for resistance to a variety of diseases. In many situations, broodstock lines are maintained because of tradition rather than because of sound genetics. If a broodstock population is infected with viruses or bacteria which can be transmitted from parents to progeny with eggs, the spread of disease can be rapid. Hatchery programs have been responsible for the transfer of a number of diseases through shipments of eggs and fish (Kimura and Awakura 1977; Busch and Lingg 1975). In the case of direct transfers of fish from hatchery to hatchery, the chances of spreading infectious diseases and parasites are greatly increased. Careful planning of production programs is needed and specific information on the disease status of all potential sources of eggs and fish is imperative.

PLANNING FOR DISEASE PREVENTION AND CONTROL TO IMPROVE FISH HUSBANDRY

Fish disease surveillance is as important as adequate rearing facilities, quality fish feed, trained personnel and good fish transport equipment. Accurate

and timely information from a fish health laboratory can be valuable when production programs are being planned and sources of eggs and fish are being identified. The avoidance of disease problems by careful planning is far less costly than problems that must be corrected after they occur. Effective fish health inspection and hatchery disease classification schemes can help administrators and fish culturists prevent the spread of serious diseases. Information derived from disease surveillance programs is also useful in directing fish disease research studies, planning the rehabilitation of facilities, and in guiding the improvement of fish cultural practices.

Planning during the development of new fish cultural facilities provides an opportunity to accommodate improved husbandry practices and fish health protection. An adequate supply of clean, fresh water which is free of resident fish is one of the most important assets a facility can have. The species, sizes, and numbers of fish to be reared must be geared to the available water supply. These factors will determine the number and design of rearing units. Requirements for rearing unit sanitation between uses must be considered in the selection of construction materials and in the isolation of one unit from another. A fish barrier at the lower end of the facility should be included in the site plan to prevent contact between cultured fish and wild fish downstream from the hatchery. These features help to provide a good cultural environment and should be addressed during the design and construction of new production facilities and also during the rehabilitation of older hatcheries. Improvements can be made in existing programs, but it is often costly and difficult to accomplish without disrupting on-going production operations.

DISEASE CONTROL AND HATCHERY MANAGEMENT

The Ontario program for 'Fish Health Protection in the Provincial Fish Culture System' (A. J. Sippel, Ontario Min. Nat. Res., Toronto, personal communication) contains a brief section dealing with disease control and hatchery management. Most elements of fish husbandry are directly related to fish health protection. Consequently, wherever healthy fish are successfully reared, sound husbandry practices must be already in use. Diligence, persistence, and a firm commitment to the protection of fish health and to the production of quality fish lie at the heart of successful fish cultural programs. Required procedures include:

1. Minimizing stresses on cultured fish
2. Prevention of the introduction of serious (designated) diseases
3. Confinement of disease outbreaks to affected rearing units
4. Minimizing losses from disease outbreaks
5. Learning from past disease outbreaks to minimize future disease losses

Many other factors and numerous inter-relationships also affect the outcome of the interactions among fish and their pathogens. Successful fish health protection is the result of the successful management of these factors.

MINIMIZING STRESS

According to Wedemeyer et al. (1976), the prevention of fish diseases through proper environmental management requires an understanding of how environmental factors and stresses affect the physiology of culture fish. Environ-

Taking routine inventory of size and weight of fish in ponds is an important husbandry practice (Ontario Min. of Nat. Resources)

mental conditions and their improvement through better fish cultural techniques can have a significant bearing on the outcome of host/pathogen/ environment interactions. Wedemeyer et al. listed five important methods for improving husbandry practices:

1. Maintain water quality characteristics within the requirements of the species being reared.
2. Keep population densities regulated at levels low enough to prevent crowding stress and thus minimizing disease problems.
3. Learn to recognize environmental stress factors. Minimize or eliminate handling and other sources of stress and use prophylactic medication to prevent the activation of latent infections.
4. When stress is unavoidable, allow sufficient recovery time, based on the physiological disturbances involved, before again handling or stressing fish.
5. Use salt solutions (0.3% for salmonids) to mitigate stresses associated with handling and transportation.

Techniques that minimize environmental stress can be applied at all installations, old or new. Aeration devices used to remove excess gasses from inflowing water are a leading example. 'Packed columns' (Owsley 1981) are simple aerators made from 1.52 m lengths of plastic pipe, packed with appropriately-sized Koch rings, through which the inflowing water tumbles and splashes to establish atmospheric equilibrium. Packed columns effectively reduce supersaturated nitrogen gas levels associated with disease problems, impaired growth, and other conditions which are often difficult to diagnose.

Grading fish leads to more equitable size distribution in ponds (Ontario Min. of Nat. Res.)

DISEASE PREVENTION

Specific methods of fish disease prevention are reviewed in detail in other chapters of this Guide. The minimization of stress is related to the environmental aspects of the host/pathogen/environment relationship so disease prevention focuses on improving the disease resistance of the host and on the reduction or elimination of virulent pathogens.

Disease cannot occur unless a pathogen is introduced into the fish cultural system. Eggs or fish from uninfected sources have the best chance to remain free of infectious diseases if they are reared in clean water and in clean facilities. It is not a good practice to expose uninfected fish to diseases to 'toughen' them for possible future encounters with infectious agents after their release from the hatchery. Ensuing outbreaks can be severe in dense hatchery populations which are more conducive to the spread of infectious disease than conditions encountered in the natural waters where fish can seek preferred environmental conditions. If a specific survival problem has been identified, the best approach is to use immunization techniques to help fish ward off future infections. Species selection, nutrition, genetic diversity, sanitation, and the judicious use of prophylactic chemicals can all be used to help prevent diseases and to enhance the post-stocking survival of hatchery fish.

At large hatcheries, an extensive crew may be required to handle fish during the administration of a vaccine or antibiotic. This crew is injecting adult lake trout (U.S. Fish and Wildl. Serv.)

DISEASE DETECTION AND CONTROL

Major improvements in the health and survival of cultured fish can be made by careful disease surveillance. Some fish culturists are hesitant to ask for

professional assistance with their fish disease problems. Some producers fear the unknown, some fear adverse publicity, while others merely consider losing a certain percentage of their stock to disease as a routine part of the cost of doing business. Severe and costly problems can be avoided by early detection, accurate diagnosis and prompt corrective action.

A systematic program of health checkups can effectively prevent serious disease outbreaks and can prevent the inadvertent transfer of diseases to other rearing units or to other fish hatcheries. An effective disease surveillance program coupled with regular professional diagnostic and inspection services can do much to identify disease problems. Appropriate control measures can then be developed that will cause the least possible disruption of fish cultural operations.

Learning from Past Experiences

A hatchery superintendent nearing retirement age commented that he did not have thirty years of experience but that he had, instead, 'one year's experience, thirty times over'. Many hatchery operations seem to be managed in a similar way. Serious deficiencies that may have been built into a facility may cause recurring disease problems. Inappropriate management decisions on the kinds and numbers of fish that must be reared at a facility can also contribute to the difficulties encountered. The fish culturist may be little more than a bystander in such situations. Many fish cultural inventions and many research studies have developed as after-the-fact remedies for ailments that can be attributed to the facility itself or to the program being carried out. Basic corrective action is required. In some situations, good progress is possible. In others, the program is pressed forward in spite of the difficulties. The purpose of fish disease control policies and improved husbandry practices is to help hatchery personnel cease doing the things that have created problems in the past, and to apply the knowledge gained from these experiences to facilitate the production of quality fish at minimal cost in the future. This may be difficult medicine to swallow because the required changes may necessitate the alteration of traditional programs or reductions in the numbers of fish produced.

CONCLUSION

Improved husbandry practices begin with basic fish cultural decisions. From the moment that a decision is made to hatch and rear fish, a succession of decisions is initiated that ultimately leads to the design and construction of facilities and to the acquisition of fish stocks. Fish culturists and fish pathologists are then called upon to meet production objectives and to prevent fish health problems that may have been irrevocably built into the program just as solidly as the concrete used to build the facility. Technological advances in disease detection, prevention, and control occasionally make it possible to successfully culture fish that are free of most serious diseases. Certain diseases, especially those that are vertically transmitted from parent to progeny through contaminated eggs, become established in fish populations and defy control, thereby vividly demonstrating the necessity for aggressive containment and prompt eradication efforts.

A multifaceted, integrated fish disease control program directed toward all stages of the disease process and sources of infection may be required. Bacterial kidny disease (BKD) in cultured coho salmon is a good example of a complex, deeply entrenched infectious disease problem in the Great Lakes basin and on the west coast of North America. In retrospect, it is interesting to speculate about the savings of fish, money, time, and effort that could have been realized if early outbreaks of BKD, whirling disease, and infectious pancreatic necrosis had been relentlessly pursued with quarantine, eradication procedures, and facility rehabilitation procedures, rather than accommodated by the rearing of additional numbers of fish to make up for disease losses. Perhaps little would have been gained. On the other hand, the fact that there exists a perceived need for this Guide is, in itself, testimony that improved husbandry practices are needed, and that certain infectious diseases threaten to cause intolerable economic losses in both the public and private sectors.

REFERENCES

Busch, R. A., and A. J. Lingg. 1975. Establishment of an asymptomatic carrier state infection of enteric redmouth disease in rainbow trout *(Salmo* gairdneri). J. Fish. Res. Board Can. 32: 2429-2432.

Buss, K.W. 1981. An approach to functional, economical, and practical fish culture through better bio-engineering, p. 227-234. In Bio-Eng. Sympos. Fish Cult., Am. Fish Soc., Fish Cult. Sec., Publ. 1, Bethesda, MD.

Davis, H.S. 1953. Culture and diseases of game fish. Univ. of Calif. Press., Berkeley, CA. 332 p.

Kimura, T., and T. Awakura. 1977. Current status of diseases of cultured salmonids in Hokkaido, Japan, p. 124-160. In Proc. Int. Symp. Dis. Cult. Salm., Tavolek, Inc., Seattle, WA.

Leitritz, E., and R. C. Lewis. 1980. Trout and salmon culture (Hatchery methods). Publ. No. 4100, Div. Agri. Sci., U. of Cal., Berkeley. 197 p.

Needham, E.A. 1977. The salmonid pathologist in 1977, p. 8-15. In Proc. Int. Symp. Dis. Cult. Salm., Tavolek, Inc., Seattle, WA.

Owsley, D.E. 1981. Nitrogen gas removal using packed columns. p. 71-82 In Bio-Eng. Symp. Fish Cult., Am. Fish. Soc., Fish Cult. Sec., Publ. 1. Bethesda, MD.

Wedmeyer, G.A., F.P. Meyer, and L. Smith. 1976. Book 5: Environmental stress and fish diseases. TFH Publications Inc., Neptune City, NJ. 192 p.

Westers, H., and K.M. Pratt. 1977. Rational design of hatcheries for intensive salmonid culture based on metabolic characteristics. Prog. Fish-Cult. 39: 157-165.

3

PLANNING A FISH HEALTH PROGRAM FOR HATCHERY OPERATIONS

D.B. GOLDTHWAITE
U.S. Department of the Interior
Fish and Wildlife Service
Newton Corner, MA

and

T.G. CAREY
Department of Fisheries and Oceans
Aquaculture and Resource Development Branch
Ottawa, ONT

As is true of most endeavors, planning, whether it is highly structured or quite informal, is the basis upon which sound fish health management programs are developed. The absence of planning can lead to indecision, hesitation, false starts and untimely changes in direction which may seriously affect the outcome and effectiveness of disease control measures.

Planning, in its broadest context, is the selection of courses of future action from a number of alternatives, and is the procedure by which a manager determines what goals are to be achieved. Planning must involve, in some form, the hands-on fish culturist as well as the fishery administrator. Elements that are generally involved in planning are summarized in Fig. 1.

Planning, as an activity, means many things to many people. It can be all-consuming from a mechanical process standpoint but this undoubtedly should be avoided. In general, it is advisable to keep the complexity of the planning process appropriate to the scope of the problem. As a general rule, planning a fish health program should not be considered an overly complex exercise. The primary objective of fish health management in any fish culture operation should be reduction of the risk of exposure of fish stocks to disease. This objective can best

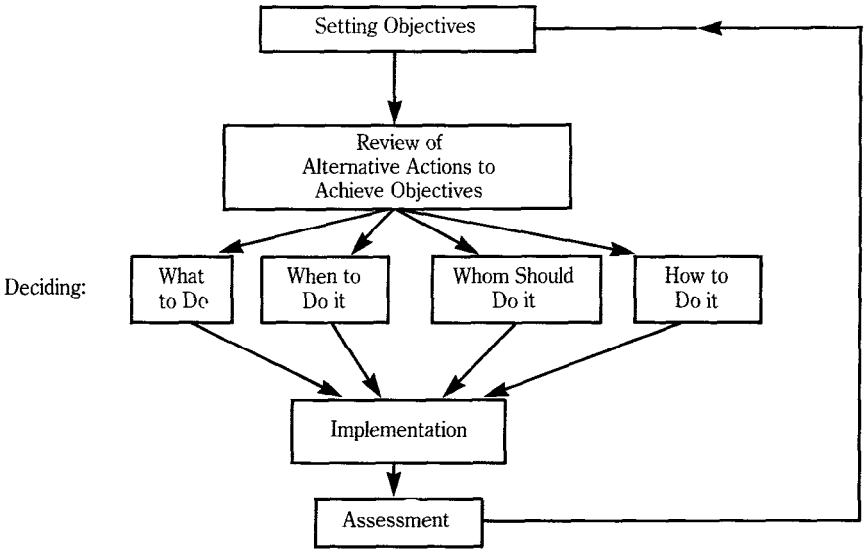


FIG. 1 General sequence of actions in planning and implementing a fish health program.

be accomplished by using a combination of strategies and by selecting those alternatives which are most effective and for which costs do not exceed financial resources available.

Maximum flexibility for fish health planning is available during the design phase for a new fish culture facility. Alternatives can be reviewed and decisions made regarding whether to use groundwater, as opposed to surface water, as the source of supply; whether water treatment facilities are required; whether quarantine facilities are needed if disease-free sources of stock are not available; or whether separate facilities should be constructed for broodstock rearing to eliminate the necessity for bringing in new stock each year. Generally, it has always proved to be less costly to incorporate disease control measures into new facilities during initial construction than after a rearing facility has been completed. Needham (1977) recommended that a fish pathologist be involved in all phases of culture operational planning, including site selection, hatchery design, selection of brood stocks, harvest methods, and quality control. Fish health considerations should be an integral part in the planning of all these activities.

For an existing fish production system, the same fish health objective applies but the approaches to be used may differ. Routine procedures and facilities already in place may have to be changed in order to reduce or eliminate potential disease risks. If diseases are already present and causing problems, plans should be developed for phased elimination of the diseases and for a reduction of further exposure to disease organisms.

The potential actions available to fish health planners can be categorized as disease prevention, control and eradication:

1. Disease Prevention Techniques - These techniques are designed to maximize the ability of cultured fish to withstand disease as well as to minimize the risks of exposure to pathogens and/or outbreaks of disease. They are used

primarily before diseases are detected and after a disease has been eradicated. In order to maximize the ability of fish to withstand disease, maintenance of optimal environmental conditions is of utmost importance, as are the proper selection of genetic strains, diets, etc. Fish that are in poor physiological condition due to one or more undesirable factors are normally more susceptible to disease. In fact, avoidance of stress is probably the single most important way to prevent disease (Avault 1981).

In order to minimize the risk of exposure to pathogens or outbreaks of a disease, the water supply can be treated to kill pathogens (disease organisms) or infected fish that may be present, facilities can be disinfected between crops or year classes, fish stocks can be vaccinated, and introduced fish placed in quarantine until it can be demonstrated that they are free of diseases of concern. It should also be noted that continuing disease prevention techniques are important in order to control the spread of diseases to new areas and other facilities after outbreaks have occurred.

2. Control Measures - These measures are employed to alleviate the impact of pathogens after a disease problem has been detected. Techniques used can be either direct in their effects on pathogens in the fish (e.g. use of bactericides and antibiotics), or indirect in that they reduce the density of pathogens available to infect fish (e.g. partial water treatment).

3. Eradication of a Disease - Eradication includes all actions required to eliminate specific pathogens from a facility, and to prevent further opportunity for expression of the disease. It is strongly recommended that fish culture program managers develop the outline of an eradication plan prior to a disease outbreak. It is far more difficult to be objective in preparing such a plan when fish may be dying in large numbers.

After considering all disease control alternatives that could be applied in a given situation, decisions must be made on the actions that should be taken, when, how and by whom. Important considerations at this stage include the formulation of contingency plans, incentives for the staff, and the full range of other management issues.

A plan or course of action is no more than an abstract exercise unless the needed resources are available and steps are taken to make it operational. Too often, procedures for fish disease control are clearly laid out but not followed. Workers may become careless if not regularly reminded, and errors made in the space of a few seconds may negate years of work in fish disease control. Implementation of a fish health plan at the operational level is therefore as important as the planning exercise itself. At higher organizational levels, planning activity should be translated directly into budgets that will support future fish health management activities.

Finally, regular assessment of the progress that has been made in implementing the plan will provide essential feedback to determine if objectives are being achieved. Objectives may have to be modified or fine-tuned based on the effectiveness and costs of different actions.

A simplified, theoretical fish health program describes the measures that can be implemented to reduce the risk of exposure to diseases at each stage of a fish cultural operation. Fish health programs will vary considerably among facilities in detail and complexity, depending on the objectives set and resources

available. It is for this reason that it is important that planning be undertaken separately for each field installation.

REFERENCES

Avault, J. W. 1981. Prevention of fish diseases: Some basics reviewed. Part two. Aquaculture 7(6): 4.

Needham, E.S. 1977. The salmonid pathologist in 1977, p. 8-15. In Proc. Int. Symp. Cult. Salmon. Tavolek, Inc., Seattle, WA. 189 p.

PART II

DISEASE CONTROL OPTIONS IN HATCHERY MANAGEMENT

4. CONSIDERATIONS IN HATCHERY DESIGN FOR THE PREVENTION OF DISEASES

5. SELECTION OF WATER SUPPLIES

6. WATER SUPPLY SANITATION

7. STOCK AND YEARCLASS SEPARATION

8. NUTRITION AND FISH HEALTH

9. GENETICS AND FISH HEALTH

10. STOCKING PRACTICES AND DISEASE CONTROL

11. ROUTINE FISH DISEASE MONITORING

12. CHEMOTHERAPY

13. IMMUNIZATION WITH VACCINES

14. HATCHERY DISINFECTION AND DISPOSAL OF INFECTED STOCKS

4

CONSIDERATIONS IN HATCHERY DESIGN FOR THE PREVENTION OF DISEASES

HARRY WESTERS
Michigan Department of Natural Resources
Fisheries Division Lansing, MI

Proper hatchery design and modes of operation can contribute significantly toward the prevention of fish diseases. Prevention techniques are aimed at maximizing the natural ability of the fish to resist disease as well as minimizing the risks of exposure to pathogens.

In the introduction to this manual, three elements were identified in fish health: the host (fish), the environment, and the pathogen. All three must also be considered in hatchery design.

Brown trout, for instance, should not be reared in water where furunculosis is endemic; brook trout should not be reared in IPN-infected water sources; etc. Although counter measures such as genetic disease resistance and fish immunization techniques are possible avenues to reduce disease problems, these techniques are not, as yet, available for most fish species and diseases.

Wedemeyer et al. (1976) consider stress to be one of the most important factors with respect to susceptibility to disease. Needham (1977) states that if a correctly designed and engineered environment is not provided for the fish, disease will strike. Since in intensive fish culture, fish are reared in very artificial situations, the hatchery design must be aimed at creating the best (least stressful) rearing conditions feasible.

The presence or absence of pathogens is a prime factor in whether or not a disease outbreak will occur, therefore design must aim at preventing the introduction of disease organisms, as well as reducing the pathogen load.

Hatchery design, in its broadest concept, includes site selection as well as providing options to apply sound hatchery management techniques and modes of operation.

DESIGN CONSIDERATIONS - GENERAL

Maximum opportunity for fish health planning is available during the design stage of new culture facilities. That is the time to incorporate appropriate measures for disease control, rather than attempting to add them after the facility has been built and is in operation.

Unfortunately, such opportunities are often not available and a manager may have to work with established facilities after constraints are already in place, firmly embedded in concrete and steel and where water sources are fixed. Design of a new facility starts with site selection and should have water concerns as the number one criterion.

The ideal is to have a source of high quality water, free of fish, and, consequently, free of obligate pathogens. A hatchery built with this type of water source has the potential to be specific pathogen-free, provided that no infected stock is brought into the facility and that appropriate safeguards are taken against contamination.

Well and spring water sources are the most likely candidates. Such sources are difficult to find, are sometimes limited in flow, and often must be pumped. If the quality is high, such water can be used up to three times, as long as there is adequate re-aeration, unionized ammonia is kept below harmful levels, and most of the suspended solids are removed (Westers and Pratt 1977).

Another option to accomplish specific pathogen-free status is by treating the incoming water to destroy pathogens that may be in the source. The technology is available, such as ozone, UV irradation, and chlorination, but they are not without risk, since they can be toxic to the fish or may fail to destroy the pathogens. At this time, water sanitizing systems are not widely used, except possibly for UV irradiation. This technique poses no direct risk to the fish, but it requires rigorous quality control and maintenance in order to remain continuously effective. The system is expensive but merits consideration, especially in situations with relatively low flow requirements, such as egg incubation and early rearing.

Since specific pathogen-free rearing is just that, fish will still be exposed to ubiquitous opportunistic pathogens. Infections by these pathogens are best controlled by reducing stresses. The same approach, however, can also be applied for obligate pathogens whenever the option of specific pathogen-free rearing is not available.

In summary, these criteria must be considered in hatchery design:

1. Site Selection (water quality)
2. Water treatment
3. Pathogen control (sanitation, disinfection, quarantine)
4. Quality environment (reducing stress)
5. Hatchery management and operation (reducing stress)

DESIGN CONSIDERATIONS - SPECIFIC

In site selection, water (quality and quantity) must be the first consideration. The guiding principle, as stated in the chapter on "Selection of Water Supplies", is to select sources that minimize risk of disease exposure and provide an environment which maximizes fish health.

A source free of obligate pathogens is, of course, the most effective way to prevent certain diseases. Selection of this type of water should always be given priority, even if it involves some sacrifice in quantity. Such sources are scarce and of relatively low volume. However, they are usually of high quality, offer stable water temperatures and yield higher production per unit flow.

WATER TREATMENT

It is likely that in the search for an ideal water source, one must accept a source that is less than ideal or even marginal. As a result, it may be necessary to pre-treat the water to improve chemical and physical characteristics and control pathogens.

Pre-treatment to improve the chemical and physical characteristics may involve oxygenation, degassing (nitrogen, hydrogen sulphide, carbon dioxide, etc.), iron removal, reduction in suspended and settleable solids, temperature control, and pH control (buffering).

For most of these, the needed technology is available in the form of aeration or de-gassing devices (packed columns, screen decks, various mechanical aerators, etc.), filtering systems (rapid sand filters) and temperature controls (well water, chillers, boilers, solar heating). Cost and economics of operation are a big factor in the selection of these systems.

PATHOGEN CONTROL MEASURES

In addition to sanitizing and disinfecting the water supply, hatchery design must consider controlling contamination from outside sources and facility disinfection.

If eggs are routinely brought into the facility, the hatchery must be equipped with an egg disinfecting room. This should be a separate, isolated room that is easy to keep sanitized with an approved water (liquid) disposal system. Ideally, hatchery rearing facilities should be zoned into operational areas. Although physical barriers are not recommended, water intakes and outflows should be zoned. Zoning could be arranged in logical areas, such as those for incubation, early rearing, indoor rearing, outdoor rearing. It may be feasible to design indoor and outdoor rearing facilities at large hatcheries so that groups or even individual rearing units can be disinfected.

Specific pathogen-free hatcheries should have a quarantine facility, especially if the hatchery routinely receives fish from outside sources, even if they are from certified stocks (the only kind to be permitted into the facility!)

Successful pathogen control depends as much on the human element as on facility design. Expediency, lack of administrative support, lack of training and discipline, and improper procedures can destroy or negate pathogen control efforts even with the best designs. Although the facility designmust allow for the most effective way to perform the needed work tasks, unless there is strict adherence to a sound plan, the disease control program is doomed. A chain is only as strong as its weakest link.

QUALITYENVIRONMENT

Quality concerns begin with the water supply. The poorer the quality, the fewer fish it can support. Incoming water must have a dissolved oxygen level at or near saturation (not less than 90%) and water temperatures should be stable. The maximum should not exceed the standard environmental temperature (SET) by one-third of its value. For example, if the SET for a species (rainbow trout) is 15°C, the maximum temperature should not exceed 15 + (⅓ x 15) or 20°C. Species with a SET of 24°C should not be exposed to temperatures over 32°C.

Toxic gases, such as hydrogen sulfide and methane, must be completely removed through a degassing device (packed column, screen decks, etc.) Nitrogen (N_2) gas should be kept at 100 % saturation or less. Supersaturation with N_2 gas can have subtle adverse effects without the classical signs of gas bubble disease. Nitrogen supersaturation can be a serious, insidious stressor and it is strongly recommended that any supersaturation be avoided.

The higher the water quality, the higher the production potential will be. As a consequence, water quality will greatly affect the design of rearing space. The objective is to obtain maximum production without seriously stressing the fish.

Two aspects of production must be considered. One is production in terms of flow, termed "loading", and expressed as weight of fish (kg) per litre per minute flow (kg/l/min); the second is expressed in terms of space, called density. Density is expressed as kg fish per cubic meter of rearing space (kg/ma). Both are interdependent with the hourly exchange rate (R) as the only variable. This relationship has been expressed by Westers (1981) as

$$L = \frac{D \times .06}{R} \text{ and } D = \frac{L \times R}{0.06}$$

Where L = Loading in kg/l/m
D = Density in kg/m^3
R = Hourly water exchange rate
and .06 = 1.01/min = 60 litres per hour = $.06m^3$.

Water temperature is the most significant factor in loadings. Lower temperatures permit higher oxygen levels and lower oxygen consumption rates by the fish (lower metabolic rates).

If a decrease of 10°C reduces the metabolic rate by half, in theory, loading can be doubled. If the density must remain the same, twice as much rearing space must be available. The effect of density as a stressor is probably the most controversial subject in the intensive culture of fish (Westers 1982). Opinions vary widely. Unless one knows what the optimum loading and density is for a particular species (optimum from both the biological and production point of view), one cannot properly determine the optimum rearing space. Further complications arise when water reuse is applied. Table 1 provides some insight into these relationships of water quality, maximum allowable loadings and densities, and optimum hourly water exchange rates in terms of hatchery design (rearing units). Water quality has been assigned values of 8, 6, 4 and 2, with the highest number representing highest quality.

According to Table 1, it is difficult, if not impossible, to have an ideal balance in hatchery design between loadings, densities, and exchange rates. The weakest area of knowledge concerns rearing density. Little is known about the point at which density becomes sufficiently stressful that it threatens the health or well-being of the fish. It seems important that the necessary research be done, since it is of such vital importance in hatchery design.

HATCHERY MANAGEMENT AND OPERATION

How well a hatchery is managed and operated depends both on the skill, knowledge, and dedication of the staff and on what the physical design allows them to do.

Fish culturists must develop a strong consciousness for the welfare of the fish under their care. In time they should cultivate, as it were, a sixth sense that allows them to recognize any stressful condition the fish are exposed to. Certain design features can aid the fish culturist in reducing stresses on the fish.

Baffles, for instance, can be placed in rectangular, flow-through rearing units to provide effective and continuous cleaning action. Such baffles are usually spaced at distances approximately equal to the width of the tank or pond. The gap between baffles and the bottom of the unit varies from 3-8 cm. The objective is to create enough velocity along the bottom to move solids to the foot (clean-out section) of the raceway. A velocity of 20-40 cm/sec directly behind a baffle is recommended. To determine the velocity, the following formula can be used:

$V = L \times R$ where V equals velocity in meters per second (m/sec), R is the hourly water exchange rate, L is the length of the rearing unit in meters, and 3600 is the number of seconds in one hour. This equation gives the velocity of the water through the rearing unit.

To determine the velocity behind the baffles, this equation is used:

$VB = \frac{D1}{D2} \times V$, where VB is the velocity in m/sec behind the baffle, and $D1$ is the baffle gap (this ratio can be expressed in either cm or m). If D_2 is the unknown, we have: $D_2 = \frac{V1}{VB} \times V$.

Baffles eliminate the need for frequent cleaning by drawdowns and broomsweeping, actions which excite and stress the fish. However, not all species can adapt to baffles. It has been our experience that lake trout and splake are stressed by baffles. Louvered baffles may provide the answer. Such baffles can be closed for a few hours each day to promote cleaning in the raceways. The design should be tailored, where applicable, to the species to be produced. Unfortunately, not enough is known about the specific requirements of most species. It is believed that requirements differ between Atlantic salmon and rainbow trout. Atlantic salmon must be provided more horizontal space (bottom area) (Peterson et al. 1972), while rainbow trout successfully occupy vertical space - in other words, rainbow trout can be "stacked". Klontz et al. (1978) provide the following density-index recommendations: rainbow trout 0.5; coho

Table 1. Relationship of rearing to flow, loadings (kg/Lpm), densities (kg/ma), and water exchange rates. The values are based on an available flow of 10,000 L/m and an individual raceway rearing volume of 60 ma.

Description of design parameters	Water Quality Ranking			
	8	6	4	2
1. Maximum allowable loading (kg/l/min)	3.00	2.25	1.50	.75
2. Total maximum production in kg	30,000	22,500	15,000	7,500
3. Rearing space (ma) needed for a maximum allowable density of 32 kg/m³	937	703	468	234
4. Number of raceways required	15	12	8	4
5. Hourly exchange rate per raceway	2.6	3.4	5.1	10.3
6. Resulting water velocity in cm/sec.	2.4	3.2	4.8	9.7
7. Resulting density for R = 4; rearing space needed is 600 ma or 10 raceways	50	38	25	13

salmon 0.4; chinook salmon and cutthroat trout 0.3. These reflect a relationship of 10:8:6. For design purposes, this means that chinook salmon and cutthrout trout require 1.66 times as much rearing space as rainbow trout, while coho salmon must be provided 1.25 times as much space as rainbows, providing loading requirements are the same. Such differences in requirements would drastically influence design of a particular facility.

Lake trout and splake require shade, but subdued lighting conditions appear to be beneficial to most species. It is recommended that serious consideration be given to the construction of roofs over raceways. In addition to shading out direct sunlight, roofs are effective in predator control, especially if the sides are screened as well. Predators not only destroy fish outright, but harassment by such predators is another stressor. Human activity can have a similar effect since public hatcheries, traditionally, are open to visitors. When designing such facilities, visitor control should be kept in mind. Design features should permit the public to observe operations without disturbing the fish. A visitor's center with a display pond can help to satisfy public interest.

Handling and harvesting, two necessary activities, are also stressful to the fish. Needham (1977) made the comment that most of the bacterial and fungal diseases he observed in fish were caused by physical damage. Proper design can help in easing the removal of fish from the rearing units. Fish should not have to be "chased around" and should be left out of water for as short a time as possible. Temporary holding facilities, where fish are concentrated for short periods of

time, must be adequately supplied with oxygen to meet the increased oxygen demand of the fish. Metabolic rates may triple if fish become excited and frightened as they are handled. This stress is unavoidable, but the effects can be reduced through proper design and technique.

Specific design features aimed at reducing and countering stresses are not "off the shelf" items. As new hatcheries are planned, biologists, engineers, and bio-engineers are challenged to come up with the best design possible. Finally, it is important also to obtain input from the experienced, practical fish culturists whenever design for a new hatchery is started.

REFERENCES

Klontz, G. W., I.R. Brock, and J.A. McNair. 1978. Aquaculture techniques: water use and discharge quality. Univ. of Idaho, Idaho Water Res. Inst., Moscow, ID. 114 p.

Needham, E.A. 1977. The Salmonid Pathologist in 1977, p. 8-15. In Proc. Int. Symp. Dis. Cult. Salm., Tavolek, Inc., Seattle, WA.

Peterson, H. H., 0. T. Carlson, and S. Johasson. 1972. The rearing of Atlantic salmon. Astro-Ewos AB, Södertälje, Sweden. 39 p.

Wedemeyer, G.A., F.P. Meyer, and L. Smith. 1976. Book 5: Environmental stress and fish diseases. TFH Publications, Inc., Neptune City, NJ. 192 p.

Westers, H. 1981. Fish culture manual for the State of Michigan. Principles of intensive fish-culture. Mich. Dept. Nat. Res., Lansing, MI. Unpublished. 101 p.

Westers, H. 1982. Facility design and operational modes to ensure quality. Proc. Workshop on Quality Improvement in Finfish Aquaculture. Am. Fish. Soc., Fish Cult. Sect., Bethesda, MD. (in press).

Westers, H., and K.M. Pratt. 1977. Rational design of hatcheries for intensive salmonid culture, based on metabolic characteristics. Prog. Fish-Cult. 39: 157-165.

5

SELECTION OF WATER SUPPLIES

J.B. DAILY

and

P. ECONOMON
Minnesota Department of Natural Resources
St. Paul, MN

Careful selection and prudent use of water sources will provide an environment which maximizes fish health. To be suitable, the water supply should be adequate to provide for the level of fish production required. Ideally, the water should be clean and moderately hard and there should be no fish in the water supply. The water should be of a uniform temperature (9-15.5°C) but warm or cool enough to promote satisfactory growth of the cultured species. The pH should be slightly alkaline and the water should be buffered to resist the effects of organic acids and ammonia. The hatchery design and operation should promote and maintain optimal water quality parameters (see Table 1).

Potential water supplies should be tested with an experimental group of fish to determine its suitability before initiating a fish culture project. The fish should be held in well-aerated tanks or troughs for an entire season or as long as seems practical to obtain the maximum test results. If the fish grow, remain healthy, and are active over the test period, the water can be considered to be suitable for raising fish.

TABLE 1. Water quality standards for fish culture (U.S. EPA 1979-80)

Alkalinity (hardness as $CaCO_3$)	*20* ppm at least
Aluminum (Al)	<.01 ppm

Ammonia (NH_3)	<.02 ppm
Arsenic (As)	<.05 ppm
Barium (Ba)	5 ppm
Cadmium (alkalinity<100)	.0005 ppm
(alkalinity>100)	.005 ppm
Calcium (Ca)	52 ppm at least
Chlorine (Cl)	<.003 ppm
Chromium (Cr)	.03 ppm
Carbon dioxide (CO_2)	1.5 ppm is best (no more than 15 ppm)
Copper (alkalinity<100 ppm)	.006 ppm
(Cu) (alkalinity>100 ppm)	.03 ppm
Dissolved oxygen (DO)	75% of saturated level, never less than 5 ppm
Fluoride (F)	<.5 ppm
Hydrogen cyanide (HCN)	<.005 ppm
Hydrogen sulfide (H_2S)	<.003 ppm
Iron (Fe)	<.1 ppm
Lead (Pb)	<.02 ppm
Magnesium (Mg)	<15 ppm
Manganese (Mn)	<.01 ppm
Mercury (Hg)	<.2 ppm
Nitrogen (N)	<100% total gas pressure, 103% nitrogen gas
Nitrate (NO_3)	<1.0 ppm
Nitrite (NO_2)	<<1.0 ppm
Nickel (Ni)	<.01 ppm
PCB	.002 ppm
PH	6.7 - 8.6
Potassium (K)	<5.0 ppm
Salinity	<5 PPt
Selenium (Se)	<.01 ppm
Silver (Ag)	<.003 ppm
Sodium (Na)	<75 ppm
Sulfur (S)	<1.0 ppm
Sulphate (SO_4)	<50 ppm
Total Dissolved Solids (TDS)	<400 ppm
Total Suspended Solids (TSS)	<80 ppm
Uranium (U)	<.1 ppm
Vanadium (V)	<.1 ppm
Zinc (Zn)	.005 ppm
Zirconium (2)	.1 Ppm

WATER CHARACTERISTICS

OXYGEN

Dissolved oxygen is one of the most critical aspects of water quality for culturing fish. It varies according to the source of the water, temperature, altitude, and biochemical activity of the water. The amount of dissolved oxygen required for fish culture should never be less than 75% of the saturation level. This level can be maintained by proper hatchery design or by the use of mechanical aeration devices.

AMMONIA

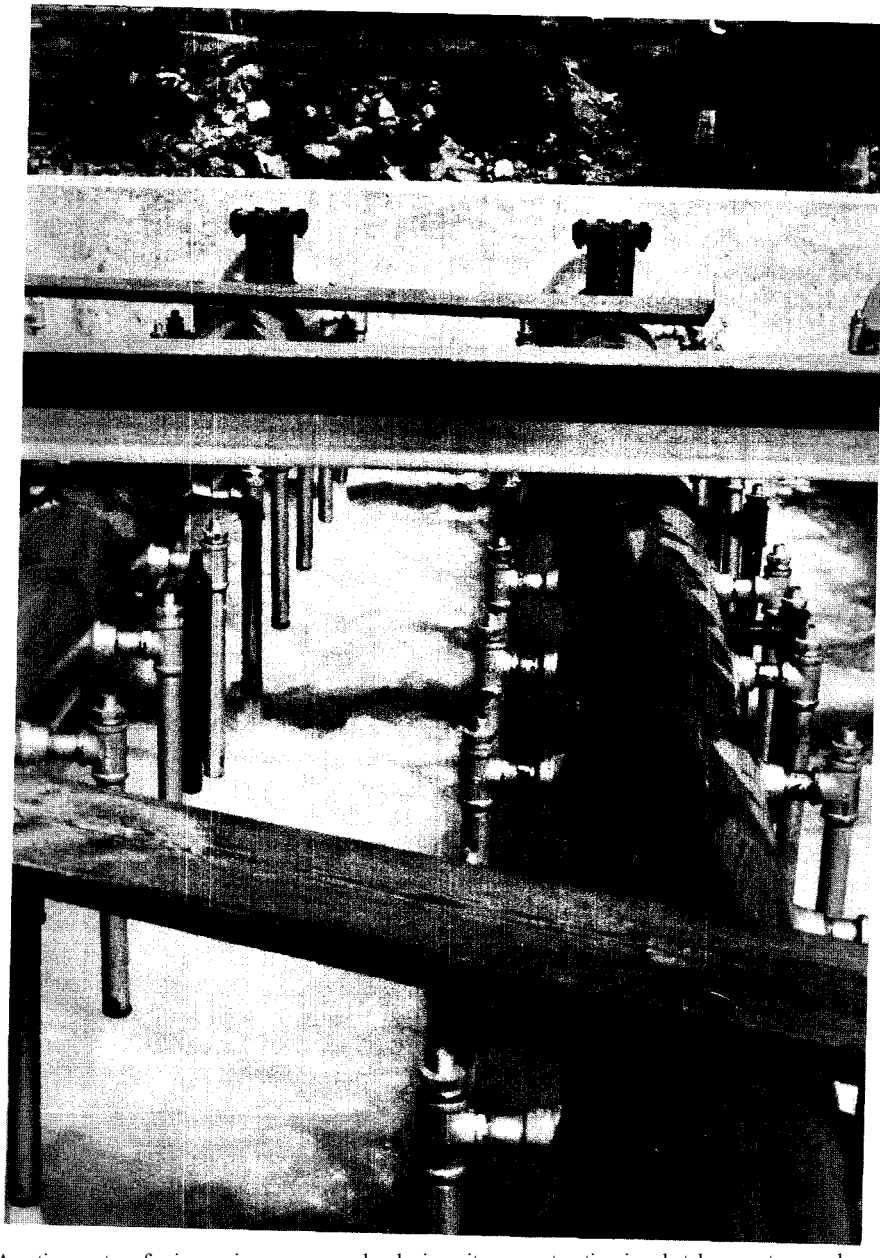
Ammonia in the water is generally a biological by-product; in its un-ionized form, it is very toxic to fish. Normally, the ammonia found in hatchery waters is produced by the fish themselves. Consequently, the production potential of a given water supply is determined by the quality of water available and by the number, size, and weight of fish present. For these reasons, the incoming water should contain little or no background ammonia to avoid or reduce potential problems.

NITROGEN

Waters in equilibrium with air contain dissolved nitrogen gas as well as dissolved oxygen in accordance with Henry's Law. Even though nitrogen constitutes about 78% of normal air by volume, its solubility in water is comparatively low. Water at sea level in equilibrium with air at 10" C contains about 14.89 ml/l of dissolved nitrogen gas - Handbook of Chemistry and Physics (Anon. 1950-51). Unlike oxygen, dissolved nitrogen is inert and is not consumed in biochemical processes, so that its content in water fluctuates only in accordance with temperature and pressure changes. Under normal conditions of less than saturation, no detrimental effects toward fish are anticipated. Supersaturation is not likely to occur in surface waters. However, if for some reason, the air over the water is under pressure, the amount of dissolved nitrogen will increase and can become a potential hazard to fish health. A review of dissolved gas supersaturation problems is provided in Weitkamp and Katz (1980).

Supersaturation of nitrogen can result from a number of causes, including the following:

1. Air drawn into the water supply due to faulty pumps, leaking pump seals, broken intake lines, and packing glands or connections which are not kept air tight.
2. Air drawn into a gravity flow water system through a partially submerged water standpipe or perforated line.
3. Air drawn into aquifers with seepage from surface precipitation (especially at low temperatures); or from subterranean pools with nitrogen-filled air domes held under pressure. Artesian wells and springs are often supersaturated with nitrogen gas (Wood 1968).



Aeration system for increasing oxygen and reducing nitrogen saturation in a hatchery water supply (T.G. Carey)

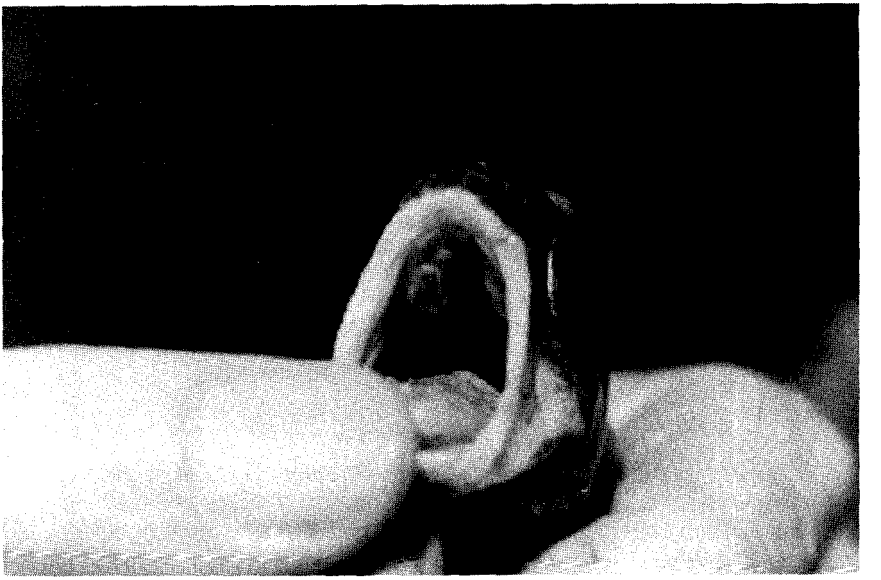
4. Plunge pools, where water passes over a dam or falls from a steep gradient, will entrain air and cause supersaturation in the water at the base of the waterfalls (Harvey and Cooper 1962).

Wood (1968) reported that the following levels of nitrogen saturation appeared detrimental or caused death in salmon of various ages:

- Advanced yolk-sac and newly "buttoned-up fry" - 103-104%
- Fingerlings and yearling - 113% (death); 105-112% (eye damage and blindness)
- Adults - 118% (eye damage)

A sudden change in the partial pressure of nitrogen gas in water, as from a rapid rise in water temperature, can be a serious threat to fish health. The essential cause being that the exchange of gases by diffusion at the gill-water interface is unable to maintain equilibrium between internal and external pressures (van Duijn 1966; Economon 1980). This temperature-induced phenomenon is not unlike the "bends disease" caused in divers due to sudden pressure changes.

Supersaturation of nitrogen in a water supply can be remedied by vigorously breaking up the water so that excess gas can escape to the atmosphere. The most practical devices, such as columns of pipe packed with Koch rings (Owsley 1981), operate by gravity flow. Other methods include spraying, dripping, formation of a thin sheet of water, or forcing air bubbles into water. Rucker and Tuttle (1948) designed a simple aerator for hatchery operations that allowed the water to flow as a shallow sheet. This was accomplished by allowing the water to flow over the sides of a series of troughs, one above the other.



In situations of a supersaturated water supply, fish may develop gross gas bubbles in the less dense tissues. Note the large bubbles in the mucous membranes lining the mouth of this fish. (U. S. Fish and Wildl. Serv.)

To accurately test for the level of nitrogen gas dissolved in water, it is necessary to analyze the water by either the Van Slyke technique, as modified by Oesting (1934), by the gas chromatography procedure of Swinnerton et al. (1962), by mass spectrometer (Benson and Parker 1961), by the Weiss saturometer (Fickeisen, et al. 1975), or by the recently developed gasometer (Bouck 1982).

HARDNESS

Hard water may be undesirable because it sometimes contains toxic gases, such as hydrogen sulfide, methane, carbon dioxide and nitrogen. For fish culture, the hardness (as $CaCO_3$) should be at least 20 ppm or more; around 120 ppm is ideal. Generally, an alkalinity or hardness greater than 20 ppm is preferred because of its capacity to buffer the effects of contaminating substances. Also, hard water is more productive, especially for pond culture.

IRON AND MANGANESE

Iron and manganese are detrimental to fish culture in both the ionized and un-ionized forms. Alkaline waters favor the development of insoluble hydroxide precipitates which can settle on the gill filaments, which, of themselves, have an alkaline reacting surface. A tenacious film may be formed that seriously limits respiratory function. In addition, saprophytic iron bacteria can proliferate on the ferric hydroxide that coats the gills, further promoting mechanical injury and blockage of the respiratory channels. Incubating eggs can be similarly affected. Wells that are drilled in areas of iron bearing deposits are the primary sources of dissolved iron (van Duijn 1966). Manganese can accumulate in the hypolimnion of reservoirs impounded over substrates containing this metal. If hatcheries allow such waters to be used as part of the water supply, manganese toxicity may result.

DISSOLVED SOLIDS

Dissolved solids represent a variety of substances including carbonates, bicarbonates, chlorides, sulphates, phosphates, nitrates and other metallic elements. At levels above 440 ppm, these dissolved solids can cause excessive stress to cultured fish and result in loss of feeding, decreased growth, poor reproduction, and mortality.

TURBIDITY

Turbidity caused by suspended solids is a measure of the particulate matter in the water column. This condition interferes with feeding and may block or seriously irritate the gills causing faulty respiration, poor growth, stress, and susceptibility to infections. Dissolved and suspended solids present additional hazards by reacting synergistically with pollutants to produce a totally unsatisfactory aquatic environment for raising healthy fish.

CONTAMINANTS

Less common contaminants, such as heavy metals, industrial chemicals, Cl, Cu, Hg, etc., are usually introduced through contamination. These water quality abnormalities are best corrected at the point of origin. Items or practices to be avoided inlcude the use of paints containing heavy metals, galvanized equipment or containers, the use of copper piping over fish culture tanks, and the use of stream water that may be contaminated from the washing of agricultural equipment or by live-stock use.

WATER SOURCES

GROUND SOURCES

1. Springs

Springs occur when a flowing aquifer is exposed at the surface of the ground or when a crack or other fault occurs in an otherwise impervious layer above a confined aquifer allowing water to escape to the surface. Water from springs reaches the surface either as a single point source or as a large area of seepage. Most springs with a large enough discharge to be useful for aquaculture are of the point source type and a sizable stream often flows from such springs.

Springs generally have high quality water of a constant temperature and require no pumping to get the water to the surface. Since pumping costs are a significant part of most production expenses, this factor can greatly reduce operating costs of a culture system. Spring water, like most other groundwater sources, generally has a low oxygen concentration and may require oxygenation. Care should be taken to quantify the discharge of a spring during dry weather. Small springs, and even some large ones, may have significant reductions in discharge during periods of dry weather. Springs exhibiting this characteristic should be avoided.

More specific help with design of a spring development as an aquaculture water source may be available from the local Soil Conservation Service (SCS). Most SCS offices have the services of a professional engineer available free of charge, or at a nominal charge to the user. Private consultants can also be retained for design purposes when necessary.

2. Wells

Except for high quality springs, wells are the best source of water for aquacultural enterprises. Well and spring waters are of comparable quality, but wells are less desirable because of the cost of pumping. Well water is usually free from pollution, although some well waters contain undesirable gases (e.g., hydrogen sulfide) or chemicals (e.g., high iron or sulfur concentrations). Well water normally travels considerable distances through the soil and may dissolve many substances along the way. Low dissolved oxygen is a common problem, but aeration can remedy this. A water table well is essentially a hole opened into an aquifer below a confining overlay of rock and water is removed by pumping. The yield from a well of this type is dependent on the permeability and vertical

thickness of the aquifer. The diameter of the well also influences yield. Generally, greater aquifer permeability and/or thickness allows greater yields. These wells, sometimes referred to as shallow wells, tend to be more susceptible to yield variations during dry weather than deeper wells.

Artesian or flowing wells are those from which water begins to flow without pumping as soon as drilling is complete. Essentially, artesian wells occur when drilling penetrates an aquifer confined between two relatively impermeable layers. The recharge area (area where water can enter the aquifer) must be higher than the outlet of the well. Water entering the recharge area flows under gravity through the aquifer toward the well. Since some energy is required to cause flow through the permeable aquifer, the recharge area must be sufficiently higher in elevation than the outlet of an artesian well to overcome head losses caused by flow through the aquifer. The head loss experienced during flow through an aquifer is dependent on flow velocity, permeability of the aquifer, and the distance water must move through the aquifer. Thus, free discharge from an artesian well is a direct function of aquifer permeability and an inverse function of velocity of flow through the aquifer and distance from well to recharge area.

SURFACE SOURCES

Surface water supplies can be divided into four major sources; streams, lakes, ponds, and salt or brackish bodies of water. All surface water supplies have the undesirable characteristic of being exposed to sources of pollution and should be used with caution. However, most surface waters are well oxygenated, which is advantageous for aquatic enterprises.

1. Streams

Streams, like other water sources, offer advantages and disadvantages that are related to stream characteristics. Because turbulence is prevalent in most streams, the dissolved oxygen content usually is high. The pH and dissolved mineral content are determined by the topography, by the type and solubility of substrate of the area through which the stream flows, and by biological activity within the stream. Latitude, altitude, season, rainfall, stream depth, width, and turbulence influence the flow, temperature, and concentrations of dissolved solids and suspended organic matter in a stream. Radiant energy and dissolved inorganic mineral concentrations affect photosynthesis, respiration, and decomposition which, in turn, determine the amount of organic material present in the water. Stream discharge characteristics are important when the stream is the sole source of water for an aquacultural enterprise. If the discharge varies greatly during the year, a stream may not furnish sufficient water during dry periods when demands for water by aquatic organisms are highest. High temperatures often occur during these same periods and increase evaporation rates and the risk of thermal death or thermal stress.

States and provinces have regulations covering water diversion. Legal problems may arise if water quality of a stream is reduced by the effluent from an aquaculture installation. All aquatic organisms add waste products to the water. Agencies concerned with environmental control may require monitoring or treatment if the effluent contains excessive levels of waste products.

2. Lakes, ponds, and reservoirs

Water drawn from lakes, ponds and reservoirs has many similarities to that in rivers. However, oxygen concentrations tend to be lower, particularly if water is drawn from below the thermocline. Temperatures of lake water and, to a lesser degree, pond waters are more stable and predictable than the temperature of water from streams. Water temperatures change markedly with seasons and this factor must be considered when designing an aquacultural facility. Other water quality parameters, such as pH and dissolved and suspended nutrients, tend to be more constant in lake and/or pond water than in stream water. The larger the body of water, the smaller and less abrupt the changes will be in all environmental parameters.

Permits may be required from the necessary federal, state, or provincial governmental agencies to pump out of or discharge into a lake, pond or reservoir.

3. City water

Another source of water for aquaculture is a city water supply. Cities supply water mainly as a source of potable water for people, and their major concern is to maintain water quality such than humans can drink without endangering their health. To maintain this water quality, chemicals such as chlorine and/or fluorine may be added. Unfortunately, the chemicals added to increase the acceptability of the water for humans may make it very unsatisfactory for aquatic organisms. The most common example is the addition of chlorine to kill bacteria. Chlorine is highly toxic to fish.

WATER TRANSMISSION

1. Materials

Supply lines are developed to move water to the hatchery and within the hatchery so it can be presented to the fish in a condition that promotes good growth and health. Before one discusses the subject of hatchery water transmission, it is necessary to consider the possible toxicity of materials used to convey or contain hatchery water.

All equipment, regardless of its material makeup, must be seasoned or leached before it can be used to hold or transport water that will be used to grow fish. For equipment, this can be done by submerging the item for several hours downstream from the hatchery. In the case of new tanks, troughs, incubators or other rearing containers, allow water to flow through the filled containers for at least 24 h before they are stocked with fish or eggs. Preferably, the water leaving the containers should be diverted to the effluent to avoid contact with fish or any other equipment utilized by the hatchery.

The following materials are nontoxic in flowing water systems if leached before initial use:

- aluminum
- asphalt
- concrete
- Plexiglas
- polyethylene
- polypropylene

- fiberglass
- glass
- gravel and rock
- heavy metal-free paints
- iron
- nylon
- polyvinyl chloride (PVC) Type 1
- rubber
- silicone and hot melt adhesives
- stainless steel
- wood

Questions concerning the toxicity of a material can best be answered with a bioassay. This involves introduction of the material in question into a container of hatchery water with a small number of fish (at least 10) and leaving it for 24 h. If the fish show abnormal behavior there is reason to suspect that the material tested is toxic.

The following lists some of the more common toxic materials that find their way into a hatchery. ***Avoid these materials:*** cadmium (Cd) - usually a plating material for tools; copper (Cu) - anything brass, an alloy of Zn & Cu; city water (chlorinated at .002 ppm); detergents; heavy metals; neoprene; PVC type II; zinc (Zn) - anything galvanized - if the water is less than 50 ppm in hardness.

PUMPS

Pumps are mechanical devices used in hatchery water supply systems to provide head pressure and flow. If improperly installed or malfunctioning, pumps can also present fish health problems such as supersaturation with gases or an unreliable water flow.

1. Centrifugal pumps

These pumps have an impeller than spins the water, forcing it against the pump housing and out the discharge port. An oil or water-sealed bearing is located between the impeller's drive shaft and pump housing. To avoid toxicity problems, water-sealed bearings are preferred in hatchery use. The discharge of a centrifugal pump decreases as the head increases. In a situation where water must be pumped to considerable heights, this requirement may dictate the use of a different pump. The outlet or discharge of a centrifugal pump can be choked without risk of damage to the power source equipment but there is a significant loss of efficiency.

2. Rotary pumps

The impeller in this category of pumps simply pulls water through the housing. This type of design is referred to as a positive displacement pump since there is no water slippage around the impeller. For this reason, a rotary pump's discharge should ***never*** be restricted or choked and an outlet relief valve is mandatory.

A bearing between the end of the impeller shaft and the housing bears the thrust created by the impeller. This force can be substantial and represents a maintenance problem.

Power requirements for rotary pumps increase as the pressure head increases. Rotary pumps are best in situations involving a low head and a high discharge.

SUMMARY

Water supplies for aquacultural enterprises must meet both quantity and quality requirements. The available water quantity must be sufficient to make up for evaporative and seepage losses, to supply the necessary amount of oxygen, and to provide adequate flushing to remove waste products. Sufficient water must also be available to supply other uses such as irrigation, domestic needs, and fire protection, if necessary. Water quality for aquaculture must meet certain high standards. The temperature, oxygen content, pH, and hardness must be at optimal levels for fish, or it must be economically feasible to alter them to needed levels. Pollutants and undesirable organisms in the water supply must be eliminated.

Groundwater is the most desirable water supply source for aquacultural enterprises if adequate precautions are taken to assure that sufficient oxygen concentrations are present in the water. Good springs are most desirable, since pumping costs are eliminated. Wells are also good but pumping may be expensive. If depression of the water table occurs, wells should be used only after careful thought and planning. Chemicals are added to the water in most city systems, so pretreatment is necessary before such water can be used in fish culture systems.

Surface water sources (e.g. streams, lakes, ponds) all exhibit greater variations in environmental parameters than groundwater sources. Surface waters are normally high in oxygen concentration when compared to groundwater but a greater risk for pollution problems exists.

REFERENCES

Anonymous. 1950-51. Handbook of chemistry and physics. 32nd ed. Chemical Rubber Publ. Co., Cleveland, OH. 2879 p.

Benson, B.B., and P.D.M. Parker. 1961. Relations among the solubilities of nitrogen, argon, and oxygen in distilled water and sea water. Physical Chem. 65: 1489-1496.

Bouck, G. R. 1982. Gasometer : An inexpensive device for continuous monitoring of dissolved gases and supersaturation. Trans. Am. Fish. Soc. 111 : 505-516.

Economon, P. 1980. Nitrogen gas sickness in hatchery reared lake trout fingerlings. Minnesota Dep. Nat. Resources, Fish Wildl. Pathol. Arch. 25, St. Paul, MN. 3 p.

Fickeisen, D. H., M. J. Schneider, and J. C. Montgomery. 1975. A comparative evaluation of the Weiss saturometer. Trans. Am. Fish. Soc. 104: 816-820.

Harvey, H.H., and A. C. Cooper. 1962. Origin and treatment of supersaturated river water. Int. Pac. Salm. Fish. Comm., Progr. Rep. 9. 19 p.

Oesting, R.B. 1934. A modified Van Slyke method for the determination of dissolved oxygen and total carbon dioxide in water. Physiol. Zool. 7: 542-549.

Owsley, D.E. 1981. Nitrogen gas removal using packed columns, p. 71-92. In Bio-Eng. Symp. Fish Cult., Am. Fish. Soc., Fish. Cult. Sec. Publ. No. 1. Bethesda, MD.

Rucker, R.R., and E.M. Tuttle. 1948. The removal of excess nitrogen in a hatchery water supply. Prog. Fish-Cult. 10: 88-90.

Swinnerton, J. W., V.J. Linnenbom, and C.H. Cheek. 1962. Determination of dissolved gases in aqueous solutions by gas chromatography. J. Anal. Chem. 34: 483-485.

U.S. Environmental Protection Agency. 1979-80. Water quality standards criteria digest: A compilation of state/federal criteria. U.S. EPA, Washington, DC.

Van Duijn, C. 1966. Diseases of fishes. 2nd ed. Iliffee Books Ltd., London. 309 p.

Weitkamp, D. E. and M. Katz. 1980. A review of dissolved gas supersaturation literature. Trans. Am. Fish. Soc. 109: 659-702.

Wood, J.W. 1968. Diseases of Pacific salmon, their prevention and treatment. Wash. Dep. Fish., Hatchery Div., Olympia, WA. 82 p.

6

WATER SUPPLY SANITATION

A. J. SIPPEL
Ontario Ministry of Natural Resources
Fisheries Branch
Toronto, Ont.

Disinfection of water used in fish culture is an effective means of disease control in fish culture situations (Burrows and Combs 1968; Vlasenko 1969; Bedell 1971; Sanders et al. 1972; Hoffman 1974; Conrad et al. 1975; Bullock and Stuckey 1977). Dupree (1981) has prepared an excellent review of the various methods of water disinfection for disease control. Several applications are listed below:

1. Disinfection can be used to permit the use of certain otherwise undesirable water supplies when alternate sources are unavailable or when fish pathogens are unavoidably present in the water supply. This reduces the risk of infecting fish or eggs in the culture station and helps avoid serious epizootics although disease may not be eliminated entirely (Vlasenko 1969; Sanders et al. 1972; Hoffman 1974). Significant results may be achieved without disinfecting the entire hatchery water supply. In the case of IPN for example, disinfection of water used during early rearing will help prevent exposure to the pathogen until the fish are past the susceptible stage.
2. Disinfection can reduce the incidence of disease transmission between units if water must be reused (Conrad et al. 1975; Bullock and Stuckey 1977). If water is reused within the same unit, disinfection can help reduce the numbers of pathogens present in the system.
3. Disinfection can also be used to prevent dissemination of pathogens in effluents from fish culture operations. Disinfection (if not sterilization) would be valuable at a fish disease research facility, at a quarantine station for imports, or when protection of native fish populations or

other fish culture facilities in/on downstream waters is desirable. This application is discussed below under "Quarantine Facilities".

Disinfection of effluent water is important because fish pathogens may reach high densities in the water. Desautels and MacKelvie (1975) detected significant levels of IPN virus in water taken from rearing troughs during an epizootic. *Aeromonas salmonicida* densities of 4,000 to 120,000 bacteria per ml were reported in the effluent from a tank of infected brown trout (Bullock and Stuckey 1977). Furthermore, some infectious pathogens can survive for a considerable time in the absence of a suitable host (Table 1). These high densities and long survival times suggest that the spread of pathogens from culture facilities can be significant.

METHODS OF DISINFECTION

The disinfection method(s) used must meet several criteria, including:

1. The method must not alter the physical-chemical properties of the water.
2. Treatment chemicals or treatment by-products must not be harmful to fish or other aquatic life or must be easily rendered safe. (The treatment must not damage any biofilters that may be in use in a re-circulating system.)
3. All equipment should be adaptable to flow through situations with minimal use of electrical or other forms of energy.
4. Every effort must be made to minimize the chance of a pathogen's escape by making the system as fail-safe as possible.
5. Most importantly, the method must be effective in eliminating the pathogenic organisms that are of concern in a particular situation.

Three methods meet some or all of these criteria. They are ozonation, ultra-violet (UV) irradiation, and chlorination. This report will not discuss designs for disinfection equipment. However, one important design feature must be used with each disinfection method. All three procedures are most effective and efficient following filtration of the water being treated. Burrows and Combs (1968) recommended UV irradiation following filtration through rapid flow sand filters to control diseases in closed or semi-closed systems. Bullock and Stuckey (1977) also found that filtration improved the effectiveness of UV radiation in destroying Gram-negative fish pathogens. Sanders et al. (1972) used a sand or Microfloc filter followed by chlorination or UV irradiation to control ceratomyxosis.

Filtration reduces the particulate organic material usually present in surface waters, re-circulation waters, and hatchery effluents. Organic material increases the amount of chlorine and ozone required to destroy pathogenic organisms. It also reduces the ability of UV radiation to penetrate the water being treated, thereby reducing its effectiveness.

Table 1. Survival time of several fish pathogens in water

Pathogen	Survival Time	Temp.	Remarks	Reference
IPN virus	*24* wks.	*4°C*	residual infectivity remained	Desautels and MacKelvie (1975)
IPN virus	10 days	4°C		Tu, et al. (1975)
IPN virus	8 wks.	10°C	no loss of titer in hard or soft water	Wedemeyer, et al. (1978)
IHN virus	*7* wks.	10°C	hard or soft water	" "
Aeromonas salmonicida	*2* wks.	*20°C*	soft lake water	Wedemeyer and Nelson (1977)
" "	*2* wks.	*20°C*	hard lake water	" "
Yersinia ruckeri	*20* days	*20°C*	survived over *20* days in hard and soft lake water	" "

Further discussions of factors affecting the use of disinfectants can be found in Dychdala (1968) for chlorination; in Hoffman (1974) and Spotte (1979) for ultraviolet irradiation; and in Farooq et al. (1977a, 1977b) and Spotte (1979) for ozonation.

There are no water disinfection standards for fish culture use. The remander of this report will review the available literature on the effectiveness of each method of disinfection in controlling fish pathogens and will recommend levels for control procedures.

CHLORINATION

Chlorination (the use of active chlorine compounds or liquids or gaseous chlorine) is used widely as a method of disinfection. Chlorination is recommended by the Great Lakes Fish Disease Control Committee (GLFDCC) as the

method of disinfection for eradication of emergency diseases. The GLFDCC recommends a two hour exposure to 200 mg/l total chlorine. This is an extremely high concentration and is designed with a 'one time only' application in mind. Because chlorine is extremely toxic to aquatic organisms, even at very low concentrations, it must be completely neutralized before re-use or discharge. Obviously, the high concentration recommended by the GLFDCC poses serious problems for routine use, particularly with large water volumes.

Bedell (1971) and Sanders et al. (1972) were able to control (but not eliminate) ceratomyxosis by treating the water supply with 1 to 5 mg/l residual chlorine. Lower concentrations may also be equally effective in controlling other disease agents and the available data (Table 2) indicate this is the case. However, destruction of Myxosoma *cerebralis* spores requires exceptionally high chlorine concentrations for prolonged exposures (Hoffman and Putz 1969). Therefore, whirling disease control may not be practical by chlorination. For water supplies and reuse systems, residual chlorine concentrations of at least 1.0 mg/l for 10 minutes should effectively control most viral and bacterial fish pathogens. However, there are conflicting data on the efficacy of chlorine against IPN virus and caution is advised if this is the target pathogen.

OZONATION

Ozone gas (triatomic oxygen -0,) has been used to disinfect fish culture water (Conrad et al. 1975; Spotte 1979) and the efficacy of ozone against certain fish pathogens has been determined (Table 3). Ozone, delivered at the rate of 90 mg/l with a minimum 20 min exposure time, should effectively control most bacterial and viral fish pathogens in water supplies and reuse systems although this level may not eliminate 100% of all pathogens. No data are available on control of *M. cerebralis* or *C. shasta* by ozonation.

Like chlorine, ozone is toxic to aquatic organisms. Furthermore, a break-down product of 0_3 is 0_2 and oxygen supersaturation may also occur. Ozonated water must be aerated in a holding tank prior to use or discharge.

An important consideration with ozonation is the energy inefficiency of current ozone generators (Spotte 1979). Therefore, ozonation is not a practical disinfection method for large water volumes.

ULTRA VIOLET (UV) IRRADIATION

UV light at wave lengths of about 254 nm has been shown to be an effective disinfectant for fish culture waters (Burrows and Combs 1968; Vlasenko 1969; Hoffman 1974; Spotte 1979). Hoffman (1974) reviewed the use

Table 2. Chlorine concentration and exposure time required to kill or inactivate certain fish pathogen

Pathogen	Concentration	Time	Water Quality	Reference
IPN virus	*25* mg/l	30 min		Desautels and MacKelvie (1975)
" "	0.2 mg/l	10 min.	soft lake water(a)	Wedemeyer, et al (1978)
" "	0.7 mg/l	2 min	hard lake water(b)	" "
IHN virus	0.5 mg/l residual	10 min	" "	" "
" "	" "	5 min	soft lake water	" "
" "	1.0 mg/l	*30 sec.*	hard lake water	" "
Aeromonas salmonicida	0.1 - 0.2 mg/l	*30-60 sec.*	hard or soft water	Wedemeyer and Nelson (1977)
Yersinia ruckeri	0.1 mg/l	2 min	" "	" "
Ceratomyxa shasta	uncertain *(0.3 - 1.5* mg/l)	100 min.	lake water	Bedell (1971)
" "	*2.2-5.3* mg/l residual		surface water	Sanders, et al. (1972)
Myxosoma cerebralis	1600 mg/l	*24 hr.*		Hoffman and Putz (1969)
	200 mg/l(c)	" "		" "

a) - Water hardness of 30 mg/l as $CaCO_3$; 10°C
b) - Water hardness of 120 mg/l as $CaCo_3$; 10°C
c) - Not proven 100% effective

Table 3. Ozone delivery rates and exposure times required to kill or inactivate certain fish pathogens

Pathogen	Delivery Rate (a)	Exposure Time	Water Quality	Reference
Aeromonas salmonicida	90 mg/h/l	5 min	hard lake water (b)	Wedemeyer and Nelson (1977)
" "	" "	15 min	soft lake water (c)	"
" "	20 mg/h/l	30 min	hard or soft	" "
Yersinia ruckeri	90 mg/h/l	10 min	" "	" "
	20 mg/h/l	25 min	" "	" "
IPN virus	90 mg/h/l	10 min	hard lake water	Wedemeyer et al (1978)
" "	" "	30 sec	soft lake water	" "
IHN virus	70 mg/h/l	10 min	hard and soft	" "

a) - rate of ozone delivery, not the residual ozone concentration
b) - water hardness of 120 mg/L as $CaCO_3$; 20°C (= 20°C)
c) - water hardness of 30 mg/L as $CaCO_3$; 20°C

of UV and its effectiveness against certain fish pathogens. Vlasenko (1969) reported on the effectiveness of UV against *Saprolegnia* and several protozoan parasites. Bullock and Stuckey (1977) found that a UV dosage of 13,100 uW/sec/cmz killed 99.99-100% of several species of Gram-negative bacterial pathogens. However, even 21,000 to 24,000 uW/sec/cm^2 did not consistently kill 100% of the test organisms (Bullock and Stuckey 1977). UV irradiation at 13,100 uW/ sec/cm^2 prevented the transmission of *A. salmonicida* from a tank of infected brown trout to Atlantic salmon fingerlings over a four week period (Bullock and Stuckey 1977).

Few data are available on the use of UV for the inactivation of fish pathogenic viruses. In a static system, MacKelvie and Desautels (1975) reported that 2000 uW/cm^2 reduced the concentration of infective IPNV particles over a six minute period but did not inactivate all viruses even after 15 min. The researchers

concluded that UV radiation could not effectively eliminate IPN virus from the water (MacKelvie and Desautels 1975).

Spotte (1979) recommended UV radiation at 35,100 uW/sec/cm2 for disinfection in re-use systems. However, 13100 uW/sec/cm2 may be sufficient to effect control of bacterial pathogens. In the case of protozoan parasites, such as *Ichthyophthirius,* 90,000 to 1,700,000 uW/sec/cm2 may be required for effective control (Vlasenko 1969). Many of the "effective" doses reported in the literature do not effect 100% kill consistently. However, 90-99% may be adequate for disease control in some circumstances. UV levels effective against viral fish pathogens are not known.

Ultraviolet systems require regular cleaning of the bulbs and annual bulb replacement. However, the systems are readily adapted to fish culture operations, no toxic products are produced, and the level of radiation can be controlled more easily than levels of chlorine or ozone.

UV irradiation appears to be the method of choice for disinfection of water supplies and reuse systems. Levels of at least 13,100 uW/sec/cm2 should be used to control bacterial diseases and increased to 35,000 uW/sec/cm2 if *M. cerebralis* or *C. shasta* are of concern. Even higher levels are required to control other protozoan parasites. The final choice of disinfectant will depend, however, on such considerations as initial capital costs, annual maintenance costs, energy requirements and the pathogen(s) to be controlled.

QUARANTINE FACILITIES

The principle of quarantine facilities is to protect against the introduction of a new pathogen to an area (i.e. fish hatchery, watershed, political jurisdiction) through the introduction of a new stock of fish that may be infected. Quarantine facilities must be separated, either by distance or physical barrier, from other fish culture activities and all effluent water must be disinfected. The water supply sanitation procedures discussed above can be applied.

Inactivation of 90-99% of pathogens may produce effective results in sanitation of water supplies and re-use systems. However, in the case of pathogen containment and quarantine facilities, 99% or even 99.99% is not acceptable. For this reason, ozonation and ultraviolet irradiation are not recommended. Chlorination at 200 mg/l total chlorine for two hours is preferred. However, some facilities have had good results using lower chlorine concentrations, particularly if a longer exposure time is used. These levels are only recommended for small water volumes as use on large volumes of water could represent a serious environmental hazard.

Quarantine failities for fish culture are not in use outside of research facilities. However, systems are being developed in Alaska, U.S.A. and the Maritime Provinces of Canada. Suggested contacts for further information are:

1. Principal Pathologist
   Fish Pathology Section
   Alaska Department of Fish and Game
   333 Raspberry Rd.
   Anchorage, AK 99502

2. Connaught Research Institute
1755 Steeles Ave. W.
Willowdale, Ontario M2N 5T8

3. Fish Disease Laboratory
Department of Microbiology
Oregon State University
Corvallis, OR 97331

4. Fish Health Unit-Scotia-Fundy Region
Dept. of Fisheries and Oceans
P.O. Box 550
Halifax, Nova Scotia B3J 2S7

5. National Fish Health
Research Laboratory
Route 3, Box 700
Kearneysville, WV 25430

A word of caution is in order. Although the principles behind the operation of quarantine facilities suggest that the potential for success is present, there has been insufficient experience to fully assess their potential.

REFERENCES

Bedell, G.W. 1971. Eradicating *Ceratomyxa shasta* from infected water by chlorination and ultraviolet irradiation. Prog. Fish-Cult. 33: 51-54.

Bullock, G.L., and H.M. Stuckey. 1977. Ultraviolet treatment of water for destruction of five Gram-negative bacteria pathogenic to fishes. J. Fish. Res. Board Can. 34: 1244-1249.

Burrows, R.E., and B.D. Combs. 1968. Controlled environments for salmon propagation. Prog. Fish-Cult. 30: 123-136.

Conrad, J.F., R.A. Holt, and T.D. Kreps. 1975. Ozone disinfection of flowing water. Prog. Fish-Cult. 37: 134-136.

Desautels, D., and R.M. MacKelvie. 1975. Practical aspects of survival and destruction of infectious pancreatic necrosis virus. J. Fish. Res. Board Can. 32: 523-531.

Dupree, H. K. 1981. An overview of the various techniques to control infectious diseases in water supplies and in water reuse aquacultural systems. pp. 83-89 In L. J. Allen and E. C. Kenney [ed.] Bio-Eng. Symp. Fish Cult., Am. Fish. Soc., Fish Cult. Sec., Publ. 1. Bethesda, MD.

Dychdala, G.R. 1968. Chlorine and Chlorine Compounds, p. 278-329. *In* C.A. Lawrence and S. S. Block (ed.). Disinfection, sterilization, and preservation. Lea and Febiger, Philadelphia, PA.

Farooq, S., R.S. Engelbrecht, and E.S.K. Chian. 1977a. Influence of temperature and ultraviolet light on disinfection with ozone. Water Res. 11: 737-741.

Farooq, S., E. S.K. Chian, and R.S. Engelbrecht. 1977b. Basic concepts in disinfection with ozone. J. Water Pollut. Control Fed. 49: 18181831.

Hoffman, G.L. 1974. Disinfection of contaminated water by ultraviolet irradiation, with emphasis on whirling disease *(Myxosoma cerebralis)* and its effect on fish. Trans. Am. Fish. Soc. 103: 541-550.

Hoffman, G. L., and R. E. Putz. 1969. Host susceptibility and the effect of aging, freezing, heat, and chemicals on *the* spores of *Myxosoma cerebralis.* Prog. Fish-Cult. 31: 35-37.

MacKelvie, R.M., and D. Desautels. 1975. Fish viruses -survival and inactivation of infectious pancreatic necrosis virus. J. Fish. Res. Board Can. 32: 1267-1273.

Sanders, J.E., J.L. Fryer, D.A. Leith, and K.D. Moore. 1972. Control of the infectious protozoan *Ceratomyxa shasta* by treating hatchery water supplies. Prog. Fish-Cult. 34: 13-17.

Spotte, S. 1979. Fish and Invertebrate culture: Water management in closed systems, 2nd ed. John Wiley and Sons, Toronto. 179 p.

Tu, K.C., R.S. Spendlove, and R. W. Goede. 1975. Effect of temperature on survival and growth of infectious pancreatic necrosis virus. Infect. Immun. 11: 1409-1412.

Vlasenko, M. I. 1969. Ultraviolet rays as a method for the control of diseases of fish eggs and young fishes. Probl. Ichthyol. 9: 697-705.

Wedemeyer, G. A., and N. C. Nelson. 1977. Survival of two bacterial fish pathogens *(Aeromonas salmonicida* and the enteric redmouth bacterium) in ozonated, chlorinated and untreated water. J. Fish. Res. Board Can. 34: 429-432.

Wedemeyer, G.A., N.C. Nelson, and C.A. Smith. 1978. Survival of the salmonid viruses infectious hematopoietic necrosis (IHNV) and infectious pancreatic necrosis (IPNV) in ozonated, chlorinated, and untreated waters. J. Fish. Res. Board Can. 35: 875-879.

7

STOCK AND YEAR CLASS SEPARATION

RICHARD POYNTER
Michigan Department of Natural Resources
Fisheries Division
Oden State Fish Hatchery
Oden, MI

A fish hatchery operation that rears brood fish for eggs, replacement stocks, and fish for stocking is more complex than an operation which produces only fish for stocking. A combination broodstock-production hatchery may have many species of fish, comprised of many year classes. The placement of various species and sizes of fish within the hatchery system can be of major importance because of water quality demands, possible disease transmittal, and the volume of waste generated.

All salmonids, large and small, require water that is high in oxygen, moderate in temperatures, and low in metabolic waste levels (Laio, 1971). Small fish and adult fish generally do not tax water quality as much as fish approaching release or market size because of the lower densities (in kilograms per cubic foot) involved. Among species, fish have different requirements for water quality. Brook trout cannot tolerate poor water conditions, rainbows are more tolerant and brown trout are the most tolerant. As fish size increases, so does their tolerance for "used" water. However, attempts to rear broodstock in re-used water in a Michigan hatchery resulted in increased disease problems, mortalities, inferior eggs, and prolonged spawning. Rainbow and brown trout broodstock being held in raceways below 500,000 production rainbows experienced mortalities of 1% per day or greater. After moving these fish into ponds above the production fish, losses began to decrease and declined to less than 1% per month.

While pathogen-free and/or disease-resistant fish should be the goal, progress toward this goal is often slow. Disease-free fish and eggs are available from certified sources. Eggs from diseased fish can be disinfected with iodophors or water-hardened in a solution of erythromycin phosphate to control pathogens for

which there is no vertical transmission. These measures are helpful when attempting to eradicate disease organisms, but they usually prove fruitless if diseased fish are present in the hatchery water supply or if "carrier" fish are present in the hatchery system.

Separation or isolation of fish stocks is one way to prevent the introduction or spread of diseases within a hatchery. If new disease-free stocks are introduced into the hatchery, or if stocks resulting from disinfected eggs and future brood stock are placed below mature, disease- carrying fish, contamination of the new stocks will occur and the disease will continue to be present in the hatchery. Isolation of infected fish from future brood stock, production fish, and eggs should help to prevent the spread of disease within a hatchery.

Depending on hatchery design, complete separation can be very simple or almost impossible to achieve. Since most fish pathogens require a host and are waterborne (Wood 1968), the placement of disease-free fish in unused, fish-free water should prevent exposure to disease organisms. Fish of unknown disease status should never be located above disease-free fish or below fish that have a known infection. Diseased fish should not be placed above fish known to be disease-free.

Isolation or separation of a fish stock can be accomplished by the following:

1. Always place disease-free fish upstream from known or suspected diseased fish.
2. Place diseased fish downstream from fish already in the hatchery.
3. Smaller fish should be upstream from larger fish.
4. If rearing units consist of a series of ponds on a two or three pass system, use a separate series of ponds to raise different year classes of a single species. If raising mixed species, place the species requiring highest quality water in the first pass, or the species showing least tolerance to disease in the first pass. Proceed through second or third pass ponds using fish more tolerant of lower quality water or with less disease susceptibility.
5. An isolated area in the hatchery system should be utilized as a quarantine area. Raising fish of unknown disease status for a period of 6 months to two years should indicate the presence of any disease organisms the new fish may have, due to mortalities or to the detection of specific pathogens during regular disease inspections.

Introductions of infected or carrier fish from other hatcheries or sources should be avoided. Introducing a new disease organism or a different strain of an existing disease can create very serious problems. Again, isolation of suspect fish from existing stocks and disinfection of fish handling equipment should prevent the spread of disease organisms to other fishes. Even so, many possibilities exist for contaminating "clean" stocks,

The utilization of "wild" fish for hybridization to broaden the gene pool, and to improve survival of stocked fishes, is on the increase. Although these fish may be free of detectable disease organisms, they usually show little resistance to pathogens that may be present in the hatchery system. Treatments often prove ineffective, and prolonged mortalities may occur until these fish develop a resistance to the pathogen.

Stress is a major factor in fish disease outbreaks and mortalities (Wedemeyer 1970; Klontz et al. 1979). Stresses are often present, even under

quality rearing conditions. Many “normal” activities stress fish and can cause serious disease outbreaks in healthy appearing fish. Monthly sampling, thinning operations, fin-clipping, pond cleaning, and spawning operations can be stressful. Mature fish are obviously stressed during spawn-taking, but fish in nearby ponds may also be affected. Extended activities associated with sorting and spawn-taking often cause fish in adjacent units to become excited and go off their feed. Birds, animals, and hatchery visitors can cause stress to fish and help to spread disease from pond-to-pond. Physical barriers are the only effective means of preventing this type of stress and spread of disease organisms (Ostergaard 1981; Salmon and Conte 1981).

The placement of various sizes of fish within the hatchery system can also be critical relative to the generation of solid wastes. Fecal materials that easily pass thorugh a 2 x 2 mesh screen at the foot of a mature brood stock pond will tend to plug a 6 x 6 mesh screen at the head of the lower pond if used to produce small production fish.

Separation of fish stocks by species, size, or use is difficult in a facility not designed for rearing and holding brood and production fish. The ideal hatchery would consist of a series of double- or triple- pass ponds with 900 to 2,400 cubic feet of rearing space. Small ponds would be used to accommodate small numbers of future brood stock (5,000 - 20,000), small lots of mature brood stock (50 +), and select production fish. Large ponds would be utilized for maturing brood stock (1,000 - 10,000), mature fish (500 - 5,000) and large lots of production fish (50,000 - 100,000). Such a pond system would allow for the segregation of fish by size, species, age, and disease status. A large number of ponds in combination with limited water reuse would produce both quality fish and eggs.

REFERENCES

Klontz, G. W., P. C. Downey, and R. Focht. 1979. A manual for trout and salmon production. Sterling H. Nelson and Sons, Inc., Murray, UT 19 p.

Laio, P.B. 1971. Water requirements of salmonids. Prog. Fish-Cult. 33: 210-212.

Ostergaard, D.E. 1981. Use of monofilament fishing line as a gull control. Prog. Fish-Cult. 43: 134.

Salmon, I? and ES. Conte. 1981. Control of bird damage at aquaculture facilities. U.S. Fish Wild. Serv., Wildl. Mgmt. Leafl. 475. Washington, DC. 11 p.

Wedemeyer, G. 1970. The role of stress in the disease resistance of fishes, p. 30-35. In S.F. Snieszko, (ed.) A symposium on diseases of fishes and shellfishes. Am. Fish. Soc., Special Publication No. 5, Bethesda, MD.

Wood, J.W. 1968. Diseases of Pacific salmon: their prevention and treatment. Washington Dep. of Fish., Hatchery Div., Olympia, WA. 82 p.

8

NUTRITION AND FISH HEALTH

C. YOUNG CHO
University of Guelph
Department of Nutrition
Guelph, Ont.

INTRODUCTION

In culturing fish in captivity, nothing is more important than sound nutrition and adequate feeding. If there is no utilizable feed intake by the fish, there can be no growth and death eventually results. Under-nourished or malnourished animals cannot maintain health and growth, regardless of the quality of the environment. Therefore, before any attempt at fish culture it would be wise to ask a fundamental question, "What and how should I feed my fish?"

Faulty nutrition impairs fish productivity and affects their health; a fact the fish culturist should always keep in mind. Clinical disease often ensues when nutritional needs are not met. The borderline between reduced growth and diminished health and overt disease is difficult to define.

Diets may hasten recovery from infection or slow the progress of an idiopathic disease or overcome environmental stress. However, diets may also cause nutrient imbalances, deficiency diseases, nutritional toxicoses, or may introduce infective agents. As a consequence, nutritionally- balanced and quality-controlled diets are of critical concern in fish production.

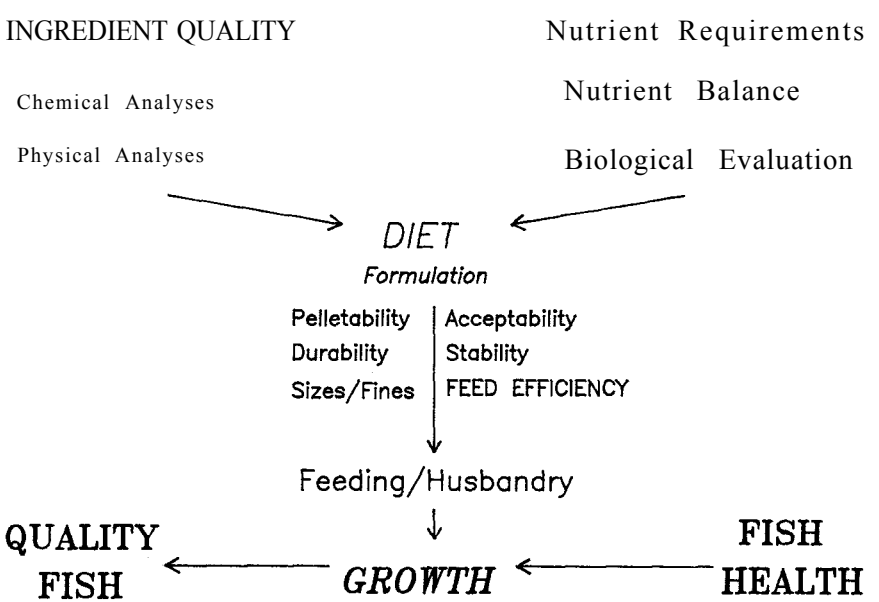
As shown in Fig. 1. many steps of research, quality control, and biological evaluations must be exercised by various individuals and groups to develop and produce nutritionally-balanced diets for fish.

NUTRIENT REQUIREMENTS AND DEFICIENCIES

ENERGY

Energy is not a nutrient. It is rather an end-product of absorbed macro-organic nutrients when they are oxidized and metabolized. All organic com-

Figure 1 NUTRITION AND FISH HEALTH



of energy. For salmonid fishes, most dietary carbohydrates, such as raw starch from plant feedstuffs, are not utilized as energy sources. Simple sugars can be utilized by salmonids but their use as energy sources in feed formulations is of no practical significance. Lipids and proteins, therefore, provide the primary dietary sources of energy. Physiologically, lipids and proteins form an important part of a structure of a fish, but the need for energy can preclude their incorporation into tissues and may involve their catabolism as a source of energy. Thus, utilization of the nutrients of each diet depends both upon the level of intake and upon the make-up of the diet. The over-riding importance of food as an energy source means that the major factor regulating the food intake of the animal is its energy value in relation to the animal's energy needs. As a consequence, the concentration of the essential nutrients which must be provided in the diet to adequately meet the animal's requirements is directly related to the energy value of the diet. Therefore, the biologically utilizable (metabolizable) energy content of a diet must be defined in relation to the other needs before one can estimate the effect of a diet upon the growth and well-being of the fish.

In practice, fish culturists must estimate the biologically available (digestible) energy content of a diet before they can determine the weight of feed that should be fed each day. A low energy diet which usually contains a high level of carbohydrate (starch and fiber) results in poor weight gain and feed efficiency in salmonids. Furthermore, the increased intake of a poorly utilizable feed results in increased excretion of feces which will pollute the aquatic system. Diets for

salmonids should contain at least 14 MJ (3.35 Mcal) total digestible energy per kg feed; of this, at least 4 MJ or 1 Mcal/kg energy must be of lipid origin (Cho 1981).

Amino Acids and Proteins

Dietary proteins are the source of essential amino acids and provide nitrogen for the synthesis of non-essential amino acids. Proteins in the body tissues are built using about 23 amino acids. Of these, 10 are essential amino acids (see Table 1) which must be supplied in the fish diet. Proteins or amino acids are necessary for maintenance, growth, reproduction and for the replacement of depleted tissues. In addition, certain amino acids are readily converted to glucose to provide an essential energy source for some critical body organs and tissues such as brain and red blood cells. Since carbohydrate is not prevalent in their natural diet, fish are more dependent upon amino acids as precursors to glucose than most other animals. Therefore, a portion of the dietary protein is always used as an energy source in fish.

Not all dietary proteins are identical in their nutritional value. To a large extent, the bio-availability of a protein source is a function of its digestibility and amino acid makeup. Some protein feedstuffs which contain a high level of crude protein (% total nitrogen x 6.25) are low in amino-nitrogen and do not contribute toward the requirement of amino acids. As a result, such materials may merely increase ammonia production into the water environment.

A deficiency of essential amino acids may lead to poor utilization of dietary protein, and may result in growth retardation, poor live weight gain, and low feed efficiency. In severe cases, amino acid deficiency lowers resistance to diseases and impairs the effectiveness of the immune response mechanism. Deficiencies of specific amino acids may also elicit clinical signs. For example, experiments have shown that tryptophan deficient fish become scoliotic, showing a characteristic curvature of the spine (Kloppel and Post 1975) and a methionine deficiency is one cause of lens cataracts (Poston et al. 1977).

The protein component generally represents the largest portion of the total cost of a diet, but protein ingredients are not necessarily the most expensive feedstuffs. Under certain market conditions, using protein, rather than fat, as an energy source may reduce feed cost. Major sources of protein for salmonid diets are marine fish meals (in Canada - herring and whole capelin meals). In most diet formulas, other protein ingredients such as soybean meal, corn gluten meal, and dairy or animal by-product meals are employed as protein sources. In formulas for fry, brood stock, and fingerling fish, 30-50% of high quality fish meal and 10-15% of fish oil are recommended in the diet. The cost of feed is a relatively small fraction of the potential economic value of the fish produced.

Most salmonid diets should contain 56-75 g of amino-nitrogen per kg of feed; this is equivalent to 35-50% crude protein. However, amino acids or protein must be supplied in relation to the needed energy content. The recommended ratio of protein to energy in salmonid diets is 3.5-4.0 g digestible amino-nitrogen per MJ digestible energy (15-17 g N or 92-105 g protein per Mcal). Ratios in excess of these values result in increased ammonia excretion. Furthermore, the dissolved oxygen requirement increases as energy efficiency is decreased.

Table 1. Recommended Nutrient Levels in Salmonid Diets

	Recommended Levels		Deficiency(a) Sign Codes
ENERGY:			
Digestible energy, total	14-17 MJ/kg feed(b) (3.3-4.1 Meal)		32, 38
Energy of lipid origin (=10-18% lipid of diet)	4-7 MJ/kg feed (1.0-1.7 Meal)		32, 38
DIGESTIBLE PROTEIN/ENERGY RATIO:			
per MJ energy	3.5-4.0 g amino-N		32 38
per Mcal energy	15-17		
AMINO ACIDS AND PROTEINS:			
Amino-Nitrogen (= 35-50% crude protein)	56-75 g/kg feed		22, 32, 38
Amino Acids(c)	(g per 16 g of amino-N)	*(g per* kg feed)	
Arginine	6.0	24	
Histidine	1.8	7	
Isoleucine	2.2	9	
Leucine	3.9	16	
Lysine	5.0	20	
Methionine + Cystine	4.5	18	09, 11, 48
Phenylalanine + Tyrosine	5.1	21	
Threonine	2.2	9	
Tryptophan	0.5	2	62
Valine	3.2	13	
FATTY ACIDS:(d)			
Longer chain, unsaturated w-3 fatty acids	20-30 g/kg feed		16,28,31,46,51 53,63

a) Deficiency sign codes - see Table 2
b) 1MJ/kg = 239 kcal/kg = 0.239 Mcal/kg
c) Amino acid recommendation - modified from NRC 1981
d) Fatty Acid recommendation - modified from Castell et al. 1972

	Recommended Levels	Deficiency(a) Sign Codes
VITAMINS: (e)		
Fat-Soluble		
Vitamin A (IU/kg feed)	3500	03,18,25,28,29,41,43,45,48
Vitamin D3	3000	62,67
Vitamin E	100	01,03,10,24,26,29,31,33,35
Vitamin K (mg/kg feed)	10	01.13
Water-soluble		
Ascorbic acid (mg/kg feed)	300	01,03,08,22,29,44,50,52,60
B12	0.02	01,35,40
Biotin	0.5	06,14,15,19,31,33,35,47,64
Choline	3000	31,43
Folic acid	5	01,14,34,50
Inositol	400	01,23,31,49
Niacin	150	01,25,45,47,49,50,57,65,67
Pantothenic acid	60	04,05,12,20,30,49,54,60,66
Pyridoxine	10	04,14,15,27,36,55,61,64,66
Riboflavin	20	09,11,14,28,41,48,57,59,68
Thiamine	10	06,09,15,16,25,27,46,50,55
MINERALS:(f)		
Calcium (g/kg feed)	0.2-0.3	32,38
Phosphorus, inorganic	5-8	17,62
Magnesium	0.5-0.7	02,07,38
Copper (mg/kg feed)	3	38
Manganese	12-13	38
Selenium	0.1-0.4	21,24
Zinc	15-30	09,11,28,48
Iodine (ug/kg feed)	0.6-l. 1	37
Iron	required	01

a) Deficiency sign codes - see Table 2
e) Vitamin recommendation - modified from NRC 1981
f) Mineral recommendation - modified from Lall 1981

Fatty Acids and Lipids

The nutritionally active components of dietary lipids are fatty acids. Fish and mammals appear to be unable to synthesize fatty acids that are unsaturated in the w-3 or w-6 positions unless a suitable precursor is supplied in the diet. Thus, the lipid component of the diet must provide an adequate amount of essential fatty acids for growth as well as for required dietary fuel. In contrast to mammals which have a major requirement for w-6 fatty acids, many coldwater and marine fishes require w-3 fatty acids. Therefore, sufficient amounts of essential fatty acids (w-3 fatty acids or longer chain members of these series) must be included in the dietary lipids. One percent linolenic acid (18:3w3) in the diet is required by rainbow trout to avoid such deficiency signs as loss of pigmentation, fin erosion, cardiac myopathy, fatty infiltration of the liver, and shock syndrome (Castell et al. 1972). Salmonids utilize lipids as a major source of energy and digest complex carbohydrates very poorly. Diets for salmonids, therefore, should contain very high levels of lipids (10-18%) in comparison to diets for other animals. Because of the high level of use, lipid quality is critical since marine fish oil is very susceptible to oxidation. In all circumstances, rancid oil must be avoided in fish feed. Fish suffering from lipoid liver disease have extreme anemia, a bronzed, rounded heart and a swollen liver with rounded edges. Histologically, the main feature is the extreme lipoid infiltration of hepatocytes and associated loss of cytoplasmic staining and distortion of hepatic muralia (Cowey and Roberts 1978). All salmonids are suceptible to lipoid degeneration of the liver, but it is a particularly significant problem in rainbow trout. Slightly affected fish are usually capable of recovery, but if severe anemia and hepatic ceroidosis have developed, the fish are rarely capable of recovery to an acceptable feed efficiency (Cowey and Roberts 1978).

Vitamins

Vitamins are micro-nutrients required for normal growth, reproduction, health and maintenance of fish metabolism. The requirements of fish depend upon the intake of other nutrients, size of the fish, and environmental stresses. Four fat-soluble and eleven water-soluble vitamins are known to be required by the fish and the roles and functions of individual vitamins have been described (NRC 1981 and 1977). Recommended dietary levels and deficiency signs are summarized in Tables 1 and 2. In the early days of fish culture, the most common nutritional deficiencies were those associated with vitamins. However, most of today's practical diets contain sufficient quantities of vitamins. In spite of the addition of excess amounts of vitamins to most fish diets, vitamin deficiency disorders still occur in fish culture. The primary reasons are related to improper manufacturing, handling, or storage of fish feed. Vitamins are very susceptible to destruction by oxidation in the presence of excessive moisture, heat, and trace minerals, particularly if rancid fat is present.

Many of the vitamin deficiency signs are non-specific (Table 2) and it is difficult and costly to analyze for most of the vitamins. Therefore, the diagnosis of vitamin deficiencies is usually accomplished by a process of eliminating other causes and reviewing information on the diet formula used, the level of supplementation of vitamins and minerals, and the manufacturing and storage conditions.

Table 2: Nutritional Deficiency Signs in Finfish

Code	Signs	Possible Nutrient Deficiencies
01	Anemia	Folic acid, inositol, niacin, pyridoxine, riboflavin, rancid, fat, vitamins B12, C, E&K
02	Anorexia (poor appetite)	Biotin, folic acid, inosito, niacin, pantothenic acid, pyridoxine, riboflavin, thiamine vitamins A, B12 and C
03	Ascites	Vitamins A, C & E
04	Ataxia	Pyridoxine, pantothenic acid, riboflavin
05	Atrophy, gills	Pantothenic acid
06	Atrophy, muscle.	Biotin, thiamine
07	Calcinosis, renal.	Magnesium
08	Cartilage abnormality.	Vitamin C, tryptophan
09	Cataract	Methionine, riboflavin, thiamine, zinc
10	Ceroid liver.	Rancid fat, vitamin E
11	Cloudy lens.	Methionine, riboflavin, zinc
12	Clubbed gills.	Pantothenic acid
13	Clotting blood, slow.	Vitamin K
14	Coloration, dark skin	Biotin, folic acid, pyridoxine, riboflavin
15	Convulsions	Biotin, pyridoxone, thiamine
16	Decoloration, skin	Fatty acids, thiamine
17	Deformation, bone.	Phosphorus
18	Deformation, lens	Vitamin A
19	Degeneration, gills.	Biotin
20	Dermatitis.	Pantothenic Acid
21	Diathesis, exudative.	Selenium
22	Disease resistance, low	Protein, vitamin C
23	Distended stomach	Inositol
24	Dystrophy, muscular	Selenium, vitamin E
25	Edema.	Niacin, pyridoxine, thiamine, vitamins A & E
26	Epicarditis.	Vitamin E
27	Equilibrium loss	Pyridoxine, thiamine
28	Erosion, fin.	Fatty acids, riboflavin, vitamin A, zinc
29	Exophthalmos.	Pyridoxine, vitamin A, C and E
30	Exudated gills.	Pantothenic acid
31	Fatty liver.	Biotin, choline, fatty acids, inositol, vitamin E
32	Feed efficiency, poor	Biotin, calcium, choline, energy, fat, folic acid, inositol, niacin, protein, riboflavin

Code	Signs	Possible Nutrient Deficiencies
33	Fragility, erythrocytes	Biotin, vitamin E
34	Fragility, fin	Folic acid
35	Fragmentation, erythrocytes	Biotin, vitamins B12 and E
36	Gasping, rapid	Pyridoxine
37	Goiter	Iodine
38	Growth, poor	Biotin, calcium, choline, energy, fat, folic acid, inositol, niacin, pantothenic acid, protein, pyridoxine, riboflavin, thiamine, vitamins A, B12, C, D, and E
39	Hematocrit, reduced	Iron, vitamins C and E
40	Hemoglobin, low	Iron, vitamins B12 and C
41	Hemorrhage, eye	Riboflavin, vitamin A
42	Hemorrhage, gill	Vitamin C
43	Hemorrhage, kidney	Choline, vitamins A and C
44	Hemorrhage, liver	Vitamin C
45	Hemorrhage, skin	Niacin, pantothenic acid, riboflavin, vitamins A and C
46	Irritability	Fatty acids, pyridoxine, thiamine
47	Lesion, colon	Biotin, niacin
48	Lesion, eye	Methionine, riboflavin, vitamins A and C, zinc
49	Lesion, skin	Biotin, inositol, niacin, pantothenic acid
50	Lethargy	Folk acid, niacin, pantothenic acid, thiamine, vitamin C
51	Lipoid liver	Fatty acids, rancid fat
52	Lordosis	Vitamin C.
53	Myopathy, cardiac	Essential fatty acids
54	Necrosis, liver	Pantothenic acid
55	Nerve disorder	Pyridoxine, thiamine
56	Pale liver (glycogen)	Highly digestible carbohydrate, biotin
57	Photobia	Niacin, riboflavin
58	Pinhead	Starvation
59	Pigmentation, iris	Riboflavin
60	Prostration	Pantothenic acid, vitamin C

Code	Signs	Possible Nutrient Deficiencies
61	Rigor mortis, rapid	Pyridoxine
62	Scoliosis	Phosphorus, tryptophan, vitamins C and D
63	Shock syndrome	Essential fatty acids
64	Slime, blue	Biotin, pyridoxine
65	Spasm, muscle	Niacin
66	Swimming, erratic	Pyridoxine, pantothenic acid
67	Tetany, white muscle	Niacin, vitamin D
68	Vascularization, cornea	Riboflavin

For more details of all deficiency signs see NRC (1977 and 1981)

Nutritional disorders caused by vitamin deficiencies can impair the utilization of other nutrients, weaken the health of the fish, and lead to disease. It is well known that pantothenic acid deficiency results in nutritional or clubbed gill disease. However, this condition may not be as specific as reported because feeding a diet containing 10 mg pantothenic acid/kg feed (NRC recommends 40 mg/kg) for 5 mg did not produce either the described deficiency signs or growth depression at our laboratory.

Ascorbic acid (Vitamin C) is the most unstable vitamin required in fish diets (Hilton et al. 1977). Therefore, the extent of destruction of ascorbic acid in a feed gives some indication of manufacturing methods and storage conditions.

In addition to losses associated with manufacturing and storage, there is some loss of vitamins due to leaching during the feeding process. However, at least for salmonids, the leaching of vitamins from properly manufactured dry pellets and granules is not a major problem. Most published data on leaching losses were obtained under very artificial conditions.

Nutritional deficiency signs usually develop gradually, and it is difficult to detect clear signs in the early stages. However, the culturist may obtain indirect clues of vitamin deficiency from such signs as poor appetite, reduced weight gain, and poor feed efficiency.

MINERALS

In fish, minerals perform important roles in osmoregulation, intermediary metabolism, and in formation of the skeleton and scales (Lall 1981). Mineral requirements of fish are difficult to study because many minerals are required in only trace amounts and others are absorbed from water in significant quantities through the gills as well as from the diet. It is also very difficult to obtain mineral-free feed ingredients for experimental diets. Most practical diets for salmonids provide the major mineral requirements through fish meal which is also a major source of protein. However, diets which rely heavily on plant protein sources must be supplemented with carefully balanced mineral premixes. The minerals required in finfish diets include calcium, zinc, manganese, cobalt, selenium,

iodone and fluorine. The functions of some of these have been described in detail (NRC 1977 and 1981). The recommended dietary levels of minerals and related deficiency signs are shown in Tables 1 and 2. The potential for toxicity of minerals must also be carefully assessed since fish are very sensitive to excess amounts of minerals.

A recent study by Paterson et al. (1981) demonstrated that an imbalance of dietary minerals in certain diets predisposes Atlantic salmon to bacterial kidney disease under specific environmental conditions. Atlantic salmon fed a diet containing high levels of iodine (4.5 mg/kg feed) and fluorine (4.5 mg/kg feed) showed a low incidence of symptomatic bacterial kidney disease.

TOXINS AND ANTIMETABOLITES

Toxins which may be present in fish feeds include mycotoxins, residues of polychlorinated biphenyls, pesticides, herbicides and other agricultural and industrial chemicals. Mycotoxins are produced by many molds on plant products such as oilseed byproducts (soybean, cotton seed, and peanut meals) and grain byproducts. In particular, allatoxin B1 (at less than 1 ppb) in the diet will produce liver cancer in rainbow trout in one year, and at 8-20 ppb will induce grossly visible hepatomas in 4-6 months (Sinnhuber et al. 1977).

Other toxins and antimetabolites in plant materials are protease inhibitors, hemagglutinins, goitrogens, cyanogens, saponins, and gossypol (NRC 1977 and 1981). However, these compounds can be destroyed or inactivated by proper processing (e.g. heating or chemical treatment). Microbial toxins which are produced by microorganisms associated with feed contamination or spoilage cause bacterial toxicoses. Mycobacteriosis (fish tuberculosis) in Pacific salmon was related to the practice of feeding raw salmon offal to hatchery stocks (Ross et al. 1959). Converting to diets containing pasteurized salmon viscera effectively eliminated the primary source of infection (Hublou et al. 1959).

CONCLUSION

All nutrients required for the well-being and normal growth of the fish must be supplied in formulated diets as available (digestible) nutrients. Otherwise, the fish cannot utilize the nutrients present in the feed ingredients. The formulated diets also must be pelleted and processed in such a manner that they are durable and water stable with a minimum amount of fines. Proper feeding of a quality diet should be considered as a high priority in the daily routine on fish culture stations. Wasted feed depletes oxygen levels, causes gill damage, and supports fungal and bacterial growth, all of which can lead to disease problems. Because it is necessary to transfer dietary nutrients into the fish through a water medium, problems occur which are unknown in terrestrial animal feeding practices. Most of the feeding charts available today are based on meat-meal diets of the past. One must be cautious in applying these tables to modem diets which have higher nutrient densities and availabilities. The main factors influencing feed intake of fish are water temperature, the energy content of the diet, and expected growth. Therefore, an estimation of feed intake needed must be based on these fundamental factors. If a group of fish is not feeding actively or growing as expected, diagnostic work is needed to determine the cause. Lack of appetite or retarded growth are often early signs of stress and disease.

REFERENCES

Castell, J.D., R.O. Sinnhuber, J.H. Wales, and D.J. Lee. 1972. Essential fatty acids in the diet of rainbow trout *(Salmo gairdneri):* growth, feed conversion and some gross deficiency symptoms. J. Nutr. 102: 77-86.

Cho, C.Y. 1981. Effects of dietary protein and fat levels on the heat increment of feeding in rainbow trout. Mini-Symp. Proc. of XII Int. Congress of Nutrition (IUNS), San Diego, CA.

Cowey, C.B. and R.J. Roberts. 1978. Nutritional pathology of teleosts, p. 216-226. In R.J. Roberts (ed.), Fish Pathology. Bailliere Tindall, London. 318 p.

Hilton, J. W., C.Y. Cho, and S.J. Slinger. 1977. Factors affecting the stability of supplemental ascorbic acid in practical trout diets. J. Fish. Res. Board Can. 34: 683-687.

Hublou, W.E, J. Wallis, T.B. McKee, D. K. Law, R. 0. Sinnhuber, and T. C. Yu. 1959. Development of the Oregon pellet diet. Ore. Fish Comm., Res. Briefs, Corvallis, OR. 7: 28-56.

Kloppel, J.M., and G. Post. 1975. Histological alterations in tryptophan- deficient rainbow trout. J. Nutr. 105: 861-866.

Lall, S.P. 1981. Minerals - a Review, *In* Biological aspects of aquaculture - nutrition. Proc. World Conf. Aquacult. Int. Trade Show. Sept. 20-23, Venice, Italy.

Nutrition Research Council. 1977. Nutrient requirements of warmwater fishes. Natl. Acad. Sci., Washington, DC. 78 p.

Nutrition Research Council. 1981. Nutrient requirements of coldwater fishes. Natl. Acad. Sci., Washington, DC. 63 p.

Paterson, W.D., S.P. Lall, and D. Desautels. 1981. Studies on bacterial kidney disease in Atlantic salmon (Salmo *salar)* in Canada. Fish Pathol. 15: 283-292.

Poston, H.A., R.C. Riis, G.L. Rumsey, and H.G. Ketola. 1977. The effect of supplemental dietary amino acids, minerals, and vitamins on salmonids fed cataractogenic diets. Cornell Vet. 67: 472-509.

Ross, A.J., B.J. Earp, and J. W. Wood. 1959. Mycobacterial infections in adult salmon and steelhead trout returning to Columbia River basin and other areas in 1957. U.S. Fish and Wildl. Serv., Spec. Sci. Rep. Fish., No. 332. Washington, DC. 34 p.

Sinnhuber, R.O., J.H. Wales, J.D. Hendricks, G.B. Putnam, J.E. Nixon, and N. E. Pawlowski. 1977. Trout bioassay of mycotoxins. In J. V. Rodricks, C. W. Hesseltine and M.A. Mehlman (eds.), Mycotoxins in human and animal health. Pathodox Publishers, Inc., Park Forest South, IL.

9

GENETICS AND FISH HEALTH

V.A. MUDRAK
Pennsylvania Fish Commission
Bellefontaine, PA

The planned selection of stocks to improve quality and production has been practiced in salmonid hatcheries for decades. However, emphasis in selection programs has been generally placed on physical characteristics such as size, coloration and growth rate, with little recognition of the long term genetic impact that such selection imposes on the stocks. Intensive selection for certain characteristics without a thorough appreciation of genetic principles can result in an eventual loss of fitness of the broodstock and the development of undesirable side effects, thus defeating the original objectives of the selection program.

Many technologies are available for brood stock development to improve the health of cultured stocks, but producers must be aware of the inherent advantages and disadvantages of each method. Such projects are, of necessity, long term with genetic gains only being made among successive generations. However, with careful planning and program control, the application of genetic principles in broodstock development can result in an improvement of fish health.

In selecting for disease resistance, one must be careful to differentiate between resistant and disease-free stocks. Resistant stocks do not develop clinical signs when infected but can be carriers of disease. Consequently, resistant fish can pose a disease risk if they are stocked in geographical areas where the disease is not already present.

BROODSTOCK MANIPULATION

Years of intensive breeding have developed lines of trout that are particularly adapted to fast growth in fish hatchery environments. Historically, selection criteria were aimed at a variety of traits, such as egg size and number, egg viability, fry survival, coloration and growth. Any quality lines that were

produced were then maintained by random mass selection; i.e. pooling the eggs of many selected females and fertilizing those eggs with sperm from many selected males. The resultant progeny would again be evaluated and the selection process repeated again.

Consistent with this approach, it seems logical to expect that disease resistance to be an end result of the natural selection process, and that this characteristic could be accelerated through a selective genetics program. However, refinement of existing methodologies to include the criterion of specific disease control requires sophisticated techniques of evaluation and control. Precautionary measures must be included to minimize inbreeding, and the program must continue to identify and select for fish stocks which display those traits which ensure efficient hatchery operations and/or a product well suited for the resource.

PROCEDURES

Whereas the philosophy of "survival-of-the-fittest" is simple, the establishment of a selective breeding regime which supports a viable fisheries program is inherently difficult. The problem rests with the myriad of performance traits which characterize a successful program. Strain performance indices must be established which describe the most important characteristics for selection, depending on the final use of stocks produced. More importantly, the selection program must recognize that trait evaluations are comprehensive, and should therefore be based upon a long-term ranking effort. The following list describes some strain performance characteristics that are commonly used:

1. Qualities relevant to husbandry
   a. Quality of eggs - volume, number, size, percent hatch.
   b. Fry considerations - percent abnormal to swim-up, percent survival, disease resistance, tolerance to therapeutic treatments.
   c. Fingerling aspects - growth rate, disease resistance, food conversion efficiency, survivability, tolerance to therapeutic treatments.
   d. Adult qualities - growth rates, food conversion efficiency, survivability, disease resistance, crowding tolerance, quality of flesh, tolerance to warm temperatures, size uniformity, aesthetic appearance, longevity, broodstock potential, (early/late spawner; fecundity).
2. Qualities relevant to the resource
   a. Rehabilitation goals - stamina, cover-seeking behavior, survival, temperature tolerance, pH tolerance, natural reproduction potential, predator avoidance, ability to coexist with other species, longevity.
   b. "Put-and-take" goals - aesthetic appearance, (body form; low fin abrasion; markings and coloration), growth rate, fishing mortality (catchability), strong fighting tendencies, quality of flesh.

Implementation of a selective breeding program begins when individual values are assigned to each trait deemed important. These values can be changed at the discretion of those in charge of the breeding program to accommodate hatchery and fishery dynamics. As individual fish lots are evaluated, each is compared to the best performing lot and assigned a relative performance value. A comparative trait performance value can then be developed for each test

lot by multiplying each trait value by the corresponding relative values (Bedell and Gall 1968). The summarization of all trait performance values for each test lot would then constitute its strain performance index. This index value, expressed as a percent, represents the provisional strain value. It should be noted that strains with some very poor performance indices might possess outstanding singular trait performance values.

In a program where there is selection for disease resistance, it would be expected that survival after a challenge by the disease of importance would be assigned a high trait value. Selection could be accomplished by a challenge involving exposure to pre-determined densities of pathogens (Ehlinger 1964) to the test lot, or by rearing the fish in a water supply contaminated by the infectious agent. However, survival is only one of the desirable traits. In the case of diseases where vertical transmission occurs, this approach could lead to development of a carrier state. This is an undesirable situation, especially if disease carriers were to be introduced into new areas where the diseases did not otherwise occur.

Caution must be exercised since one can over-select for one trait at the expense of genetic variability. Spawning should also include at least 60 pairs (see Bedell and Gall 1968; Kincaid 1976a, 1976b; Ryman and Stahl 1980). Continued selection and cross breeding of those fish displaying the highest strain performance indices should result in greater resistance to the disease. When resistance has been established, future breedings should be planned to improve other selected traits without substantially reducing the performance level of any other important trait, and to continue to maintain maximum genetic variability within brood stocks.

HATCHERY MANAGEMENT IMPLICATIONS

The principle of disease control through avoidance continues to be utilized by hatchery managers as a means of circumventing epizootics. The avoidance mechanisms generally used include a mixture of the following: control of the water supply (wells, UV treatment, etc.); control of the fish stocks (disease-free stock, surveillance, eradication); limiting the pathways to infection (segregate downstream culture activities from upstream activities); and disinfection of fish eggs and/or contaminated culture facilities. Such efforts are generally directed towards controlling the influx of any new diseases not normally associated with the station or region, and simultaneously, minimizing the effects of. ubiquitous disease agents.

A difficulty arises in determining which of the disease avoidance mechanisms will pay the biggest dividends. Many program managers start by ensuring a clean hatchery site. They then obtain eggs or fish from hatchery stocks certified to be free of specific pathogens (Loftus 1975). This approach works well if other avoidance mechanisms continue. However, should the culture system become contaminated with a reportable infectious agent, there would be cause for alarm inasmuch as the disease-free stocks could possibly lack genetic resistance to the disease. Moreover, new stocks may be unsuited to the existing environmental conditions (water quality, bacteria, etc.) associated with the fish culture station. Experience has demonstrated that the hatchery manager may experience some devastating mortalities if isolated disease-free stocks are

The dilemma of whether or not to use disease-free stocks is further hampered by the hatchery classification system. Because classification is based upon periodic inspections of hatcheries, it is possible fish health diagnosticians may miss an infection. This speculation is supported by recent authors (Mitchell and Hoffman 1981) who state that "attempts to isolate bacteria and viruses may be futile unless clinical signs of an infection are apparent." Furthermore, in their conclusion they relate that most diagnosticians do not certify fish to be disease free of specific diseases; they only examine the fish and report what they do or do not see. However, the agents of BKD, IPN, and IHN can be isolated by qualified inspectors in the absence of disease signs.

The bottom line of this discussion relates to those alternatives that examine the question: "Where can we go from here?" It has already been established (Dill 1973; Yamamoto and Kilistoff 1979) that disease control by avoidance has considerable potential benefit, as does the use of disease-free stock; but what about the use of selective stock manipulation to develop disease-free or disease-resistant brood lines? Snieszko, as early as 1953, reported that, for the control of viruses in cultured fish, "the long-range approach is the breeding of resistant strains of fish'. Recent literature (Dill 1973; Wolf 1976; McIntyre and Amend 1978) also supports this philosophy; however, the use of avoidance and vaccines may be equally or more effective in the control of diseases.

Moreover, it has been suggested (Ehlinger 1964; Fujihara and Tramel 1968; Wolf 1953; Winter et al. 1980) that the severity of common bacterial diseases can be reduced through controlled selection, wherein the host is challenged by the infectious agent. Therefore, if a specific disease becomes important enough to warrant the effort, a regimented breeding scheme could be established to select for those characteristics which reinforce survival after infection. Examples follow which demonstrate alternative approaches for the control of IPN. Modifications of this approach would be necessary for each disease, depending upon its etiology.

EXAMPLE 1

DEVELOPING IPN DISEASE-FREE BROODSTOCK

RATIONALE

Wolf et al. (1968), reported that the most effective method for controlling IPN epizootics lies in the capability to utilize virus-free broodstock, and to rear resultant fry and fingerlings in IPNV-free water. These brood fish could either be acquired through a tedious selection process or by direct transfer from a virus-free station. This technique suggests that one could recruit broodstock or eggs from an IPNV-free hatchery, and then successfully rear the progeny in virus-free water. Field experience, however, has demonstrated that this practice does not always accomodate the hatchery program. Recruited strains may not be suited for those environmental factors associated with a particular hatchery and severe mortalities may occur. From a production viewpoint, a more difficult approach might be to derive IPNV-free stock from those hatchery strains which already demonstrate desirable performance characteristics. If this is the chosen pathway it follows that the primary objective of such a program must be twofold: first, to

maintain the genetic variability of the strain which characterizes high quality performance; and second, to isolate those hatchery fish which are free of IPNV.

PROCEDURES

The development of IPNV-free broodstock will require a multi-faceted, regimented program. A standard protocol for selective breeding must also be included to maintain the genetic variability of the brood lines. Virological testing must determine which fish are carriers and presumptively IPNV-free fish must then be isolated and maintained in virus-free water. Spawning of single pairs (one male + one female) followed by egg/fry/fingerling isolation within separate but comparable rearing units (and virus-free water) will permit long-term evaluation to confirm that resultant progeny are indeed free of IPNV. Requirements for implementation of such a program are listed below (see Wolf et al. 1968):

1. Tag/mark adult fish and rear in virus-free water.
2. Screen prospective brood fish for IPNV using fecal examination.
3. Eliminate individuals carrying IPNV
4. Screen sex products for IPNV.
5. Eliminate IPNV carriers.
6. Conduct pair matings and maintain each family lot as a separate entity in virus-free water. Be certain to select a minimum number of adults to supply at least 50 pairs of disease-free fish from each hatchery strain (Kincaid 1976a, 1976b; Ryman and Stahl 1980).
7. During egg, fry and fingerling stages, each individually spawned lot must be kept isolated from the other lots (within a virus-free water supply).
8. Each lot should be observed for IPN signs; suspect lots would be screened and eliminated if IPNV is verified.
9. If no signs develop, screening should be conducted 6-8 wk after the start of feeding; those lots confirmed to be virus-free could then by combined to form the initial virus-free complement from the original hatchery strain.

PRECAUTIONARY CONSIDERATIONS

While these principles seem simple and straightforward, practical application could be hampered by the logistics associated with the number of strain replicates required (Falconer 1960; Hynes et al. 1981) and the required number of individual rearing units. Since the progeny are still susceptible to IPN infection, the isolation of virus-free stocks at fish culture stations may be impractical in those regions where IPNV is enzootic. Also there are many strains of IPNV, some of which are more virulent than others. Fish that appear resistant to some strains may be susceptible to the virulent strains. Furthermore, managers should be aware that resistant fish may carry subclinical infections and that their stocking into waters where IPNV is not endemic creates a definite risk of introducing the disease to wild, native stocks.

EXAMPLE 2

DEVELOPING IPN DISEASE-RESISTANT STOCK

RATIONALE

Resistance to viral diseases has been shown to be an heritable characteristic in a number of plants and animals. Correspondingly, comparisons of various strains of fish and IPNV has indicated that IPNV affects some fish more severely than others. Since this observation suggests IPN resistance, a breeding program could be initiated to measure differences in IPN resistance and to assign this performance characteristic a high trait value for use in selection. Continued selection might then develop lines of fish which would demonstrate a high degree of resistance to IPNV.

PROCEDURES

Initial contact with IPNV has been historically catastrophic with sudden and massive mortalities. However, at some fish culture stations which have encountered IPN disease for many years, a disease tolerance has developed to the point where hatchery managers feel they can "live with" the disease. By applying the principles of genetic selection, it seems possible that the natural selection process could be accelerated and improved upon.

Using the premise that IPNV disease normally kills the fastest growing fish, the main selection criterion should be the isolation of the largest surviving individuals since these fish may possess greater resistance to IPNV. While this process might minimize the effects of IPNV, it must be cautioned that the selection will be genetically very restrictive, so a large initial population of individuals must be utilized as future broodstock to maintain an adequate gene pool. A wise approach to this selection process would be to utilize the previous breeding program (Example 1) where performance characteristics are assigned trait values. This would permit the production manager to control the rate of selection by working out a methodology which conforms to the needs of the hatchery/fishery program. Taking this further, it may be possible to segregate non-carrier broodstock so that resultant eggs and fry would be both IPN resistant and free. A procedural outline follows:

1. Establish appropriate selection criteria which conform to management needs; assign a high trait value to those larger individuals that survive an IPN epizootic.
2. Continue to select for the resistance trait as long as other important traits are not appreciably impaired. Water supplies should harbor IPNV carriers.
3. A subgroup of fish which possess little natural resistance to IPNV should be maintained. These could then be used to challenge the developing resistant lines. Moribund fish from the susceptible sublot, which displays symptomatic IPN disease, could be ground and fed to those resistant families in order to intensify their chances of infection and to accelerate the selection process.

4. Maintain accurate and complete reference records for future comparison.
   a) Start with a population of 15,000 swim-up fry. After seven days, reduce the lots to 10,000 feeding fry (this will compensate for early differential mortality rates that occur between strains). Rearing units should be exact replicates with equal flows of virus-contaminated water.
   b) Record daily mortalities, water flow and temperatures, feed and feeding rates, etc.
   c) Summarize and graph the number of survivors at the end of each week.
   d) Summarize and graph the weekly accumulated mortalities, as a percent of those survivors at the beginning of each week.
5. When clinical signs of IPN develop, sample the fish and check for IPNV infection.
6. If no IPN signs develop, sample fish 6-8 wk after the start of feding and check for the presence of IPNV. If these fish are negative, challenge them with IPNV-infected ground fish and recheck them after 2 wk.
7. The rate at which further IPNV resistance develops may eventually diminish to a very low level. At that point in time, and if further selection is deemed appropriate, more sophisticated testing using anti-IPN serum may prove meaningful. Taking this further, it may be possible to segregate non-carrier broodstock so that resultant eggs and fish would be both IPNV resistant and free (see Wolf 1953, 1976).

WILD BROODSTOCK - HATCHERY AND FISHERIES MANAGEMENT IMPLICATIONS

It has been postulated (Hynes et al. 1981) that captive hatchery fish stocks may undergo unintentional detrimental genetic changes as a result of selective breeding, and that subsequently, the planted species may not meet the expectations of a given fisheries management program. Consideration of this domestication effect is especially critical when management objectives include the rehabilitation of depleted recreational or commercial fish stocks. Fisheries managers must, therefore, consider the total needs of the fishery and communicate with aquaculturists so that an integrated approach can be established to maximize the genetic integrity of managed stocks. To accomplish this end, progeny from wild broodstock should be reared within facilities that simulate the constraints associated with natural conditions.

The philosophy of matching the fish to the fishery is not new, and many authors (Allendorf and Phelps 1980; Barns 1972; Donaldson and Menasveta 1961; Flick and Webster 1962, 1964, 1976; Fraser 1981; Greene 1952; Horak 1972; Ihssen and Tai 1974; Krueger and Menzel 1979; Moyle 1969; Ryman and Stahl 1980; and Vincent 1960) have studied differences in performance among wild and domestic stocks. While fishery managers agree that wild stocks could contribute more to the fishery than captive hatchery stocks, the procurement and culture of wild broodstocks may only delay domestication. Because fish culture practices inadvertently result in genetic selection for the hatchery environment, the practice of utilizing truly wild fish stocks, as maintained within

the confines of traditional fish culture facilities, may only be a concept. Nevertheless, stock improvement toward accomplishing this goal would be possible if certain criteria can be accommodated. These are as follows:

1. Methods for identifying genetic changes within fish stocks.
   a) Identify the genetic composition of the original stocks to be preserved.
   b) Determine the changes in the genetic composition of both wild and captive stocks.
2. Methods of identifying phenotypic changes within fish stocks.
   a) Establish methods to differentiate between genotypic and phenotypic changes.
   b) Develop methods to determine whether phenotypic changes are the result of environmental or genetic factors.
3. Establishment of fish culture methods which preserve the genetic integrity of the stocks.
   a) Locate, identify, and characterize available fish stocks. Careful consideration must be given to closely match the genetic make-up of the stock to be used to rehabilitate the fisheries program (Krueger et al. 1981).
   b) Efficient management of specific stocks will require an increased number of highly versatile fish culture stations. These should be located as close as possible to those water bodies that contain the target stock (Hynes et al. 1981).
   c) Random sampling of the founding parents is essential; the sampling must cover the complete range of sizes, ages, spawning times, and spawning sites (Bedell and Gall 1968).
   d) Random mating procedures using a minimum of 60 males and 60 females should be an established protocol (Bedell and Gall 1968; Kincaid 1976a, 1976b; Ryman and Stahl 1980).
   e. Techniques such as rotational line crossing should be established as a protocol to reduce genetic inbreeding (Kincaid 1977).
   f. Periodic infusions of genetic material from wild stocks may be considered as a means of maintaining genetic variability, although this practice carries with it inherent risks of introducing diseases (Hynes et al. 1981).

While fisheries management has been traditionally characterized by typological (no difference between stocks) thinking (Schreck 1979), it is now apparent that administrators are aware of the stock concept (Hynes et al. 1981; Loftus 1976) and are ready to integrate the necessary disciplines into their management schemes. To implement this philosophy, managers must take a more holistic approach whereby elements are comprehensively perceived as dependent variables to some management goal.

Administratively, this effort must start at the top and then permeate throughout the system. Program development should not overemphasize one concept to the point where ideas become so polarized that the original goals may actually be hindered (Kutkuhn 1979). Ideally, the program should be flexible, open-ended, and above all, practical.

CONCLUSION

In summary, today's fish producer has many options available for broodstock development to improve fish health. Depending on what he wants to do with his

fish (e.g. restoration program, put-and-take fishery, direct food market, etc.), he must use discretion and implement those options that best suit his needs. For this reason, no specific recommendations have been provided; rather, a selection of alternative approaches has been offered. The final decision rests in the hands of the manager who must match available resources with those approaches that will most effectively achieve the desired goals.

REFERENCES

Allendorf, ES., and S.R. Phelps. 1980. Loss of genetic variation in hatchery stock of cutthroat trout. Trans. Am. Fish. Soc. 109: 537-543.

Barns, R.A. 1972. A quantitative evaluation of survival to the adult stage and other characteristics of pink salmon (Oncorhynchus gorbuscha) produced by a revised hatchery method which simulates optimal natural conditions. J. Fish. Res. Board Can. 29: 1151-1167.

Bedell, J., and G. A. Gall. 1968. Rainbow trout broodstock selection. Calif. Dep. Fish Game Report. Sacramento, CA. 12 p.

Dill, W.A. (ed.). 1973. Symposium on the major communicable fish diseases in Europe and their control. EIFAC, T17, Suppl. 2, FAO, Rome, Italy. 255 p.

Donaldson, L.R., and D. Menasveta. 1961. Selective breeding of chinook salmon. Trans. Am. Fish. Soc. 90: 160-164.

Ehlinger, N.F. 1964. Selective breeding of trout for resistance to furunculosis. N.Y. Fish Game J. 11: 78-90.

Falconer, D.S. 1960. Introduction to quantitative genetics. Oliver and Boyd, Edinburgh. 365 p.

Flick, W.A., and D.A. Webster. 1962. Problems in sampling wild and domestic stocks of brook trout (Salvelinus fontinalis). Trans. Am. Fish. Soc. 91: 140-144.

Flick, W.A., and D.A. Webster. 1964. Comparative first year survival and production in wild and domestic strains of Brook Trout (Salvelinus fon-*tinalis*). Trans. Am. Fish. Soc. 93: 58-69.

Flick, W. A., and D.A. Webster. 1976. Production of wild, domestic and inter-strain hybrids of brook trout *(Salvelinus fontinalis)* in natural ponds. J. Fish. Res. Board Can. 33: 1525-1539.

Fraser, J.M. 1981. Comparative recoveries of paired plantings of domestic and semi-wild stocks of brook trout in lakes of Algonquin Park, Ontario. Can. J. Fish. Aquat. Sci. 38: 1672-1684.

Fujihara, M.P., and R.L. Tramel. 1968. Columnaris exposure and antibody production in seaward and upstream migrant sockeye salmon. Pac. NW Ann. Rep. for 1967 to the USAEC. 1: 9.16 - 9.21.

Great Lakes Fishery Commission. 1975. Recommendations of the Great Lakes Fishery Commission for the control of fish diseases in the Great Lakes basin: model Great Lakes fish disease control program. GLFDCC recommendations, as approved at GLFC Annual Meeting, 1975. Ann Arbor, MI.

Greene, C.W. 1952. Results from stocking brook trout of wild and hatchery strains at Stillwater Pond. Trans. Am. Fish. Soc. 81: 43-52.

Horak, D. L. 1972. Survival of hatchery-reared rainbow trout *(Salmo gairdneri)* in relation to stamina tunnel ratings. J. Fish. Res. Board Can. 29:

Hynes, J.D., E.H. Brown, Jr., J.H. Helle, N. Ryman, and D.A. Webster. 1981. Guidelines for the culture of fish stocks for resource management. Can. J. Fish. Aquat. Sci. 38: 1867-1876.

Ihssen, I?, and J.S. Tait. 1974. Genetic differences in retention of swimbladder gas between two populations of lake trout *(Salvelinus namaycush).* J. Fish. Res. Board Can. 31: 1351-1354.

Kincaid, H. L. 1976a. Inbreeding in rainbow trout populations *(Salmo gairdneri).* J. Fish. Res. Board Can. 33: 2420-2426.

Kincaid, H. L. 1976b. Effects of inbreeding on rainbow trout populations. Trans. Am. Fish. Soc. 105: 273-280.

Kincaid, H.L. 1977. Rotational line crossing: An approach to the reduction of inbreeding accumulation in trout brood stocks. Prog. Fish-Cult. 39: 179-181.

Krueger, C. C., A. J. Gharrett, T.R. Dehring, and F. W. Allendorf. 1981. Genetic aspects of fisheries rehabilitation programs. Can. J. Fish. Aquat. Sci. 38: 1877-1881.

Krueger, C. C., and B. W. Menzel. 1979. Effects of stocking on genetics of wild brook trout populations. Trans. Am. Fish. Soc. 108: 277-287.

Kutkuhn, J.H. 1979. Great Lakes lake trout: Have we really lost what we are trying to restore? p. 15-20. In Proceedings of the 1979 Wild Trout II Symposium, Yellowstone National Park., Sept. 1979. Trout Unlimited and Federation of Fly Fishermen.

Loftus, K.H. 1976. Science for Canada's fisheries rehabilitation needs. J. Fish. Res. Board Can. 33: 1822-1857.

McIntyre, J. D., and D.F. Amend. 1978. Heritability of tolerance for infectious hematopoietic necrosis in sockeye salmon *(Oncorhynchus* nerka). Trans. Am. Fish. Soc. 107: 305-308.

Mitchell, A. J., and G.L. Hoffman. 1981. Preparation of live channel catfish for shipment to states requiring a health permit. Aquaculture. 7: 28-29.

Moyle, PB. 1969. Comparative behavior of young brook trout of wild and hatchery origin. Prog. Fish-Cult. 31: 51-56.

Ryman, N. and G. Stahl. 1980. Genetic changes in hatchery stocks of brown trout *(Salmo trutta).* Can. J. Fish. Aquat. Sci. 37: 82-87.

Schreck, C.B. 1979. Research perspectives for management of wild steelhead trout, p. 26-31. In Proceedings of the 1979 Wild Trout II Symposium, Yellowstone National Park, Sept. 1979. Trout Unlimited and Federation of Fly Fishermen.

Snieszko, S.E 1953. Virus diseases in fishes: outlook for their treatment and prevention. Prog. Fish Cult. 15: 72-74.

Vincent, R.E. 1960. Some influences of domestication upon three stocks of brook trout (Salvelinus *fontinalis* Mitchill). Trans. Am. Fish. Soc. 89: 35-52.

Winter, G.W., C.B. Schreck, and J.D. McIntyre. 1980. Resistance of different stocks and transferrin genotypes of coho salmon *(Oncorhynchus kisutch)* and steelhead trout *(Salmo gairdneri)* to bacterial kidney disease and vibriosis. Oreg. Fish Comm. Fish. Bull. 77: 795-802.

Wolf, K. 1976. Fish viral diseases in North America, 1971-75, and recent research of the Eastern Fish Disease Laboratory, USA, Fish Pathol. 10: 135-154.

Wolf, K., M.C. Quimby, C.P. Carlson, and G.L. Bullock. 1968. Infectious pancreatic necrosis: selection of virus-free stock from a population of carrier trout. J. Fish. Res. Board Can. 25: 383-391.

Wolf, L.E. 1953. Development of disease resistant strains of fish. Trans. Am. Fish. Soc. 83: 342-349.

Yamamoto, T., and J. Kilistoff. 1979. Infectious pancreatic necrosis virus: quantification of carriers in lake populations during a 6-year period. J. Fish. Res. Board Can. 36: 562-567.

10

STOCKING PRACTICES AND DISEASE CONTROL

ROBERT H. GRIFFITHS
New York Department of Environmental Conservation
Albany, NY

Wild stocks of fish generally are free of epizootic diseases. Outbreaks of serious contagious diseases are normally associated with the intensive culture of fish in a hatchery environment. The release of hatchery reared fish should be programmed to reduce the risk of spreading disease to fish in wild environments.

Discontinuance of stocking diseased fish has resulted in the apparent disappearance of hatchery disease since monitoring failed to detect those pathogens in fish from natural waters. Yamamoto and Kilistoff (1979) and Rosenlund (1977) have shown that planting IPNV-free fish in water previously planted with IPNV-carrier fish, over several years, resulted in a decline in the incidence of IPNV detected in the fish population. Herman (1970) noted that disease management plans should include prevention of contact between pathogen and host. Sonstegard and McDermott (1972) stated that "planting IPNV-infested fish in natural waters creates a potential source of infection and that if egg collections are to be made from wild stock, it is important to maintain fish in the natural environment as free of disease as possible". It is the goal of the Great Lakes Fishery Commission "to restrict the spread of certifiable fish diseases, to contain them within their known geographic range and to strive for their elimination". The several techniques and practices designed to accomplish this goal include encouragement of each member, provincial or state agency, to prevent the release of seriously diseased fish and to prevent the movement of fish infected with certain certifiable disease into or within the Great Lakes basin.

PROCEDURES

The Great Lakes Fish Disease Control Committee recommends that several procedures be adopted as policy by all agencies that might stock fish in the

Great Lakes basin. These procedures include:

1. Avoid the release of diseased fish which are suffering an active epizootic, regardless of the pathogen or parasite involved. These fish could be a potential source of infection through horizontal transmission of disease agents to wild populations. The added stress of loading, transporting and stocking could also lead to high post-stocking mortalities if the fish are carrying disease.
2. Prevent any releases of fish that have been exposed to or are known carriers of a certifiable disease.
3. Hatcheries using surface water supplies usually cannot produce disease-free stocks. Hatcheries with closed water supplies (wells and/or springs) avoid introducing diseases assuming that no fish or eggs from other locations are allowed in the hatchery and eradication can be carried out should a disease be introduced.
4. No matter what the nature of a hatchery water supply, it is recommended that fish never be released in hatchery headwaters. Even if the risk seems remote, it is not advisable. Carrier fish could infect the water supply and continue to reinfect hatchery stocks for as long as they remain in the water supply system.

If agency program requirements prohibit destruction of fish with emergency or certifiable disease, stocking sites should be selected in areas where the disease is already endemic, in remote areas, or in areas not intended to provide wild broodstock. Bacterial kidney disease is not classified as a certifiable disease although it should also be included in these stocking practice guidelines.

REFERENCES

Herman, R. L. 1970. Prevention and control of fish disease in hatcheries, p. 3-15. *In* S.F. Snieszko (ed.) A symposium on diseases of fishes and shellfishes. Am. Fish. Soc., Spec. Publ. 5. Bethesda, MD.

Rosenlund, R.D. 1977. Infectious pancreatic necrosis virus at the Willow Beach National Fish Hatchery, Nevada, in rainbow trout stocked into adjacent Lake Mohave. Fish Health News 6: 1O-13.

Sonstegard, R. A., and L. A. McDermott. 1972. Epidemiological model for passive transfer of IPNV by homoiotherms. Nature. 237: 104-115.

Yamamoto, T. , and J. Kilistoff. 1979. Infectious pancreatic necrosis virus: quantification of carriers in lake populations during a 6-year period. J. Fish. Res. Board Can. 36: 562-567.

11

ROUTINE FISH DISEASE MONITORING

L.L. PETTIJOHN
U.S. Department of the Interior
Fish and Wildlife Service
Leetown, WV

The purpose of a fish disease monitoring program is to obtain information relative to the health status of stocks of fish, and the suitability of conditions under which fish are maintained at a production installation. A well-designed monitoring program should enhance the efficiency of hatchery operations and minimize the impact of fish diseases by providing the following:

1. Information need to plan a fish health program.
2. A quick response to disease outbreaks (diagnosis, therapy, prevention).
3. Reduction of the mortality rate in infected lots of fish by providing diagnosis and subsequent therapy. Food conversion ratios should improve in response to lowered mortality.
4. Information as to whether or not a disease problem is attributable to poor management or is primarily due to the presence of a fish pathogen.
5. Incentives for the adoption of measures to control or prevent the introduction of fish disease agents from outside sources, via the transfer of fish or fish eggs or exposure to other contaminated sources.
6. Enhanced market opportunities by the provision of stock that is free, or relatively free, of specific fish disease agents. The producer can, therefore, supply markets located in geographical areas protected by fish disease regulations.

PERSONNEL AND EQUIPMENT REQUIRED

Ideally, every hatchery should have at least one person trained in basic fish disease diagnosis, therapy, control, and prevention, and in the collection, preservation, and shipment of specimens for laboratory analysis.

At the present time, a number of short courses (1 to 2 wk duration) oriented toward the basics of fish disease problems are conducted by federal, state, and university departments. Attendance at such courses will provide basic fish disease information that will be a valuable asset in planning for and conducting a fish health program.

The procedures involved in the diagnosis of many important fish diseases are neither complicated nor time consuming. The presence of bacterial gill disease, perhaps the most common and debilitating infection of hatchery fish, can usually be detected by an individual with a minimum of instruction and access to a suitable microscope. Prompt, on-site identification of a fish disease permits the immediate application of chemotherapy or remedial measures to control or eradicate the disease. As a result, losses are kept to a minimum.

The following equipment is sufficient for diagnosing most disease situations in a hatchery:

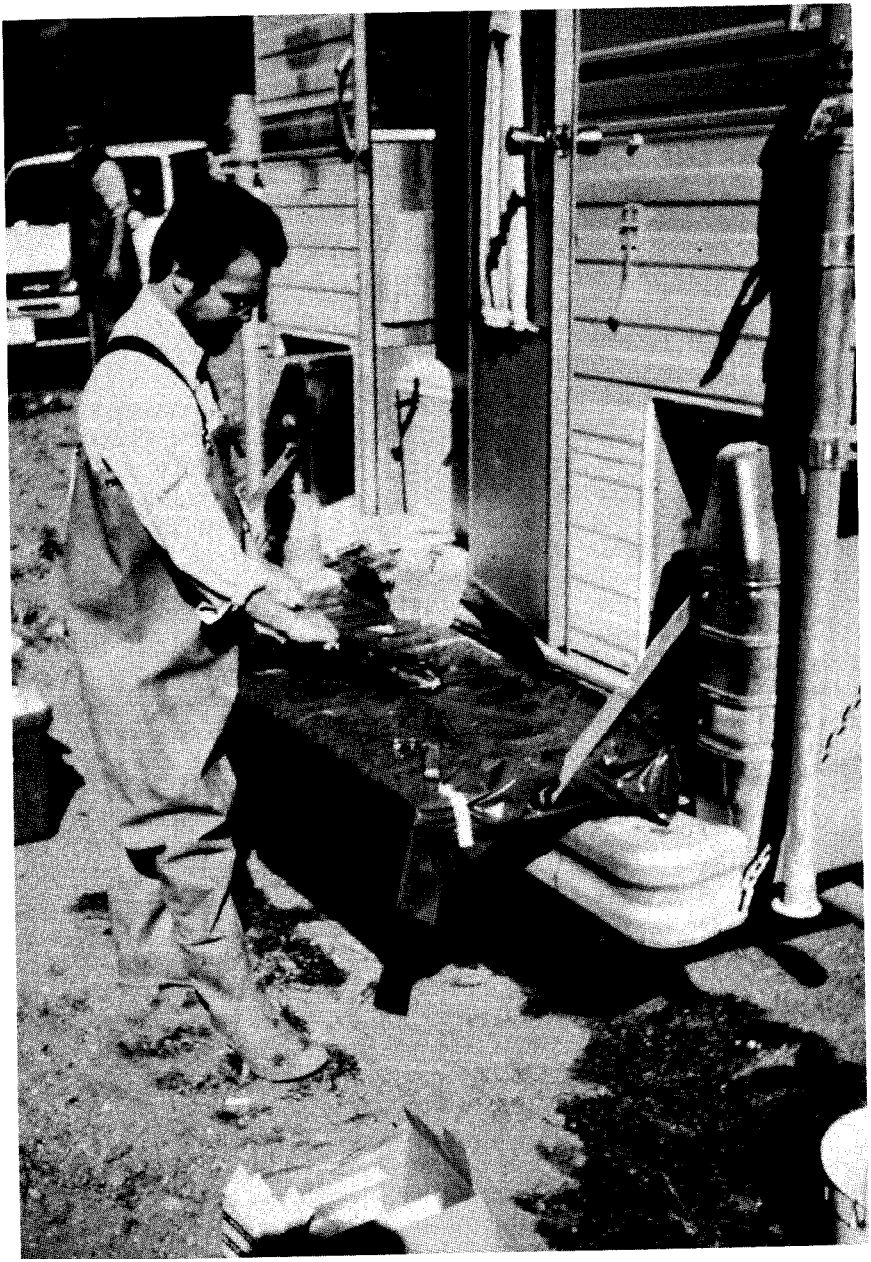
1. Compound microscope (binocular, oil immersion capability, 1000x magnification).
2. Set of dissecting tools.
3. Set of selected bacteriological stains.
4. Microscope slides and cover glasses.
5. Selected bacteriological media.
6. pH meter.
7. Oxygen meter or kit.
8. Bunsen burner or propane torch.
9. Bacteriological inoculating loop.
10. Media sterilizer (pressure cooker or autoclave).

PROCEDURES

Many fish disease problems are seasonal in nature due to factors such as water temperature, spawning, stress, and other conditions. Accordingly, a fish disease monitoring program should encompass the entire year in order to provide information relative to the status of fish health under all phases of production.

Ideally, a hatchery should be inspected by a qualified biologist for the presence of fish disease agents at least twice a year. The survey should be conducted using procedures that will detect the presence of parasitic, viral, and bacterial pathogens. All lots of fish present at the time of the survey should be included. A lot of fish is generally defined as a group of fish of the same age, derived from the same brood stock, and held in a common water supply.

Following determination of the number and location of lots present, a decision must be made concerning the number of fish to be sampled. Considering that a survey is usually conducted to detect the presence of fish disease organisms (in asymptomatic carrier fish) as well as the actual presence of diseased fish, the number of specimens required for sampling is based on a postulated 5% incidence and a 95% probability that carriers or infected specimens will be detected. In general, a 60-fish sample per lot will be required. However, in some situations, such as the presence of valuable brood stock, or other circumstances, it may be necessary to reduce the sample size for particular lots. The usual practice is that the individual in charge of the survey determines the number of specimens required.



A mobile fish disease laboratory for inspecting fish in a multi-hatchery program (British Columbia Min. of Env.)

To be meaningful, a complete survey must be supervised by a qualified biologist who has the expertise and access to laboratory facilities for processing collected samples. The individual conducting the survey must cooperate with personnel of the involved hatchery in pre-planning and in conducting the survey. Depending on circumstances, surveys may be conducted for the presence of specific fish disease organisms only. If conducted according to established procedures, a disease survey should provide all necessary information concerning the present disease status of the production unit surveyed.

MONITORING

One way to develop information on the current fish disease status at a hatchery is to implement a disease monitoring program. A monitoring program should be so scheduled that specimens will be examined at various intervals throughout the year to ensure that fish will be examined under all production conditions and at all ages. A monitoring program can be conducted either by on-site examinations by hatchery personnel or by prior arrangements with a selected laboratory, or a combination of both. Live or preserved specimens, slides of specific tissues, and media inoculated from specific organs can be delivered or forwarded to the laboratory for subsequent examination and evaluation.

Ideally, sample specimens or material should be collected and examined during every month of the year. Care should be exercised in the selection of specimens for examination. In situations where a disease problem is suspect, only those specimens exhibiting symptoms of distress should be selected. Live moribund specimens are preferred but, if necessary, freshly-dead specimens may be collected.

When it is necessary to send specimens away for examination and circumstances prohibit live delivery, specimens may be forwarded by preserving them on wet ice, but not frozen. Specimens for parasitology examinations may be preserved in a 5-10% formalin solution. If the specimens are longer than 7.5 cm (3 in) an incision should be made in the body wall to allow the preservative to reach the internal organs. Specimens should be completely immersed for 24 h in the preservative mixture, using a ratio of at least five volumes of preservative per volume of fish. Preserved samples can then be shipped in plastic bags containing just enough preservative to keep them moist. If possible, include solid materials that collected in the bottom of the original vessel in which fish were preserved. Glass containers should not be employed for shipment because of possible breakage and the hazardous nature of the preservative.

Slides of selected tissues and organs can be made on-site by merely smearing the material on a slide. Slides can then be stained and examined on-site or forwarded for processing and examination. Bacteriological media can be inoculated with material from various organs by employing aseptic techniques. The media may then be incubated on-site for subsequent examination or forwarded to a laboratory for evaluation.

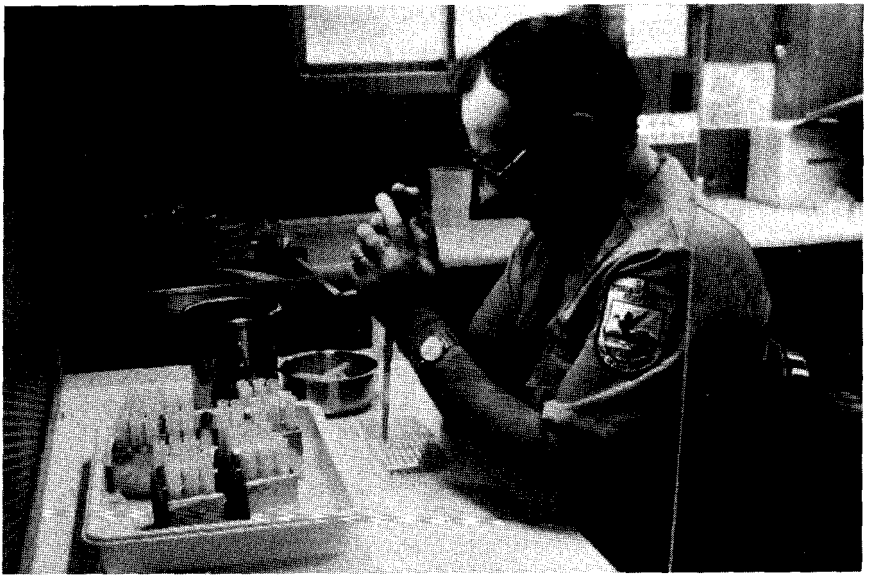
The presence of many common internal and external parasites can be determined on-site if a suitable microscope is available. Wet mounts of gill material, body and fin scrapings, or pieces of internal organs are made by placing a drop of water on a microscope slide, adding the excised material to the slide,

and placing a cover glass over the material. The material may then be examined by microscope at various magnifications.

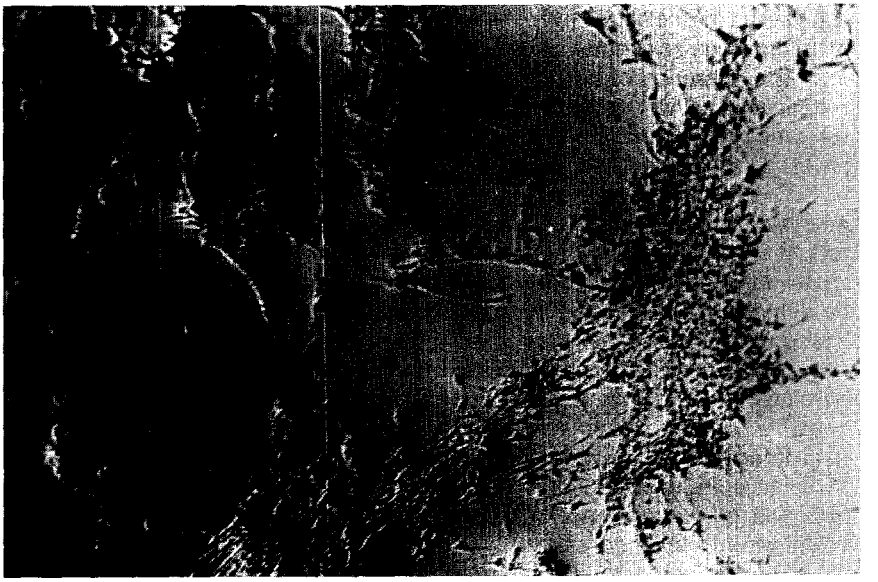
The presence of bacterial gill disease can be detected by a microscopic examination of wet mounts of gill tissue. Suspect gill tissues also should be smeared on a microscope slide, stained with a simple stain, and examined for the presence of the causative myxobacteria.

The presence of a systemic bacterial infection can often be determined by microscopic examination of stained slides prepared from excised material removed from various organs of suspect specimens. The presence of bacterial kidney disease *(Renibacterium salmoninarum),* a Gram-postive bacterium, can be detected and identified by this method. The presence of a Gram-negative bacterium such as *Aeromonas salmonicida,* the causative agent of furunculosis, can also be detected by this method, but no positive diagnosis can be made. Serologic methods are required for the positive identification of most Gram-negative bacteria. Fluorescent antibody techniques have been developed that help determine the presence and identity of bacterial organisms. However, such techniques are usually utilized only in well-equipped laboratories. For the confirmation of the presence of specific organisms, a complete bacteriological examination is still necessary.

The presence and identification of viral agents or viral diseases must be determined at a laboratory that maintains fish tissue cultures and specific antisera. Station personnel can collect the needed specimens and forward them to a laboratory for testing. Whenever specimens are to be submitted for viral testing, the diagnostic laboratory should be contacted inadvance for instructions on sample collection and shipment.



Good laboratory technique, modern equipment, and sound technical training are required for the accurate diagnosis of fish diseases. This fish health specialist is preparing samples to check for the presence of fish virus. (U.S. Fish and Wildl. Serv.)



Tissue cultures are used to check for the presence of fish viruses. The viruses destroy the normal continuous sheet of cells. This photo shows the extensive damage (cytopathic effect) caused by IPN virus (U.S. Fish and Wildl. Serv.)

Certain clinical signs may suggest the presence of viral disease in a particular lot of fish. In situations where an active viral epizootic is suspected, only relatively few specimens (15-20) will be required for examination. The specimens selected for analysis should be moribund and exhibit typical symptoms associated with the suspected disease. In viral infections of fish, usually only small size (2.5-7.5 cm) fish are involved; therefore, whole specimens can be forwarded for examination. Whole fish or kidney and spleen tissues should be packaged in sealed plastic bags, placed on wet ice immediately following collection, and promptly forwarded to the diagnostic laboratory. It is imperative that collected samples be kept on ice prior to testing so careful planning and timing is required in order to expedite shipment delivery and to assure that samples arrive in satisfactory condition.

In addition to the careful selection of specimens or tissues, any pertinent information such as clincial signs, abnormal behavior, stress factors, lot number, species, mortality rates, water temperatures, loading densities, age, and water chemistry data should be included when shipping materials as they can aid diagnosticians in assessing the disease situation.

When information is desired as to whether or not viral agents are present, all lots maintained at the hatchery must be surveyed. Testing techniques are highly sensitive and can usually detect the presence of viral agents in carrier fish even though there are many situations where viral agents are present but do not give rise to disease. The usual survey procedure involves selecting 60 fish from each lot on hand. The fish are anesthetized and killed, and the kidneys and spleen excised. Excised material from five specimens is pooled in a plastic tube containing normal saline solution. Thus, there will be 12 tubes representing the

material excised from one lot. In situations where it is necessary to use 60 fish from a lot, e.g. valuable broodstock and selected stocks of fish, or where it is not practical to sacrifice 60 specimens, ovarian fluid samples may be collected for survey purposes. This practice should be used only when necessary because ovarian fluid is not as reliable a source of viral agents as the kidneys or spleen. The decision about number of specimens to be sampled to assure that adequate and reliable information is obtained is generally left to the judgment of the individual conducting the survey. Certification requirements may dictate the minimum numbers of fish allowed during testing.

NUTRITIONAL DISORDERS

Nutritional disorders, in general, are not considered to be disease problems. However, nutrition plays an important role in the prevention of disease by providing essential nutrients. Nutritional disorders can result in significant losses of fish stock, can be the cause of poor conversion rates, and can have a profound effect upon a stations production capability and quality of the fish produced. Certain parameters provide useful information for monitoring or evaluating the condition of fish stocks in relation to their diet. Parameters such as eye condition, excess fat, body configuration, anemia, sluggishness, abnormal coloration, fin erosion, nervousness, pale liver coloration, and poor conversion rates are valuable indicators in the surveillance for nutritional disorders.

Problems of a nutritional nature do not, for the most part, lend themselves to easy solution and may require an in-depth investigation, In the event that nutritional disorders are suspected and the immediate cause of factors involved cannot be readily determined, it is advisable to consult trained individuals or laboratories familiar with fish pathology and fish nutrition. The resolution of nutritional problems often involves diet tests or trials, designed to provide specific information and may require several years before results can be fully evaluated. Depending upon the nature of the problem, the cooperation of fish food manufacturers, biologists, and laboratories may be required to determine the nature of the problem. In many instances, a histological examination of vital organs is necessary for obtaining information concerning the nature and origin of observed tissue changes.

ENVIRONMENTAL CONDITIONS

Environmental conditions under which stocks are maintained can have a profound effect on the well-being of fish. A complete monitoring program should include tests to provide information concerning characteristics of the environment. Routine tests for oxygen, ammonia, nitrogen and other dissolved gases, temperature, and pH will provide necessary and valuable information that may prevent unfavorable environmental conditions. Additional testing may be required in problem situations.

Monitoring environmental conditions can alert hatchery personnel to the presence of stress factors that may cause mortality or give rise to serious disease problems. Water chemistry kits that employ “cook book” techniques will provide relatively accurate information. Such kits permit hatchery personnel to conduct on-site tests that will provide data concerning critical environmental conditions.

An instrument known as the Weiss gas saturometer can be employed to determine whether or not gas supersaturation exists. Water supplies that are supersaturated can give rise to a relatively common condition known as "gas bubble disease". The condition can arise when dissolved gas pressures exceed atmospheric pressures. When a state of supersaturation exists, gas bubbles form in fish tissues causing conditions which are similar to the "bends" in deep sea diving. The Weiss saturometer measures the sum total of all dissolved gases and provides useful information within minutes. The instrument is portable, designed for field use, and is simple to operate. It can detect low levels of supersaturation which subject fish to stress but do not elicit overt signs of distress. The instrument is also useful for determing whether or not remedial measures to alleviate or reduce gas pressures are actually effective.

RECORD KEEPING

Record keeping is an integral part of any monitoring program and must include data concerning current and past information relative to the status of fish health at the facility and the conditions under which fish stocks are were maintained. The availability of good records provides a valuable reference source for future investigations concerning the health status and well being of fish stocks, and provides data that can be used to evaluate and enhance the efficiency of management procedures. The following data should be included:

1. Diet (amount, size, type, source of food fed each lot or holding unit).
2. Mortality (number daily for each holding unit).
3. Therapy (chemicals and drugs employed, dosage rate, duration of treatment, results).
4. Environment (water volumes, flow rates, water chemistry data, loading densities, etc.).
5. Conversion rates used.
6. Notes concerning observations relative to abnormal behavior, presence of disease, nutritional disorders, stress factors, and other pertinent factors.

SUGGESTED READING

Bullock, G.L., D.A. Conroy, and S.E Snieszko. 1971. Book 2A: Bacterial diseases of fishes. TFH Publications, Inc., Neptune City, NJ. 151 p.

Elliott, D. 1978. Fish disease inspection and certification. Mar. Fish. Rev. 40: 69-71.

Fickeisen, D.H., M.J. Schneider, and J.C. Montgomery. 1975. A comparative evaluation of the Weiss saturometer. Trans. Am. Fish. Soc. 104: 816-820.

Groberg, W. J. 1979. Collection and preparation of samples for viral examination, p. 32-36. In Proceedings from a conference on disease inspection and certification of fish and fish eggs. Oregon State Univ. Sea Grant Coll. Program., Publ. ORESU-W-70-001. Corvallis, OR.

Hedrick, R.P. 1979. Viral diagnosis: isolation and identification, p. 36-38. *In* Proceedings from a conference on disease inspection and certification of fish and fish eggs. Oregon State Univ. Sea Grant Coll. Program. Publ. ORESU-W-70-001. Corvallis, OR.

Hoffman, G. L., and F. P. Meyer. 1974. Parasites of freshwater fishes: a review of their control and treatment. TFH Publications, Inc., Neptune City, NJ. 224 p.

Lannan, C.N. 1979. Diagnosis of parasitic diseases-whirling disease and certatomyxosis, p. 29-31. In Proceedings from a conference on disease inspection and certification of fish and fish eggs. Oregon State Univ. Sea Grant Coll. Program. Publ. ORESU-W-70-001. Corvallis, OR.

McDaniel, D. (ed.). 1979. Proceedings for the detection and identification of certain fish pathogens. Fish Health Sec. Am. Fish. Soc. Bethesda, MD. 118 p.

Ransom, D. 1979. Sampling and necropsy of fish for parasitic and bacterial diseases, p. 23-24. In Proceedings from a conference on disease inspection and certification of fish and fish eggs. Oregon State Univ. Sea Grant Coll. Program. Publ. ORESU-W-70-001. Corvallis, OR.

Snieszko, S. E, editor. 1970. A symposium on diseases of fishes and shellfishes. Am. Fish. Soc., Spec. Publ. 5. Bethesda, MD. 526 p.

Snieszko, S.E 1974. The effects of environmental stress on outbreaks of infectious diseases of fishes. J. Fish Biol. 6: 197-208.

Warren, J. W. 1981. Diseases of hatchery fish -A fish disease manual. U.S. Fish Wildl. Serv., Twin Cities, MN. 91 p.

Winton, J. 1979. The diagnosis and identification of bacterial fish pathogens. p. 25-28. In Proceedings from a conference on disease inspection and certification of fish and fish eggs. Oregon State Univ., Sea Grant Coll. Program. Publ. ORESU-W-70-001. Corvallis, OR.

Wolf, K. 1966. The fish viruses. p. 35-101. In K.M. Smith and M.A. Lauffer (ed.). Advances in virus research. Vol. 12. Academic Press,.New York, NY.

12

CHEMOTHERAPY

R.W. HORNER
Illinois Department of Conservation
Division of Fish and Wildlife Resources
Manito, IL

Chemotherapy is defined as the use of drugs and chemicals for the treatment of infectious disease. To be useful, the chemicals must be effective against the pathogen without significant adverse effects on the fish host.

The first successful chemical was probably salt, used as a dip treatment to reduce pathogens on external surfaces (Rucker 1972). Formalin, a commonly used parasiticide, was first used in 1909 (Schnick 1973). In the U.S., drugs and chemicals used on cultured fishes must be approved by the Food and Drug Administration. Unfortunately, the list of chemicals approved for use in U.S. fish culture is very short. Table 1 lists chemicals approved for use in fish culture.

Therapeutants seldom eradicate a disease. Successful disease control involves a careful program of fish health management that will remove infected stocks, prevent reinfection, reduce stress, and maintain optimal conditions. Therapeutants give the fish only a temporary edge over the pathogens. Unless an effective fish health management program is promptly initiated, disease will reoccur whenever stresses develop making the fish more susceptible. The best treatment of all is good animal husbandry

TABLE 1. COMPOUNDS REGISTERED FOR FISHERY USES (After Schnick, Meyer, and Van Meter, 1979)

Chemicals	Fishery Use	Comments
REGISTERED		
Antimycin	Fish toxicant- 5-10 ppb (ug/l)	Nonfood fish use only
Bayer 73	Lampricide- Rate not to exceed 2% of TFM applied; as a sampling tool, 100 lb/A(112 kg/ha)	Nonfood fish use only; restricted to use by Great Lakes Fishery Commission (GLFC), Federal, or State personnel
Calcium hypochlorite (HTH)	Disinfectant - 5-10 ppm (mg/l) for 12-24 h for control of algae and bacteria; 200 ppm (mg/l) for 1 h to sanitize	Food fish use
Casoron (Dichlobenil)	Herbicide- 7-15 lb/A(7.8-16.8 kg/ha)	Nonfood fish use only
Copper (from basic carbonate)	Herbicide- 14 lb/150-250 gal water/A (15.7 kg/l, 403-2338 l/ha)	Food fish use
Copper sulfate	Algicide- Rate dependent on water chemistry; 2.3-4 ppm (mg/l)	Food fish use
2,4-D	Herbicide- 6-40 lb/A(6.7-45 kg/ha)	Food fish use
Dichlone	Algicide- 0.055 ppm (mg/l)	Nonfood fish use only

Chemicals	Fishery Use	Comments
Diquat	Herbicide and algicide-5.4 ppm (mg/l) in water for submerged weeds; 3 lb/A (3.4 kg/ha) for floating weeds	Food fish use
Endothall	Herbicide 6.8-9.5 lb/A ft (2.5-3.5) g/m3)	Food fish use
Fenac	Herbicide 15-19.5 lb/A (16.8-21.9 kg/ha)	Nonfood fish use only
Fluorescein sodium	Dye to check water flows or dilution-O. 1 ppm (mg/l)	Food fish use; exempted from registration
Furanace	Antibacterial drug for myxobacteria-O. 05-O. 1 ppm (mg/l) for an indefinite period; 1.0 ppm (mg/l) for 5-10 min	Nonfood fish use only
Lime	Pond sterilant-1338 lb/A (1165 kg/ha) of quick lime; 1784 lb/A (2002 kg/ha) of slaked lime	Food fish use; generally regarded as safe (GRAS)
Masoten	Parasiticide for copepods-0.25 ppm (mg/l) active ingredient	Nonfood fish use
MS-222	Anesthetic-15-66 ppm (mg/l) for 6-48 h for sedation; 50-330 ppm (mg/l) for l-40 min for anesthesia	Food fish use, but 21-day withdrawal after use
Potassium permanganate	Oxidizer and detoxifier-2 ppm (mg/l)	Food fish use; exempted from registration
Rhodamine B	Dye to check water flows or dilution rates-20 ppb (ug/l)	Food fish use; exempted from registration

Chemicals	Fishery Use	Comments
Rotenone	Fish toxicant- 1-5 ppm (mg/l)	Nonfood fish use only
salt	Osmoregulatory enhancer- 0.5-1% for indefinite period; 3% for 1O-30 min	Food fish use; GRAS
Silvex	Herbicide- Granular, 40 lb/A (45 kg/ha); liquid, 6 lb/A ft (2.2 g/m^3) for submerged weeds; 8 lb/A (9 kg/ha) for emerged weeds	Nonfood fish use only
Simazine	Herbicide and algicide- 1.4-6.8 lb/A (0.5-2.5 g/ma)	Food fish use
Sulfamerazine	Antibacterial against furunculosis-10 g/100 lb (22 g/100 kg) of fish per day for 14 days in feed	Food fish use in salmonids
Terramycin (oxytetracycline)	Antibacterial against *Aeromonas* and *Pseudomonas*- 2.5-3.75 g/100 lb (5.5-8.25 g/100 kg) of fish per day for 10 days in feed	Food fish use
TFM	Lampricide- l-10 ppm (mg/l), depending on water quality	Nonfood fish use only; restricted to GLFC, Federal, or State personnel
TFM: Bayer 73	Lampricide- 98 TFM: 2 Bayer 73, TFM at l-10 ppm (mg/l)	Nonfood fish use only; restricted to GLFC, Federal of State personnel
Xylene	Herbicide- 100 gal/A (935 l/ha)	Nonfood fish use only

Chemicals	Fishery Use	Comments
STATUS INDEFINITE		
Acetic acid	Parasiticide	Declared as GRAS by FDA as feed additive; not labeled for fishery use
Carbon dioxide	Anesthetic	Declared as GRAS by FDA as feed additive; not labeled for fishery use
Formalin	Parasiticide- 25 ppm (mg/l) in ponds; up to 250 ppm (mg/l) for lh in tanks and raceways	All requirements completed; final action by FDA completed
Sodium bicarbonate	Anesthetic	Declared GRAS by FDA as feed additive; not labled for fishery use

coupled with avoidance of pathogens. If an animal is given a good environment and adequate nutrition, the risk of infection by any pathogen is greatly reduced.

Drugs and chemicals are often used to correct errors in management. While this practice can be used as a stop-gap, it cannot be used to prop up poor culture programs. Sound husbandry is the best approach to disease control. Indiscriminant uses of therapeutic agents should be avoided.

Questionable practices include the continuous feeding at low levels of antibiotics in the diet as a proyhylactic measure against outbreaks of bacterial disease during periods of stress, or to improve growth rates. Indiscriminant feeding of low levels of antibiotics will remove only those bacteria most sensitive to the drug and can lead to the development of drug resistant strains. Drug resistant bacteria can transmit this resistance to different species of bacteria that have never been exposed to the drug (Burrows 1973). Therefore, treatment with antibiotics is recommended only when needed, and then only at prescribed treatment levels. If a decision to use antibiotics is made, treatment should be conducted for the full time period required. Shorter treatments will also encourage the development of drug resistance and can lead to the need for elevated drug levels, and eventually, to loss of

effectiveness. Information on drug resistence is provided by Shotts et al. (1976a, 1976b).

The casual use of therapeutics on a routine basis is not without possible adverse effects on the general health of the fish and is not recommended. Whenever possible, seek a positive diagnosis of any disease problem by a professional fish health specialist. Avoid "shotgun" approaches in which "hit or miss" techniques are used. Start treatment with the correct drug at the recommended level of treatment. If it is determined that a chemotherapeutant is needed, treat quickly and effectively. Users are advised to proceed with caution and to follow label directions. Recommended rates of treatment are based on the levels that researches have found to be necessary and that various fishes will tolerate. Although there is a built-in safety factor, using more than the recommended rate is not necessarily better, may be harmful, and may be illegal.

Culturists who rear anadromous fish must also exercise care when treating fingerlings at the time of smoltification. It has been shown, for example, that salmon smolts treated with diquat for bacterial gill disease just before they are released have difficulty initiating salt excretion from the gills. As a result, the smolts die when they enter salt water or may remain in the estuary and never go to sea (G.A. Wedemeyer, USFWS, Seattle, WA personal communication). Wedemeyer et al. (1980) recommend a 2-wk withdrawal from all chemotherapeutic treatments before the intended release date of smolting salmon. Table 2 shows the effects of 12 other drugs and chemicals commonly used in fish culture on the salt pump of coho salmon smolts (Bouck and Johnson 1979).

Lastly, time requirements (identified on container labels) for clearance of drug residues from treated fish should be strictly observed. If fish are being reared for human food, sale of the carcasses may not be allowed if drug residues are found in the fish.

"Treatment Tips" by Fred P. Meyer (1968) is a useful guide for calculating dosage levels for treatments. Metric conversion tables are included.

GENERAL TREATMENT GUIDELINES

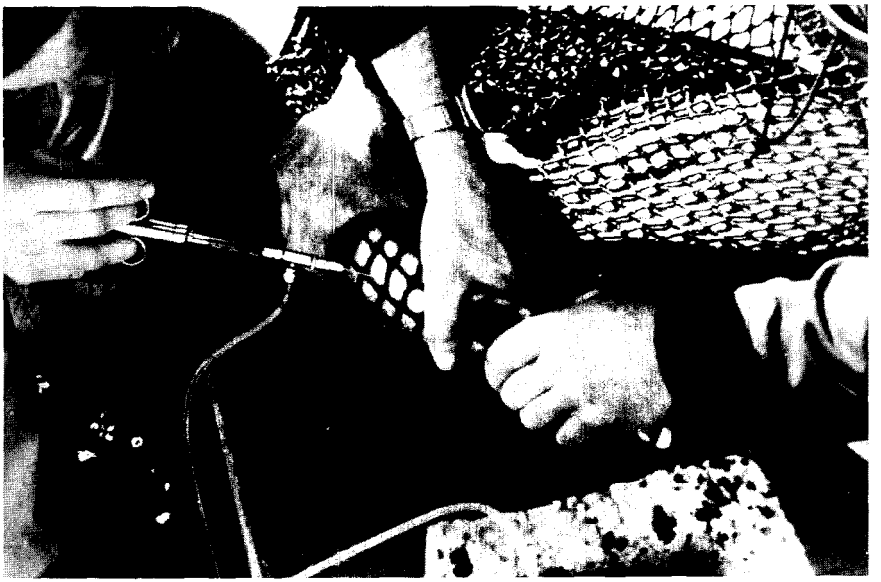
Before applying any chemotherapeutic treatment, ensure that information on chemical characteristics of the water supply is available, and ascertain how they will affect the toxicity and efficacy of the treatment. What will work at one place may not be effective elsewhere because of differences in water chemistry. Before using any chemical, be sure to test it first on a small number of sick fish. Keep in mind that healthy fish can tolerate chemical treatment more readily than sick fish and that treatment levels may need to be reduced if the fish are weak or in poor condition.

Table 2. Summary of seawater survival of coho salmon smolts following standard treatments with drugs and chemicals commonly used in fish culture (Bouck and Johnson 1979).

	Treatment			Total mortality (%) during 10d in seawater -	
	Concentration of active ingredients (mg/l)	Length of daily exposure (min)	Consecutive days treatment was given	Direct transfer into seawater following treatment	Treatment, 4d in fresh water, then 4h acclimation to seawater
Controls				0	0
Copper sulfate	37	20	1	100	20
Endothal	5	60	1	100	4
Formalin	167	60	1	12	0
Hyamine 1622	2	60	4	68	4
Malachite green	1	60	1	44	12
MS-222	100	100	6	1	100
Nifurpirinol	1.5	60	4	8	0
Oxytetracycline	1	60	1	20	12
Potassium permanganate	2	60	3	80	12
Quinaldine	2.5	10	1	0	0
Simazine	2.5	60	1	4	0
Trichlorofon	0.5	60	1	0	0

Poupard (1978) established a set of guidelines of things to do before, during, and after treatment. These guidelines are:

1. Ensure that rearing facilities are clean before treating. Dirty raceways or tanks may contain organic matter that will absorb part of the treatment chemical and reduce its effectiveness.
2. If the fish density is excessive, it should be reduced, if possible, prior to static treatment. Supplemental aeration should be provided, if needed.
3. During hot weather, treatments should be made during the coolest part of the day using chemicals that create the least environmental hazard or stress.
4. Starving fish l-2 d prior to treatment will reduce their oxygen consumption, will reduce ammonia production, and will increase resistance to scale loss. Never treat within 4 h after feeding.
5. Always observe fish during treatment to watch for signs of stress or unexpected toxicity.
6. Any parasitism of the gills should be treated first since such parasites may affect respiratory capability of the fish.
7. Monitor dissolved oxygen levels before and during treatment. Fish are stressed during treatment and increase their need for oxygen.
8. Before treating with a new compound or formulation or using a product for the first time on an installation, always treat a small group of fish first and watch for unexpected mortality.
9. Always check your calculations (0.1X will be ineffective; 1.0 is effective; but 10X will be fatal). If possible, have someone check your figures.
10. Keep records of all treatments, their purpose, and the results for future reference.



Antibiotics and biologics can be administered to large fish through the use of an automatic syringe (U.S. Fish and Wildl. Serv.)

TREATMENT IN THE DIET

If available, commercial feed with antibiotic additives is cheap and easy to use. Medicated feed stores well and can be used in place of the regular diet.

If commercially medicated feed is not available, medicated feed can be prepared on site. Antibiotics, such as oxytetracycline (Terramycin), which are water soluble may leach out of the feed unless preventive steps are taken. It is best to suspend such drugs in oil when preparing medicated feed (cod liver oil seems to have better palatability than soy bean or corn oils, but any of these will do). The daily ration of feed can then be coated with the oil/ antibiotic mixture while the pellets are tumbled in a small cement mixer.

Once treatment has been started, keep rigidly to the recommended dose and treatment schedule. Do not try to save money stopping treatment when mortalities stop, by using lesser amounts, or by treating for shorter periods (Johnson 1971).

LOCALIZED APPLICATIONS

EXTERNAL

Localized skin applications are feasible only for broodstock and other valuable fish. Herwig (1979) recommends that the drug used should be relatively insoluble in water, act on contact, and either be denser than water or so adhesive that the fish can't rub it off.

INTERNAL

For small numbers of valuable fish, injections of antibiotics can be used but may be very expensive and labor intensive.

Intraperitoneal injection are superior to subcutaneous or intramuscular injections. If possible, the fish should be immobilized by holding it in the web of a large mesh net. If struggling causes scale losses or injury, or if the fish are simply too large to handle, it may be best to anesthetize the animals. Both MS-222 (tricaine methanesulfonate) and carbon dioxide have been used with success for this purpose. Injections of drugs require small syringes and use a 20 to 26 gauge needle depending on the size of the fish. Insert the needle through the body wall just posterior to the pelvic girdle at a shallow angle until resistance suddenly ceases. Avoid puncturing the intestine or gonads by inserting the needle too far (Herwig 1979).

BATH/DIP TREATMENTS

Dipping the fish involves a short bath treatment with a duration varying from only a few seconds to 5 min, depending on the chemical and concentration used. Leitritz and Lewis (1976) recommend the use of wood tubs but plastic avoids possible chemical reactions between galvanized metal and treatment chemicals, which may be toxic to the fish.

Dip treatments are often used on broodstock. They are effective but can be highly stressful. After treatment, the fish should be rinsed in clean water before they are returned to the holding facility to avoid transfer of chemical to the tank (Poupard 1978). Treated fish should be placed in water that is free of parasites or pathogens (Leitritz and Lewis 1976).

SHORT BATHS

For treatments up to 1 h, when fish are held in facilities where fresh water is available and adequate oxygen levels can be maintained, short baths are useful because high concentrations of chemicals can be used. Extreme care is required to avoid chemical overdoses or overly long contact times.

INDEFINITE TREATMENTS

This method is suitable only for treating ponds. Low concentrations of chemical are used and allowed to dissipate in the pond. Treatments may have adverse effects on the biota or on dissolved oxygen levels. The degradation of formalin uses 1 ppm oxygen for each 5 ppm formalin as it decomposes. Formalin is also algicidal and can lead to depressed dissolved oxygen levels due to loss of photosynthetic activity and cellular decay. Pond applications require a boat and motor. A boat bailer can be used to pump the chemical into the water.

FLUSH TREATMENTS

In treatments of this type, a measured amount of concentrated chemical is added at the inlet and allowed to flush through a pond or raceway. Amounts

of chemical used must be accurately determined for your hatchery. Lowering the water level in the holding unit can be used to reduce the amount of chemical needed and also facilitates rapid dilution of the treatment when fresh water is added to restore normal levels (Poupard 1978). This technique is also useful when using indefinite treatments in ponds (see above).

CONSTANT FLOW

In constant flow treatments, the chemical is metered into the water inflow at a constant rate to maintain a given concentration for a given period of time. This treatment method requires accuracy and is expensive in terms of the amount of chemical needed. The method requires no special attention to oxygen or ammonia levels, since the water flow remains unchanged. Application equipment may vary from a controlled drip bottle to a 135 liter (30 gal.) plastic garbage can with a siphon, depending on the amount of chemical required.

REFERENCES

Bouck, G. R., and D.A. Johnson. 1979. Medication inhibits tolerance to seawater in coho salmon smolts. Trans. Am. Fish. Soc. 108: 63-66.

Burrows, W. 1973. Textbook of microbiology. p. 204-206. W. B. Saunders Co., Philadelphia, PA.

Her-wig, N. 1979. Handbook of drugs and chemicals used in the treatment of fish diseases. Charles C. Thomas Publications, Springfield, IL. 272 p.

Johnson, S.K. 1971. Oral administration of antibiotics to fishes. Tex. A&M Univ., Agric. Ext. Serv., Fact Sheet L-1018, College Sta., TX. 4 p.

Leitritz, E., and R.C. Lewis. 1976. Trout and salmon culture. Calif. Dep. Fish Game, Fish. Bull. 164, Long Beach, CA. 197 p.

Meyer, EI? 1968. Treatment tips. U.S. Fish and Wildl. Serv., Fish Farming Expt. Sta., Stuttgart, AR. 17 p.

Poupard, C.J. 1978. Therapy of fish diseases, p. 269-275. In R.J. Roberts (ed.) Fish Pathology. Bailliere Tindall, London. 218 p.

Rucker, R.R. 1972. Fish disease therapy: past, present and future, p. 135-140. In R. W. Moore (ed.) Progress in fishery and food science. Univ. Wash., Seattle, WA.

Schnick, R.A. 1973. Formalin as a therapeutant in fish culture. U.S. Fish Wildl. Serv., La Crosse, WI. NTIS No. PB-237 198. 145 p.

Schnick, R.A. and EF! Meyer. 1978. Registration of thirty-three fishery chemicals: status of research and estimated costs of required contract studies. U.S. Fish Wildl. Serv., Invest. Fish Cont. No. 86. 19 p.

Schnick, R.A., EI? Meyer, and H.D. Van Meter. 1979. Announcement of compounds registered for fishery uses. Prog. Fish-Cult. 41: 36-37.

Shotts, E.B. (Jr.), V. L. Vanderwork, and L.M. Campbell. 1976a. Occurrence of R factors associated with *Aeromonas hydrophila* isolates from aquarium fish and waters. J. Fish. Res. Board Can. 33: 736-740.

Shotts, E.B. (Jr.), V.L. Vanderwork and W. J. Long. 1976b. Incidence of R factor associated with *Aeromonas hydrophila* complex isolated from aquarium fish. p. 143-151. In L.A. Page (ed.) Wildlife diseases. Plenum Publishing Corp. New York, NY.

Wedemeyer, G.A., R.L. Saunders, and W.C. Clarke. 1980. Environmental factors affecting smoltification and early marine survival of anadromous salmonids. Marine Fish. Rev. 42: 1-14.

13

IMMUNIZATION WITH VACCINES

B.W. SOUTER
Department of Fisheries and Oceans
Fisheries Research Branch
Winnipeg, Man.

World demand for high quality protein foods has stimulated a rapid development of intensive fish culture techniques. Inherent with the intensive rearing of fish are related problems of high loading densities, declining water quality, adequate diets, handling, and disease control. Man has only recently recognized the threat imposed by disease and its limitation on economic development of the aquaculture industry (Roberts 1978).

When confronted with a disease problem, the producer has had essentially only two expensive and potentially devastating options at his disposal: a) antibiotic prophylaxis and treatment of fish (which could affect palatability of food and may result in the development of resistant strains of bacteria); and b) destruction of all fish at the station followed by thorough hatchery disinfection and reintroduction of disease-free stock. Both the producer and the fish health specialist appreciate that while some chemotherapeutants for controlling disease have proved very successful, the availability of registered drugs and chemotherapeutics is decreasing.

In recent years, a new technique for the prevention of fish diseases is rapidly emerging as a result of research into the development of fish vaccines. Fish immunology has a more recent history than human and veterinary immunology but the techniques used are similar. However, methods of administering vaccines to fish differ and are dependent upon species, pathogen, temperature, and environment (Anderson 1974).

Immunity is an important physiological mechanism in animals for protection against infectious disease agents and the maintenance of internal homeostasis (Ingram 1979). Work by Duff (1942) involving the oral immunization of cutthroat trout against furunculosis provided the first evidence that fish possess an immune

response system. Subsequent oral immunization studies by Post (1963), Krantz et al. (1963), Ross and Klontz (1965), Spence et al. (1965) and Klontz and Anderson (1970), using various salmonid species, substantiated Duffs earlier work.

In very general terms, fish are protected from infectious diseases by non-specific barriers such as the mucus, scales and epidermis (Anderson 1974); by non-specific factors which include complement, interferon, and lysozymes (Anderson 1974; Ellis 1978; Ingram 1979); and by specific defense mechanisms such as antibody production (Anderson 1974; Ellis 1978; Ingram 1979). An antibody is a specific immunoglobulin (modified protein) produced in response to and that reacts specifically with an antigen. An antigen is any foreign substance which is capable, under appropriate conditions, of stimulating the formation of antibodies and reacting with the produced antibodies, in a detectable manner (Davis et al. 1967). Vaccines or bacterins contain antigens that are generally attenuated or killed disease agents which, when administered to a host, stimulate the production of specific antibodies or non-specific resistance to that particular disease agent. The protection conferred to an immunized animal by the production of antibodies and other factors will enhance its chances of survival when subjected to a natural challenge by the pathogen.

Intensive research into vaccine development commenced in the early 1970's and has resulted in production of bacterins for three salmonid diseases: enteric redmouth *(Yersinia ruekeri),* vibriosis *(Vibrio anguillarum)* and furunculosis *(Aeromonas salmonicida).* All three bacterins have been licensed by the United States Department of Agriculture/Animal and Plant Health Inspection Service (U.S.D.A./A.P.H.I.S.) and are commercially available. Vaccines for the salmonid viruses (IPN, IHN, VHS), and a bacterin for bacterial kidney disease *(Renibacterium salmoninarum)* are still in the developmental stages. Biologics for diseases of warmwater fishes, as well as other vertebrates and invertebrates raised in aquaculture, are also being planned (G. Tebbit 1981, Wildlife Vaccines, Wheatridge, CO, personal communication). Ambient water temperature, size and species have a direct effect on the immune response in fish and should always be considered at the time of immunization. Research has demonstrated that fish respond faster immunologically and retain immunity longer in a manner directly proportional to increasing water temperature and fish size (Fender and Amend 1978; Amend and Eshenour 1980).

DELIVERY SYSTEMS

Fish can be vaccinated by a variety of methods. Two delivery systems that have been used successfully to immunize fish include: (a) immersion/spray-shower vaccination and b) vaccine injection.

Immersion/Spray-Shower Vaccination

The immersion method is a fast, efficient and economical way to vaccinate. It is particularly suited to small fish (1-4 g) (Antipa and Amend 1977; Egidius and Anderson 1979; Amend and Eshenour 1980). Spray-shower vaccination is a variation of immersion vaccination designed and recommended for fish larger than 4 g. An added advantage of this system over the immersion method is the

Vaccinating large rainbow trout (1O-15 cms) in the field (British Columbia Min. of Env.)

fact that greater weights of fish can be vaccinated more economically with less effort (Amend and Eshenour 1980). Under optimum conditions, following antigenic stimulation, 2-4 wk are required before protective immunity develops. Therefore, the producer should allow sufficient time for immunization before the first expected outbreak of disease.

PROCEDURES

Commercially available fish vaccines or bacterins should always be used according to the recommendations included with the product. The procedures have been substantiated by the manufacturer and approved by the U.S.D.A./ A.P.H.I.S.

1. Immersion Vaccination

   This procedure involves the following steps:

   a. Determine the amount of vaccine required by referring to the manufacturer's reference chart or labels.

   b. Prepare the vaccine solution based on the total weight of fish to be immunized.

   c. Crowd the fish into a confined area.

   d. Weigh the fish or inventory by displacement, if necessary.

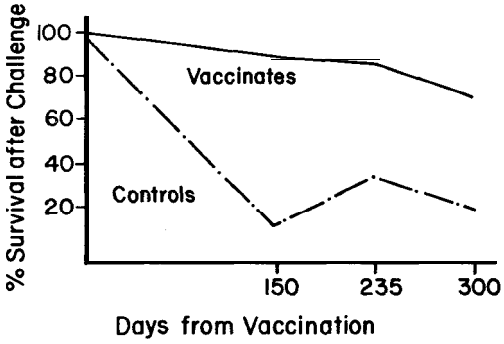
   e. Immerse fish in the vaccine solution.

   f. Expose the fish for the proper length of time.

   g. Return fish to the rearing area.

To insure immunization efficacy, do not exceed the manufacturer's recommended number of immersed lots of fish. When this number has been reached discard the vaccine and prepare a fresh solution.

Fig. I. Graph showing high levels of protective immunity in salmon after vaccination with Wildlife Vaccines' Vibrio anguillurum bacterin.*



Reprinted with permission from Wildlife Vaccines Inc. ERM and Vibrio Bacterin Product Literature.

2. Spray-shower Vaccination
   This involves the following steps:
   a. Prepare the bacterin as recommended by the manufacturer.
   b. Place the bacterin solution in the reservoir of a system calibrated to deliver a prescribed volume of vaccine per minute through a fine spray-shower nozzle.
   c. Place fish in the immersion vaccination unit and allow a contact exposure time of 2-5 s from a distance of 30-50 cm.
   d. Return fish to the holding area.
   e. Discard bacterin solution after recommended use.

DEGREE OF SUCCESS

Field and laboratory studies have reported survival rates of 80-97% in terms of relative percent survival (Table 1, Fig. 1) (Antipa and Amend 1977; Croy and Amend 1977; Sawyer and Stout 1977; Lannan 1978; Gould et al. 1979; Antipa et al. 1980; Amend and Eshenour 1980; and G. Tebbit 1981, Wildlife Vaccines, Wheatridge, CO, personal communication).

Table 1. Documented results from field vaccinations conducted from February 1978 to August 1980.”

	Starting Population	ERM Mortality	Medication Fed-(kg)	Conversion Rates(a)
Vaccinated	22,959,329	299,815 (1.3%)	91,295	1.9
Non-vaccinated	4,272,728	342,642 (8.0%)	74,600	2.2

a) Kg of food fed per kg of fish gained.

*Reprinted with permission from Wildlife Vaccines Inc. ERM and Vibrio Bacterin Product Literature.

Under optimal conditions, fish can retain their immunity for well in excess of 300 days if temperatures are favourable (Amend and Eshenour 1980).

Problems and Precautions

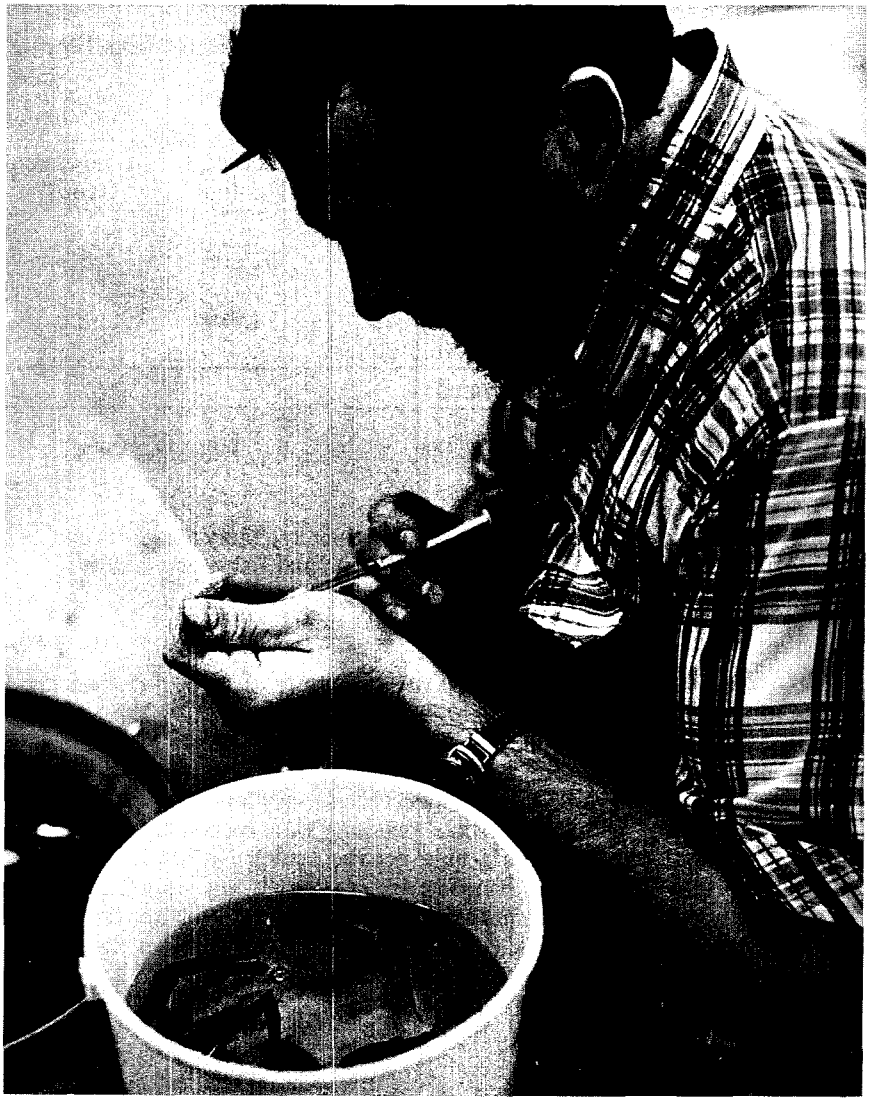
To date, few problems have been encountered if immersion and spray-shower vaccination are administered according to the manufacturer’s recommended procedures. Potential problem areas of which the producer should be aware include: treatment stress, especially when vaccinating fish less than 1 g in weight (greater than 450/lb); improper vaccine dilution; and exceeding the prescribed number of immersed lots resulting in a reduction of vaccination efficacy. Precautions to be taken include: not vaccinating fish during an epizootic; not feeding fish 12 h prior to handling and treatment; not storing the diluted vaccine solution; and not vaccinating fish within 21 d of slaughter because the bacterins contain oxytetracycline hydrochloride as a preservative (Tavolek Inc. 1978, and Wildlife Vaccines Inc., 1981).

Useful Features

Immersion and spray-shower vaccinations provide rapid administration, easy adaptation to different fish culture situations, cost effective treatment, reduced treatment stress, less dependence on fish size at vaccination, and allow the administration of several vaccines in combinations.

Injection

Vaccines also can be administered by injection. Subcutaneous, intraperitoneal, and intramuscular inoculations can be used but intraperitoneal injection is preferred because of the rapid development of protection and ease of administration (Anderson 1974).



Injecting individual fish with vaccine (U. S. Fish and Wildl. Serv.)

Equipment

- Vaccine and diluent (sterile 0.85% saline)
- Automatic Cornwall-type repeating syringe with stainless steel needles
- Nets and seines
- Plastic containers
- Anesthetic
- Balance or scale
- Pencil and paper

Procedure

Vaccines for injection purposes are prepared and standardized to deliver a prescribed amount of antigen per unit-weight of fish. Experienced personnel in this area should be consulted prior to the use of this technique.

Degree of Success

Factors such as handling costs, treatment stresses, processing time, and fish size, limit injection as a method of choice for vaccine administration. However, use of an automatic repeating syringe allows an experienced individual to inject up to 1,000 fish per hour at minimal cost to the producer (Antipa 1976). Studies have shown that intraperitoneal injection provided protection slightly superior to immersion vaccination (W. Paterson 1981, Connaught Labs, Toronto, Ont., personal communication).

Problems and Precautions

Vaccine injection is now recognized as a safe, effective, economical means of vaccinating fish. Some potential problem areas of which the producer should be aware include: excessive treatment stresses which can result in mortalities or predisposition of the fish to infection by ubiquitous, water-borne, opportunistic pathogens; poor inoculation technique which can cause excessive tissue damage at the inoculation site and result in necrosis, infection, and/or internal organ damage and death; and improper vaccine preparation or failure to maintain vaccine in constant suspension resulting in a reduced introduction of antigen and a concomitant low level of protection (Anderson 1974). Precautions to be taken are similar to those for immersion and spray vaccination.

Useful Features

The advantages of injection include the incorporation of adjuvants into the vaccine which enhance the immune response and the option to include multiple antigens that can be injected in combination. Injectable vaccines have been used primarily to protect valuable broodstocks or genetic strains that are limited in numbers. In these cases, injection may prove more feasible than immersion vaccination. Furthermore, intraperitoneal injection may be required for vaccines developed in the future and, as such, the development of advanced methods will overcome disadvantages of the injection delivery system.

A number of other significant benefits have been demonstrated as a direct result of vaccination, including reduced need for antibiotic prophylaxis, faster growth rates, and improved feed conversion (G. Tebbit, Wildlife Vaccines, Wheatridge, CO, personal communication). Although the benefits of fish vaccination are substantial, the hatchery operator should not view vaccination as a panacea or as a substitute for effective husbandry practices, but rather as one of many disease preventative measures available for controlling fish diseases and ultimately maximizing fish survival and monetary return.

REFERENCES

Amend, D.F., and D. C. Fender. 1976. Uptake of bovine serum albumin by rainbow trout from hyperosmotic solutions: a model for vaccinating fish. Science. 192: 793-794.

Amend, D.F., and R. W. Eshenour. 1980. Development and use of commercial fish vaccines. Salmonid. Vol. III, No. 6: 8-12.

Anderson, D.P. 1974. Diseases of Fishes. Book 4: Fish Immunology. TFH Publications, Neptune City, NJ. 239 p.

Antipa, R. 1976. Field testing of injected *Vibrio anguillarum* bacterins in pen-reared Pacific salmon. J. Fish. Res. Board Can. 32: 1291-1296.

Antipa, R., and D. F. Amend. 1977. Immunization of Pacific salmon: comparison of intraperitoneal injection and hyperosmotic infiltration of *Vibrio anguillarum* and Aeromonas *salmonicida* bacterins. J. Fish. Res. Board Can. 34: 203-208.

Antipa, R., *G.* Gould, and D. F. Amend. 1980. *Vibrio anguillarum* vaccination of sockeye salmon, *Oncorhynchus nerka* (Walbaum), by direct and hyperosmotic immersion. J. Wildl. Dis. 3: 161-165.

Croy, TR., and D.F. Amend. 1977. Immunization of sockeye salmon (Oncorhynchus nerka) against vibriosis using the hyperosmotic infiltration technique. Aquaculture. 12: 317-325.

Davis, B.D., R. Dulbecco, H.N. Eisen, H.S. Ginsberg and W.B. Wood, Jr. 1968. Introduction to the immune response, p. 358-364. In Principles of Microbiology and Immunology, Harper and Row, Publishers, New York, NY.

Duff, D.C.B. 1942. The oral immunization of trout against *Bacterium salmonicida.* J. Immunol. 44: 87-94.

Egidius, E. C. and K. Anderson. 1979. Bath immunization - a practical and non-stressing method of vaccinating sea farmed rainbow trout, *Salmogairdneri* Richardson, against vibriosis. J. Fish Dis. 2: 405-410.

Ellis, A.E. 1978. The immunology of teleosts, p. 92-104. *In* R.J. Roberts (ed.). Fish Pathology. Balliere Tindall, London.

Fender, D. C., and D. F. Amend. 1978. Hyperosmotic infiltration: factors influencing uptake of bovine serum albumin by rainbow trout *(Salmo gairdneri).* J. Fish. Res. Board Can. 35: 871-874.

Gould, R. W., R. Antipa, and D.E Amend. 1979. Immersion vaccination of sockeye salmon *(Oncorhynchus nerka)* with two pathogenic *strains* of *Vibrio anguillarum.* J. Fish. Res. Board Can. 36: 222-225.

Ingram, G.A. 1980. Substances involved in the natural resistance of fish to infection - a review. J. Fish Biol. 16: 23-60.

Klontz, G.W., and D.P. Anderson. 1970. Oral immunization of salmonids: a review, p. 16-20. In S. F. Snieszko (ed.) A symposium on diseases of fishes and shellfishes. Am. Fish. Soc., Special publication No. 5, Bethesda, MD.

Krantz, G.E., J.M. Reddecliff, and C.E. Heist. 1963. Development ofantibodies against *Aeromonas salmonicida* in trout. J. Immunol. 91: 757-760.

Lannan, J. E. 1978. Vibriosis vaccination of chum salmon by hyperosmotic infiltration. Prog. Fish-Cult. 40: 43-45.

Post, G. 1963. The immune response of rainbow trout *(Salmo gairdneri)* to *Aeromonas hydrophila.* Utah Dep. Fish Game, Publ. No. 63-7. Salt Lake City, UT 82 p.

Roberts, R.J. 1978. Preface, p. ix-x. In R.J. Roberts (ed.) Fish Pathology, Balliere Tindall, London. 218 p.

Ross, A. J., and G. W. Klontz. 1965. Oral immunization of rainbow trout *(Salmo gairdneri)* against an etiological agent of "redmouth disease". J. Fish. Res. Board Can. 22: 713-719.

Sawyer, E. S., and R. G. Strout. 1977. Survival and growth of vaccinated, medicated and untreated coho salmon *(Oncorhynchus kisutch)* exposed to *Vibrio anguillarum.* Aquaculture. 12: 311-315.

Spence, K. D., J. L. Fryer, and K. S. Pilcher. 1965. Active and passive immunization of certain salmonid fishes against *Aeromonas salmonicida.* Can. J. Microbiol. 11: 397-405.

Tavolek, Inc. 1978. Salmonid vaccination procedure. Tavolek Inc., Redmond, WA. 3 p.

Wildlife Vaccines Inc. 1981. Product information on fish vaccines and salmonid vaccination procedures. Wildlife Vaccines, Wheatridge CO.

14

HATCHERY DISINFECTION AND DISPOSAL OF INFECTED STOCKS

J.G. HNATH
Michigan Department of Natural Resources
Fisheries Section
Mattawan, MI

O'Donnell (1944) wrote: "Within recent years, with the increase in production of legal trout at a comparatively high cost, the necessity of preventing and controlling fish diseases has assumed major importance. Our knowledge of the methods of preventing and eliminating disease has increased considerably but there is still much to be learned. Some diseases are controlled quite easily, while others, such as furunculosis, caused by *Bacterium salmonicida (Aeromonas salmonicida)* has failed to yield to experimental treatments. The only known method of absolute control of *B. salmonicida* involves complete elimination of all fish from a hatchery, through disinfection of the hatchery, the rebuilding of a new stock of disease-free fish, and the maintenance of disease-free conditions throughout future operations. Unfortunately, however, this method is practical only at those hatcheries having a controlled water supply, that is, originating in wells or springs that can be kept free of fish. The disinfection of hatcheries utilizing river water, or other public waters, would be inadvisable because of the constant danger of new infections from fish in these waters".

This introduction to a paper written nearly 40 years ago provides a critical reminder to us that while our "knowledge?" may be increasing, we are slow to put what we know into practice. Our most current information confirms what O'Donnell said in 1944, namely that the control of certain fish diseases can be achieved only through disinfection and eradication of contaminated stocks. The purpose of this paper is to provide guidelines for hatchery disinfection.

WHY DISINFECT

Disinfection should be done whenever it becomes desirable to rid a facility of an infectious agent because of production problems that agent has caused, or because of the implications of rearing and shipping infected fish. The time to disinfect is whenever the facility can be taken out of production because effective disinfection will kill fish. Whenever a particularly serious disease problem occurs at a facility, it may be necesssary to destroy all stocks of fish in order to prevent further spread of the infectious agent - see "Disposal of Infected Stocks".

CONSTRAINTS ON DISINFECTION

Under some circumstances, disinfection is not practical. One such situation occurs when there are infected fish, which serve as reservoirs of infection, that cannot be removed from the hatchery water supply. In this case, it may be necessary to install special equipment and effectively treat all of the incoming water to remove the pathogens before a disinfection operation is practical. In other situations, it may be appropriate to drill wells or to seek alternative pathogen-free water sources to avoid contaminated water supplies entirely.

Disinfection may be impractical if economic consequences of the disease are less than the costs of disinfection, or where the probability of reinfection from nearby waters or fish farms is unavoidably high. On the other hand, when serious diseases are encountered a major disinfection operation may be called for. The situation that has occurred in Denmark (Jorgensen 1977) is a good example of how effective disease control can be achieved through disinfection and eradication. Such programs insure that a fish farm which undergoes disinfection is relatively sure that it will not be recontaminated by its neighbors. In Denmark, viral hemorrhagic septicemia (VHS) had spread to the majority of the 540 Danish trout farms by 1974. Through a rigorous disinfection program, approximately 380 of these farms had been registered as free of VHS by 1977 (Jorgensen 1977).

DISPOSAL AND/OR UTILIZATION OF INFECTED STOCKS

In any situation where the disinfection of facilities is contemplated, ways should be explored for the maximum utilization of the fish stocks on hand in ways that do not aggravate the disease problem or contribute to its spread to non-enzootic areas. For a commercial operator, first consideration should be given to controlled marketing in ways which do not exacerbate the disease problem. Diseased live fish should obviously not be sold to another fish farmer who intends to raise them. However, market-sized fish may be sold commercially under certain circumstances (check with regulatory agencies on this aspect). For example, it has recently been demonstrated (Wolf and Markiw 1982) that hot smoking of fish infected with whirling disease will effectively destroy infectivity of the parasite. It may even be possible to utilize small fish in a pet food industry if the fish product would be pasteurized during processing State and federal hatcheries may be able to utilize infected stocks in a controlled stocking program in which infected fish could be planted in a known enzootic area where there would be little danger of spreading the disease to other fish or outside of the enzootic area.

The final option when direct utilization of the fish stocks is not applicable, requires destruction of the fish stocks through burial or incineration. Either method will eliminate the pathogens.

Burial is the least expensive, and the only practical method for large amounts of fish. For this method of disposal, a site should be selected that is remote from fish cultural areas and without drainage into natural waters. Local regulatory agencies often have special requirements which should be observed. A pit should be dug large enough to accommodate all the fish to be disposed of and provide for several feet of clean fill over the top. The fish should be buried in the pit over a layer of quicklime (Hoffman 1976) and each layer of fish should have another layer of quicklime spread over it. Since there are no apparent literature references to this type of disposal the relative amounts of quicklime to fish must be left to the judgement of the person supervising the disposal operation. In Michigan, it was found that a layer of clean uncontaminated soil 0.7 m deep over carcasses infected with whirling disease effectively prevented reinfection of fish at a disinfected hatchery.

Cooking

Thorough cooking followed by any utilization is satisfactory and acceptable (G. Hoffman, USFWS, Stuttgart, AR, Personal Communication).

Incineration

Since heat effectively destroys all fish pathogens, any incineration which reduces the carcasses to ash is adequate. It should be kept in mind, though, that fish carcasses contain much water, and an incinerator to be used for this purpose must be capable of handling the water content involved.

Steam Sterilization

Pathogens in small amounts of fish could effectively be destroyed by steam sterilization under pressure (autoclaving) at 6.8 kg steam pressure at 121°C for *20* min.

PREPARATION FOR CHEMICAL DISINFECTION

Note: The following material has been adapted from U.S. Fish and Wildlife Service and Great Lakes Fishery Commission recommendations for the disinfection of fish cultural facilities.

Sequence of Events

1. A preliminary planning conference should be convened at the hatchery for the purpose of relating planning to the actual physical facilities and situation.
2. Needed chemicals and materials should be obtained.
3. Cleaning and pre-disinfection preparation at the hatchery should be done several weeks prior to the actual disinfection.

4. A final planning and preparedness conference should be held and attended by participants at the first meeting.
5. Final acquisition of chemicals, supplies, and equipment should be completed.
6. The hatchery disinfection should be carried out.
7. Success of the process must be reviewed and evaluated. Before starting actual disinfection of a hatchery, a number of preliminary steps are necessary. It is important that hatchery blueprints be carefully reviewed at this time to see that all unexposed piping (supply and drains) are included for disinfection at appropriate times. All raceways and troughs must be measured at full capacity and calculations made for the quantity of chlorine needed to produce a concentration of 200 ppm (available chlorine). Additional chlorine will be needed for preparation of the 1600 ppm spray solutions to be used on the interior of hatchery buildings and on walkways, etc. around raceways. A 200 ppm level of chlorine is produced by adding 285 gm of dry HTH powder (70% active) to 1,0001 of water.

Safety Precautions

Although chlorine applied as a dry chemical according to label specifications should not be harmful to the applicators, it can pose a human health hazard if misused. Always follow manufacturer's precautions as indicated on the label. A sample label from Olin HTH Chlorine Granular is given in Table 1.

If the chlorine treatment solution will enter fish-bearing waters upon leaving the hatchery, neutralization will be necessary to inactivate the chlorine. Commercial sodium thiosulfate (Hypo) is used as a neutralizer; 1.5 gm is required to neutralize each liter of 200 ppm chlorine solution with a safety factor of 4. The amount of thiosulfate needed should be calculated and ordered to be on hand with the supplies of chlorine and Hypo for the actual disinfection.

Table 1. Label Description from Olin HTH Chlorine Granular

AVAILABLE CHLORINE	65%
Active Ingredient: Calcium hypochlorite	65%
Inert Ingredients	35%

Application of Olin HTH Dry Chlorinator Granular in Fish Hatchery Facilities and Fish Ponds

1. Sanitizing Fish Tanks, Raceways, and Utensils

(a) Clean thoroughly with soap and water to remove scum and dirt. Then rinse with clean water.
(b) Apply Olin HTH Dry Chlorinator Granular to tank and raceways (filled with water) to provide 200 ppm available chlorine (1 ounce HTH Granular for every 25 gallons of water will provide approximately 200 ppm).
(c) Allow one hour of exposure to this concentration of HTH solution and then thoroughly rinse with clean water. NOTE: For utensils such as nets, after

thoroughly cleaning, allow to remain in an HTH solution (200 ppm available chlorine) for one hour, and then thoroughly rinse with clean water.

2. Fish Pond

To control growth of algae and kill many bacteria in fish ponds. (NOTE: this also kills all remaining fish).

(a) Remove all fish from fish pond.

(b) Superchlorinate to establish a chlorine residual of 5-10 ppm (1 ounce per 500-100 gallons of water). Allow 5 minutes for the Olin HTH Dry Chlorinator to disperse and then test the chlorine residual with a pool test kit. Repeat this dosage as needed to maintain the 5-10 ppm residual for 12 to 24 hours. Afterwards allow the pond to stand until the chlorine residual drops to 0 (usually 1-2 days). Assure yourself that there is no chlorine residual in the pond before fish are allowed in the fish pond.

DANGER! FATAL OR HARMFUL IF SWALLOWED, MAY PRODUCE SEVERE CHEMICAL BURNS. DO NOT ALLOW CONTACT WITH EYES, SKIN, MUCOUS MEMBRANES, OR CLOTHING. STRONG OXIDIZER, CONTACT WITH OTHER MATERIAL MAY CAUSE FIRE OR EXPLOSION.

Keep from contact with clothing and other combustible materials. Remove and wash contaminated clothing promptly. While HTH by itself is not a combustible material, it must not be mixed or contaminated with any foreign materials such as household products, Olin pool stabilizer, soap products, paint products, garbage, solvents, acids, pool chemicals, vinegar, beverages, oils, pine oil, dirty rags, etc. Contamination or mixing with these types of chemicals and products may result in fire or explosion and the fire can be of great intensity. Prevent any burning material such as a lighted cigarette from falling into product. Drench fires with water. Flush spilled product by flushing with large amounts of water. Keep in a cool dry place in original container. Always replace lid. Wash empty container thoroughly with water and discard. Do not reuse empty container.

FIRST AID: External - flood skin or eyes with plenty of water for 15 minutes. If irritation to skin persists, get medical attention. For eyes - call a physician immediately. Internal - drink milk, gelatin solution, or egg whites. Follow with milk of magnesia or vegetable oil. Call physician immediately.

HTH is toxic to fish. Keep out of lakes, streams, or ponds. Do not contaminate water by cleaning of equipment, or disposal of waste. Apply this product only as specified on the label.

OLIN CORPORATION
120 Long Ridge Road
Stamford, Connecticut 06904

Each member of the disinfection team should be provided with complete rubber outfits, including boots, coat, hat, and gloves. The outer garments must be removed and left on the hatchery grounds at the end of each day's work if the crew does not remain until the disinfection has been completed, and should be thoroughly disinfected before removal.

The following equipment should be obtained by the hatchery manager for a disinfection operation:

1 high pressure spray unit (car washing sprayer)
2 100 ft. lengths of hose for sprayer
- pairs of rubber boots
- pairs of wet weather gear (coats, hats, pants)
1 pickup truck
2 wire brushes
2 heavy brooms
2 pails, 12-16 quart
3 large sponges
- respirators (gas masks)
2 pairs of safety goggles
3 heavy duty extension cords
- electric frypans
- all other equipment and supplies as needed

Prior to disinfection, all loose equipment should be throughly scrubbed with warm soapy water or 600 ppm Hyamine, if possible, and left near a raceway for later disinfection. Alternatively, such items may be reserved after cleaning for fumigation with paraformaldehyde gas. Such equipment includes buckets, pans, small troughs, tubs, screens, and seine nets. Hatching and rearing troughs should be scrubbed clean. Side walls of all raceways should be scrubbed and the bottoms raked. Particular attention should be given to removing any left-over fish food, pond scum, or other organic detritus.

Any building to be fumigated with formaldehyde gas should be measured and the entire volume of space therein calculated. Paraformaldehyde powder should than be ordered at the rate of 10.0 gm per cubic meter of space.

After disposing of all fish, hatchery personnel should clean, drain, and dry all rearing facilities. During the cleaning process, the person in charge should tour the entire hatchery facility to plan and schedule disinfection work to make sure that all facilities are in the proper state of preparation for effective disinfection.

During the cleaning process, all fish rearing facilities should be scrubbed clean of algae, dirt, and organic wastes. Concrete tanks, incubators, troughs, raceways, water supply headboxes and tailraces should be thoroughly cleaned. Earthen ponds must be drained and the entire area cleaned of plant growth and debris.

When cleaning operations have been completed, all equipment and facilities should be readied for disinfection. All interior surfaces of hatchery buildings should be saturated with an effective disinfectant solution. A solution of 1600

ppm chlorine or 200 ppm of Roccal, Hyamine, or other suitable disinfectants should be applied with a power sprayer. A backpack spray pump can be used for small areas. Sufficient solution should be applied so that it will penetrate every crevice and destroy infectious organisms. All concrete, metal, plastic, fiberglass, or wooden rearing facilities and all fish cultural equipment including nets, screens, and distribution equipment should receive similar disinfection.

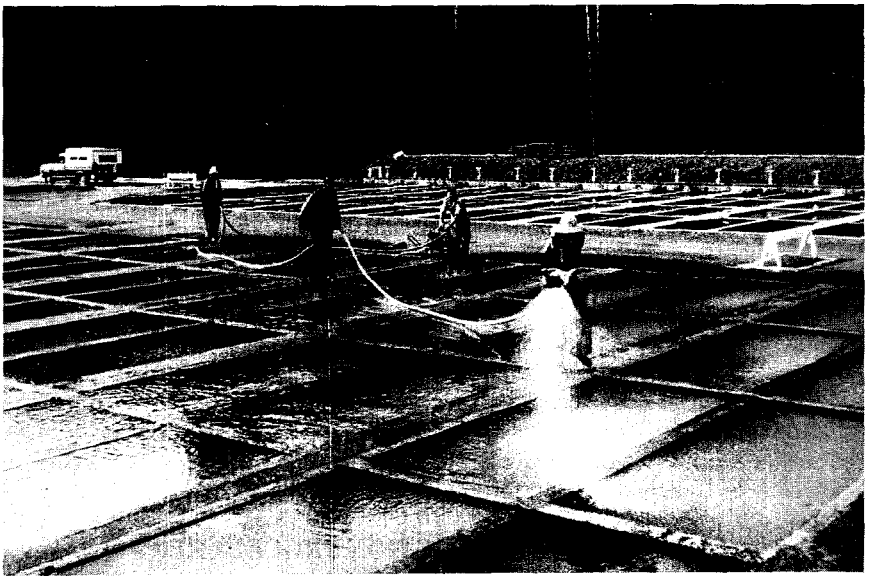
When dealing with whirling disease, drained (but wet) earthen ponds should be disinfected by applying slaked lime at a rate of about one ton of lime per acre of wet pond bottom. As earthen ponds are the most dangerous source of whirling disease spores, special attention must be given to these rearing units. Several treatments may be required to thoroughly disinfect earthen ponds because most chemicals are not effective in mud.

Drying and plowing of the pond bottom is advisable between successive treatments. On successive applications, the lime should be worked into the earth of the pond bottom, to a depth of 25 cm (10 in). If the earth is dry, the pond should be soaked with water to activate the lime and gradually drained after about 2 d, then allowed to dry in the sun for at least 30 d.

CHEMICAL DISINFECTION PROCEDURES

It is desirable that the actual disinfection be carried out in such a manner that the full strength of chlorine (200 ppm) will be maintained for at least 1 h and that a concentration of 100 ppm or more be maintained for several hours. Chlorine dissipates rapidly and is inactivated by organic matter. Most hatcheries are so large that complete disinfection cannot be completed in one day. As a consequence, treatment must be carried out in areas or blocks starting at the upper end of the hatchery. Before adding chlorine, all ponds, raceways, and troughs must be drained completely. Additional splash-boards should be provided if needed to cause the water level to rise to the top of each section. Rearing troughs should be plugged so that they will over-flow. After the initial draining, the splash-boards should be installed and the water allowed to rise until the particular unit is half full. Half the calculated amount of the concentrated chlorine to be added is then emptied into the raceway, and stirred thoroughly, When the water has risen to within 5 cm (2 in) of the top, the remaining chlorine should be added and again stirred. The same general procedure is continued for each raceway unit until the entire raceway system, including all pipe lines, has been completely filled with chlorinated water.

If the hatchery must be disinfected in sections, the work should be carefully planned and timed so that all sections will contain the maximum chlorine level at the same time. This is necessary to insure that no contaminated waters flow through part of the system after it has already been disinfected, and to prevent dilution of the chlorine solution. When planning a hatchery disinfection, due consideration must be given to the sequence of events to ensure complete removal of resident fish and thorough disinfection of drain pipes, since these are frequently the source of continuing hatchery problems. The sequence of chemical addition and neutralization can be critical to an effective disinfection. While the maximum concentration of chlorine is being maintained in the raceway system all loose equipment such as pails, tubs, trays, boards, and other material may be placed in raceways for disinfection. Since chlorine is very corrosive,



Spraying ponds with chlorine during total hatchery disinfection (Ontario Min. of Nat. Res.)

certain metal tools and equipment should be disinfected in Hyamine or Roccal to prevent damage. Care must also be taken to see that wooden equipment is kept submerged. Throughout the course of disinfection, checks should be made on the approximate chlorine content of the water using orthotolidine reagent, or preferably by iodometric titration. If any section shows less than 100 ppm Cl, before 1 h has elapsed, additional chlorine should be applied.

FORMALDEHYDE FUMIGATION PROCEDURES

Buildings or rooms which can be sealed and made air-tight are most easily and effectively disinfected of all known disease organisms by fumigation with formaldehyde gas. Clothing, laboratory equipment, electronic gear, or other articles which may be harmed by various wet disinfectant solutions can be safely disinfected using this gas.

The most important consideration in preparation for this type of disinfection is that gases have poor penetrating powers so all surfaces must be clean of debris. Also, all equipment and materials to be disinfected must be mounted or hung in such a way as to allow free air circulation in and around them, because only direct contact with the gas will effect disinfection.

Formaldehyde gas is the active disinfecting agent. It can be produced by heating paraformaldehyde powder in an electric frypan set on the highest temperature. The recommended application is 10 gm of paraformaldehyde per cubic meter of space.

Although there is ordinarily no danger of fire by this method, formaldehyde gas is highly flammable as it is generated and care should be taken to ensure that no open flame or sources of sparking are present in the area.

In as much as formaldehyde gas begins to escape soon after heating begins (it may take several hours - up to 8 h to sublime all the powder in the frypan), a long extension cord should be used to connect the pan to a source of power outside of the building or room being disinfected. It is mandatory that the room be prepared and everything be in readiness prior to commencing. All windows and doors should have been taped, all holes, vents, louvers, etc. covered to prevent escape of the gas. It is helpful to have one or more windows and a door adjusted so that they can be opened from the outside and a couple of fans set so they may be turned on from outside, in order to flush the interior with fresh air after disinfection has been completed. The room should be disinfected for a minimum of 8 h after all of the gas has been generated, then allowed to air wash for 24-48 h before entry. The room is safe to work in when individuals can work for an hour or more without eye and nose irritation.

Formaldehyde gas is most effective in moist air at 18°C. When necessary, rooms should be heated to at least 18°C prior to and during fumigation.

Although formaldehyde gas is toxic, it provides its own warning signal as it is extremely irritating to the eyes and mucous membranes and one can not tolerate enough exposure to be harmful. After a treated building has been sufficiently air washed to permit one to work inside without irritation, it is safe. The State of New York no longer uses formaldehyde gas because of reported effects and hazards associated with chronic exposure to this chemical.

DISINFECTION OF EARTHEN PONDS

Earthen ponds are considerably more difficult to disinfect than hard surface ponds or raceways. This is because organic material in the pond mud readily inactivates chlorine and can severely limit its effectiveness. The choice of disinfectant to be used will depend upon the specific disease agents to be eliminated.

For bacteria, viruses, and non-specific protozoa, Finlay (1978) recommended a solution of 1% sodium and 0.1% Teepol (a detergent). The presence of the detergent enhances penetration of the disinfectant through soil and the combination is not affected by organic matter. The effectiveness of this disinfectant is dependent upon the maintenance of a high pH (11 or above); this can be easily checked with pH comparator paper.

Earthen ponds should preferably be treated in the summer when the surface is dry. The sodium hydroxide solution (with Teepol) must be applied at the rate of 1/2 gallon/m^2 and left to react for several days before refilling the pond. Although the solution has low toxicity to fish, fresh water should be used to flush the pond for several days before fish are introduced.

When applying the disinfectant with a sprayer, protective clothing and a complete face mask should be worn to prevent inhalation and/or contact with the skin or clothing since the substance is very caustic.

Finlay (1978) suggested that the effluents from ponds treated in this way present " . .no danger to wildlife in rivers receiving them." Even so, it is best to check with local authorities before commencing such a treatment if it may result in toxic materials being released through the effluent into receiving waters.

If earthen ponds are infected with whirling disease, *(Myxosoma cerebralis),* Hoffman (1976) recommends the replacement of the earthen ponds with con-

crete raceways because of the great difficulty in effectively disinfecting mud to any great depth. If an attempt at disinfection of an earthen pond is to be made, Hoffman cautions that the disinfection procedures may have to be repeated several times to ensure total eradication of the parasite. Under such circumstances, he recommends that the ponds first be drained and thoroughly cleaned. The quicklime at 380 g/m² or calcium cyanamide at 500 g/ma should be spread evenly over the drained, wet pond bottoms and dikes. This procedure should be repeated several weeks or months later. It is then desirable to put a small number of "test" fish (rainbow trout fry or fingerlings less than 7 cm and less than 8 months of age) in the pond for 2-4 months to see if they contact the infection before restocking the pond with large numbers of valuable fish.

MAINTENANCE OF HATCHERIES

After a hatchery has been completely disinfected and placed on a disease-free water supply, the prevention of recontamination is of utmost importance.

The movement of any live fish into a disinfected hatchery should be absolutely forbidden, and production should be started only with disinfected eggs from certified disease-free stocks. All fish eggs should be disinfected with a solution of iodophor. It is important to protect the hatchery from contamination via the water, packing material, or cases of shipped eggs, and from the hands of carriers of eggs as well as the shipping truck.

All equipment coming into the hatchery should be thoroughly disinfected before coming into contact with clean hatchery equipment or water. All equipment at the hatchery and all employees hands should be cleaned and disinfected at frequent intervals. The liberal use of warm water and soap is recommended. All trucks and equipment coming into the hatchery to transport fish should be disinfected before entering the hatchery property. See guidelines on Transport Unit Disinfection.

The spread of disease can be prevented only by complete adherence to rigid standards of cleanliness similar to those used in operating rooms at hospitals. A KEEP IT CLEAN attitude must be instilled in all employees, with the realization that even a minor slip in procedure may undo all previous efforts to eliminate disease.

TRANSPORT UNIT DISINFECTION USING CHLORINE

It is good sanitary practice to disinfect transport units and associated equipment after hauling fish from one station and before entering another hatchery. To inadvertently introduce a disease to a station where it is absent may have serious consequences. Here are some suggested procedures for fish transport unit disinfection:

1. All equipment utilized in handling fish at one station should not be allowed to enter any other fish rearing station until it has been sanitized. Equipment involved includes all vehicles, scap nets, seines, fish pumps, waders, raingear, boots, or anything else which might come in contact with fish or fish cultural waters.

2. All personnel directly involved in fish planting operations should wear outer protective garments when handling fish or fish cultural water. At the end of fish planting operations at a station, these protective garments should be disinfected.
3. Transport equipment disinfection must be thorough, complete, and rigidly enforced to be effective. A suitable site must be chosen where facilities are available, and where the chlorine treatment solution may be safely dumped.
   (a) Vehicles should be disinfected with a solution of 1O-20 ppm Cl, sprayed over the exterior surfaces and circulated through the fish transport tanks for a minimum of 30 min. The 20 ppm Cl, solution can be prepared by adding 3.5 g of dry HTH powder (70% active) to 100 1 of water. A solution of 2.21 of household bleach (5.25% sodium hypochlorite) per 5680 1 of water will also give a 20 ppm Cl, solution.

   After disinfection the solution should be dumped in a safe site away from direct drainage into natural waters and where there will be no harmful effects of Cl,. If necessary, the chlorine solution can be neutralized with sodium thiosulfate before release. The chassis and all underparts of vehicles should be thoroughly cleaned and sprayed with the chlorine solution, and rinsed.

   All surfaces which come into contact with Cl, must be thoroughly rinsed immediately after disinfection with copious amounts of fresh water because chlorine is corrosive. The fish holding tanks and recirculation equipment should be rinsed with fresh water containing enough sodium thiosulfate to effectively neutralize any remaining Cl,. Normally 50 g of thiosulfate should neutralize any Cl, in 100 1 of rinse water, within a ten minute period of recirculation. The effectiveness of neutralization should be checked by using orthotolidine test solution after this ten minute period.

   Orthotolidine can be used to test for residual chlorine by taking identical samples of water to be tested in clean glass containers (approximately 125 ml of water each) and adding a dropperful of the test solution to one of the samples. Keep both samples in the shade and examine after 5 min. Any formation of yellow color in the solution to which orthotolidine has been added will indicate residual Cl,, and means that the neutralization was not complete. If this occurs add additional thiosulfate to the transport unit and repeat the orthotolidine test until no color develops.

   Only after complete Cl, neutralization has occurred should the tanks be emptied. They should be refilled with enough fresh water to rinse out the remaining thiosulfate solution and then dumped. Thiosulfate is not toxic to fish in the low levels used.

   (b) Interior surfaces of vehicles should not be disinfected with Cl, because of its strong oxidative and corrosive nature. If these areas require disinfection, use a 600 ppm solution of Hyamine or Roccal. Such solutions can be prepared by adding approximately 1.5 ml of the 50% stock solution per liter of water.

   (c) All other equipment, as defined in statement (1), shall be immersed, sprayed, scrubbed, or otherwise covered with a solution of 20 ppm Cl,. Porous or otherwise “difficult to clean” items should be soaked for a minimum of 30 min before rinsing. Other items, such as waders, and

raingear, may be thoroughly scrubbed with the disinfectant and then rinsed well with fresh water. Some caution is needed as the disinfectant is somewhat caustic. Natural fabrics such as wool and cotton can be severely damaged by Cl_2 and should be disinfected with a solution of Hyamine or Roccal at 600 ppm and soaked for 30 min.

4. The State of New York does not use chlorine for transport unit disinfection because of the corrosive nature of chlorine and its adverse effects on aerators and pumps. They have switched to Roccal at 800 - 1000 ppm.

   According to technical information provided by the Hilton-Davis Chemical Company, these levels should provide adequate disinfection (J. Schachte, NYDEC, Rome, NY, personel communication).

OTHER DISINFECTANTS AND APPLICATIONS

An excellent summary of other disinfectants was prepared by J. Finlay (1978). The reader is referred to that paper for more specific information. Table 2 provided is mostly taken from that reference. In addition to those mentioned in Table 2, the following disinfectants have been used:

1. Roccal (50% active) or Hyamine 3500 (50% active) (quaternary ammonia compounds) - 70 ml/l for dipping nets, brushes, boots, and seines, or same concentration for 30 min for fish transport unit disinfection.
2. 1,000 ppm of quaternary ammonium compounds spray for disinfecting hard surfaced rearing units (J. Schachte, NYDEC, Rome, NY, personal communication).
3. 2% formalin (200 g commercial formalin water) may be used as a spray to disinfect nearly all apparatus used for fish culture (FAO 1977).
4. Iodophors - 50 to 100 ppm free iodine may be used as a spray on previously cleaned and dried equipment and machines (FAO 1977).
5. Sodium hydroxide (NaOH) - a 0.2% solution is recommended for cleaning hatchery equipment, rearing units, transport equipment, and buildings prior to disinfection with any of the above (FAO 1977).
6. Steam cleaning method of disinfecting ponds recommended in New Zealand (Hewitt 1972) is cleaning with live steam at temperatures of 115 - 130° C. Since normal steam cleaning equipment is set to operate at 104° C, it will be necessary to be sure that the required temperatures are produced in order to assure disinfection. It is also important that the steaming process proceed slowly enough that all surfaces to be disinfected also reach the higher temperature range. Some advantages of steam over chlorine are that no toxic chemical is used and therefore there need be no concern over leakage of toxic materials through the effluent into receiving waters and streams, and it is not corrosive to metal.

Table 2. Other Disinfectants and Their Application

Disinfectant	Working Strength	Application*	Effective against
Chlorine	1-2%	Concrete Fiberglass Butyl-lined ponds Nets, etc. Footbaths	Bacteria Fungi Viruses (Whirling Disease (G.L. Hoffman, J. J. O'Grodnick, 1977)
Sodium Hydroxide	1% with 0.1% Teepol 1/2 gallon/m^2	Earthen ponds Concrete Fiberglass Butyl-lined ponds Nets, etc. Footbaths	Bacteria Viruses Protozoa
Iodophors	250 ppm	Concrete Fiberglass Butyl-lined ponds Nets, etc. Angling Equipment Clothing, hands	Bacteria Viruses
	100 ppm	Ova	
Quaternary ammonium compounds (Hyamine, Roccal, etc.)	As manufacturers' instructions	Nets Clothing, hands	Bacteria
Calcium Oxide	As powder 380 g/m^2	Earthen ponds Fiberglass Concrete Butyl-lined ponds	Protozoa (Whirling Disease)

* All surfaces should be scrubbed clean before the disinfectant is applied and, if necessary, the disinfectant should be scrubbed into the surface.

REFERENCES

Anonymous 1977. Control of the spread of major communicable fish diseases. FAO Fish. Rep. No. 192. United Nations, Rome. 44 p.

Anonymous 1982. Fish disease control policy. Appendix I: Approved disinfection methods. Wash. Dep. Fish. Game, Olympia, WA. Unpublished draft.

Clary, S.D. 1978. Realistic control of fish diseases through hatchery sanitation and water conditioning. Salmonid. Vol. l(5): 1O-11.

Davis, H.S. 1938. The use of chlorine for disinfecting fish ponds. Prog. Fish-Cult. 42: 24-29.

Finlay, J. 1978. Disinfectants in fish farming. G.B. Ministry Agric. Fish. Food, Fishery Notes, No. 59. Lowestoft, England. 7 p.

Hagen, W. Jr. 1940. Sterilizing hatchery water supplies. Prog. Fish- Cult. 48: 19-24.

Hewitt, G.C. 1972. Hygiene in New Zealand trout hatcheries. N.Z. Mar. Dep. Fish. Tech. Rep. No. 86, 12 p.

Hoffman, G.L. 1976. Whirling disease of trout. U.S. Fish Wildl. Serv. Fish Dis. Leafl. 47. Washington, DC. 10 p.

Hoffman, G. L., and J. J. O'Grodnick. 1977. Control of whirling disease *Myxosoma cerebralis:* effects of drying, and disinfection with hydrated lime or chlorine. J. Fish. Biol. 10: 175-179.

O'Donnell, D. J. 1944. The disinfection and maintenance of trout hatcheries for the control of disease, with special reference to furunculosis. Trans. Am. Fish. Soc. 74: 26-34.

Vestergard-Jorgensen, PE. 1977. Surveillance and eradication of diseases from hatcheries, p. 72-73. In Proc. Int. Sympos. Dis. of Cult. Salm., Tavolek, Inc., Seattle, WA.

Wolf, K., and M. E. Markiw. 1982. *Myxosoma cerebralis:* Inactivation of spores by hot smoking of infected trout. Can. J. Fish. Aquat. Sci. 39: 926-928.

PART III

ADMINISTRATIVE OPTIONS IN DISEASE CONTROL

15

REGIONAL CONTROL OF COMMUNICABLE DISEASES OF FISH

T.G. CAREY
Aquaculture and Resource Development Branch
Department of Fisheries and Oceans
Ottawa, Ont.

The watershed of the Great Lakes basin covers a vast area, exceeding 350,000 Km^2, and traverses eight states (Illinois, Indiana, Michigan, Minnesota, New York, Ohio, Pennsylvania and Wisconsin), one province (Ontario) and an international boundary (U.S.A. and Canada). Fishery resources in the Great Lakes, after experiencing a period of major decline due to sea lamprey predation and water pollution, are rebounding and supporting major recreational and commercial fisheries. In addition to native fish species, the successful introduction of Pacific salmon for development of sport fisheries has added further diversification to the total fish fauna of the Great Lakes.

Within this complex system comprising large multi-species fisheries managed by many agencies, the culture of salmonids in both public and private sectors has expanded dramatically in recent years to support restoration and augmentation programs for many recreational and commercial fisheries, and for investment purposes in aquaculture business. There are now over 100 government-operated fish culture stations and broodstock collection sites in the Great Lakes watershed, and probably more than 200 privately operated fish culture facilities. These hatchery operations have involved international, inter-regional and intra-regional transfers of live fish and eggs, and massive releases of hatchery-reared fish into rivers and lakes are common occurrences.

The introduction and dissemination of communicable fish diseases through these activities is a potential hazard that could ultimately damage the salmonid fishery resources and affect major business investments if movements of stocks are allowed to take place indiscriminately. Communicable diseases include oblig-

ate pathogens that are not ubiquitous in surface waters and are transmitted primarily through contact between infected and non-infected fish. Hazards of communicable diseases may also be created by the movement of causative organisms or health-related agents, such as research materials, feed ingredients and biologics. Similarly, drugs or therapeutic agents that may affect the character of pathogens or overall health of fish should be subject to regulated usage.

The effective control of communicable diseases on a regional basis requires mutual understanding by public and private sectors of their accountability for disease control, and the need for development of cooperative procedures and mechanisms which minimize the risks of exposure to communicable diseases. Beneficiaries of such action will include all those who depend on aquatic resources, including fish culturists, sports and commercial fishermen, and those businesses whose economic welfare is dependent on fish culture activities.

This section will summarize the acknowledged levels of accountability of various sectors for controlling communicable diseases, mechanisms for implementing control measures, and recommended procedures for application of regional fish disease control interventions in the Great Lakes basin.

ACCOUNTABILITY FOR CONTROL OF COMMUNICABLE FISH DISEASES

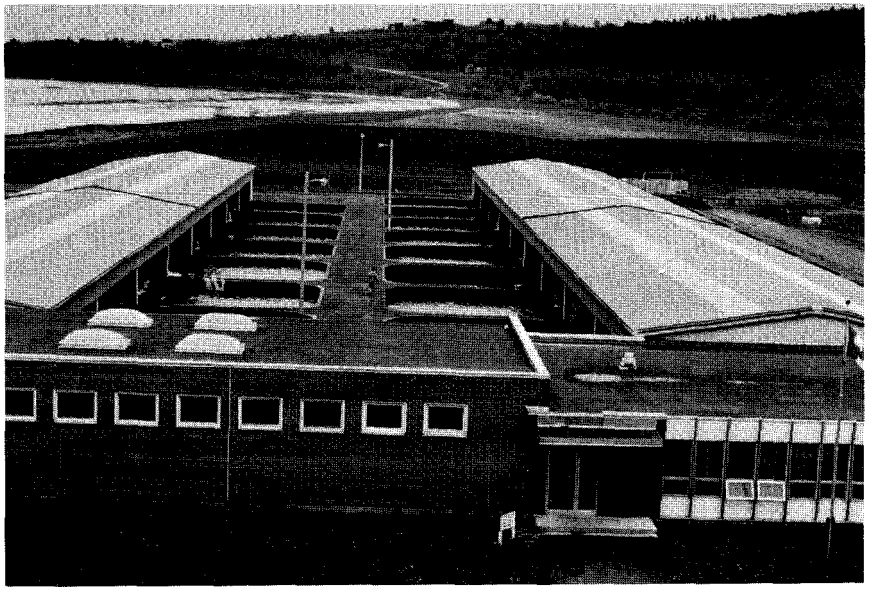
The term "accountability" in this context can be described as the person, office, or unit on which onus is placed to undertake certain actions, and for which they are answerable if those actions are not properly discharged. It is also used here in the ethical as opposed to legal sense, the intention being to ensure that all those involved in or with fish culture are aware of their obligations to participate in the control of communicable diseases.

HATCHERY OPERATIONS

The culture of salmonids in hatcheries in the Great Lakes basin is undertaken to restore naturally spawning populations, to supplement the reproduction of valuable stocks to support populations that do not reproduce naturally (e.g. Pacific salmon species), and to produce fish for market by the private sector. With the growing importance and value of salmonid fisheries, and the steady growth of a fish culture industry within the private sector, the level of production of cultured stocks and the numbers and size of production facilities have increased significantly in recent years.

Fish culture facilities tend to be the primary sites for disseminating fish diseases, because of increased susceptibility of fish to diseases when reared in artificial environments. Owners and managers of these facilities must therefore recognize the potential for dissemination of diseases to the natural environment from facilities under their control. Diseases can be disseminated through hatchery effluents, poor hatchery sanitation procedures, or through inter-station transfer or release of infected eggs and fish. Other hatcheries in the same watershed and, in some cases, natural fish populations can be affected.

Acknowledgement of this accountability in the operation of hatcheries, and in the development of procedures to minimize the risk of spreading disease, will contribute significantly to overall efforts for control of communicable fish dis-



A modem government hatchery used for production of Atlantic salmon smolts for stocking in rivers (T. G. Carey)

eases. It will also enhance public support for aquaculture activities and help foster further expansion of the industry.

STATE/PROVINCIAL GOVERNMENTS

The responsibility for protection of aquatic resources for the benefit of the common interest is vested in governments. This includes governmental administration of fisheries and activities that impact on them. In the U.S.A., this authority has been given to individual states. In Canada, the authority for administration of certain inland fisheries has been delegated to provincial governments (e.g. Ontario).

Within this framework, maintaining the health of natural fish populations is an important factor for which state and provincial authorities are accountable within their state or province. This applies equally to the control of communicable fish diseases as it does to ensuring retention of genetic integrity of stocks, maintaining good environmental quality, and rational exploitation of stocks.

With respect to control of communicable diseases, the objective is to minimize risks of introduction and dissemination of diseases to natural stocks from potential disease sources. Those with the administrative authority must take necessary measures to assure this protection using regulatory options implemented as acts, regulations, policies or guidelines to balance the need for control with the need to operate efficient and economical fish culture units.

State and provincial authorities are also accountable for the protection of innocent parties that might be affected by the actions of others. For example, water is a fluid environment that can quickly and easily carry fish pathogens

Releasing hatchery-reared salmon to supplement natural populations (T. G. Carey)

across considerable distances from a disease source. Consequently, a fish culturist who has worked hard to minimize risk of disease outbreaks in his facility may have his efforts negated by a less conscientious operator upstream of his facility. Similarly, the production of vaccines, fish feeds, and therapeutic chemicals is beyond the control of individual hatchery operators. Therefore, the fish health risks introduced through the use of such products should be reviewed and minimized by governments.

Finally, state/provincial authorities are accountable for the conduct of their own fish culture programs, and for setting an example for the private sector. In many instances, production by government stations and scope of distribution to rivers and lakes far exceeds that in the private sector; thus, the risk of disease dissemination through release of stocks is high. Health protection programs and the use of procedures of control the introduction and spread of communicable diseases must be of major importance to state and provincial governments.

FEDERAL GOVERNMENTS

As well as assuming some of the responsibility for aquatic resources within each state or province, federal governments in U.S.A. and Canada are responsible for control of interprovincial or interstate and international activities affecting these resources. In effect, they maintain a national perspective by monitoring internal and external factors that influence the aquatic environment in the respective countries. Federal agencies also provide national leadership in research and the establishment of standards related to fish health and the control of communicable diseases.

Accountability of the two federal governments with respect to the control of communicable diseases is similar to those of state/provincial governments, but on a much broader scale. Federal governments are accountable for the health of aquatic resources in each nation as a whole (including transboundary shipments), for the protection of innocent parties, and for the federal hatchery programs throughout each country.

MECHANISMS FOR IMPLEMENTING DISEASE CONTROL MEASURES

To fulfill their obligations for disease control, accountable authorities have basically three mechanisms through which control measures can be implemented. These options vary in the degree of control that can be applied, although each can be as effective as the others depending on the circumstances in which they are used.

GUIDELINES

Guidelines recommend voluntary compliance to stated procedures and practices which, if adhered to, will reduce the risk of exposure to diseases. Guidelines can be formulated and implemented at any level in a program structure.

POLICIES

Formal policies are statements of intent, usually approved at the highest level of authority. They are developed as a result of forecasting, planning, and decision-making and must aim at achieving conformity in conception and realization. While not enforceable, policies require strict adherence within the organization which initiated them and can also provide protection from external influences.

STATUTES

Statutes are enforceable acts and regulations promulgated by governments and written into law. They are the least flexible of the three mechanisms available for initiating disease control interventions and are costly to maintain. However, they provide the greatest potential for achieving uniform compliance with disease control procedures because of their legal status.

The legality of statutes demands that they be judiciously planned and formulated. They set the standards for fish health and disease control, and provide details of enforcement procedures as well as designate penalties and incentives for compliance. Finally, with the potential for litigation, statutes are likely to be utilized and tested in court actions.

DEVELOPMENT OF REGULATORY OPTIONS TO CONTROL COMMUNICABLE DISEASES IN THE GREAT LAKES

Since the active salmonid culture industry in the Great Lakes basin involves both public and private sectors, a wide variety of culture strategies, and uses of

cultured stocks, it is sometimes necessary to make broad interventions to reduce the risk of introducing and disseminating communicable diseases. A range of measures is available to hatchery operators to control diseases, but individual efforts are not always sufficient to provide the protection needed against communicable diseases.

Prevention should be given priority in developing effective measures to reduce the risk of exposure to communicable diseases. In the absence of disease agents, there can be no threat from diseases. A second, but still important, consideration involves control and eradication after pathogens have been detected. Consideration must also be given to the development of regional disease control measures that will achieve the necessary control, to moderation of direct and indirect impacts on industry, and to assessment of the cost of administration and enforcement activites. Resolution of conflicts and generation of public acceptance for disease control regulations will require co-operative effort and understanding if the proposed measures are to achieve their objectives.

Implementation of regulatory options can have negative as well as positive effects. On the negative side are the costs of services for administration, enforcement, and inspection of facilities and disease diagnostics; trade disruptions; the need for indemnification and insurance programs when eradication and rehabilitation are involved: and the potential for litigation. Alternatively, well-planned regulatory programs increase awareness of fish health considerations and provide assurance to new and expanding fish culture enterprises. They can generate confidence in stocks originating from disease-free operations and expand the market potential for products. Finally, they set an example which can be emulated by others, contributing to the overall improvement in fish health status.

The planning, design, implementation, evaluation and updating of controls are important components in the use of regulatory options, and it is logical that these procedures should be undertaken sequentially as described below.

Step One: Review of Activities for Potential Disease Risks

Opportunities for the introduction and dissemination of communicable diseases are always present in salmonid culture programs undertaken in the Great Lakes region. Potential hazards exist in what might otherwise be considered routine hatchery procedures, and it is important that the potential disease impact of these activities be understood and acted on.

1. Transportation of Fish

   The movement of fish stocks from one region or country to another has been recognized as one of the major factors involved in dissemination of diseases. This applies to transfers of wild, as well as cultured, fish because wild fish also can be carriers of disease agents.

   In the context of the Great Lakes, communicable diseases of salmonids could be transmitted when:

   a. Eggs, juveniles or broodstock are imported from sources outside the Great Lakes watershed to support hatchery production programs;
   b. Stocks at any stage are transferred between hatchery facilities within a state/province or between regions;

c. Hatchery-reared fish are released into rivers and lakes to create fisheries or to enhance natural production of wild stocks.

The basic premise in most controls on movements of fish is to apply the principles of risk management and to permit transfer only of those stocks that are considered free of specific fish pathogens. Uncontrolled traffic of salmonids into and within the Great Lakes basin would present a high level of risk for the introduction and dissemination of communicable diseases.

2. Introduction of Exotic Fish Species

The impacts of introducing non-indigenous fish species into new waters have been well documented (Courtenay 1973; Regier 1968; Vooren 1972) and include the potential risk of transferring diseases with exotic species. "Introduction of a pathogen via transplants of an exotic species to an area where evolutionary adaptation and partial immunity have not been acquired among native stocks, has the potential to destroy native populations. Conversely, the success of the planned introduction of an exotic species could be limited by the impact of local diseases on the introduced stocks" (Anon 1979). Other factors that must also be considered when introducing exotic species include competition with indigenous species, habitat destruction, genetic implications, and assessment of the commercial value of the introduced exotic species with its potential impact on indigenous species and the environment.

Coho, chinook, and pink salmon, and steelhead trout have all been introduced to the Great Lakes basin in recent years and several species have established naturally spawning populations. Coho and chinook salmon depend on artificial propagation for recruitment, and their populations are controllable to some degree. With fisheries managers continually striving to expand and diversify fisheries to meet increasing demands from recreational and commercial fishermen, the tendency is to search for additional new species and sources of broodstock that might result in improved fisheries. Strict regulatory control and detailed assessment of the impact of introductions of exotic species are therefore desirable to minimize the potential risk of introducing and disseminating communicable fish diseases through this avenue.

3. Emergency Disease Outbreaks

If regulatory measures are promulgated to control the movement of fish stocks, the introduction of disease agents could still occur. If serious fish diseases are involved that could threaten cultured and natural fish stocks, and that have not been recorded in a region or country despite extensive surveys, they could be considered as "emergency" diseases.

Prompt eradication of a disease can be used to prevent further dissemination to new areas. Speed of action is an essential factor if eradication procedures are to be effective. This depends largely on the degree of planning and preparation that precedes the outbreak of an emergency disease, and on the cooperation and collaboration that can be generated at short notice.

Important considerations that should be examined when developing regulatory programs for the eradication of emergency diseases include

naming of the diseases and their causative agents, responsibility for co-ordination and decision-making, establishment of quarantine zones, assessment of potential success of eradication procedures, post-eradication monitoring to determine success, availability of replacement stocks, and compensation for losses (indemnification) suffered by the private sector. These factors require input from and supervision by skilled professionals, and the overall costs of eradication procedures and indemnification should not be under-estimated.

A recent example of the introduction and spread of what might be classified as an emergency disease was documented for *Myxosoma cerebralis (whirling* disease) by Hoffman (1970) in the U.S.A. This disease, which can cause high mortalities during the early life cycle of certain salmonid species, has now been recorded in the Great Lakes states of Michigan, Ohio and Pennsylvania, having been first introduced to the U.S.A. in 1956. Although whirling disease does not lend itself easily to normal eradication procedures, such as the destruction of stocks and disinfection of facilities, these measures, together with establishment of quarantine or buffer zones, could have slowed or prevented dissemination of the disease as widely as it is now recorded.

4. Production and Use of Biologics

The use of biologics, specific antisera, and vaccines for pathogen identification and control of fish diseases has increased as the aquaculture industry has expanded. Use of non-indigenous pathogens for research and live vaccines are also potential hazards for spreading communicable diseases. Regulatory mechanisms and procedures for the control of similar biologics are already well-developed in the agricultural and human health sectors, and can be used as models for developing appropriate controls for biologics related to communicable fish diseases.

5. Other Hazards

Although not related directly to communicable diseases, interventions by government agencies should be considered for control of the production and use of chemotherapeutics in fish feeds. Routine inspection and certification procedures, and requirements for labelling and efficacy and safety testing of these products should be part of any fish health protection program affecting the Great Lakes region.

Step Two: Quantification of Risks and Assessment of Impact of Interventions

The objectives of any intervention to control communicable diseases should be to minimize risks at the least cost and to create as little disruption of the industry as possible. Control measures should be workable, acceptable and implementable within the constraints of budgets, manpower allocations and existing technology, and should focus on those activities that present the greatest degree of risk.

Not all activities of concern present the same degree of risk for introduction and spread of communicable diseases. The risk of transferring diseases is lowest in dead fish destined for the consumer market. In live organisms, egg stages present the least risk, especially since procedures have been developed for

treatment of broodstock and disinfection of eggs to reduce the potential for vertical transmission of disease from brood stock to progeny. The greatest risk, on the other hand, occurs in the transfer of broodstock because stresses of sexual maturation significantly increase the possibility of disease.

Fish that are known to be carriers of disease agents need not necessarily exhibit overt signs of disease. Bacterial kidney disease, for example, might be detected in very low incidence in the carrier state at a hatchery yet the facility may never experience an epizootic (M. Campbell, Canada Department of Fisheries and Oceans, Halifax, N. S., personal communication). Similarly, many wild fish are carriers of pathogens that will only cause an epizootic if the fish are held in a stressful environment. Consideration should be given, then, to whether control measures must be applicable to carriers of the diseases as well as those in which overt signs have been found.

Different species of fish are more disposed to infection by certain disease agents than others. Enteric Redmouth Disease, caused by *Yersinia ruckeri*, is more prevalent in rainbow trout than in any other salmonid species. On the other hand, furunculosis has been detected in a wide-range of hosts from cold, cool and warm waters and measures to control this disease would have to apply to more than just a few target species.

A primary requirement for quantifying risks of disease introduction is the need for an extensive and detailed data base in the geographical region of concern. A considerable body of knowledge on disease distribution has been developed in the Great Lakes region by state, provincial and federal fish pathologists. In addition, sensitive diagnostic protocols are available and disease control procedures have been developed. This provides a strong base for developing programs considered necessary for controlling communicable diseases in the region.

In assessing the impact of potential disease control interventions, consideration must be given to the state of development of the aquaculture industry, its economic importance, and aquatic resource usage. These factors can influence decisions on whether controls are necessary at all, and on the types of interventions required to control communicable diseases.

When information on potential disease threats has been analyzed and quantified, long-term projections can be prepared to anticipate where the risks of disease exposure are greatest and what types of control options are available, if needed. The distribution of pathogens of concern can be mapped, disease-free zones established, and control measures can be formulated which reflect the concerns of fish culturists and other users of the aquatic environment.

Step Three: Mechanisms for Reducing Perceived Risks

A. GENERAL PRINCIPLES

The need for controls to prevent the introduction and dissemination of communicable diseases is not disputed. The dilemma is to decide on the degree of control required and to select an appropriate strategy to achieve the desired results. It is also important to ensure that there is good communication between public and private sectors, to develop a common perception and knowledge of the problems to be addressed, and to formulate interventions that are equally applicable to public and private fish culture programs and operations.

Some of the alternative interventions that can be made for disease control include bans, zoning, and establishment of quarantine areas and facilities. Bans can control factors such as the movements of fish and use of drugs, although their benefits are most apparent when used as interim measures to react to unforeseen or emergency situations. As a long-term option, bans are restrictive and rigid, and reduce the potential to provide flexibility needed for growth in a diverse aquaculture industry.

The concept of zoning is well recognized in the agriculture sector as a means of controlling the spread of animal and plant diseases. This is based on the variable distribution of diseases that are found in some areas and not others. Thus, there is little risk of transferring a specific disease if animals or fish are moved from one clear zone to another. The zoning concept is equally applicable to fish diseases, provided that there is an adequate history of testing for disease prevalence on which designation of disease-free zones can be based.

Quarantine procedures, used to control the introduction of diseases, involve retention of newly-imported fish stocks in quarantine facilities for prescribed periods to ascertain whether they are carriers of disease agents. This strategy is most relevant for controlling communicable diseases that have never been recorded in a country or region, and requires use of sophisticated holding facilities with capabilities for total disinfection of any effluents. Adoption of this principle places a heavy cost burden on the regulating agencies for the constuction and maintenance of quarantine facilities, and is most appropriately used for special situations such as introduction of exotic species.

B. CONTROL OF FISH DISEASES IN THE GREAT LAKES BASIN

The mechanisms that have been used for implementing controls related to communicable fish diseases in the Great Lakes basin are summarized below:

1. Guidelines

Within the Great Lakes region, the use of voluntary compliance to guidelines is best exemplified in the Great Lakes Fishery Commission's model fish disease control program (Appendix III>. This program relies on voluntary adherence to guidelines related to traffic in eggs and fish, releases of fish, and routine monitoring of diseases in government fish culture facilities within the Great Lakes basin. The guidelines are designed to reduce the risk of introduction and spread of diseases within the basin.

Similarly, formation of the Great Lakes Private Fish Health Protection Cooperative represents a new initiative to control fish diseases within the private sector. This Cooperative has allied itself with the Great Lakes Fish Disease Control Committee, and will endeavor to use guidelines to control communicable diseases of concern in the region. Representation of the Cooperative on the GLFDCC will also lead to improved communication and understanding with respect to disease control problems and help direct effort towards common goals.

2. Policies

For example, in 1977 the Ontario provincial government reaffirmed its long-standing position that no importation of salmonids would be allowed for commercial purposes. A major consideration in this stance was the prevention of

introduction and spread of communicable fish diseases. Only special lots of salmonids have been imported since then for research purposes, when appropriate disease control safeguards have been incorporated in the holding facilities.

3. Statutes

In the U.S.A., national regulations have been promulgated (Title 50) pertaining to the control of communicable fish diseases, requiring that all salmonids imported into the country be certified free of whirling disease (Myxosoma cerebralis) and viral hemorrhagic septicemia (VHS). The degree of regulation related to communicable diseases in individual states in the Great Lakes basin varies considerably according to the perceptions and understanding of respective disease risks by the several state fishery agencies.

In Canada, Fish Health Protection Regulations implemented in 1977 apply to the importation of salmonids into the country as well as to their shipment between provinces. These regulations were designed to prevent the introduction and spread of infectious salmonid diseases through inspection of production sources of fish stocks (rather than individual shipments of fish), and to control the movement of infected fish stocks. Complementary to these regulations, a Manual of Compliance was prepared to provide guidelines for producers; to explain the roles of administering officers and inspection officials; and to outline the sampling, handling, and diagnostic procedures that constitute inspections leading to certification (Anon 1977). These "national" regulations governing movement of salmonids are supplemented by provincial regulations in Ontario which deal specifically with the introduction and transfer of fish stocks within that province.

Step Four: Evaluation and Improvement of Control Measures

An important aspect of administering interventions to control communicable diseases is to establish mechanisms for evaluation of the success of measures implemented. Active feedback and assessment of effectiveness provide the basis for modification and improvement of controls and provide an opportunity for the identification of new risks. It is also desirable to provide mechanisms for regular communication between private and public sectors to stimulate discussion and mutual understanding of disease concerns.

These functions can be provided by individuals or groups of people designated as focal points to coordinate regular review and evaluation of interventions. In the context of the Great Lakes, the Great Lakes Fish Disease Control Committee and the Canadian National Registry of Fish Diseases undertake the functions of review and evaluation of the effectiveness of control measures. These institutions collect, collate, and distribute verified information and regularly evaluate the Great Lakes model fish disease control program and Fish Health Protection Regulations, respectively. In addition, by taking advantage of their central status related to fish health concerns in the basin and the information they receive, they are able to:

1. Maintain a close watch on the geographic distribution and incidence of diseases, and to assess their biological and economic impact;
2. Serve as a coordinating center in the event of fish health emergencies;

3. Identify and evaluate new risk situations for the introduction and dissemination of communicable diseases;
4. Provide periodic reports, analyses, and assessments on the state of fish health in their respective regions/jurisdictions;
5. Provide health histories of sources of live fish and eggs: 6. stimulate and support research related to fish health and disease control.

REFERENCES

Anonymous, 1977. Fish health protection regulations: manual of compliance. Can. Dep. Fish. Oceans, Misc. Spec. Publ. 31. Ottawa, Ont. 32 p.

Anonymous, 1979. Considerations and feasibility studies relevant to the introduction of Pacific salmon to Atlantic waters. Can. Dep. Fish. Oceans, CAFSAC Committee, Halifax, N.S. 37 p.

Courtenay, W. R., and C. R. Robins. 1973. Exotic aquatic organisms in Florida with emphasis on fishes: a review and recommendations. Trans. Am. Fish. Soc. 102(1): 1-12.

Hoffman, G. L. 1970. Intercontinental and transcontinental dissemination and transfaunation of fish parasites with emphasis on whirling disease (Myxosoma cerebralis), p. 69-81. In S.E Snieszko (ed.)., A symposium on disease of fishes and shellfishes. Am. Fish. Soc. Spec. Publ. No. 5. Bethesda, MD. 526 p.

Regier, H. A. 1968. The potential misuse of exotic fish as introductions. Can. Comm. Freshw. Fish. Res., Jan. 1968: 92-111.

Vooren, C.M. 1972. Ecological aspects of the introduction of fish species into natural habitats in Europe, with special reference to the Netherlands. A literature review. J. Fish. Biol. 4: 565-583.

PART IV

INTEGRATING FISH HEALTH MANAGEMENT OPTIONS

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SYNTHESIS OF A FISH HEALTH MANAGEMENT PROGRAM

J.W. WARREN
U.S. Department of the Interior
Fish and Wildlife Service
La Crosse, WI

Fish health protection is an art and science that can be guided by both public and animal health programs and by agricultural pest control techniques. Fish suffer from the same types of diseases as other animals and plants. Some of these diseases infect only fish. These are caused by obligate fish pathogens that do not survive indefinitely in the aquatic environment unless diseased or carrier fish are present. Bacterial kidney disease (BKD) is a typical example. On the other hand, the majority of infectious diseases of fish are caused by facultative (opportunistic) fish pathogens usually present in the environment. Outbreaks of disease caused by facultative fish pathogens often occur when the fish have been rendered susceptible by stress. Examples include bacterial gill disease, columnaris, and infections caused by *Aeromonas hydrophila.*

Strategies for preventing or controlling fish diseases must consider the nature of the disease in question and when and where it occurs. If a disease outbreak among cultured fish can be prevented, economic losses can be avoided and complex disease control and clean-up procedures need not be applied. The first portion of this discussion will be devoted to a brief review of measures that can be taken to prevent fish diseases. This will be followed by a discussion of how to control diseases caused by obligate fish pathogens along with material on the development of an integrated fish health management program.

FISH DISEASE PREVENTION

Prevention is the cornerstone of any health protection program and can be as challenging and complex as the actual control of existing diseases. Key

elements of disease prevention include the reliable detection of disease carriers, knowledge of how pathogens are transmitted, development of effective methods to limit the entry of pathogens or carriers into clean fish cultural facilities, and the capability to provide environmental conditions conducive to good fish health. Herman (1970) discussed progress made in several areas of fish disease prevention and control. Since his 1970 review, important advances have been made in mass immunization of fish, detection of fish pathogens in carriers, enhancement of environmental conditions, and fish nutrition.

Regulatory and Cooperative Measures

Avoidance of disease is a fundamental part of programs developed to protect the health of man and domestic animals. Regulatory and cooperative measures can be effective in preventing exposure to physical, chemical and biological disease agents. Regulations are developed to provide organizational structure and to assure the execution of specific procedures designed to contain diseases and their pathogens and to guide the action to be taken when outbreaks occur. Cooperative efforts also have a role. In the poultry industry, programs have been organized to improve flocks, to control serious diseases, and to raise quality standards. Cooperative programs are only beginning in the fish cultural field, however.

Regulations for fish health protection are most useful in the control of those diseases clearly identified as being caused by obligate fish pathogens. It is essential to have the capability to accurately diagnose these diseases and to have both governmental and industry support behind any effort to develop regulations. Properly designed regulatory programs can help solve certain problems that cannot be effectively dealt with by other, less restrictive, methods but there are many other important elements of fish health management that should be considered before regulations are drafted.

Facilities, Water Supplies, and Environmental Manipulation

Successful fish culture is the result of effective environmental manipulation of circumstances dictated by the design of the facility and the nature of its water supply. The occurrence of infectious disease is often related closely to environmental stress (Wedemeyer et al. 1976). Needham (1977) pointed out that "in salmonid culture *far more fish are lost from systems failure than disease."* Environmental conditions imposed on fish are determined by site selection, water supply characteristics, facility design, fish handling and transport systems, and the efficiency of waste removal. Disease prevention in fish culture is, to a large degree, a function of the nature of a facility and how it is managed.

Nutrition and Feeding

Proper feeding of a nutritious diet is important, not only for growth and prevention of nutritional deficiencies, but also for the overall health and vigor needed to cope with a variety of disease agents. Fish under intensive culture rely entirely upon the nutritive quality of artificial feeds. Diet selection, feeding frequency, and quantities fed are controlled by the fish culturist. "Demand"

feeders, triggered by the fish themselves, are being widely adopted for use in the culture of salmonids. These labor-saving devices permit the fish to feed when they desire and appear to be practical and useful in minimizing environmental stresses associated with feeding behavior and accumulated metabolic wastes.

Nutritional problems, arising from dietary imbalances, continue to cause problems in cultured fish even though great advances have been made in the knowledge of the nutrient needs of fish. Work by Paterson et al. (1981) shows that fish nutrition plays a significant role in determining the host's ability to resist infectious disease. Paterson's work demonstrated BKD to be least serious in Atlantic salmon fed high levels of trace minerals and low levels of calcium. BKD probably cannot be eliminated by simply adjusting the mineral composition of the diet, but dietary manipulation is an interesting concept that broadens the diversity of methods that can be integrated into a fish health management program to reduce the severity of complex disease problems.

Genetic Resistance to Disease

The idea of genetically enhancing the resistance of fish to disease has tantalized workers for many years (Snieszko et al. 1959). Field and laboratory results periodically raise new optimism for selection of stocks with increased resistance to disease in cultured salmonids (McIntyre 1977). Fish are known to adapt to disease in nature, and these traits of resistance can be measured experimentally.

There are several problems involved in the process that make it clear that the development of disease-resistant strains of salmonids may be a difficult objective to attain. Ehlinger (1977) found that some brook trout strains that were selected for their resistance to furunculosis, had acquired a greater susceptibility to bacterial gill disease during the selection progress. According to Winter et al. (1980), some strains of West coast steelhead trout, that were resistant to BKD, were those that were also most susceptible to *Vibrio anguillarum* infections encountered on migration to the sea. The loss of genetic diversity in a selection process makes it difficult to develop strains of fish that are resistant to several diseases at once. Generally, by maintaining a high level of genetic diversity in a stock and by developing hybrid vigor, there should be potential for breeding fish strains with an enhanced ability to withstand stress and infectious disease agents (P. Ihssen, Ontario Min. Nat. Res., Maple, Ontario, personal communication, 1982).

The process of selecting strains of fish that are resistant to a specific disease can create another problem. Disease-carrying populations of fish have been maintained at some installations to allow for "natural selection" in survivors and as a practical method of challenging selected stocks to measure any increases in resistance. Fish strains to be tested were held in water that already had passed through an infected population. Survivors of ensuing epizootics were retained for breeding purposes. Unfortunately, this process requires the perpetuation of virulent fish pathogens and long-term maintenance of infected carriers. The risk is not severe if the pathogen involved is not transmissible from the parent stock to their progeny via eggs produced by carriers. However, vertically transmissible diseases, such as infectious pancreatic necrosis and BKD, could be spread to any location to which the eggs of the disease-resistant stock are

shipped. Until better procedures are available, other methods should be used to solve fish disease problems which have fewer complications than the process of selecting survivors of disease outbreaks.

IMMUNIZATION

Rapid progress has been made in research on the immune responses of fish and in the development of immunization procedures. As a result, licensed vaccines are now available against vibriosis, enteric redmouth, and furunculosis diseases. These vaccines do not provide absolute protection from infection but do help fish combat infections sufficiently to make immunization cost-effective in many situations where these specific diseases cause repeated problems. The significant points are that salmonids have a sensitive immune response system that can be stimulated by immunization and that vaccine delivery systems have now been developed to efficiently immunize large numbers of fish with a minimum of labor and stress.

Vaccines are still needed to reduce the impact and spread of bacterial kidney disease, infectious pancreatic necrosis, and infectious hematopoietic necrosis. These diseases are caused by obligate fish pathogens that are difficult to control by therapeutants and may be transmitted from place to place with eggs taken from infected adults. Immunization has been a highly successful tool for controlling viral diseases in humans and domestic animals. Fryer et al. (1976a) successfully vaccinated sockeye salmon against infectious hematopoietic necrosis virus. On the other hand, early work on the development of a vaccine for the control of BKD was not highly promising (Fryer et al. 1976b).

FACILITY SANITATION AND DISINFECTION

Little information has been published regarding fish cultural facility sanitation methods, disinfection procedures, or tests determining which measures are most effective in meeting the needs of different types of facilities. Herman's 1970 review cited only the work of O'Donnell (1947) as a source of written information.

The goal of a sanitation program is to prevent the transfer of fish pathogens from one point to another. Egg disinfection strives to prevent the vertical transmission of pathogens from the parent stock to the progeny and to prevent horizontal transmission from the egg facility to the rearing facility (Bullock et al. 1978). During the rearing of fish, sanitation measures can be helpful in maintaining different stocks of fish in isolation from one another. In the event that infectious disease or parasites are detected in one group of fish, sanitary procedures can confine pathogens to a single group of fish or to a single rearing unit. To be effective, measures of this kind require proper equipment, appropriate chemicals, and discipline.

The methodical and thorough use of disinfectants can remove fish pathogens from an entire facility provided that re-entry through the water supply or other source has been blocked. Disinfection can be carried out in phases or in a single, facility-wide operation. Phased disinfections (McDaniel 1971) can be performed whenever a facility cannot be de-populated and disinfected in a single operation. Total facility disinfection disrupts fish production but it is easier to carry out. There is also a better chance of success in a total facility disinfection

than in a phased operation because re-contamination is less of a problem in a total disinfection operation. Many fish cultural facilities incorporate a routine program for the phased disinfection of rearing units to prevent transmission of pathogens from older year-classes to younger fish. Even if these methods of fish disease prevention are faithfully applied, there is no absolute guarantee that the facility will be free of fish health problems. Some methods may be more useful than others and each fish cultural operation must develop its own sanitation policies and practices. When disease outbreaks occur, however, prompt technical assistance is needed for an accurate diagnosis and prompt corrective action. The containment measures to be taken depend upon the kind of causative agent and the circumstances involved. If an obligate fish pathogen is responsible, steps may be taken that could eliminate the problem entirely. On the other hand, if a common facultative disease agent is found, long-term management of the host-pathogen relationship may be the only feasible approach.

THE CONTROL OF FISH DISEASES CAUSED BY OBLIGATE FISH PATHOGENS

Infectious disease occurs when a virulent pathogen, whether obligate or facultative, is able to overwhelm the defense mechanisms of a susceptible host under environmental conditions that are conducive to the disease process. The assignment of fish pathogens to obligate or facultative categories implies an understanding, albeit imperfect, of the pathogens involved and the epizootiology of the diseases they cause. As this understanding increases, so do the possibilities for development of control strategies.

There are three kinds of control measures for coping with infectious diseases: (1) those that reduce or eliminate the source of infection; (2) those that break the connection between the source of infection and susceptible fish populations; and, (3) those that reduce the susceptibility of fish that are exposed to disease. These procedures are most pertintent to the control of diseases caused by obligate pathogens but they also have application in the control of other diseases. In the control of disease in higher animals, the measures are applied in programs that define the distribution of the disease, restrict its known range, and work within that range to eliminate the disease or minimize its impact.

To reduce or eliminate sources of infection, accurate disease diagnostic techniques and sensitive pathogen detection methods are basic requirements. How diseases are spread from fish to fish and from place to place must be determined. As more is learned, steps may be taken to prevent the spread of disease by controlling the transfer of infected fish or eggs in areas believed free of disease. Quarantine measures have been useful in containing outbreaks of disease in new areas after a disease control program has been put into operation. The elimination of infected carriers from a facility's water supply and specific therapy programs can reduce disease problems to the point where eventually they can be eradicated from a facility, a watershed, or an entire river basin system.

Breaking the connection between the source of infection and susceptible fish populations is the second measure that may be applied. This step can be initiated as soon as research findings show which methods might be effective even though significant sources of infection still exist. Broodstock populations

which carry disease agents can be treated or eliminated. Stream water supplies may harbor infected carriers but the connection between the sources of infection and the cultured fish can be broken through the use of water sterilization equipment. When "wet" diets that included raw fish products were fed to Pacific salmon fingerlings, the diet exposed them to fish tuberculosis and other diseases. After the development of the Oregon moist pellet, fish 'were fed only pasteurized fish products instead of raw salmon viscera and carcasses. Pasteurization broke the link between the source of infection and susceptible fish. Fish tuberculosis is now seldom detected (Wood 1974). Disinfection of rearing facilities between uses by different year-classes of fish can also break the connection between an infected stock and the next group of fish to be reared.

If it is assumed that the ability of a pathogen to cause disease (virulence) is an intrinsic characteristic of a given microbe, then procedures that help tilt the balance in favor of the host fish will be those that reduce their susceptibility. Susceptibility to disease is governed, not only by factors from within the fish, such as species and strain of fish, immunity, and age, but also by the fish's ability to adjust physiologically to changes in the external environment. Fish culturists often can help by adjusting environmental conditions to reduce adverse affects. Ingenious methods have been found to regulate water temperatures, alter oxygen and other dissolved gas levels, reduce ammonia and nitrite levels, reduce population densities, and to improve handling methods to protect the integrity of the skin, scales and mucous membranes of fish. These measures also help in the control of diseases caused by facultative fish pathogens. However, these agents often are widespread in the environment and all available techniques may have to be integrated into a broad fish health protection program before long-term fish disease control is attained.

INTEGRATED FISH HEALTH MANAGEMENT

Many diseases caused by facultative, opportunistic disease agents are often closely related to fish cultural stresses or to adverse environmental conditions. These deficiencies must be corrected before chemotherapy will be successful for any length of time. At most facilities, there is a familiar variety of diseases that must be managed on a regular basis and "lived with." This is not unlike the situation faced by agriculturists who must control crop pests. To cope with crop pests, a system of integrated pest management (IPM) has evolved in agriculture that may have value in the culture of fish. In fish culture, as in the raising of crops, there is little potential for totally eliminating ubiquitous disease agents and a program to manage them must be established.

Sawyer (1979) reviewed the history, principles, and application of integrated pest management when he described how IPM techniques might be used in control of the sea lamprey in the Great Lakes Basin. In his article he stated that:

> "The approach is essentially one of applied systems ecology. It should be useful in almost any situation in which a complex natural or semi-natural system (hatchery) is to be managed by manipulating biological and abiotic factors that are controllable and monitoring those that are not.... There are occasional claims that IPM is what economic entomologists have been practicing for decades. While many of its compo-

nent tactics have, indeed, been used throughout the 20th century, it is only in recent years that IPM has emerged as a structured discipline with a coherent philosophical basis."

The parallels in fish culture are obvious. A form of integrated fish health management has been practiced since fish culture began.

According to Sawyer (1979), great progress has been made in developing sophisticated agricultural programs for integrated pest management that incorporate a "transdisciplinary viewpoint in which all classes of pests and their interrelationships are jointly considered, and in which crop protection is seen as contributing to the overall management of an agroecosystem. Second, it extends the early concept of integrated control as the combination of chemical and biological control to the integration of all available management techniques. This does not merely imply simultaneous, but independent, application of two or more control methods, but rather their coordination into a unified program which seeks optimal control." These same principles are directly applicable to controlling fish diseases, especially those caused by facultative fish pathogens.

In practice, IPM techniques are used in crop protection programs to keep pests below a population density that will cause an unacceptable economic loss. In fish culture, fish species and strain selection, immunization, environmental manipulation, nutrition, and chemotherapy work toward this end. There comes a point, however, when the economic threshold of fish disease control has been reached and further efforts to control or eliminate pathogens will become more costly than the benefits derived. An obvious exception would be those circumstances in which certain dangerous diseases caused by obligate pathogens cannot be tolerated at any level and eradication is the goal of the control program. The economic threshhold for diseases caused by facultative pathogens, on the other hand, may be more difficult to determine. Through experience with varying environmental conditions and changing fish populations, the fish culturist learns to recognize early warning signals and to make timely corrections. Through experience, the economic impact of a disease can be more clearly identified and a better understanding of the merits of additional control measures then can be developed.

SUMMARY

The control of obligate fish pathogens is based on identifying sources of infection, breaking the connection between such sources and susceptible fish populations, and on reducing the susceptibility of exposed fish. A broader strategy that integrates *all* available management techniques is required for the control of diseases caused by facultative fish pathogens. A fully integrated fish health management program is an orchestration of all available preventive and control tactics rather than a reliance upon a few specific techniques such as chemotherapy, immunization, or developing genetic resistance. The collective strength of the various elements in an integrated program, each working in synchrony with the others, can potentially achieve better results than would be obtained by use of any single method. Continued progress in integrated fish health management is dependent upon the continuous development of new tools and techniques through research to replace those lost through changes in needs

and changes in the kinds of problems to be solved. Progress is dependent also upon the willingness of fish culturists, fish pathologists, and conservation agency administrators to take an interdisciplinary viewpoint. This requires close cooperation between public and private sectors of fish culture and between the various operational levels in fish culture, research, and extension programs.

REFERENCES

Bullock, G.L., H.M. Stuckey, and D. Mulcahy. 1978. Corynebacterial kidney disease: egg transmission following iodophore disinfection. U.S. Fish Wildl. Serv., Kearneysville, WV Fish Health News 7: 51-52.

Ehlinger, N.E 1977. Selective breeding of trout for resistance to furunculosis. N.Y. Fish Game J. 24: 26-36.

Fryer, J. L., K.S. Pilcher, J.E. Sanders, J.S. Rohovec, J.L. Zinn, W.J. Groberg, and R. H. McCoy. 1976a. Temperature, infectious diseases, and the immune response in salmonid fish. EPA/ERS, Publ. No. ^{600}/3-76-021. Duluth, MN. *72* p.

Fryer, J.L., J.S. Rohovec, G.L. Tebbit, J.S. McMichael, and K.S. Pilcher. 1976b. Vaccination for control of infectious disease in Pacific salmon. Fish Pathol. 10: 155-164.

Herman, R.L. 1970. Prevention and control of fish diseases in hatcheries, p. 3-15. In S.E Snieszko, (ed.). A Symposium of Diseases of Fishes and Shellfishes, Am. Fish. Soc. Spec. Pub. No. 5. Bethesda, MD. 526 p.

McDaniel, D. W. 1971. Hager-man redmouth.. . a new look at an old problem. Am. Fishes and U.S. Trout News. 15: 14-28.

McIntyre, J.D. 1977. Heritable tolerance of disease in salmonids, p. 87-90. In Proc. Int. Symp. Dis. Cult. Salm. Tavolek, Inc., Seattle, WA.

Needham, E.A. 1977. The salmonid pathologist in 1977, p. 8-15. In Proc. Int. Symp. Dis. Cult. Salm. Tavolek, Inc., Seattle, WA.

O'Donnell, D.J. 1947. The disinfection and maintenance of trout hatcheries for the control of disease, with special reference to furunculosis. Trans. Am. Fish. Soc. 74: 26-34.

Paterson, W.D., S.P. Lall, and D. Desautels. 1981. Studies on bacterial kidney disease in Atlantic salmon *(Salmo salar)* in Canada. Fish. Path. 15: 283-292.

Sawyer, A. J. 1980. Prospects for integrated pest management of the sea lamprey *(Petromyzon* marinus). Can. J. Fish. Aquat. Sci. 37: 2081-2091.

Snieszko, S.E, C. E. Dunbar, and G.L. Bullock. 1959. Resistance to ulcer disease and furunculosis in eastern brook trout, Salvelinus fontinalis. Prog. Fish-Cult. 21: 111-116.

Wedemeyer, G.A., EI? Meyer, and L. Smith. 1976. Diseases of Fish, Book 5: Environmental stress and fish diseases. TFH Publications, Neptune City, NJ. 192 p.

Winter, G.W., C.B. Schreck, and J.D. McIntyre. 1980. Resistance of different stocks and transferrin genotypes of coho salmon *(Oncorhynchus kisutch)* and steelhead trout *(Salmo* gairdneri) to bacterial kidney disease and vibriosis. Oreg. Fish. Comm., Fish. Bull. No. 77: 795-802.

Wood, J. W. 1974. Diseases of Pacific salmon: their prevention and treatment. 2nd ed. Wash. Dep. Fish. Olympia, WA. 82 p.

PART V

IMPORTANT DISEASES OF SALMONID FISHES

INTRODUCTION

17. INFECTIOUS HEMATOPOIETIC NECROSIS

18. INFECTIOUS PANCREATIC NECROSIS

19. VIRAL HEMORRHAGIC SEPTICEMIA

20. BACTERIAL GILL DISEASE

21. BACTERIAL KIDNEY DISEASE

22. COLDWATER DISEASE

23. COLUMNARIS DISEASE

24. ENTERIC REDMOUTH DISEASE

25. FURUNCULOSIS

26. CERATOMYXOSIS

27. WHIRLING DISEASE

INTRODUCTION

J.W. WARREN
U.S. Department of the Interior
Fish and Wildlife Service
La Crosse, WI

Fish culturists have opportunities to control many of the environmental and host characteristics that are important determinants in the occurrence of fish diseases. These "producer options" have been discussed in earlier parts of this guide where emphasis was placed upon providing suitable rearing facilities, clean and adequate water supplies, good nutrition, immunization, sound genetics, and other factors that contribute to the welfare of the host and the aquatic environment. Throughout these earlier chapters, many diseases were mentioned but no attempt was made to provide detailed information or to identify and compare prevention and control techniques that could be more effective for one disease than another.

The following portion of this guide contains twelve chapters that describe the important diseases of salmonids. Similar outlines have been used to discuss each disease to assure that essential information was provided and to facilitate comparisons among diseases. Each chapter opens with a short introductory summary, a brief description of external and internal signs of the disease, and methods of diagnosis. A discussion of the epizootiology of the disease follows. Like epidemiology in public health, the epizootiology of fish diseases involves the collection and analysis of information that helps to explain when, where, why, and how diseases are introduced or transmitted, and why diseases may vary in severity. Each section on epizootiology includes information on the geographic and host ranges, sources and reservoirs of infection, modes of transmission, susceptibility and resistance factors, incubation period, and seasonal incidence. Methods of control and eradication, together with some of the problems involved, conclude each chapter. Finally, a list of references is given to provide access to the literature. Wherever possible, key review articles have been cited.

The practical value of the disease chapters goes beyond providing ready access to descriptive information and the literature. In contrast to earlier chapters, they provide a different viewpoint on the need for, and methods of, fish disease control with specific reference to the nature of the pathogen and the

help to balance earlier material related to the host and the environment. Knowledge of the dynamic relationships between virulent pathogens and susceptible hosts in fish cultural environments is essential to the development and integration of effective fish health protection procedures.

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INFECTIOUS HEMATOPOIETIC NECROSIS

J.W. WARREN
U.S. Department of the Interior
Fish and Wildlife Service
La Crosse, WI

Infectious hematopoietic necrosis (IHN) is an acute systemic viral disease of Pacific Coast rainbow and steelhead trout and sockeye and chinook salmon fry and fingerlings (Amend et al. 1969). IHN can quickly kill more than 90% of a population of young fish. More than 100 million cultured and wild trout and salmon fry have died of this disease during the past 10 yr (Mulcahy 1981a). Infectious hematopoietic necrosis is caused by a bullet-shaped virus described by McAllister et al. (1974). The pathogen can be transmitted from fish to fish and from parent to progeny via seminal fluids or infected eggs (Amend et al. 1969; Wolf et al. 1973). Asymptomatic adult carriers shed infective viral particles when spawning (Amend 1975). According to Amend and Pietsch (1972), the parent to progeny transmission of IHN virus can be interrupted by organic iodine disinfection of contaminated eggs. Mulcahy (D. Mulcahy, National Fishery Research Center, Seattle, WA, personal communication) reports, however, that there have been several outbreaks of the disease among fish hatched from disinfected eggs that had been spawned by adult carriers. No therapy will halt an outbreak once it has started.

SIGNS OF INFECTION

Infectious hematopoietic necrosis infections usually are characterized by a sudden, lethal onset but the signs of infection and the times of occurrence of epizootics vary with the host species. Wild sockeye salmon fry are most severely affected at emergence from the spawning bed and during the next two months. In chinook salmon, and steelhead and rainbow trout, losses may occur from the sac fry stage through yearlings. With the possible exception of Idaho rainbow trout, older fish rarely die from IHN. After the first signs of IHN appear, sockeye fry

generally survive for only a few hours; but chinook salmon, steelheads, and rainbows may live for a day or more.

EXTERNAL SIGNS

1. IHN should be suspected if sockeye salmon fry appear dark, lethargic, whirl through the water and suffer a sudden acute mortality. Chinook salmon and steelhead and rainbow trout may reach 100 to 30/lb before IHN strikes. Infected fish usually become dark, exophthalmic, avoid the water current, drift against the tail screen and die.
2. In quiet water, long, opaque, off-white fecal casts can be observed trailing from the vents of rainbow trout fingerlings.
3. Abdominal swelling and pale gills are additional signs commonly observed.

INTERNAL SIGNS

1. In infected sockeye salmon fry, the kidney is often translucent and speckled with pigment cells. Other organs are usually pale except for the spleen which is cherry red.
2. A clear, straw-colored fluid accumulates in the body cavity of some fish infected with IHN, but most internal signs are similar to those caused by other viral diseases.

DIAGNOSIS

During IHN outbreaks, a generalized systemic viremia develops in which the virus can be detected in almost any tissue (Amend 1974). A confirmed diagnosis requires isolation of the IHN virus from fish with typical signs of the disease and the identification of the virus by neutralization tests with anti-IHNV serum (Amend 1970a). In Southern Idaho and Oregon, rainbow trout have been found to carry dual infections of IHNV and infectious pancreatic necrosis virus (Mulcahy and Fryer 1976) so care must be taken to check for other viruses.

EPIZOOTIOLOGY

GEOGRAPHIC AND HOST RANGES

Infectious hematopoietic necrosis is common in cultured and wild salmonid populations on the Pacific coast of the U.S. and Canada from California to Alaska and inland to Idaho. The disease is ubiquitous in sockeye salmon (Amend and Wood 1972; Grischkowsky and Amend 1976; Williams and Amend 1976). In wild sockeye salmon stocks, significant natural outbreaks may kill more than 40% of the emergent fry (Williams and Amend 1976).

In Idaho, IHN has become one of the most serious fish disease problems facing fish culturists in the large commercial rainbow trout hatcheries. Fry losses commonly exceed 40% in badly infected lots. As the disease becomes established, outbreaks may occur among rainbow trout of almost any age. Records show that this disease is now a threat to much older rainbows than in the past (D.

Mulcahy, National Fishery Research Center, Seattle, WA, personal communication).

Isolated IHN outbreaks have been reported in Colorado, Minnesota, New York, South Dakota, West Virginia, and possibly elsewhere (Wolf et al. 1973; Carlisle et al. 1979). In most instances, these epizootics have been associated with the shipment of infected rainbow trout eggs.

Sources and Reservoirs of Infection

Infected adult salmonids shed IHN virus at spawning time in ovarian and seminal fluids. The proportion of adults with detectable IHN virus increases as the spawning season progresses (Amend 1975; Mulcahy 1981a). During spawning, a higher proportion of sockeye salmon females than males have been found to be IHN virus carriers (Mulcahy and Fryer 1976). Ovarian fluid, collected from spawned-out females within a week after spawning, often carries more IHN virus than fluid collected from the same fish when eggs were taken. Although Amend (1975) considered IHN virus to be an external contaminant of eggs, outbreaks of the disease among fish hatched from eggs disinfected with an organic iodine disinfectant suggests that the virus may occur within eggs or as a surface contaminant (D. Mulcahy, National Fishery Research Center, Seattle, WA, personal communication). This hypothesis merits further research since egg disinfection is sometimes assumed to be an effective preventive measure.

Modes of Transmission

Egg-associated transmission of IHN virus is the primary mode of transmission but the disease has been transmitted by several other means. Early work by Guenther et al. (1959) showed that the disease can be introduced by feeding raw, frozen salmon viscera from canneries. Amend et al. (1969; 1975) found that the virus can also be transmitted by adding infected cell culture medium to tanks containing susceptible experimental fish, by placing healthy fish in a screened-off section of a tank next to infected fish, and by feeding raw, ground, infected fry to healthy fish.

Susceptibility and Resistance Factors

Sockeye and chinook salmon and rainbow and steelhead trout are severely affected by IHN. Although other species of salmonids may occur in the same waters as these species, it appears that coho salmon and other trout species are more resistant. Early work on Sacramento River chinook disease (SRCD) (now recognized as a member of the IHN group of viral diseases) showed that SRCD did not occur at water temperatures over 15°C (Amend 1970b). This may indicate the existence of several geographic strains of IHN virus because this phenomenon applies only to chinook salmon in the Sacramento River basin (D. Mulcahy, National Fishery Research Center, Seattle, WA, personal communication). Work by Hetrick et al. (1979) showed that rainbow trout were readily infected by IHN virus at 15 and 18°C. These differing responses could be caused by interactions between different geographical strains of virus, amounts of virus present, and the strain, species, and age of the host.

INCUBATION PERIOD

Experimental work with rainbow trout fingerlings has shown that the incubation period of IHN is directly related to water temperature (Hetrick et al. 1979). Hetrick and his co-workers found the mean number of days to death following exposure to the virus ranged from as little as 5.5 d at 21°C to as long as 15.8 d at 3°C. In sockeye salmon and steelhead trout the incubation period is similar. Increasing the water temperature to 18°C for 4-6 d protects chinook salmon fingerlings against the Sacramento River strain of IHN virus rather than shortening the incubation period (Amend 1970). If these same fingerlings are exposed later at lower water temperatures, they become infected.

SEASONAL INCIDENCE

IHN disease coincides with the occurrence of susceptible stages of host fishes thereby suggesting seasonality of the disease in some species and locations. Seasonal increases in population densities and the accompanying stresses also give rise to what appear to be seasonal changes in the incidence of IHN. Since trout and salmon are spawned over most of the year, fish culturists must be continually alert to the possible occurrence of IHN.

METHODS OF CONTROL

PREVENTION

Avoiding the introduction of IHN-infected eggs and fish is the only sure method of prevention. The transfer of salmon or trout eggs or fish from California, Oregon, Idaho, Washington, British Columbia and Alaska into areas where the disease is not known to occur should be done with caution. If eggs must be obtained from these areas, they should be obtained from properly inspected stocks and should be thoroughly disinfected with an organic iodine disinfectant both prior to and after shipment (Amend and Pietsch 1972). An organic iodine disinfectant solution that provides 100 ppm iodine at pH 6.0 or higher for 10 min has been recommended (Amend 1974). Organic iodine disinfection is not a guarantee against transfer of the virus.

The infectious hematopoietic necrosis virus can be reliably detected in carrier fish only at spawning time and during epizootics (Amend 1970a, 1975). Repeated inspections employing thorough virological sampling are the best means of detecting carriers of IHN virus. However, even in carefully inspected populations, the disease can appear unexpectedly (Mulcahy 1981b).

Temperature modification to prevent losses in chinook salmon is not recommended because of the frequent occurrence of temperature-resistant virus strains, high energy costs, and the post-exposure persistence of IHN carriers (Amend, 1974).

THERAPY

No drugs or chemicals are known that will halt outbreaks of IHN.

KEY STEPS TO REMOVE THE DISEASE AND/OR AGENT FROM FISH POPULATIONS

IMMEDIATE

Certain hatchery practices will aggravate an existing IHN problem. According to Mulcahy (National Fishery Research Center, Seattle, WA, personal communication), heavy loading of eggs or fry in incubators is an important factor contributing to the severity of outbreaks. Water reuse, handling stresses and other adverse conditions increase the impact of the disease on older fish.

When an IHN outbreak is suspected, immediate isolation (quarantine) of the affected lot of fish will limit spread of the disease to other rearing units or to other lots of fish. If the number of eggs or fry in individual rearing units can be reduced at the outset, fewer fish will contract the disease if an outbreak occurs. Upon confirmation of IHN, prompt destruction of the affected fish or lot, followed by intense chlorine disinfection of all associated incubators, rearing units and equipment, is recommended (Amend 1974).

LONG TERM

Procedures for the elimination of IHN in cultured rainbow trout differ from those that might be used in wild anadromous populations of sockeye and chinook salmon and steelhead trout. When a coldwater fish hatchery has been effectively disinfected and is supplied with an uninfected water source, the key to long-term IHN control lies in obtaining reliably inspected stocks of eggs or fish only from sources shown to be free of this disease. The importation of thoroughly disinfected eggs is probably safer than importing fish, but stocks originating from the West coast of North America and from Idaho should be avoided.

The elimination of IHN from anadromous fish populations is very difficult. There is no way to effectively deal with transmission of the disease in naturally spawning fish. Adults that return to hatcheries can be inspected for the presence of IHN and other diseases, but there is seldom enough time between the arrival of adults at a facility and spawn taking to allow for tests to be able to distinguish between uninfected fish and carriers. In any event, artificially spawned eggs from anadromous stocks should always be disinfected and incubated at the lowest feasible density. If hatcheries are stream-fed, a coordinated effort should be made to prevent carrier fish from migrating into the water supply stream. IHN-eradication procedures should begin with the uppermost hatchery in the watershed. As long as eggs are available from sources free of IHN, this disease should not be considered a problem that must be “lived with”, especially in rainbow trout programs (D. Mulcahy, National Fishery Research Center, Seattle, WA, personal communication).

REFERENCES

Amend, D. F. 1970a. Approved procedure for determining absence of infectious hematopoietic necrosis (IHN) in salmonid fishes. U.S. Fish Wildl. Serv., Fish Dis. Leafl. No. 31. Washington, DC. 4 p.

Amend, D.E 1970b. Control of infectious hematopoietic necrosis virus disease

Amend, D.E 1974. Infectious hematopoietic necrosis (IHN) virus disease. U.S. Fish Wildl. Serv., Fish Dis. Leafl. No. 39. Washington, DC. 6 p.

Amend, D.E 1975. Detection and transmission of infectious hematopoietic necrosis virus in rainbow trout. J. Wildlife Dis. 11: 471-478.

Amend, D.E, and J.P. Pietsch. 1972. Viricidal activity of two iodophors to salmonid viruses. J. Fish. Res. Board Can. 29: 61-65.

Amend, D. F., and J. W. Wood. 1972. Survey for infectious hematopoietic necrosis (IHN) virus in Washington salmon. Prog. Fish-Cult. 34: 143-147.

Amend, D.E, W.T. Yasutake, and R. W. Mead. 1969. A hematopoietic virus disease of rainbow trout and sockeye salmon. Trans. Am. Fish. Soc. 94: 796-804.

Carlisle, J. C., K. A. Schat, and R. Elston. 1979. Infectious hematopoietic necrosis in rainbow trout, *Salmo gairdneri* Richardson, in a semi-closed system. J. Fish Dis. 2: 511-517.

Grischkowsky, R. S., and D. F. Amend. 1976. Infectious hematopoietic necrosis virus: Prevalence in certain Alaskan sockeye salmon, Oncorhynchus nerka. J. Fish. Res. Board Can. 33: 186-188.

Guenther, R. W., S. W. Watson, and R.R. Rucker. 1959. Etiology of sockeye salmon "virus" disease. U.S. Fish Wildl. Serv., Spec. Sci. Rep. -Fish. No. 296. Bethesda, MD. 10 p.

Hetrick, EM., J.L. Fryer, and M.D. Knittel. 1979. Effect of water temperature on the infection of rainbow trout, *Salmo gairdneri* Richardson, with infectious hematopoietic necrosis virus. J. Fish Dis. 2: 253-257.

McAllister, P.E., J. L. Fryer, and K. S. Pilcher. 1974. Further characterization of infectious hematopoietic necrosis virus of salmonid fish (Oregon strain). Arch. Gesamte Virusforsch. 44: 270-279.

Mulcahy, D. 1981a. Detection of infectious hematopoietic necrosis virus in salmon ovarian fluid is related to stage of ripeness and sampling time within run. U.S. Fish Wildl. Serv., Res. Inf. Bull. No. 81-11. Bethesda, MD. 1 p. (mimeo.)

Mulcahy, D. 1981b. IHN virus found in all populations of Pacific Coast sockeye salmon, potentially in most kokanee populations regardless of location, and in chinook salmon from the Sacramento River drainage. U.S. Fish Wildl. Serv. Res. Inf. Bull. No. 81-10. 1 p. (mimeo.)

Mulcahy, D.M., and J.L. Fryer. 1976. Double infection of rainbow trout fry with IHN and IPN viruses. Fish Health News. 5(1): 5-6.

Williams, I.V., and D.F. Amend. 1976. A natural epizootic of infectious hematopoietic necrosis in fry of sockeye salmon (Oncorhynchus nerka) at Chilko Lake, British Columbia. J. Fish. Res. Board Can. 33: 1564-1567.

Wolf, K., M.C. Quimby, L.L. Pettijohn, and M.L. Landolt. 1973. Fish viruses: isolation and identification of infectious hematopoietic necrosis in eastern North America. J. Fish. Res. Board Can. 30: 1625-1627.

18

INFECTIOUS PANCREATIC NECROSIS

J. G. HNATH
Fisheries Division
Michigan Department of Natural Resources
Mattawan, MI

Infectious pancreatic necrosis (IPN) is a viral infection primarily of trout and salmon, but the virus has also been isolated from a wide variety of other fish species. The infection is characteristically seen in trout as an acute disease causing high mortality in fry and fingerlings. However, it may also occur as a benign and inconspicuous infection (Bullock et al. 1976). Because high fish losses are often associated with IPN outbreaks, it is considered one of the major fish disease problems in the United States, Canada, and Europe (Desautels and MacKelvie 1975).

SIGNS OF INFECTION

The first sign of a typical IPN epizootic is a sudden increase in mortality. The largest and most vigorous fry or fingerlings usually are affected first. A whirling behavior is typical when the mortality rate is high; affected individuals swim in a rotating manner about their long axis. Abnormal movements may be slow and feeble or rapid and frantic. When not otherwise obvious, the whirling response may sometimes be elicited by a sharp rap on the trough. Moribund behavior may alternate between periods of quiescence, during which victims lie on the bottom and respire weakly, and convulsive frenzies. Whirling is a terminal sign, and death usually ensues within an hour or two although this characteristic behavior may be absent among very young fish or fish of poor quality (Wolf 1966). Other signs of infection that may be observed include an overall darkening of individal fish, exophthalmia, abdominal distension, and hemorrhages in ventral areas. Tiny hemorrhages may occur among the pyloric caeca, and the liver and spleen are usually pale. The digestive tract is usually devoid of food. A clear or milky

mucus may be found in the stomach and anterior intestine, a distinctive characteristic of IPN.

DIAGNOSIS

If young trout suffer a rapidly increasing mortality, exhibit some of the signs described, and are free of pathogenic bacteria and parasites, there is a possibility that the fish have IPN (Wolf 1966). Confirmation of IPN requires isolation of the virus in cell culture and identification by a serum neutralization test using polyvalent, anti-IPN virus serum. IPN diagnostic and inspection procedures and the methods for IPN confirmation are described in the Canadian Fish Health Protection Regulations "Manual of Compliance" (Anon. 1977), in "Procedures for the Detection and Identification of Certain Fish Pathogens" (McDaniel 1979), and in Ljungberg and Jorgenson (1973).

EPIZOOTIOLOGY

Geographic and Host Ranges

IPN virus has been isolated from fish in the United Kingdom (Wolf 1972), Scandinavia (Ljungberg and Jorgenson 1973), Europe, Japan, and North America (Hill 1977). IPN virus has been isolated in brook, brown, rainbow and cutthroat trout; Atlantic, coho, chinook, amago, and himemasu salmon; and eels. IPN-like viruses have also been isolated from carp, perch, roach, bream, pike, and white suckers (Hill 1977).

Sources and Reservoirs of Infection

Infected fish serve as reservoirs of infection. During epizootics, virus particles are shed into the water with feces, eggs, and seminal and ovarian fluids. High levels of virus are present during IPN outbreaks. Surviving fish become carriers and intermittantly shed virus over a long period of time (Wolf 1966). It has also been demonstrated that viable virus can remain with eggs in spite of disinfection (Bullock et al. 1976). Yamamoto and Kilistoff (1979) have determined that brook trout infected with IPN virus at the time of stocking will harbor virus over a period of several years.

Susceptibility and Resistance Factors

Several factors affect the overall mortality rate in a population of salmonid fry infected with IPN virus. The susceptibility of salmonid fishes to IPN disease decreases with increasing age, the most susceptible fish being first-feeding fry. High resistance is usually, but not invariably, achieved at an age of 4-6 months. Another major factor determining the overall mortality from an IPN infection is the particular strain of IPN virus involved. It has been found that strains of IPN virus may differ in virulence and may produce mortalities among trout ranging from under 10% to over 90%. Most strains are able to produce high mortality but the susceptibility varies from species to species with higher mortality occuring in brook and rainbow trout than in brown trout and Atlantic salmon. The virus is also

pathogenic to amago and himemasu salmon, both species showing high mortalities (48-62%) in six and eight week old fry (Sano 1973). Although coho may serve as carriers of IPN virus, there is no report of a mortality caused by IPN in this species (Wolf and Pettijohn 1970). Other factors such as the dose of the virus, the route of infection, density of the fish population, water temperature, and the presence or absence of "stress" conditions all affect mortality (Hill 1977).

Modes of Transmission

"An important feature of IPN disease, from an epizootiological point of view, is the fact that most survivors of infection become life-long virus carriers and thus shed varying quantities of virus over a long period. This results in a typical transmission of the disease from parents to progeny via the egg, and is probably one of the main factors for the geographical spread of IPN" (Hill 1977).

According to Wolf (1966): "Quite likely, egg transmission is the normal means by which the virus is passed from one generation to another."

Incubation Period

The incubation period of IPN infection is temperature dependent, ranging from 6 d at 125°C to several weeks at 4°C (Wolf 1966).

METHODS OF CONTROL

Prevention

Avoidance is the most effective control measure. This requires the incubation of virus-free eggs and the propagation of IPN-free stock in an uncontaminated water supply. This approach is the method of choice. Success depends on a rigorous fish health inspection program to prevent the introduction or inadvertant spread of IPN.

Under circumstances where avoidance is not possible, the mortality associated with IPN may be reduced by rearing fry for 4 months at colder water temperatures (less than 7°C). Even so, the problem of the carrier state in surviving fish persists. This procedure may reduce loss rates because the cold water temperatures during the period of greatest fry susceptibility are cold enough to prevent mortality. Cold water temperatures also appear to have a depressing effect upon infectivity and/or transmission of the virus (Frantsi and Savan 1971).

If a hatchery must operate with water from streams containing IPN virus carriers, the water should be treated to eliminate the IPN virus. Ozone appears to be effective for this (Wedemeyer et al. 1978).

Therapy

There is no effective treatment for IPN but Economon (1972) has reported some success with povidone iodine treatments.

KEY STEPS TO REMOVE THE DISEASE AND/OR AGENT FROM FISH POPULATIONS

IMMEDIATE

As there is no effective therapy, the only immediate control of the virus is to eliminate infected stocks.

LONG TERM

Inspect all egg sources and hatchery fish populations at least annually for the presence of IPNV.

Phase out all IPN virus infected broodstock and, in the interim, do not transfer eggs from infected broodstocks to any hatcheries which are free of the disease.

Under no circumstances should infected fish be stocked into lakes, reservoirs, or streams that serve as water sources for hatcheries or that serve as broodstock sources. There is evidence that planting only IPN virus-free fish into areas previously planted with IPN virus carriers may ultimately lead to a carrier-free state (Yamamoto and Kilistoff 1979).

Any fry or fingerlings suffering from clinical IPN disease should be incinerated or buried with unslaked lime.

REFERENCES

Anonymous 1977. Fish health protection regulations manual of compliance. Can. Dep. of Fish. and Env., Fish. and Mar. Serv., Misc. Spec. Publ. 31. Ottawa, Canada. 36 p.

Adair, B.M., and H. W. Ferguson. 1981. Isolation of infectious pancreatic necrosis (IPN) virus from non-salmonid fish. J. Fish. Dis. 4: 69-76.

Bucke, D., J. Finlay, D. McGregor, and C. Seagrave. 1979. Infectious pancreatic necrosis (IPN) virus: its occurrence in captive and wild fish in England and Wales. J. Fish. Dis. 2: 549-553.

Bullock, G.L., R.R. Rucker, D. Amend, K. Wolf, and H.M. Stuckey. 1976. Infectious pancreatic necrosis: transmission with iodine-treated and non-treated eggs of brook trout (Salvelinus fontinalis). J. Fish. Res. Board Can. 33: 1197-1198.

Desautels, D., and R.M. MacKelvie. 1975. Practical aspects of survival and destruction of infectious pancreatic necrosis virus. J. Fish. Res. Board Can. 32: 523-531.

Economon, P.P. 1972. Experience paper: Polyvinyl pyrrolidone iodine as a control for infectious pancreatic necrosis of brook trout. FAO, United Nations, EIFAC 72/SC II-Symp. 12. Rome, Italy 8p.

Frantsi, C. and M. Savan. 1971. Infectious pancreatic necrosis virus - temperature and age factors in mortality. J. Wildl. Dis. 7: 249-255.

Hill, B.J. 1977. Present status of IPN virus, p. 116-119. In Proc. Int. Symp. Dis. Cult. Salm., Tavolek, Inc., Seattle, WA.

Ljungberg, O., and P.E.V. Jorgensen. 1973. Infectious pancreatic necrosis (IPN) of salmonids in Swedish fish farms, p. 67-70. In W.A. Dill (ed.) Symposium on the major communicable fish diseases in Europe and their control. FAO, United Nations, EIFAC/T17 (Suppl. 2) FAO, Rome, Italy.

McDaniel, D. (ed.). 1979. Fish health bluebook. Procedures for the detection and identification of certain fish pathogens. Am. Fish. Soc., Fish Health Sec. Bethesda, MD. 119 p.

Nicholson, B.L., G.W. Thorne, C. Janicki, and A. Hanson. 1979. Studies on a host range variant from different isolates of infectious pancreatic necrosis virus (IPNV). J. Fish Dis. 2: 367-379.

Sano, T 1973. Studies on viral diseases of Japanese fishes - IV Infectious pancreatic necrosis of rainbow trout: Susceptibility of fresh water salmons of genus Oncorhynchus. Bull. Jap. Soc. Sci. Fish. 39: 117-120.

Stephens, E.B., M. W. Newman, A.L. Zachary, and EM. Hetrick. 1980. A viral aetiology for the annual spring epizootics of Atlantic menhaden *Brevoortia tyrannus* (Latrobe) in Chesapeake Bay. J. Fish Dis. 3: 387-398.

Wedemeyer, G. A., N. C. Nelson, and C. A. Smith. 1978. Survival of the salmonid viruses infectious hematopoietic necrosis (IHNV) and infectious pancreatic necrosis (IPNV) in ozonated, chlorinated and untreated waters. J. Fish. Res. Board Can. 35: 875-879.

Wolf, K. 1966. Infectious pancreatic necrosis (IPN) of salmonid fishes. U.S. Fish Wildl. Serv., Fish Dis. Leafl. No. 1. Washington, DC. 4 p.

Wolf, K. 1972. Advances in fish virology: A review 1966-1971, p. 305-331. *In* L. E. Mawdesley-Thomas (ed.). Diseases of fish. Symp. Zool. Soc. Lond., No. 30. Academic Press. 380 p.

Wolf, K., and L. L. Pettijohn. 1970. Infectious pancreatic necrosis virus isolated from coho salmon fingerlings. Prog. Fish-Cult. 32: 17-18.

Yamamoto, T. 1975. Frequency of detection and survival of infectious pancreatic necrosis virus in a carrier population of brook trout *(Salvelinus fontinalis)* in a lake. J. Fish. Res. Board Can. 32: 568-570.

Yamamoto, T. 1979. Infectious pancreatic necrosis virus: Quantification of carriers in lake populations during a 6-year period. J. Fish. Res. Board Can. 36: *562-567.*

19

VIRAL HEMORRHAGIC SEPTICEMIA

J. W. WARREN
U.S. Department of the Interior
Fish and Wildlife Service
La Crosse, WI

Viral hemorrhagic septicemia (VHS) is an acute to chronic viral disease of salmonids that causes serious economic problems in rainbow trout cultured in several European countries. Although this disease has not been detected in North America, it is included here as a disease of significant concern. The disease was first recognized by Schaeperclaus in Germany in 1938. In 1949, the disease was named Egtved disease after an outbreak in Denmark near a village of that name. In 1966, the Office Internationale d'Epizooties recommended that the name be changed to viral hemorrhagic septicemia to reduce confusion due to the names used in the various European countries where it occurs (Roberts 1978).

Viral hemorrhagic septicemia is caused by a bullet-shaped virus similar in size and shape to the virus that causes infectous hematopoietic necrosis (IHN) in North American salmonids. The VHS virus can be distinguished from IHN by specific serum neutralization tests (McAllister et al. 1974). Losses to VHS among infected rainbow trout fingerlings often exceed 90%. If fish are exposed for the first time at older ages, the resultant disease is more chronic and has a more prolonged course. Losses can be severe in cold water under crowded, stressful situations, even among older fish. The disease is transmitted by contact and from fish to fish through the water (Rasmussen 1965). If parent to progeny transmission occurs, it is suspected that the virus is spread as a contaminant during spawning operations rather than by the virus being carried within the egg (Roberts 1978). Outbreaks of VHS are most common and most severe during the winter. Losses taper off in the spring as water temperatures rise, cease during the summer, then recur sporadically in the fall. As for other viral diseases, there is no therapy for VHS. Avoidance is the only successful control technique (Ghittino 1965).

SIGNS OF INFECTION

Viral hemorrhagic septicemia appears first in fingerlings which have been feeding for 40 d or more. Early clinical signs can easily be confused with other viral, bacterial or parasitic infections. Later in the course of the disease, signs of excitability become apparent, including erratic swimming similar to the behavior of trout dying of whirling disease caused by the parasite *Myxosoma cerebralis* (Roberts 1978).

External Signs

1. Rainbow trout involved in acute outbreaks of VHS are dark in color, lethargic, and exhibit hemorrhages in the fin sockets. Exophthalmia (popeye) is common and persists throughout the course of the disease.
2. As the disease progresses, affected fish become nearly black. An acute anemia develops, and the gills are pale in color.
3. After several months, the mortality finally may cease and some of the remaining fish often display whirling behavior, erratic swimming and nervousness.

Internal Signs

1. During acute outbreaks, small hemorrhages are common in the musculature, gills, and visceral organs. Massive hemorrhages can often be found in the abdominal cavity of freshly dead fish (Roberts 1978).
2. During mid-stages of a disease outbreak, internal organs become very pale. Visceral, intramuscular, and gill hemorrhages develop as distinctive signs of the disease.
3. In late stages of the disease, kidneys become swollen and discolored.

DIAGNOSIS

A confirmed diagnosis of VHS can be made only by isolating and serologically identifying the causative virus in an appropriate cell culture system (Jorgensen 1974). The VHS virus is similar to the agent of IHN in that it can be isolated only from fish during an active epizootic or from ovarian fluid from adult carriers at the time of spawning or shortly thereafter. The virus can seldom be isolated from asymptomatic fish at other times.

EPIZOOTIOLOGY

Geographic and Host Ranges

Numerous, severe outbreaks of VHS have occurred in Denmark, Germany, France, Italy, Switzerland and the Scandanavian countries. It has not been detected in the British Isles, in North or South America, or in the Far East (Roberts 1978).

VHS is especially serious among hatchery-reared rainbow trout. Cultured Atlantic salmon and brown trout have suffered VHS outbreaks. Brook trout have

been experimentally infected in the laboratory. All reported cases of VHS occurred among cultured fish. No natural outbreaks have been observed in wild fish populations (Roberts 1978).

Sources and Reservoirs of Infection

There is strong circumstantial evidence that survivors of VHS epizootics become asymptomatic carriers and serve as reservoirs of infection. The primary sources of VHS, therefore, involve the transfer of asymptomatic carriers to new areas, shipments of eggs contaminated by virus-laden ovarian fluids, or water-borne infectivity from upstream epizootics.

Modes of Transmission

Experimental transmission of VHS has been accomplished by the injection of infected cell culture fluid (Jensen 1965), by brushing infected fish tissue suspensions on the gills of healthy fish (Jorgensen 1970), by injecting filtrates of organ homogenates, and from fish to fish through flowing water (Rasmussen 1965). The virus survives for more than 24 hr in 14°C water and for at least a week in the dry state (Jorgensen 1974). Potential transmission can occur through virus-contaminated nets, boots, egg crates or other equipment.

Jorgensen (1970) demonstrated that VHS virus could be transmitted as an external contaminant of eggs. Ghittino (1965) attributed a 1964 outbreak he observed to VHS-infected eggs. These findings are similar to those of Amend (1975) in his work with IHN virus.

Susceptibility and Resistance Factors

Although VHS is predominantly a disease of rainbow trout, other species can be affected. The course and severity of VHS appears to be age-dependent (Roberts 1978; Bellet 1965). Losses to VHS are most severe among fingerling rainbow trout 2-6 months of age; yearling fish often have a milder case and many survive. Rainbow trout older than two years are almost completely refractile to infection (Bellet 1965).

Stress plays a significant role in the development of epizootics. Overcrowding, malnutrition, handling, and transportation can lead to high losses due to VHS. Under optimal fish cultural conditions, few fish will show signs of the disease until a stressful condition finally occurs (Jorgensen 1974).

Water temperature also plays an important role in the course of VHS and the disease is a more serious problem at water temperatures colder than 15-16°C (Bellet 1965). This helps to explain why VHS is so severe in fingerling trout during their first winter (Roberts 1978).

Incubation Period

Losses to VHS occur 6 d after gill exposures at 15°C and at 8-11 d if the virus is added to the water at 10°C (Jorgensen 1974). Losses usually continue for several months thereafter (Rasmussen 1965).

On Danish trout farms, the incubation period is reported to vary from 7-15 d. If fish cultural conditions are favorable for the fish, incubation periods of up to a year or more may occur following exposure to VHS virus through the water supply from upstream epizootics (Jorgensen 1974).

Seasonal Incidence

Viral hemorrhagic septicemia is a winter and spring time disease. Outbreaks are most severe when water temperatures are below 8-10°C. At hatcheries supplied with cold spring water or well water, the disease can be a year-round problem (Rasmussen 1965).

METHODS OF CONTROL

Prevention

Avoidance of VHS is by far the best approach to control (Ghittino 1965). No fish or eggs should be introduced from areas where VHS has been detected.

Therapy

There is no cure for VHS. Before discovery of the viral etiology of VHS, several European veterinarians believed the disease was the result of nutritional deficiencies (Rasmussen 1965; Ghittino 1965). The finding of a viral agent explained why nutritional supplements and antibacterial treatments failed to control losses (Rasmussen 1965; Bellet 1965).

KEY STEPS TO REMOVE THE DISEASE AND/OR AGENT FROM FISH POPULATIONS

Immediate

Viral hemorrhagic septicemia has not been recognized outside of Europe. If VHS is detected in new areas, an immediate quarantine should be placed on the affected facility. Water flows should be turned off to prevent release of a contaminated effluent and possible spread of the virus downstream. Destruction and proper disposal of the fish, followed by prompt, thorough disinfection of all facilities and associated equipment should commence as soon as possible. Valuable time should not be wasted on deliberations on what to do when the disease has been confirmed. A serious problem associated with possible spread of the disease exists until the water supply has been turned off, the fish buried, and the facility disinfected. The source of the infection must be identified and corrective measures must be undertaken to ensure that reinfection does not occur.

Long term

Danish authorities, prompted by catastrophic fish losses due to VHS, initiated a voluntary disease eradication plan in 1965. According to Jorgensen (1977), hatcheries were inspected and a register of disinfected or clean farms

was established. In 1969, the Danish Parliament made the voluntary program mandatory. Between 1965 and 1976, a total of 481 farms were depopulated of fish, disinfected, and repopulated with VHS-free fish. Reinfection, after disinfection, was common in Danish hatcheries unless neighboring facilities had also been treated. By June 1977, 380 farms had been registered as free of VHS.

According to Ghittino (1965), "The best prophylaxis against VHS is the perfect isolation of healthy fish from any possible source of infection." To date, hatcheries and fisheries resources in the Americas have been free of VHS. Vigilence, understanding, and strict control over introductions of salmonid eggs or fish from Europe are essential if this status is to be maintained.

REFERENCES

Amend, D.E 1975. Detection and transmission of infectious hematopoietic necrosis virus in rainbow trout. J. Wildl. Dis. 11: 471-478.

Bellet, R. 1965. Viral hemorrhagic septicemia (VHS) of rainbow trout in France. Ann. N.Y. Acad. Sci. 126(1): 461-467.

Ghittino, P. 1965. Viral hemorrhagic septicemia (VHS) of rainbow trout in Italy. Ann. N.Y. Acad. Sci. 126(1): 468-478.

Jensen, M.H. 1965. Research on the virus of Egtved disease. Ann. N.Y. Acad. Sci. 126(1): 422-426.

Jorgensen, P.E.V. 1977. Surveillance and eradication of diseases from hatcheries, p. 72-73. In Proc. Int. Symp. Dis. Cult. Salm., Tavolek, Inc., Seattle, WA.

Jorgensen, I? E. V. 1974. A study of viral diseases in Danish rainbow trout, their diagnosis and control. Ph.D. thesis. Danish Royal Vet. and Agri. Univ. Copenhagen. 101 p.

Jorgensen, P.E.V. 1970. The survival of viral hemorrhagic septicemia (VHS) virus associated with trout eggs. Riv. Ital. Piscic. Ittiopat. 5: 13-14.

McAllister, P.E., J.L. Fryer, and K.S. Pilcher. 1974. An antigenic comparison between infectious hematopoietic necrosis virus (OSV strain) and the virus of hemorrhagic septicemia of rainbow trout, *Salmo gairdneri* (Denmark strain) by cross neutralization. J. Wildl. Dis. 10: 101-103.

Rasmussen, C.J. 1965. A biological study of the Egtved disease (INUL). Ann. N.Y. Acad. Sci. 126(1): 427-460.

Roberts, R. J. 1978. The virology of teleosts, p. 128-130. In R. J. Roberts (ed.) Fish pathology, R. J. Roberts (Ed.), Balliere Tindall, London.

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BACTERIAL GILL DISEASE

JOHN H. SCHACHTE
New York Department of
Environmental Conservation
Rome, NY

Bacterial gill disease (BGD) is a common external infection of hatchery-reared salmonids and occasionally of warm water species reared under intensive conditions. As defined by Wood (1974), the name of the disease describes the clinical signs of bacterial infections on the gills. The etiological agent of the disease is considered to be one or more species of filamentous bacteria including *Flavobacterium* sp. as most recently described by Wakabayashi et al. (1980). BGD is characterized by the presence of large numbers of filamentous bacteria on the gills accompanied by fusing and clubbing of the gill filaments. Acute or chronic forms of the disease may occur and acute outbreaks may involve daily mortality rates approaching 20% (Warren 1981). The onset of bacterial gill disease usually follows a deterioration of environmental conditions associated with overcrowding and increases in toxic metabolic waste products. According to Wood (1979), fish smaller than 90-100/lb are most susceptible to the disease. Successful treatment can only be accomplished through prompt therapy and alleviation of poor environmental conditions.

SIGNS OF INFECTION

Affected fish are usually lethargic and a loss of appetite occurs. Large numbers of diseased fish gather near the screen or outlet of the pond. Acute epizootics may result in a 20 to 50% mortality in 24 h (Warren 1981; McDaniel 1979). Microscopic examinations of wet mounts of the gills usually reveal extensive clubbing of the gill filaments, lamellar fusion, excess mucus, and detritus. Large numbers of filamentous bacteria adhering closely to the gills are evident but necrosis of the gill tissue is seldom present. This is in sharp contrast to

columnaris disease on the gills which may cause extensive necrosis and erosion of gill filaments.

DIAGNOSIS

Diagnosis of bacterial gill disease depends on the detection of large numbers of filamentous bacteria on the gills. Along with the clinical signs described above, this observation represents diagnostic evidence of the disease (Snieszko 1981). In the absence of large numbers of bacteria under gill wet mount examination, gills with signs of clubbing, filament fusion, etc., should be homogenized on a slide, streaked, stained with methylene blue, and examined under oil immersion. This technique will frequently reveal the presence of the filamentous organisms that might not have been observable in a wet mount. Isolation of a pure culture of the bacteria is not necessary for diagnostic purposes (Snieszko 1981).

EPIZOOTIOLOGY

Geographic and Host Ranges

Snieszko (1981) reported that bacterial gill disease is probably cosmopolitan in its distribution and that nearly all species of intensively cultured freshwater fish are potential hosts. All species of cultured salmonids are susceptible, as are such coolwater species as fingerling walleyes and tiger muskies (northern x muskellunge hybrids).

Sources and Reservoirs of Infection

Little is known about the biology and survival of the etiological organism. Carrier fish or contaminated water are considered to be sources of infection.

Mode of Transmission

Carrier fish in the rearing units or a contaminated water source probably introduce the disease. However, epizootics are almost always associated with a deterioration of environmental conditions. Wood (1974) reported that bacterial gill disease in chinook salmon appears to be associated with pond loadings in excess of 5-6 lb of fish per gpm of inflow but he was unable to correlate outbreaks in fall chinooks to levels of unionized ammonia. This is contrary to information reported by Follet and Grischkowsky (1981) and Burrows (1964) who found that unionized ammonia was clearly a factor in epizootics.

Susceptibility and Resistance Factors

No species of freshwater fish are known to be resistant to the disease. Fingerlings less than 100/lb in size are particularly susceptible. However, Davis (1926) and Bullock (1972) reported that previously-infected fish and salmonids over one year of age, respectively, seldom develop the disease. Fingerling fish infected with bacterial gill disease will frequently become reinfected if crowding or other poor environmental conditions persist after treatment. Klontz (1979)

has proposed that certain chemical treatments, such as formalin and quaternary ammonium compounds, may have deleterious effects that predispose susceptible fish to bacterial gill disease.

INCUBATION PERIOD

Snieszko (1981) reported that the incubation period for bacterial gill disease may vary from a few days to several weeks. This required period depends upon environmental conditions, age of the fish, and virulence of the infecting organisms.

SEASONAL INCIDENCE

Bacterial gill disease is most prevalent in salmonids during the spring and early summer months. This coincides with the period when young fish are most abundant, when feeding rates are increasing, and crowding develops.

METHODS OF CONTROL

PREVENTION

Maintenance of a high quality environment is of utmost importance in the prevention of bacterial gill disease. According to Wood (1974), fish should be reared in a system with no reuse of water until they reach a size of at least 100 fish/lb. Population level should be kept at lowest feasible levels to reduce the effects of crowding. The applicaton of good sanitation practices is important. Clean ponds provided with an adequate flow of clean water coupled with prompt removal of dead or weak fish will reduce incidence of the disease.

THERAPY

A number of compounds are effective for the treatment of bacterial gill disease. Wood (1974) recommended potassium permanganate ($KMnO_4$) with certain precautions at l-2 ppm. Snieszko (1981) listed Hyamine 1622 and 3500, as well as Roccal, to be used at l-2 ppm calculated on the basis of active ingredient. Diquat, at 8.4-16.8 ppm of the formulation, has also been recommended. Another quaternary ammonium compound, Purina Four Power, used at 3-4 ppm as one-hour flush treatments, has been found effective in situations where hardness has limited the use of other compounds. Chloramine-T used in a single treatment at 10 ppm in a one-hour flush has given good results in Europe, but its effectiveness and toxicity are greatly affected by water quality (From 1980). However, due to the carcinogenicity of Chloramine T, its use in this country is unlikely (Snieszko 1981). Most compounds require multiple applications for effective results.

KEY STEPS TO REMOVE THE DISEASE AND/OR AGENT FROM FISH POPULATIONS

IMMEDIATE

Avoid crowding and handling of fish during periods of high susceptibility. Practice good sanitation and keep waste products as low as possible. Avoid rearing fish smaller than 100 fish/lb in reused water or in ponds supplied with water from a source harboring carrier fish.

LONG TERM

Where possible, use closed, one-pass, water use systems to minimize the development of predisposing conditions. Disinfection of water by ultraviolet irradiation has also been shown to be effective in intensive culture conditions.

REFERENCES

Bullock, G. L. 1972. Studies on selected myxobacteria pathogenic for fishes and on bacterial gill disease in hatchery reared salmonids. U.S. Fish Wildl. Serv., Tech. Pap. 86. Washington, DC. 16 p.

Burrows, R.E. 1964. Effects of accumulated excretory products on hatchery-reared salmonids. U.S. Fish Wildl. Serv., Res. Rep. No. 66. 12 p.

Davis, H. S. 1926. A new gill disease of trout. Trans. Am. Fish. Soc. 56: 156-160. Follet, J. E. and R. S. Grischkowsky. 1981. Bacterial Gill Disease, p. 371-378. In R.A. Dieterick (ed.) Alaskan Wildlife Diseases. University of Alaska, Fairbanks, Alaska.

From, J. 1980. Chloramine-T for the control of bacterial gill disease. Prog. Fish-Cult. 42: 85-86.

Klontz, G. W. 1979. Fish health management. Vol. II. Concepts and methods of fish disease epidemiology. Fish. Res. and Off. Cont. Ed., Univ. of Idaho, Moscow, ID. 142 p.

McDaniel, D. (ed.). 1979. Fish health bluebook. Procedures for the detection and identification of certain fish pathogens. Am. Fish. Soc. Fish Health Sec. 118 p. Bethesda, MD.

Snieszko, S. F. 1981. Bacterial gill disease of freshwater fishes. U.S. Fish Wildl. Serv., Fish Dis. Leafl. No. 62. Washington, DC. 11 p.

Wakabayashi, H., S. Egusa and J. L. Fryer. 1980. Characteristics of filamentous bacteria isolated from a gill disease of salmonids. Can. J. Fish. Aquat. Sci. 37(10): 1499-1504.

Warren, J. W. 1981. Diseases of hatchery fish. A fish disease manual. U.S. Fish Wildl. Serv., Reg. 3, Twin Cities, MN. 91 p.

Wood, J. W. 1974. Diseases of Pacific salmon: their prevention and treatment. Wash. Dept. Fish., Olympia, WA. 82 p.

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BACTERIAL KIDNEY DISEASE

J.W. WARREN
U.S. Department of the Interior
Fish and Wildlife Service
La Crosse, WI

Bacterial kidney disease (BKD) of salmonid fishes is a slowly progressive, systemic infection with a protracted course and an insidious nature. Characteris-

Bacterial kidney disease in trout and salmon is sometimes characterized by large abscesses in the kidney. (G. Camenisch Missouri Dept. of Conserv.)

tics of the causative organism, the disease, its epizootiology, and methods of control were recently reviewed by Fryer and Sanders (1981). The bacterium causing BKD is a small Gram-positive diplobacillus named Renibacterium *salmoninarum* (Sanders and Fryer 1980). The pathogen can be transmitted from fish to fish (Mitchum and Sherman 1981) or from adults to their progeny via eggs (Bullock 1980; Bullock et al. 1978). Infected fish may take months to show signs of disease. Water temperature, water hardness, numbers of organisms present, diet, and the species and strain of fish all affect the course and severity of outbreaks (Sanders and Fryer 1981; Paterson et al. 1981a; Bullock 1980). Bacterial kidney disease is one of the most difficult bacterial diseases of fish to treat (Wolf and Dunbar 1959; Snieszko et al. 1955). Research is underway to develop methods to minimize the effect of BKD infections and to improve control measures (Paterson et al. 1981a).

SIGNS OF INFECTION

Acute outbreaks occur only occasionally. Sub-clinical cases are common and can lead to acute losses if affected fish are stressed.

EXTERNAL. SIGNS

1. Infected fish may appear normal. In rainbow and brown trout, there may be a "buckshot" appearance due to the presence of numerous small, open ulcers in the skin that expose the underlying musculature.

2. In brook trout and coho salmon fingerlings and yearlings, large "boils" filled with a pinkish, creamy fluid that contains massive numbers of *Renibacterium salmoninarum* bacteria can be found on the sides of the fish.

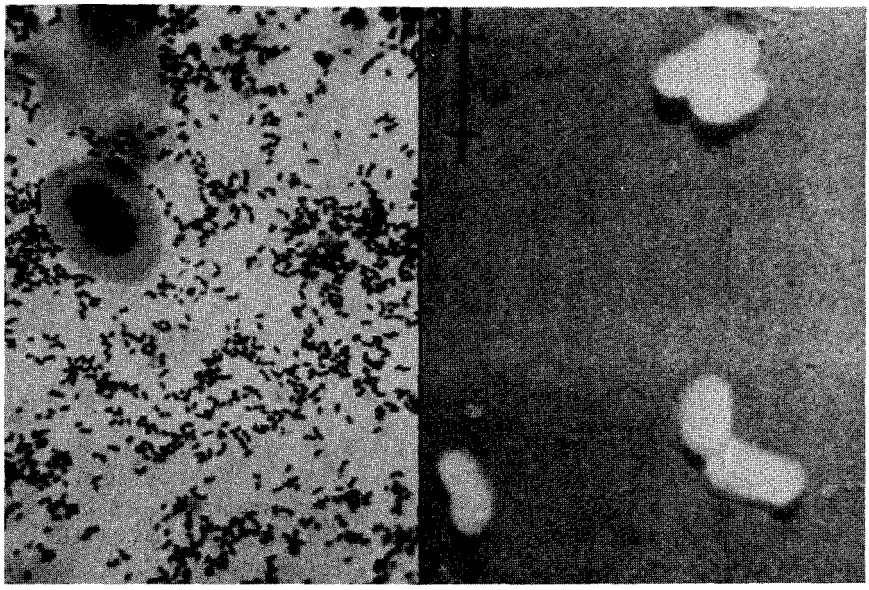
3. Exophthalmia (popeye), due to osmo-regulatory disruption, is a common sign of BKD. However, this sign is not diagnostic since it may also be due to other causes, such as gas supersaturation (gas bubble disease), enteric redmouth (ERM) infections, certain viral diseases, or parasites.

INTERNAL SIGNS

1. As indicated by its name, BKD severely affects the kidneys, and, to a, lesser extent, the spleen and liver. The kidneys are usually swollen, convex, and have a corrugated or lumpy surface in sharp contrast to the smooth, concave surface of healthy kidneys. Creamy, soft, off-white cysts represent massive colonies of the causative organism. Such cysts are common in the posterior kidney and may vary in size and number. They should not be confused with normal stanneous bodies located in the mid-kidney or with nephrocalcinosis (kidney stones) that may fill excretory tubules of the kidney.

2. A bloody, turbid, or yellow-brown fluid often accumulates in the abdominal cavity and around the heart.

3. Other internal organs and visceral fat may appear normal or appear unusually white. The intestinal tract may contain a white or yellow viscous fluid.



Bacteria that cause fish diseases require special techniques for their detection. In the left photo, cells of *Renibacterium salmoninarum* have been stained with crystal violet. The right-hand photo is an electron photomicrograph of the same organism. (J. Fryer, Oregon State University)

DIAGNOSIS

The diagnosis of BKD in salmonids can be confirmed by the detection and identification of *Renibacterium salmoninarum* organisms in the tissues. Fluorescent antibody techniques applied to fresh, frozen, or formalin-fixed infected posterior kidney tissue will usually reveal the presence of large numbers of small "bean-like" organisms (Mitchum and Sherman 1981; Paterson et al. 1979; Bullock and Stuckey 1975). Gram stains of similar material will reveal many tiny, Gram-positive diplobacilli which often occur in pairs. Fish that are apparently healthy can carry small numbers of the causative organism that are difficult to detect in Gram-stained smears (Sanders et al. 1978). Smears prepared from scrapings of the posterior intestinal tract of asymptomatic carriers may contain more organisms than smears of kidney tissue taken from the same fish (Mitchum and Sherman 1981). Diagnosis of BKD cannot be based, with any certainty, on the observation of a few isolated BKD-like organisms seen in a few microscope fields (Fryer and Sanders 1981). If this situation is encountered, more detailed sampling of the population in question should be initiated with particular attention to yearling trout or the oldest salmon fingerlings available at the site (Paterson et al. 1979; Snieszko et al. 1955).

EPIZOOTIOLOGY

Geographic and Host Ranges

The geographic range of BKD generally follows the range of certain salmonid fishes. The disease has been found to occur only in the salmon, trout and char of the sub-family Salmoninae of the family Salmonidae (Sanders and Fryer 1980). Bacterial kidney disease is common in hatcheries along the western slope of the Cascade Range on the west coast of North America, around the Great Lakes, and along the Appalachians from Georgia to the Canadian Maritime provinces. The disease has also been reported from Japan, the British Isles, Iceland, and several European countries (Fryer and Sanders 1981; Bullock 1980).

Sources and Reservoirs of Infection

Renibacterium salmoninarum is an obligate pathogen of salmonid fishes. To date, the bacterium has not been found to infect other species of fish or other animals. Salmon, trout, and char inhabiting hatchery water supplies must be considered as possible reservoirs of infection (Mitchum and Sherman 1980; Frantsi et al. 1975). Under good environmental conditions, carriers harbor subclinical infections throughout their lives. Eggs taken from infected broodstocks and adult salmon have been identified as a major source of introduction of BKD at hatcheries previously free of the disease. Outbreaks of BKD can occur a year or more after receipt of contaminated eggs. The slow progress of infections is disarming and makes it difficult to prevent the spread of the infection to other stocks in the hatchery, to other hatcheries, or to new geographic areas.

Modes of Transmission

Bacterial kidney disease is easily transmitted from parent to progeny with eggs. The bacterium is so intimately related to the egg or to the developing embryo that egg disinfection procedures using organic iodine disinfectants do not prevent transmission of the disease via eggs (Bullock et al. 1978). This seriously complicates containment of BKD and explains why egg and fish exchanges have played a significant role in the historical spread of the disease in North America and around the world.

Fish to fish (horizontal) transmission of BKD has been reported both in hatcheries and in the wild. Frantoi et al. (1975) reported hatchery-reared Atlantic salmon fingerlings contracted BKD from “naturally” infected wild fish in the hatchery water supply. Mitchum et al. (1979) showed that wild trout could transmit the disease to stocked hatchery trout that had been previously free of the disease.

Many of the early studies on the transmission of BKD dealt with the feeding of infected fish products. Until the mid-1960’s, salmon fingerlings were fed a wet diet containing raw ground salmon viscera, ground carcasses of spent broodfish, salmon eggs, and other fresh fish products together with beef liver, spleen and other animal organs. The raw salmon products often carried *R. salmoninarum* and other fish disease agents. These wet diets helped spread and establish BKD and other serious diseases in many Pacific Northwest salmon hatchery stocks (Wood 1974).

Susceptibility and Resistance Factors

In hatcheries rearing Pacific salmon, bacterial kidney disease is detected most frequently in spring chinooks, cohos and sockeyes (Sanders and Fryer 1980). A cross between pink and chum salmon (called a "chumpy") was found to be extremely susceptible (Wood 1974). According to Winter et al. (1980), some strains of steelhead trout are resistant to BKD. Unfortunately, however, BKD-resistant strains also showed the greatest susceptibility to *Vibrio anguillarum,* another bacterial pathogen often encountered by migrating smolts in estuaries. Michigan workers report fewer problems with BKD in the culture of salmon than in trout (J. Hnath, Wolf Lake State Fish Hatchery, Mattawan, MI, personal communication). Among trout and char, the brook trout is probably the most susceptible species. Rainbow and brown trout would rank next in susceptibility while steelhead trout are the most resistant (Mitchum and Sherman 1981).

Environmental conditions also play a role. Work by Warren (1963) showed that BKD is most severe in hatcheries supplied with soft water and less severe in hard water hatcheries. Many interrelated factors, including mineral metabolism, must be considered. Recent work by Paterson et al. (1981a) showed that, at one hatchery, the lowest incidence of BKD occurred in yearling Atlantic salmon fed diets with increased levels of trace minerals (Fe, Cu, Mn, Co, I, and F) and low levels of calcium. On the other hand, a high incidence of BKD occurred in similar fish fed a standard commercial ration. The commercial feed had a calcium content ranging from 2.0 - 2.7% of the diet. This calcium level may have actually increased trace element requirements in the fish because of increased mineral mobility.

Incubation Period

Fingerlings hatched from infected eggs seldom show signs of the disease until they are at least 6 months of age or about 3 inches in length. As water temperatures rise in the spring, yearling trout may suffer serious losses (Paterson et al. 1981a; Wolf and Dunbar 1959). Sanders et al. (1978) found that the experimental incubation period varied with the water temperature. At temperatures above 11°C, initial losses occurred 30 to 35 d after exposure. At temperatures between 7 and 11°C, the incubation period was 60 to 90 d or longer.

Seasonal Incidence

Outbreaks of BKD occur in cultured trout populations more often in the spring than at any other time of the year. Reported cases more than double in frequency in March, peak in May, and nearly cease in July. Rising temperatures greatly influence the onset of BKD. Warm summer temperatures may also enhance the production of immunological factors that suppress the disease in the fish. Some of the apparent decline in the number of summertime cases of BKD is due to the stocking of yearling trout which removes many of the infected fish from the hatchery. The increased spring time prevalence of BKD outbreaks in cultured trout should be considered when schedules are established for fish health inspections or for routine monitoring of mortalities for fish pathogens.

METHODS OF CONTROL

PREVENTION

Once established, BKD is an extremely difficult disease to manage. Avoidance is the first and strongest line of defense. Firm policies, careful planning, a good understanding of the etiology of BKD, and a thorough monitoring program are essential to a successful program of prevention and control. If these efforts fail, a number of other steps can be taken to contain the spread and minimize the overall effect of BKD.

Immunization of salmonids to prevent BKD or to reduce the effects of the disease is a new technique now under development. Paterson et al. (1981b) reviewed this subject and assessed the immunological response of Atlantic salmon to BKD antigens. In testing the efficacy of vaccine preparations, they found that injections of killed *R. salmoninarum cells* alone did not stimulate the production of protective antibodies in the fish. Immunization of Atlantic salmon younger than one year of age was not as effective in reducing the incidence of BKD in two-year-old smoltifying fish, as was the immunization of yearling fish during their second summer. Yearling Atlantic salmon immunized with a single injection of killed *R. salmoninarum cells,* emulsified in Freund's complete adjuvant, showed significantly fewer clinical signs of BKD. However, when the vaccinated and control groups were examined by means of indirect fluorescent antibody techniques, there was no difference in the prevalence of infected carriers among the two groups. Further studies are needed to evaluate what significance these results may have in terms of the severity of BKD in adult fish returning to the hatchery at spawning time.

In ongoing programs where BKD is already present in the adult spawning stock, there is a strong likelihood that the pathogen will be transmitted with the eggs they produce. In Pacific salmon hatcheries, erythromycin phosphate has been used in attempts to prevent this vertical transmission of the disease agent. Two delivery methods are being evaluated.

Erythromycin phosphate has been injected subcutaneously into the median dorsal sinus just anterior to the dorsal fin of adult salmon and steelhead trout at the time returning fish enter hatchery holding ponds. The adults are given additional injections at 30-d intervals until 30 d prior to spawning. Each fish is given erythromycin phosphate at 11.0 mg/kg of body weight. Eggs taken from infected females have been shown to carry drug levels that are inhibitory to *R. salmoninarum* in laboratory tests (Bullock 1980).

A second technique involves water-hardening freshly-fertilized eggs in a 2 ppm bath of erythromycin phosphate for up to one hour (Bullock 1980). The effectiveness of this method as a deterrent to BKD has not been verified in closely-controlled studies. Steve Leek (S. Leek, USFWS, Cook, WA, personal communication) reports a 94.9% eye-up in treated eggs versus an 88.7% eye-up in untreated controls.

Although active work on prophylactic measures to minimize the effects of BKD includes studies on immunization, chemotherapeutics, and nutrition, these techniques have not been integrated with effective inspection programs and improvements in fish cultural management. The key to controlling bacterial kidney disease rests with a fully integrated disease control approach that uses all available techniques.

THERAPY

At present, no drugs are known that will cure fish of BKD. Erythromycin and sulfamethazine control the disease for as long as the drugs are administered. However, when drug treatment is withdrawn, mortality due to BKD often resumes within a few weeks.

KEY STEPS TO REMOVE THE DISEASE AND/OR AGENT FROM FISH POPULATIONS

IMMEDIATE

Because BKD cannot be cured by chemotherapeutics, the only effective short-term cleanup procedure is to remove all infected or exposed fish from a facility and to disinfect the water supply and all rearing units. Pathogen-free eggs or fish can then be introduced and reared free of BKD if the cleanup operation was effective and the disease agent has not been inadvertently re-introduced. Wild stocks are difficult to adequately inspect; some have been found to be a source of BKD (Mitchum et al. 1979).

LONG TERM

Hatchery operations that are involved with anadromous fish species carrying BKD must incorporate every known approach in efforts to minimize the effect of the disease and to eventually eliminate it. The preventive measures discussed above can be used as part of a strategy of "dilution" of the infected population to result in an increasing number of healthy fish. As more healthy fish are stocked, the proportion of infected fish in wild populations can be reduced. Although Mitchum and Sherman (1981) found evidence of horizontal transmission of BKD in the wild, this does not appear to be a widespread problem in most wild salmonid populations. Population densities, stress, water characteristics, and the severity of the BKD infections undoubtedly play important roles. The same factors that influence the transmission and severity of BKD in hatcheries can be expected to influence the occurrence of BKD in wild populations. All things considered, the "dilution" strategy remains the only practical approach available for the eventual reduction of the disease in free-ranging fish populations.

REFERENCES

Bullock, G.L. 1980. Bacterial kidney disease of salmonid fishes caused by *Renibacterium salmoninarum.* U.S. Fish Wildl, Serv., Fish Dis. Leafl. No. 60. Washington, DC. 10 p.

Bullock, G.L., H.M. Stuckey, and D. Mulcahy. 1978. Corynebacterial kidney disease: egg transmission following iodophore disinfection. U.S. Fish Wildl. Serv., Kearneysville, WV. Fish Health News. 7: 51-52.

Bullock, G. L., and H.M. Stuckey. 1975. Fluorescent antibody identification and detection of the corynebacterium causing kidney disease of salmonids. J. Fish. Res. Board Can. 32: 2224-2227.

Frantsi, C., T.C. Flewelling, and K.G. Tidswell. 1975. Investigations on corynebacterial kidney disease and *Diplostomum* sp. (eye fluke) at Margaree hatchery, 1972-1973. Fish. Mar. Serv., Res. Dev. Br., Dept. Environ. Can., Marit. Reg. Tech. Rep. Ser. No. Mar/T-75-9, 30 p.

Fryer, J. L., and J. E. Sanders. 1981. Bacterial kidney disease of salmonid fish. Ann. Rev. Microbiol. 35: 273-298.

Mitchum, D.L., and L.E. Sherman. 1981. Transmission of bacterial kidney disease from wild to stocked hatchery trout. Can. J. Fish. Aquat. Sci. 38: 547-551.

Mitchum, D. L., L. E. Sherman, and G. T. Baxter. 1979. Bacterial kidney disease in feral populations of brook trout *(Salvelinus fontinalis),* brown trout *(Salmo trutta),* and rainbow trout (S. *gairdneri).* J. Fish. Res. Board Can. 36: 1370-1376.

Paterson, W. D., S. P. Lall, and D. Desautels. 1981a. Studies on bacterial kidney disease in Atlantic salmon *(Salmo salar)* in Canada. Fish Pathol. 15: 283-292.

Paterson, W.D., D. Desautels, and J.M. Weber. 1981b. The immune response of Atlantic salmon, *Salmo* salar L., to the causative agent of bacterial kidney disease, *Renibacterium salmoninarum.* J. Fish Dis. 4: 99-111.

Paterson, W.D., C. Gallant, D. Desautels, and L. Marshall. 1979. Detection of bacterial kidney disease in wild salmonids in the Margaree river system and adjacent water using an indirect fluorescent antibody technique. J. Fish. Res. Board Can. 36: 1464-1468.

Sanders, J.E., and J.L. Fryer. 1980. *Renibacterium salmoninarum* gen. nov., sp. nov., the causative agent of bacterial kidney disease in salmonid fishes. Int. J. Syst. Bacterial. 30: 496-502.

Sanders, J. E., K. S. Picher, and J. L. Fryer. 1978. Relation of water temperature to bacterial kidney disease in coho salmon *(Oncorhynchus kisutch),* sockeye salmon (O. *nerka),* and steelhead *(Salmo gairdneri).* J. Fish. Res. Board Can. 35: 8-11.

Snieszko, S.E, P. J. Griffin, H.A. Delisle, C.E. Dunbar, S.B. Friddle, and A. G. Sanderson. 1955. Kidney disease in brook trout and its treatment. Prog. Fish-Cult. 17: 3-13.

Warren, J. W. 1963. Kidney disease of salmonid fishes and the analysis of hatchery waters. Prog. Fish-Cult. 25: 121-131.

Winter, G. W., C. B. Schreck, and J. D. McIntyre. 1980. Resistance of different stocks and transfer-r-in genotypes of coho salmon *(Oncorhynchus kisutch)* and steelhead trout *(Salmo gairdneri)* to bacterial kidney disease and vibriosis. Oreg. Fish Comm., Fish. Bull. 77: 795-802.

Wolf, K. E., and C. E. Dunbar. 1959. Test of 34 therapeutic agents for control of kidney disease in trout. Trans. Am. Fish. Soc. 88: 117-124.

Wood, J. W. 1974. Diseases of Pacific salmon: their prevention and treatment. 2nd. ed. Wash. Dep. Fish., Olympia, WA. 82 p.

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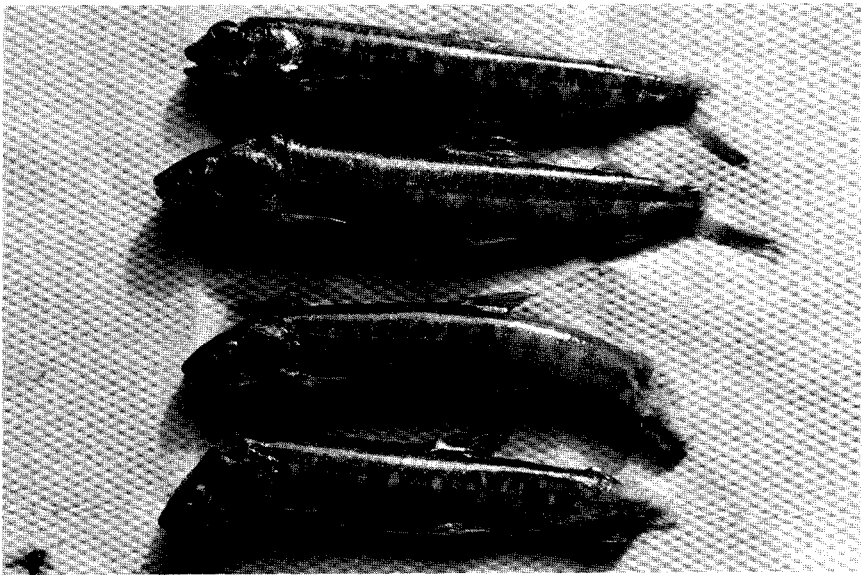
COLDWATER DISEASE

JOHN H. SCHACHTE
New York Department of
Environmental Conservation
Rome, NY

Coldwater disease, sometimes also referred to as peduncle or low temperature diseases, is a serious disease of coho salmon fry and yearlings (Amend 1970)



Lake trout fingerlings with progressive degrees of tail rot and caudal peduncle erosion due to coldwater disease, Cytophaga *psychrophila* (J. H. Schachte, Jr.)

Section (x 1000) of lake trout muscle tissue infected with Cytophaga *psychrophila*. Arrows show typical long, rod-shaped bacterial cells associated with the disease (J. H. Schachte, Jr.)

and of other species of salmon and trout (Rucker et al. 1954; Bullock et al. 1971). In recent years, the disease has become a problem in cultured fingerling lake trout in the northeastern United States (Schachte 1980). The etiologic agent of coldwater disease is the bacterium *Cytophaga psychrophila*. The disease may cause high losses of salmon and trout fry and fingerlings. In the case of fingerling fish, effective treatment is difficult. Once the disease has become established in a hatchery population, extensive treatment and the removal of grossly-infected individuals frequently yields only limited results.

SIGNS OF INFECTION

Coldwater disease is first manifested as a grey, patchy, discolored area on the caudal peduncle. In infected fish, the peduncle darkens and, as the disease progresses, the caudal fin becomes frayed and eroded. In advanced stages, clinical signs of coldwater disease may include open lesions and erosion of the skin on the caudal peduncle, complete erosion of the musculature of the peduncle, and loss of the caudal fin.

DIAGNOSIS

The coldwater disease bacterium is a long, slender Gram-negative rod measuring approximately .75 mm x 1.5 to 5 nm (Pacha and Ordal 1970). Its size and morphology is similar to *Flexibacter columnaris* which may also cause fin erosion and "tail rot" signs. The pathogen causes disease at low temperatures (Pacha and Ordal 1970); the optimum range is between 4.4-10.0°C. However,

Wood (1974) has pointed out that the optimum temperature for isolation on culture media is 15-20°C, which is considerably higher than the range at which clinical disease occurs. Even at appropriate temperatures, however, the isolation of C. *psychrophila* can be difficult. Diagnosis of the disease is achieved by isolation of butyrous yellow colonies of long, thin bacteria on Ordal's cytophaga medium. When time is short and isolation is difficult, the bacterium can be identified by the indirect fluorescent antibody test (IFAT) using smears taken directly from caudal lesions. The disease may also be systemic. However, as is frequently the case with columnaris disease, demonstration of the bacterium in tissues other than the skin and muscle of the caudal peduncle may not be possible (Wood 1974).

EPIZOOTIOLOGY

Geographic and Host Ranges

Bullock et al. (1971) reported that the geographic range of peduncle (coldwater) disease is confined primarily to the northwestern and northeastern United States, with a much higher incidence of the disease in the Pacific northwest. Coho salmon are the most susceptible species but significant infections occur in sockeye and chinook salmon. Bullock et al. (1971) report that the disease is confined primarily to brook trout in the northeast and that the disease is less of a problem in this species. Recent outbreaks of the disease in fingerling lake trout in New York (Schachte 1980) have provided additional concern for the disease in hatcheries in the northeastern United States.

Sources and Reservoirs of Infection

According to Bullock and Snieszko (1970), coldwater disease may be waterborne or transmitted from carrier fish. In the lake trout infections in New York, seriously infected fish with necrotic caudal peduncle areas were undoubtedly releasing tremendous numbers of organisms to infect other fish in the pond. Wood (1979) has indicated that the transovarian route may also be a factor.

Susceptibility and Resistance Factors

Coldwater disease is very serious in salmon sac fry. Fingerling coho salmon are readily affected and may suffer considerable mortality from the disease. Epizootics in the lake trout in New York State have been responsible for losses approaching 25% in 1O-15 cm (4-6 in) fingerlings reared in hatchery raceways.

Modes of Transmission

According to Wood (1979), the transmission of the disease is associated with the presence of carrier fish, vertical transmission of the disease to the offspring may also be a possibility. The recent diagnosis of coldwater disease during the winter of 1981 in the Cayuga Lake strain of landlocked salmon provides circumstantial evidence that New York epizootics may be tied to contaminated eggs of Finger Lake strains of lake trout. This information may provide further evidence

of vertical transmission as has been suggested by Wood (1979). Infected fish in a raceway with gross lesions probably release sufficient numbers of organisms to spread the disease throughout the population in that raceway. Asymptomatic carriers may also be responsible for some infections.

INCUBATION PERIOD

Coldwater disease develops at water temperatures between 7 and 10°C. The incubation period is considered to be less than 10 d (Bullock and Snieszko 1970). However, if as in the case of lake trout, the source of disease is related to contaminated eggs, the incubation period may be longer than that previously reported, or a latent period is involved.

METHODS OF CONTROL

PREVENTION

All efforts should be made to avoid contaminated stocks. If vertical transmission proves to be true, rigorous egg disinfection procedures should be administered. This can be accomplished by using organic iodine compounds at 100 ppm for 10 min (Amend 1974).

THERAPY

External infections may be treated early with water soluble Terramycin at 1O-50 ppm or with quaternary ammonium compounds at 2 ppm (Hyamine 3500, Roccal, etc.). Oral administration of sulfonamides at 8-10 gm or oxytetracycline (Terramycin) at 2.5-3.5 gm per 100 lb of fish fed per day is also effective.

In the case of the lake trout epizootics, when the disease had progressed to the point where significant numbers of fish exhibited eroded peduncles, quaternary ammonium and/or antibiotic therapy did not provide control. At such advanced stages, antibiotics and a potassium permanganate ($KMnO_4$) flush at 2 ppm combined with removal of seriously infected fish were required to control mortality. $KMnO_4$ apparently will not only reduce the bacterial load but also hasten the demise of infected individuals that would otherwise die later, thereby facilitating the removal of these carriers from tanks or raceways.

KEY STEPS TO REMOVE THE DISEASE AND/OR AGENT FROM FISH POPULATIONS

IMMEDIATE

A strict program of egg disinfection should be applied. Wood (1979) suggests that fish should be reared in raceways at densities of no more than 4-5 pounds per gpm to minimize oxygen deficiency problems. According to Wood (1974), such limits on rearing environments reduced the incidence of the disease. Pacha and Ordal (1970) reported that raising the water temperature to above 12.8°C (55°F) generally caused a decrease in the severity of infection. However, Wood (1974) stated that temperature manipulation had little effect on an estab-

LONG TERM

Long term measures should include careful screening and selection of broodstock that are free of the disease. Also, water sources that are free of the disease and of potential carrier fish should be developed.

REFERENCES

Amend, D.E 1970. Myxobacterial Infections of Salmonids: prevention and treatment, p. 258-265. *In* S. F. Snieszko (ed.) A symposium on diseases of fishes and shellfishes. Am. Fish. Soc., Spec. Publ. No. 5. Bethesda, MD.

Amend, D. F. 1974. Comparative toxicity of two iodophors to rainbow trout eggs. Trans. Am. Fish. Soc. 103: 73-78.

Bullock, G. L., and S. F. Snieszko. 1970. Fin rot, coldwater disease and peduncle disease of salmonid fishes. U.S. Fish Wildl. Serv., Fish Dis. Leafl. No. 25. Washington, DC. 3 p.

Bullock, G.L., D.A. Conroy and S.E Snieszko. 1971. Book 2A: Bacterial diseases of fishes. TFH Publications, Inc. Neptune City, NJ. 151 p.

Pacha, R. E. and E. J. Ordal. 1970. Myxobacterial diseases of salmonids: prevention and treatment, p. 243-257. In S.F. Snieszko (ed.) A symposium on diseases of fishes and shellfishes. Am. Fish. Soc., Spec. Publ. No. 5. Bethesda, MD.

Rucker, R.R., B.J. Earp, and E.J. Ordal. 1954. Infectious diseases of Pacific salmon. Trans. Am. Fish. Soc. 83: 297-312.

Schachte, J.H., Jr. 1980. An epizootic of coldwater disease *(Cytophaga psychrophila)* in Adirondack and Finger Lake strains of lake trout at the Chateaugay fish hatchery during the winter of 1979-80. Bur. Fish., NY. Dep. of Environ. Cons. Unpublished report. 8 p.

Wood, J. W. 1974. Diseases of Pacific salmon: their prevention and treatment. Wash. Dep. of Fish., Hatchery Div. Olympia, WA. 82 p.

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COLUMNARIS DISEASE

JOHN H. SCHACHTE
New York Department of
Environmental Conservation
Rome, NY

Columnaris disease, caused by the bacterium *Flexibacter columnaris,* may result in acute or chronic infections in both coldwater and warmwater fishes. It occurs both as external or systemic infections that result in significant losses of hatchery-reared fish, particularly at warm summer temperatures (Pacha and Ordal 1970; Becker and Fujihara 1978). Epizootics of columnaris disease frequently occur in natural populations and high losses of fish may be observed. Wood (1974) describes strains of high and low virulence; highly virulent forms attack gill tissue and the latter strains are primarily responsible for cutaneous infections. Some disagreements still exist concerning proper taxonomic placement of this organism (Snieszko and Bullock 1976). However, Bergey's Manual of Determinative Bacteriology (8th ed.) classifies it as a flexibacterium.

SIGNS OF INFECTION

In many species of fish, the first sign of the disease may be the appearance of discolored gray, patchy areas in the area of the dorsal fin. These characteristic "saddleback" lesions may progress until skin erosion exposes underlying muscle tissue. These lesions may become yellow and cratered and are often prominent in the mouth and head regions (Wood 1979). Virulent strains of *F. columnaris* may attack gill tissue and cause a "gill rot" condition (Wood 1974). Systemic infections due to less virulent strains may occur with no apparent external signs. However, cutaneous infections seem to be more prevalent in most species of fish.

DIAGNOSIS

A presumptive diagnosis of columnaris disease can be made by the detection of long, slender Gram-negative rods in smears of gills or scrapings obtained from cutaneous lesions. Frequently, material scraped from such lesions and examined under phase contrast microscopy in a wet mount will reveal the presence of unique characteristic "haystack" colonies that are of diagnostic significance. Isolation of the organism on cytophaga medium (Annacker and Ordal 1959) can be accomplished from gill or cutaneous lesions or from the kidneys of chronically infected fish. Colonies of *F. columnaris* exhibit a rough, rhizoid-margined growth that tends to extend into the agar (Snieszko and Bullock 1976). The organism may be differentiated from bacterial gill disease and coldwater disease on the basis of several characteristics. First, the causative organisms responsible for bacterial gill disease are not easily isolated on culture media. Unlike columnaris disease, bacterial gill disease causes no macroscopic gill necrosis. In the case of coldwater disease, the morphology of colonies on culture media is in the form of smooth, yellow colonies as compared to the rough-edged or rhizoid colonies of columnaris disease. Additionally, the columnar-is organism differs in cell size, i.e. 0.5-0.7x4.0-8.0 nm as compared to 0.75x1.5-5.0 nm for the coldwater disease bacterium.

EPIZOOTIOLOGY

Geographic and Host Ranges

Flexibacter columnaris is a ubiquitous soil and water-borne bacterium and natural epizootics of the disease are common. Columnar-is disease is found worldwide and infects practically all species of freshwater fishes and some amphibians (Snieszko and Bullock 1976; Becker and Fujihara 1978). It has been reported only in freshwater fishes. However, some marine fish are infected by myxobacterial diseases that are similar to columnaris (Bullock et al. 1971). Most hatchery-reared salmonids, coolwater species (such as the tiger muskellunge and the walleye), catfish and baitfish are highly susceptible under intensive culture conditions.

Sources and Reservoirs of Infection

When fish are under stress due to elevated temperatures, crowding, etc., *F. columnaris* may attack the fish and cause disease. Infected animals with gill or cutaneous lesions serve as a source of infection. In hatcheries with open water supplies, any species of infected fish in the water supply may serve as a reservoir of infection for the disease. Pacha and Ordal (1970) demonstrated that fish, such as catostomids, coregonids and cyprinids, may serve as reservoirs of infection.

Susceptibility and Resistance Factors

There appears to be little or no species resistance to columnaris disease. The role of stressors is considered a key factor in outbreaks of disease. Stress may be provided by crowding, by holding fish at above normal temperatures, as

well as by physical injury due to handling (Wedemeyer 1974). In general, however, temperature seems to be the primary determining factor as to when infection may occur. Experimental studies by Holt et al. (1970) revealed that temperatures in excess of 12.2°C (54°F) were required to induce mortality in fish infected with *F. columnaris.*

Modes of Transmission

Research has shown that *Flexibacter columnaris* can be transmitted from fish to fish directly through the water when virulent strains are used. Individual infected fish within a population harbor the bacteria over winter (Wood 1974) and serve as sources of infection during the following summer months when stresses occur due to overcrowding and water temperatures above 12.2°C (54°F), etc. Microcysts formed by *F. columnaris* have been shown experimentally to remain viable over a period of several years. Some uncertainty still exists as to the possibility that these forms are sources of infection under natural conditions (Wood 1979).

Incubation Period

The period between exposure to *F. columnaris* and the outbreak of clinical disease varies, depending upon the virulence of the strain of bacteria and the ambient water temperature. Strains of high virulence may induce acute disease within 24 h, whereas less virulent forms may require from 48 h to several weeks (Warren 1981). Holt et al. (1975) have shown experimentally that a high degree of correlation exists between clinical disease and high water temperatures. Their studies also revealed that host species differ in the time from exposure to death. Existing data reveal that the disease has a pronounced seasonal occurrence. Both natural and hatchery epizootics are concentrated during the warm summer months.

METHODS OF CONTROL

Prevention

Avoidance of exposure to the disease is a primary method of prevention. This can be accomplished by the use of disease-free water or by the use of U. V. disinfected water supplies. The elimination of wild fish in an open water supply may be helpful when feasible. If water temperature manipulation is available, temperatures above 12.8°C (55°F) should be avoided since they favor development of the disease. Crowding or handling during these periods should be delayed when possible. If the fish must be handled or crowded, certain prophylactic treatments may be administered. These include copper sulfate ($CuSO_4$) baths for 20 min at 33 ppm or potassium permanganate ($KMnO_4$) at 2 ppm for indefinite periods (Snieszko and Bullock 1976). Copper sulfate should be used with care since it is highly toxic to fish in soft water. Similarly, $KMnO_4$ should be used with caution since it may also be toxic to certain species, particularly in soft waters with low levels of organic matter.

THERAPY

Most investigators have determined that treatment for columnaris disease should include both external bath and antimicrobial feed additive therapy to combat both cutaneous and systemic infections (Amend 1970). Compounds such as copper sulfate, potassium permanganate (Snieszko and Bullock 1976) and Diquat (Wood 1979) have been used. Quaternary ammonium compounds such as Roccal, Hyamine and Purina Four Power used at 2-3 ppm in one-hour flow-through treatments have been effective. However, one should consider water quality when making a choice among these compounds. Hardness, for example, may render some treatments ineffective at recommended levels or, in the case of copper sulfate, may be toxic at water hardnesses below 50 ppm $CaCO_3$. In waters of the northeast, for example, hurnic acid levels may be high and a permanganate demand of several ppm may have to be satisfied before any beneficial effects can be expected from potassium permanganate treatments.

Oxytetracycline (Terramycin) incorporated into the food at the rate of 4 gm/100 lbs of fish fed at 3% body weight per day is the usual antibiotic treatment used to accompany the chemical bath treatment (Wood 1974).

KEYS STEPS TO REMOVE THE DISEASE AND/OR AGENT FROM FISH POPULATIONS

Unless there is a closed or disinfected water supply, there is little likelihood of eliminating this pathogen from a culture facility. In the event that an open water supply exists, measures should be taken to prevent the introduction or immigration of any wild fish into the hatchery, If a closed water supply exists, steps should be taken to ensure that resident hatchery fish that may be carriers cannot migrate into the hatchery water supply. If cool water is available during periods of warm weather it should be used.

REFERENCES

Amend, D. F. 1970. Myxobacterial infections of salmonids: prevention and treatment. p. 258-265. *In S.E* Snieszko (ed.) A symposium on diseases of fishes and shellfishes. Am. Fish. Soc., Spec. Publ. No. 5, Bethesda, MD. 526 p.

Anacker, R. L., and E. J. Ordal. 1959. Studies on the myxobacterium *Chondrococcus columnaris*. I. Serological typing. J. Bact. 78: 25-32.

Becker, CD., and M.P. Fujihara. 1978. The bacterial pathogen Flexibacter *columnaris* and its epizootiology among Columbia River fish. Am. Fish. Soc., Monogr. No. 2, Bethesda, MD. 92 p.

Bullock, G.L., D.A. Conroy, and S.E Snieszko. 1971. Book 2A: Bacterial diseases of fishes, T.F.H. Publications, Inc., Neptune City, NJ. 151 p.

Holt, R.A., J.E. Sanders, J.L. Zinn, J.L. Fryer, and K.S. Pilcher. 1975. Relation of water temperature to *Flexibacter columnaris* infection in steelbead trout *(Salmo gairdneri),* coho *(Oncorhyncus kisutch)* and chinook *(O. tshawytscha)* salmon. J. Fish. Res. Board Can. 32: 1553-1559.

Pacha, R.E. and E.J. Ordal. 1970. Myxobacterial diseases of salmonids. p. 243-257. *In* S.E Snieszko (ed.) A symposium on diseases of fishes and shellfishes. Am. Fish. Soc., Spec. Publ. No. 5, Bethesda, MD.

Snieszko, S.E, and G. L. Bullock. 1976. Columnaris disease of fishes. U.S. Fish Wildl. Serv., Fish Dis. Leafl. No. 45, Washington, DC. 10 p.
Warren, J. W. 1981. Diseases of hatchery fish. A fish disease manual. U.S. Fish Wildl. Serv., Reg. 3, Twin Cities, MN. 91 p.
Wedemeyer, G.H. 1974. Stress as a predisposing factor in fish diseases. U.S. Fish Wildl. Serv., Fish Dis. Leafl. No. 38, Washington, DC. 8 p.
Wood, J.W. 1974. Diseases of Pacific salmon: their prevention and treatment. Wash. State Dep. Fish. Olympia, WA. 82 p.

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ENTERIC REDMOUTH DISEASE

J. W. WARREN
U.S. Department of the Interior
Fish and Wildlife Service
La Crosse, WI

Enteric redmouth (ERM) is an acute to chronic systemic bacterial disease of salmonids. It is especially severe among intensively cultured rainbow trout reared in water warmer than 13°C (Dulin et al. 1976). The disease is caused by a motile, Gram-negative, rod-shaped bacterium identified as Yersinia *ruckeri* (O'Leary et al. 1979; Ewing et al. 1978). This pathogen is readily transmitted from fish to fish by contact and through the water (Rucker 1966). Where ERM has become established, it usually causes sustained low-level losses. Occasionally, severe epizootics occur with mortalities exceeding 50% if corrective measures are not taken (Ross et al. 1966; Bullock et al. 1976). Surviving fish frequently become asymptomatic carriers which can spread the disease if these fish are transferred to new locales (Busch and Lingg 1975; Hunter et al. 1980). Enteric redmouth disease can be successfully controlled by a combination of environmental improvement and antibacterial drugs (Rucker 1966; McDaniel 1971). Vaccines have been developed that can reduce the impact of the disease (Busch et al. 1978).

SIGNS OF INFECTION

Acute cases of ERM seldom go undetected. The clinical signs, however, are similar to those of other common bacterial infections. *Aeromonas hydrophila* and *A. salmonicida* infections are indistinguishable from ERM if the diagnosis is based solely upon clinical signs (Dulin et al. 1976). Enteric redmouth causes different signs at different stages of the disease and a wide variety of signs can be observed among the victims of a single epizootic (Wobeser 1973; Rucker 1966).

Rainbow trout infected with enteric redmouth bacterial disease are frequently dark in color and exhibit lesions associated with mucous membranes of the mouth and eyes. (U.S. Fish and Wildl. Serv.)

EXTERNAL SIGNS

1. Dark, lethargic fish that do not feed and appear to isolate themselves from the main population are evident in the early stages of an ERM outbreak. This condition will also be common among carriers after losses have subsided. Affected fish often have missing eyes or exophthalmia (popeye) and are blind and sluggish with little avoidance reaction.
2. During the acute stage of the disease, small bright hemorrhages occur along the gum line of the mouth and on the tongue, which, together with general inflammation, give the "redmouth" appearance for which the disease is named.
3. The normally white ventral body surface (belly) may be speckled with small hemorrhages that may also be evident at the base of the fins.

INTERNAL SIGNS

1. Flaccid stomachs filled with clear fluid are common in fish with ERM. This is an *important sign!* Enteric redmouth should be suspected whenever rainbow trout with hemorrhages in the mouth are found to also have flaccid, fluid-filled stomachs.
2. Enteric redmouth victims often have enlarged, dark spleens, hemorrhagic specks on the air bladder and pyloric cecae, and reddening of the posterior intestinal tract.

DIAGNOSIS

The diagnosis of enteric redmouth disease depends upon the detection isolation and identification of the causative bacterium, *Y. ruckeri.* Bacteriological samples from the lower intestine and posterior kidney should be collected from dark, sluggish fish and from any fish with hemorrhages in the mouth and flaccid, fluid-filled stomachs. A quick presumptive diagnosis can be made by conducting fluorescent antibody tests on smears prepared from these samples (Bullock and Snieszko 1979; Johnson et al. 1974). One method for detecting *Y. ruckeri* in asymptomatic carriers is to apply fluorescent antibody tests on smears prepared from scrapings of the posterior intestinal tract (Busch and Lingg 1975; Hunter et al. 1980). Confirmation of the presence of Y. *ruckeri,* however, requires the bacteriological isolation of Gram-negative, motile rods which have the following biochemical characteristics: cytochrome oxidase negative; acid, but no gas from glucose; positive for ornithine and lysine decarboxylase but not arginine dihydrolase; and, a positive agglutination with rabbit anti-Y. *ruckeri* serum (Bullock et al. 1978).

EPIZOOTIOLOGY

Geographic and Host Ranges

In the USA, ERM has occurred in at least 18 of the lower 48 states and in Alaska (Bullock et al. 1978). In Canada, the disease or its agent has been detected in British Columbia, Saskatchewan, Ontario and Nova Scotia (Bullock et al. 1978). The disease is most prevalent in rainbow trout. It has also been reported from cutthroat trout and from coho, chinook, and Atlantic salmon (Bullock and Snieszko 1979). The principal factor involved in the spread of the disease has been the shipment of infected carriers (Dulin et al. 1976; Busch and Lingg 1975). The causative bacterium, Y. *ruckeri,* occurs naturally in some areas and ERM can occur when susceptible fish are subjected to stress (Rucker 1966; Hunter et al. 1980). There are no reports of natural outbreaks of ERM among free-ranging wild fish.

Sources and Reservoirs of Infection

Asymptomatic carriers are the primary source and reservoir of *Y. ruckeri* (Rucker 1966; Busch and Lingg 1975). After a population of fish has recovered from an ERM outbreak, carriers in the group serve as a major source of reinfection, An infection/recovery/re-infection cycle of 36-40 d was reported by Busch and Lingg (1975) in rainbow trout held at 14.5°C. Their work showed that more than 25% of the survivors carried localized infections in their lower intestine. Invertebrates, such as crayfish, that inhabit the water supplies of hatcheries where ERM persists have been found to harbor Y. *ruckeri,* but the role of invertebrates in the etiology of this disease is unknown (Dulin et al. 1976).

Modes of Transmission

Fish to fish transmission has been frequently reported from both hatchery

1980). Direct fish-to-fish contact is not necessary. If water from infected fish passes through rearing units containing susceptible fish, these fish readily become infected (Ross et al. 1966). No evidence of the transmission of *Y. ruckeri* from parent to progeny with eggs has been reported.

Susceptibility and Resistance Factors

Rainbow trout are severely affected by *Y. ruckeri* infections and enteric redmouth disease causes serious losses in raceway-cultured fish, especially in commercial hatcheries in southern Idaho (Dulin et al. 1976). Water temperature, crowding, excess ammonia, low oxygen, obesity, and handling stresses are important factors that influence the timing and severity of epizootics (Rucker 1966). Since these factors can be controlled by fish culturists, the disease can be managed by maintaining good environmental conditions (DuIin et al. 1976; McDaniel 1971).

Several workers report that fish size affects the susceptibility of fish (Rucker 1966; DuIin et al. 1976). while the disease is seldom a problem in rainbowtrout less than 7.5 cm in length, size alone may not be the determining factor. In many hatcheries, fish must be periodically weighed, moved to new rearing units, or sorted to harvest market-sized fish. These activities often take place when the fish population has reached or exceeded the optimal carrying capacity for the unit. Under these circumstances, stress from handling can trigger outbreaks of ERM, especially if water temperatures exceed 13°C (Dulin et al. 1976; Busch and Lingg 1975). At these temperatures rainbow trout grow rapidly, oxygen demands are high, and economics losses due to ERM will often be high among large fish (Dulin et al. 1976).

Incubation Period

At 15°C, enteric redmouth disease kills 7.5-10.0 cm rainbow trout in 5-19 d (DuIin et al. 1976; Ross et al. 1966). In 12.5°C water, Atlantic salmon 6.0 cm in length, began to die 9 dafter exposure (Bullock et al. 1976). Cumulative losses of 52% occurred in three separate sets of experiments in which the fish were not medicated. After mortality ceases, Y. *ruckeri can* be detected in the survivors for another 60 d, but after 80 d the bacteria may be no longer detectable (Hunter et al. 1980).

Seasonal Incidence

No reports suggest that the occurrence of enteric redmouth is seasonal If water temperatures are constant, stress-mediated epizootics may occur at any time (Hunter et al. 1980).

METHODS OF CONTROL

Prevention

The transfer of carriers to hatcheries previously free of ERM has been well documented as the primary way this disease has been spread (Dulin et al. 1976;

Busch and Lingg 1975). Thorough inspections of hatchery fish populations prior to the shipment of fish to other hatcheries can help to prevent the inadvertant introduction of ERM. Persistent monitoring of mortalities for cause of death coupled with annual fish health inspections are invaluable in developing a reliable history of the absence of ERM disease at a facility. These measures are necessary because the detection of *Y. ruckeri* in apparently healthy carrier fish can be difficult, especially when the fish are being reared in good environmental conditions. This fact, however, demonstrates the effectiveness of good fish cultural conditions in reducing the overall impact of ERM in hatcheries where it has been troublesome in the past. Other important preventive steps include the use of chlorine to disinfect water supply springs, ditches, pipelines and headboxes to remove possible disease-carrying fish, invertebrates and bacteria (McDaniel 1971).

Immunization of cultured trout against ERM can also be beneficial (Busch et al. 1978). Commercial vaccines are now available which improve the ability of fish to ward off the disease. While immunization does not provide total protection against ERM, it apparently contributes sufficiently to the well-being of the fish to be worthwhile. A word of caution is in order, however. Care should be taken to starve the fish for 24-72 h prior to handling and prophylactic treatments should be given to rid the fish of sub-clinical cases of bacterial gill disease or external parasites. If precautionary measures are neglected, stresses associated with the immunization process can elicit outbreaks of other diseases (Busch et al. 1978).

THERAPY

Sulfamerazine and oxytetracycline (Terramycin) have been used extensively to control ERM (Dulin et al. 1976). Regardless of the effectiveness of antibacterial drugs, they alone cannot be relied upon for the control of this disease. Adverse environmental factors and excessive handling stresses must be eliminated or the disease may recur shortly after drugs are withdrawn. Neither sulfamerazine, oxytetracycline, nor any other drugs used in the past against ERM are currently approved by the U.S. Food and Drug Administration for use in food fish to control ERM. Research studies needed for the registration of compounds effective against ERM should be given a high priority.

KEY STEPS TO REMOVE THE DISEASE AND/OR AGENT FROM FISH POPULATIONS

IMMEDIATE

No procedures have been developed that will completely free fish populations of Y. *ruckeri* bacteria once they have become infected.

LONG TERM

Although prevention is the key to minimizing the affect of ERM, several steps must be followed after the disease has been detected. A clean water supply that is free of fish and invertebrates is essential. Rearing facilities should be

depopulated and disinfected. This can be accomplished all at once, or in carefully-orchestrated phases while fish production continues (McDaniel 1971). Strict control over all fish brought into the hatchery or over the associated geographic area will help to protect ERM-free hatcheries and watersheds.

REFERENCES

Bullock, G.L., and S.E Snieszko. 1979. Enteric redmouth of salmonids. U.S. Fish and Wildl. Serv., Fish Dis. Leafl. No. 57. Washington, DC. 7 p.

Bullock, G.L., H.M. Stuckey, and E.B. Shotts, Jr. 1978. Enteric redmouth bacterium: comparison of isolates from different geographic areas. J. Fish Pathol. 1: 351-356.

Bullock, G.L., H.M. Stuckey, and R.L. Herman. 1976. Comparative susceptibility of Atlantic salmon (Salmo salar) to the enteric redmouth bacterium and *Aeromonas salmonicida.* J. Wildl. Dis. 12: 376-379.

Busch, R.A., and A.J. Lingg. 1975. Establishment of an asymptomatic carrier state infection of enteric redmouth disease in rainbow trout (Salmo *gairdneri).* J. Fish Res. Board Can. 32: 2429-2432.

Busch, R.A., N.E. Burmeister, and A.L. Scott. 1978. Field and laboratory evaluation of a commercial enteric redmouth disease vaccine for rainbow trout, p. 67. In Proceedings of Joint 3rd Biennial Fish Disease Workshop. Am. Fish. Soc., Fish Health Sec. and 9th Annual Midwest Fish Dis. Comm., Kansas City, MO. August 15-18, 1978.

Dulin, M.I?, T. Huddleston, R.E. Larson, and G.W. Klontz. 1976. Enteric redmouth disease. Univ. of Idaho, College of Forestry, Wildl. and Range Sci., Bull. No. 8. Moscow, ID. 15 p.

Ewing, W.H., A.J. Ross, D.J. Brenner, and G.R. Fanning. 1978. Yersinia *ruckeri* sp. nov., the redmouth (RM) bacterium. Int. J. Syst. Bacterial. 28: 37-44.

Hunter, VA., M.D. Knittel, and J. L. Fryer. 1980. Stress-induced transmission of *Yersinia ruckeri* infection from carriers to recipient steel-head trout, *Salmo gairdneri* Richardson. J. Fish Dis. 3: 467-472.

Johnson, G. R., G. Wobeser, and B.T. Rouse. 1974. Indirect fluorescent antibody technique for detection of RM bacterium of rainbow trout *Salmo gairdneri.* J. Fish. Res. Bd. Can. 31: 1957-1959.

McDaniel, D. W. 1971. Hagerman redmouth . . . A new look at an old problem. Am. Fishes and U.S. Trout News. 15(5): 14-28.

O'Leary, P. J., J. S. Rohovec, and J.L. Fryer. 1979. A further characterization of *Yersinia* ruckeri (enteric redmouth bacterium). Fish Pathol. 14(2): 71-78.

Ross, A.J., R.R. Rucker, and W.H. Ewing. 1966. Description of a bacterium associated with redmouth disease of rainbow trout (Salmo *gairdneri).* Bull. Off. Int. Epizoot. 65: 825-830.

Wobeser, G. 1973. An outbreak of redmouth disease in rainbow trout *(Salmo gairdneri)* in Saskatchewan. J. Fish. Res. Bd. Can. 30: 571-575.

25

FURUNCULOSIS

JOHN H. SCHACHTE
New York Department of
Environmental Conservation
Rome, NY

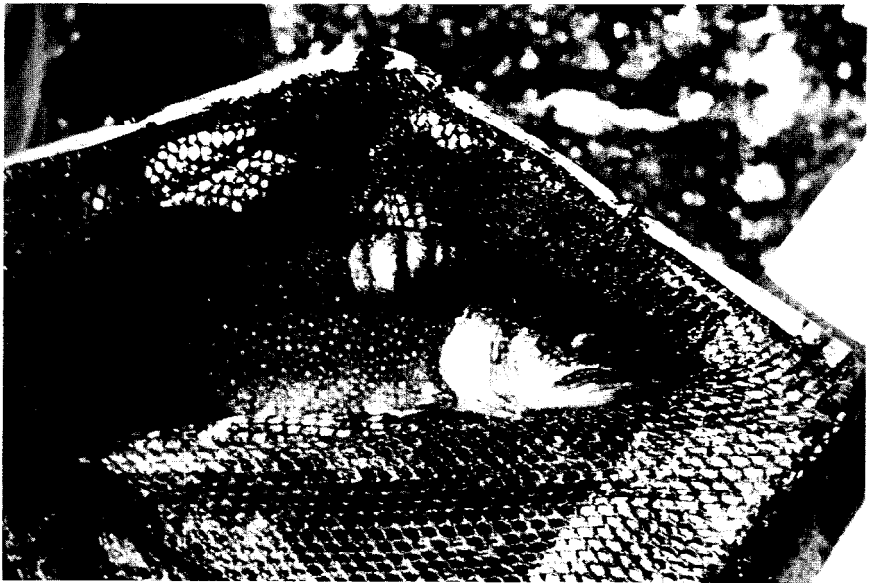
Furunculosis is a serious, septicemic, bacterial disease found principally in salmonid fishes, but it may also occur in goldfish and other cyprinids. The common name of the disease is derived from the presence of "blisters" or furuncules on the surface of chronically infected salmonids (Snieszko and Bullock 1975). However, this sign is not diagnostic of this disease inasmuch as it may be encountered in fish infected with other pathogens. It should be pointed out that, in acute cases of furunculosis, the furuncules may not be present.

The disease is caused by a Gram-negative bacterium, *Aeromonas salmonicida,* described by Griffin et al. (1953). It has recently been demonstrated (Paterson et al. 1980) that the ulcer disease attributed to *Hemophilus piscium* is, in fact, caused by a strain of *A. salmonicida.* Numerous reports in the literature describe the epizootiology and control of the disease (McGraw 1952; Herman 1968; and Bullock et al. 1971). Furunculosis is found worldwide with few exceptions and causes disease in many species of coldwater and warmwater fishes. In trout hatcheries in North America, it accounts for a high percentage of the fish losses attributable to infectious diseases.

SIGNS OF INFECTION

Clinically-infected fingerlings will usually exhibit hemorrhages at the base of fins and erosion of the pectoral fins. Bloody or hemorrhagic vents and petechial hemorrhages on the ventral surface are frequently observed. In chronically infected adults, typical "furuncules" or blisters on the skin containing an amorphous yellow substance and blood may be present. This is rarely seen in small or fingerling fish since an acute infection frequently causes massive bacteremia and death before gross lesions develop (Snieszko and Bullock 1975). Internal exam-

inations frequently reveal a bloody fluid in the body cavity. Petechial hemorrhages are commonly observed in the body wall and viscera.



Furunculosis, a bacterial fish disease, may cause large lesions on the external body surface such as those on this lake trout. (U.S. Fish and Wildl. Serv.)

DIAGNOSIS

Positive diagnosis of furunculosis depends upon isolation and identification of the causative agent, *A. salmonicida.* The organism is typically a Gram-negative, non-motile rod that ferments selected carbohydrates, produces cytochrome oxidase, and produces a water soluble brown pigment on several types of isolation agar. Care must be exercised, however, in the identification of non-motile cytochrome oxidase-positive, Gram-negative rods since a number of atypical and achromogenic variants have been reported (Elliot and Shotts 1980a; Paterson et al. 1980) in several species of fish. If an atypical *A. salmonicida,* such as that encountered in ulcerative disease of goldfish is suspected, enriched isolation media may be required. Elliot and Shotts (1980a) reported that either chocolate agar or tryptic soy agar plus 5% defibrinated sheep's blood was required for adequate growth of isolates. Isolates from suspect variants of *A. salmonicida* should be given sufficient culture time to allow for those strains which slowly produce a brown, water soluble pigment to do so (Elliot and Shotts 1980b). In such cases, or when rapid identification is needed, the fluorescent antibody technique (FAT) may be used (McDaniel 1979).

It is generally accepted that asymptomatic carriers are very difficult to detect. If it is necessary to establish the absence of carriers in a potential broodstock population, the use of serum agglutination techniques or corticosteroid techniques described by Bullock and Stuckey (1975) might be employed.

Sampling of intestinal contents has also been suggested for carrier detection but information is lacking on the reliability of this technique.

EPIZOOTIOLOGY

Geographic and Host Ranges

Furunculosis is primarily a disease of salmonid fishes. However, epizootics have been diagnosed in numerous cool and warmwater species, including Esocids and Cyprinids. With the exception of Australia and New Zealand, the disease is distributed worldwide wherever trout and salmon are reared (Snieszko and Bullock 1975).

Sources and Reservoirs of Infection

Aeromonas salmonicida is considered to be an obligate pathogen of fish. It has not been found when diseased or carrier fish are absent. The organism may survive for days or weeks in water but cannot persist indefinitely in the absence of carrier fish (Snieszko and Bullock 1975).

Susceptibility and Resistance Factors

Most species of salmonids and many cool and warmwater species are susceptible to the disease. In New York State, for example, the intensive culture of tiger muskies has encountered significant problems with furunculosis. In addition, forage minnows fed to muskellunge have been shown to be carriers and the sources of infection.

Various state fisheries research groups have been working to develop resistant strains of fish through selective breeding. Ehlinger (1964) in New York, reported the first successful work in achieving resistance in brook and brown trout to furunculosis. Field testing in New York, Pennsylvania, and Minnesota has shown that both species possess a high degree of resistance to the disease. Pennsylvania and Missouri have been progressing steadily toward the development of IPN and BKD resistant strains of trout. It should be kept in mind, however, that in this method of fish disease management, resistant stocks are generally considered to be carriers of the disease. As such, they might serve as sources of infection for other susceptible stocks with which they might come in contact.

In addition to species susceptibility to the disease, the problem of antibiotic resistance by the furunculosis bacterium exists. Resistance to both Terramycin and sulfamerazine has become widespread. This is believed to be primarily due to the questionable use of low levels of these compounds as prophylactic measures in the absence of acute disease outbreaks.

Modes of Transmission

Transmission generally occurs as a result of contact with diseased or carrier fish, but can occur through water passed from one pond or raceway to another. Contaminated clothing or equipment may also transfer the disease from one

culture unit to another. The possibility also exists that fish-eating birds may transfer the disease either by contact or by dropping infected fish into an uninfected pond (Snieszko and Bullock 1975). If eggs from carrier broodstocks are not disinfected prior to incubation, the organisms may be transferred on the surface of the eggs (Wood 1974). Japanese investigators have conducted studies that indicate *A. salmonicida* is not an invasive pathogen. According to their work, infection occurs experimentally only when the pathogen is ingested or has access to external injuries on the fish (Sakai 1979).

Incubation Period

The incubation period for acute cases of furunculosis is probably from 2-4 d. However, in chronic cases, particularly at lower temperatures, the period may be extended by several weeks (Groberg et al. 1978). Furunculosis is usually seasonal with the highest incidence of disease during the midsummer months of July and August. Incidence is related to temperature; the disease is most prevalent in the range from 12.8°C (55°F) to 21.1°C (70°F). At low temperatures, chronic furunculosis has been observed in landlocked salmon cultured in the Adirondack Mountains of New York at water temperatures of 0.5°C (33°F) to 1.6°C (35°F).

METHODS OF CONTROL

Prevention

A basic step in the prevention of serious communicable fish diseases is the adherence to a sound program of hatchery inspections and a disease classification system. As a minimum, all lots of fish in a hatchery should be inspected at least once per year for the presence of disease. Utilizing the data generated from these inspections, transfers of suspect or known carrier fish from hatchery to hatchery should be avoided. All eggs from susceptible species should be routinely disinfected using organic iodine compounds at 100 ppm of active iodine for 10 min (Amend 1974) on water hardened eggs. The hatchery water supply should be kept free of fish. Barriers should be provided to prevent the introduction of potential wild carrier fish into the hatchery. Resistant strains of fish should be utilized as a disease management tool where appropriate. If eggs must be imported from outside of the hatchery system, insist that only eggs supplied from inspected and certified furunculosis-free sources be used.

Therapy

Epizootics of the disease may be treated through the addition of drugs to the fish feed. Terramycin (oxytetracycline) should be added to feed at the rate of 3.0 g/100 lb fish, administered daily for 10 d to affected fish. Sulfamerazine should be administered at the rate of 5-10 g/100 lb fish and fed for 10 or 15 consecutive days. Care should be taken to determine if the strain of furunculosis involved is resistant to either or both of the compounds used for therapy.

KEY STEPS TO REMOVE THE DISEASE AND/OR AGENT FROM FISH POPULATIONS

IMMEDIATE

Use only stocks certified free of furunculosis as sources of eggs for introduction into the hatchery. This practice must be accompanied by the disinfection of all eggs brought into the hatchery. In those cases where hatcheries must use open water supplies, stock only disease-free fish in the headwaters above the hatchery.

LONG TERM

If complete eradication of the disease is required, removal of all fish and complete disinfection of the contaminated hatchery may be necessary. This approach should be complemented by an annual disease inspection program which will provide the basic information for disease classification of hatcheries. With these tools, it will then be possible to restrict transfers of fish, thus avoiding contamination of facilities which do not already have the disease.

REFERENCES

Amend, D. I? 1974. Comparative toxicity of two iodophors to rainbow trout eggs. Trans. Am. Fish. Soc. 103: 73-78.

Bullock, G.L., D.A. Conroy, and S.E Snieszko. 1971. Book 2A. Bacterial diseases of fishes. TFH Publications, Neptune City, NJ. 151 p.

Bullock, G. L., and H. M. Stuckey. 1975. *Aeromonas salmonicida:* detection of asymptomatically infected trout. Prog. Fish-Cult. 37: 237-239.

Ehlinger, N.F. 1964. Selective breeding of trout for resistance to furunculosis. N.Y. Fish Game J. 11: 78-90.

Elliot, D.G., and E.B. Shotts, Jr. 1980a. Aetiology of an ulcerative disease in goldfish, *Carassius auratus* (L): microbiological examination of diseased fish from seven locations. J. Fish Dis. 3: 133-143.

1980b. Aetiology of an ulcerative disease in goldfish, *Carassius auratus* (L): experimental induction of the disease. J. Fish Dis. 3(2): 145-151.

Griffin, I? J., S.E Snieszko, and S.B. Friddle. 1953. A more comprehensive description of *Bacterium salmonicida.* Trans. Am. Fish. Soc. 82: 129-138.

Groberg, W.J., Jr., R.H. McCoy, K.S. Pilcher, and J.L. Fryer. 1978. Relation of water temperature to infections of coho salmon (Oncorhynchus *kisutch),* chinook salmon (0. *nerka)* and steelhead trout *(Salmo gairdneri)* with *Aeromonas salmonicida* and *A. hydrophila.* J. Fish Res. Board Can. 35: l-7.

Herman, R.L. 1968. Fish furunculosis 1952-1966. Trans. Am. Fish. Soc. 97: 221-230.

McGraw, B.M. 1952. Furunculosis of fish. U.S. Fish and Wildl. Serv., Spec. Sci. Rep. Fish. No. 84. Washington, DC. 87 p.

McDaniel, D. (ed.). 1979. Fish health bluebook: procedures for the detection and identification of certain fish pathogens. Am. Fish. Soc., Fish Health Sec. Bethesda, MD. 119 p.

Paterson, W.D., D. Douez, and D. Desautels. 1980. Relationships between selected strains of typical and atypical *Aeromonas salmonicida, Aeromonas hydrophila* and *Haemophilus piscium.* Can. J. Microbiol. 26: 588-598. Sakai, D. K. 1979. Invasive routes *of Aeromonas salmonicida* subsp. *salmonicidae.* Abstract in: Fish Health News (1980). 9: 25.

Snieszko, S.E and G.L. Bullock. 1975. Fish furunculosis. U.S. Fish and Wildl. Serv., Fish Dis. Leafl. No. 43. Washington, DC. 10 p.

Wood, J.W. 1974. Diseases of pacific salmon: their prevention and treatment. Wash. Dep. Fish. Olympia, WA. 82 p.

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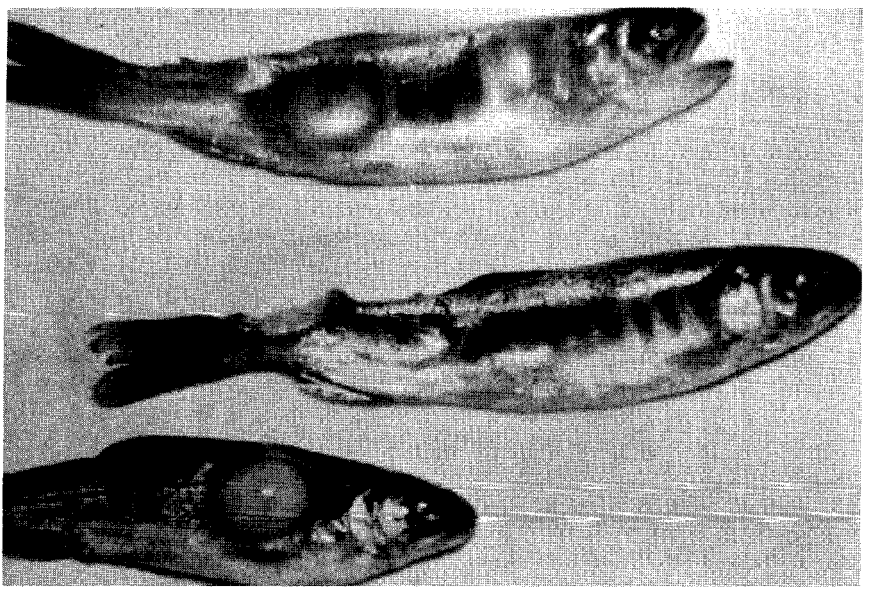
CERATOMYXOSIS

J.G. HNATH
Fisheries Section
Michigan Department of Natural Resources
Mattawan, MI

Ceratomyxosis is caused by *Ceratomyxa shasta,* a tissue-invading protozoan parasite. It is an infectious disease capable of causing serious losses of



The protozoan parasite, *Ceratomyxa shasta,* causes large, spore-filled cysts in young salmonids. (J. Conrad, Oregon Fishery Commission)

both prespawning adults and in hatchery and wild juvenile salmonids (Johnson et al. 1979). *C. shasta* is unique in that it has never been reported outside of the Pacific Northwest in North America (Johnson et al. 1979). In fact, the only known method of initiating an infection with C. shasta is by exposure of fish to water containing the infectious stage (Schafer 1968). Since the disease has never been detected outside the Pacific Northwest, every effort should be made to contain it within its present range.

SIGNS OF INFECTION

Signs of *Ceratomyxa* infections vary in different species of salmonids. Clinical signs include loss of appetite, a distended abdomen, popeye, dropsy, emaciation, swelling in the area of the vent, and hemorrhages and swelling along the digestive tract. Other signs include nodules in the gut of adult chinook salmon, large abscesses in the musculature, and gross lesions in the kidney, liver, and spleen. Juvenile steelhead may also show infectious material in the entire digestive tract, liver, spleen, gonads, kidney, heart, gills, and skin (Johnson et al. 1979).

DIAGNOSIS

The disease is confirmed by the microscopic demonstration of typical spores in scrapings from the lower intestinal wall, gall bladder, or lesions. Spores are bicapsulate with broadly rounded ends and shell valves that are strongly arched posteriorly (in other words, somewhat kidney-bean shaped). The spores measure 14-23 nm long and 6-8 nm wide at the middle (Johnson et al. 1979).

EPIZOOTIOLOGY

GEOGRAPHIC AND HOST RANGES

Infected fish have been found along the northwest coast of North America in Oregon, California, Washington, British Columbia, and Idaho. The disease is unique, however, in that it has been transmitted to other susceptible fish only in certain river systems in Oregon, Washington, and California. The geographic distribution of the organisms has been well described by Johnson et al. (1979) as follows:

> "There are two principal features concerning the geographic distribution of *C. shasta* that must be emphasized. Fist, there are waters that contain the infective stage of the organisms. Infectivity is generally demonstrated by exposure of susceptible animals and later examination for the organisms. Second, there are waters that lack the infective stage of *C. shasta,* but do contain infected salmonids. Diseased fish found in such areas are assumed to have contracted the organism while living in or migrating through waters containing the infectious stage. These animals do not seem to transmit the disease to other susceptible salmonids. An important aspect about the distribution of *C. shasta* is its confinement to salmonids along the Pacific Coast. The parasite has not been reported elsewhere during the period of almost 30 years since the

disease was first described. This restricted distribution is in marked contrast to that of many other fish pathogens, which have a wider distribution, often as a result of the shipment of eggs or fish to new locations. Such ecological specificity is compatible with the idea that some, as yet unknown, specific factor is required for infection of salmonids by C. *shasta".*

The overwhelming evidence suggests that the parasite cannot establish itself outside of the waters presently known to contain infective stages -hence it is assumed that the parasite cannot be spread through transfer of infected fish or eggs. However, until this hypothesis is established beyond doubt, the importation of fish or eggs from enzootic areas is not recommended unless the fish have been inspected and certified to be free of C. *shasta.*

The following species have been infected with C. *shasta* (either naturally or experimentally): coho, chinook, sockeye, chum, and Atlantic salmon; and rainbow, cutthroat, brown and brook trout. Of these, rainbow trout, cutthroat trout, chum salmon, and coho salmon are the most susceptible; brown trout, brook trout, and Atlantic salmon are much less susceptible (Johnson et al. 1979).

Sources and Reservoirs of Infection

The only known sources and reservoirs of the infectious agent are waters that contain the infective stage. The mechanism of transmission is unknown since diseased fish do not transmit the disease directly to other susceptible salmonids. As long as the mode of transmission remains an unknown, there is the chance that the disease might occur elsewhere. Thus, the movement of fish from enzootic to non-enzootic areas is not recommended.

Schafer (1968) reports that standing (lake or pond) water is required in a system for C. *shasta* to become infective. This observation has been substantiated by the work of others (Harlan Johnson, former USFWS hatchery biologist at Little White Salmon Hatchery, Washington) and by epizootiological studies on the Deschutes River system in Oregon which has headwaters in infected lakes. It is apparent from hatcheries operated by the Oregon Department of Fish and Wildlife that C. *shasta* is no problem at hatcheries fed by fast flowing streams with no lakes or reservoirs in the headwaters.

Susceptibility and Resistance Factors

All species of salmonids tested were found to be susceptible (Zinn et al. 1977), although only rainbow trout, cutthroat trout, and chum salmon are highly susceptible (Johnson et al. 1979). Coho and chinook salmon are somewhat less susceptible, brook trout and brown trout are much less so (Johnson et al. 1979).

There is considerable variation in susceptibility among different strains of rainbow trout and chinook salmon. Those strains originating from areas with a long history of exposure to the parasite are generally more resistant than newly-exposed strains (Johnson et al. 1979).

Temperature also plays a role in susceptibility, In a report by Udey et al. (1975), juvenile rainbow trout mortalities averaged 80% and were independent of water temperature between 6.3 and 23.3°C. However, in coho salmon, mor-

talities increased progressively from 2% at 9.4°C to 22% at 15.0°C to 84% at 20.5°C. No deaths occurred in either rainbow or coho at 3.9°C nor in coho at 6.7°C.

MODES OF TRANSMISSION

Natural transmission occurs only when susceptible salmonids come in contact with water containing the infective stage. A 30 min or longer exposure to such water is necessary to initiate the disease. Fish to fish infection does not occur. According to Johnson et al. (1979): "Transmission has not occurred as a result of feeding of infected tissues, cohabitation of infected fish with susceptible fish, or holding susceptible salmonids in water containing a mixture of mud and infected tissues".

INCUBATION PERIOD

The incubation period, defined as the length of time from infection to death varies with fish species and water temperature. The infection will develop only at water temperatures above 3.9°C. Deaths occur in coho at 12.5 d at 23.3°C and losses do not occur until 146 d at 9.4°C. Deaths in rainbows occur in 14 d at 23.3°C to 15 d at 6.7°C or colder (Udey et al. 1975).

SEASONAL INCIDENCE

Infections first appear in the spring when waters reach 10°C, and fish continue to become infected until water temperatures cool in late fall or early winter (Schafer 1968).

METHODS OF CONTROL

PREVENTION

The disease may be prevented in areas where it is endemic by avoiding the use of infected water supplies or by treatment of infected water with a combination of filtration followed by chlorine or UV irradiation (Sanders et al. 1972).

The disease can be prevented from reaching geographic regions where it does not presently occur by not transferring fish or water into these areas from locations where the disease occurs.

Resistant stocks should be used to reduce the incidence of C. shasta. Use of resistant stock in known infected waters should reduce the number of infective units being returned to the "mud" (Zinn et al. 1977)

THERAPY

There is no known treatment.

KEY STEPS TO REMOVE THE DISEASE AND/OR AGENT FROM FISH POPULATIONS

IMMEDIATE

There is no known way to rid infected fish of the parasite.

LONG TERM

In three areas where C. shasta occurs, the use of non-infected water sources in hatcheries has proven effective. Treatment of infected water by a combination of filtration followed with chlorine or UV irradiation has prevented outbreaks of the disease (Sanders et al. 1972).

REFERENCES

Johnson, K.A. 1975. Host susceptibility, histopathologic, and transmission studies on *Ceratomyxa shasta,* a myxosporidan parasite of salmonid fish. Ph.D. Thesis, Oregon State Univ., Corvallis. 145 p.

Johnson, K.A., J.E. Sanders and J.L. Fryer. 1979. *Ceratomyxa shasta* in salmonids. U.S. Fish and Wildl. Serv., Fish Dis. Leafl. No. 58. Washington, DC. 11 p.

Ratliff, D. E. 1981. *Ceratomyxa shasta:* Epizootiology in chinook salmon of central Oregon. Trans. Am. Fish. Soc. 110: 507-513.

Sanders, J.E., J.L. Fryer, and R. W. Gould. 1970. Occurrence of the myxosporidan parasite, *Ceratomyxa shasta,* in salmonid fish from the Columbia River basin and Oregon coastal streams, p. 133-141. In S. F. Snieszko (ed.) A symposium on diseases of fishes and shellfishes. Am. Fish. Soc. Spec. Publ. 5. Bethesda, MD.

Sanders, J.E., J.L. Fryer, D.A. Leith, and K.D. Moore. 1972. Control of the infectious protozoan *Ceratomyxa shasta* by treating hatchery water supplies. Prog. Fish-Cult. 34: 13-17.

Schafer, W.E. 1968. Studies on the epizootiology of the myxosporidan *Ceratomyxa shasta* Noble. Calif. Fish and Game. 54: 90-99.

Udey, L. R., J. L. Fryer, and K. S. Pilcher. 1975. Relation of water temperature to ceratomyxosis in rainbow trout *(Salmo gairdneri)* and coho salmon *(Oncorhynchus kisutch).* J. Fish. Res. Board Can. 32: 1545-1551.

Zinn, J.L., K.A. Johnson, J.E. Sanders, and J.L. Fryer. 1977. Susceptibility of salmonid species and hatchery strains of chinook salmon *(Oncorhynchus tshawytscha)* to infections by *Ceratomyxa shasta.* J. Fish. Res. Board Can. *34: 933-936.*

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WHIRLING DISEASE

J. G. HNATH
Fisheries Section
Michigan Department of Natural Resources
Mattawan, MI

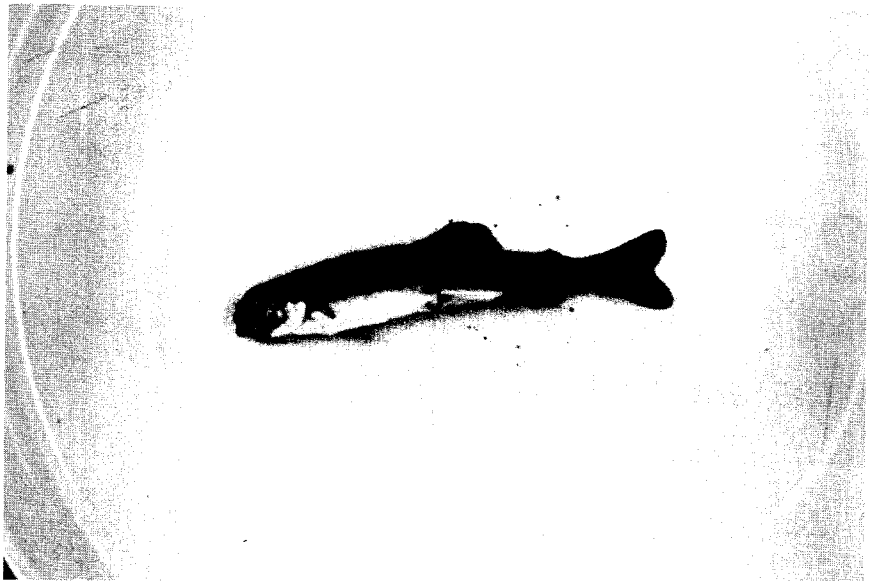
Whirling disease is a parasitic infection of trout caused by the protozoan Myxosoma *cerebralis.* Signs of the disease are the result of the parasite feeding on the cartilage of young host fish. A common sign of the disease is rapid, tail-chasing behavior when fish are frightened or trying to feed. The course of the disease ranges from sub-clinical infections to acute disease with mortalities of fry and fingerlings.

SIGNS OF INFECTION

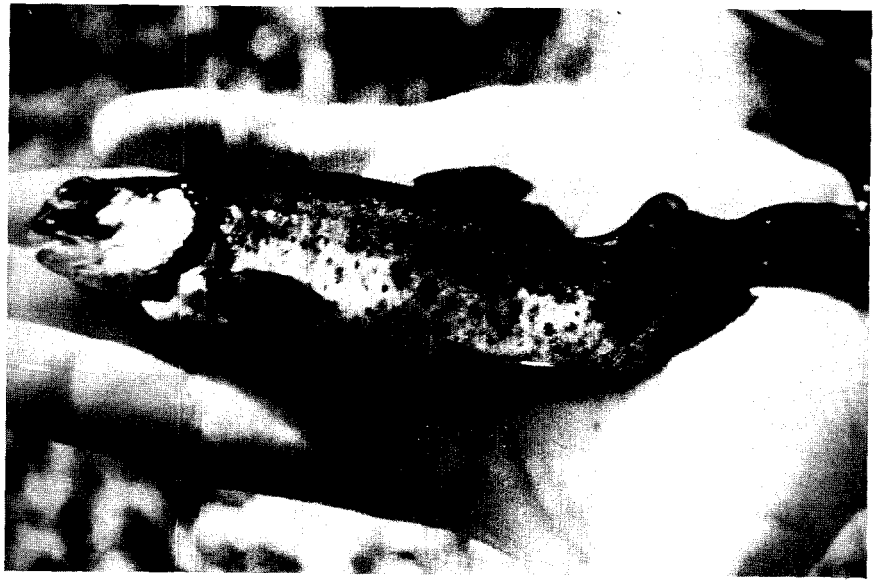
The characteristic sign from which the disease gets its name is the rapid tail-chasing behavior observed in some species and sizes of trout. In surviving fish, as the disease progresses, skeletal deformation, including misshapen heads, jaws, and gill covers, and spinal curvature may develop. Trout exposed early in life sometimes develop a "blacktail" due to loss of control of chromatophores on the caudal penduncle. Acutely- infected fry reared in contaminated water may show no signs before they suffer a high mortality. When older fish are exposed, they exhibit less whirling behavior and blacktail, but any skeletal deformities will remain for life. Fish with light infections show none of these signs, but will carry spores throughout their life.

DIAGNOSIS

Infection by *M. cerebralis* should be strongly suspected whenever signs of whirling, blacktail, or skeletal deformities are seen. Infections are confirmed either by the demonstration of spores, or by demonstration of the immature forms of the parasite in histological sections (see McDaniel 1979, for specific



An early sign of whirling disease caused by Myxosoma *cerebralis* is a loss of nerve control over the posterior one-third of the body. This young trout shows the typical "black tail" that results. (U.S. Fish and Wildl. Serv.)



Myxosoma cerebralis infections cause extensive damage to the skull of developing salmonids. Resulting pressures on nerves may destroy control of muscle groups and lead to grossly deformed bodies in surviving fish. (U.S. Fish and Wildl. Serv.)

EPIZOOTIOLOGY

Geographic and Host Ranges

The disease was first reported in Europe about 1904. Its present range, as defined by Halliday (1976), includes Africa, North and South America, Asia, New Zealand, and Europe. In North America, it has been reported from the following states: California, Connecticut, Massachusetts, Michigan, Nevada, New Jersey, Ohio, Pennsylvania, West Virginia, Virginia, and New Hampshire (K. Wolf, USFWS, Kearneysville, WV, personal communication).

Susceptible hosts include all species of salmon, trout, and grayling. Although M. *cerebralis* has been reported in several non-salmonid species (Halliday 1976), there is reason to question the accuracy of these reports.

Sources and Reservoirs of Infection

Infected trout or salmon, contaminated water supplies, and contaminated mud are known to be sources and reservoirs of infection. It has been reported (Christensen 1972) that the spores may survive for 1O-15 yr in contaminated mud. Yoder (1972) reported the spread of M. *cerebralis* from a contaminated hatchery by approximately 6 miles in 28 months following the introduction of the disease to the hatchery. In spite of repeated attempts to destroy the infected fish population and to disinfect the stream, the infection persists and presents a constant threat for further spread of the disease. Bogdanova (1970) reported that, in the USSR, infection rates in natural waters may reach 100% in non-anadromous salmonids, but that the intensity of infection was so low that no clinical signs were observed. Even so, asymptomatic carriers present a threat of infection to hatcheries.

Modes of Transmission

The exact route of infection has yet to be demonstrated, but it is suspected that spores are released from dead and decaying fish or shed by living fish. Freshly-shed spores are not immediately infective and must spend time (4-5 months) aging in mud before they develop infectivity (Hoffman and Putz 1969). Transmission of whirling disease with fertilized eggs is highly unlikely if care is used to avoid contaminated water and mud during the spawning and egg-packing procedures. Spores can survive as long as two months in frozen infected fish.

Other recent work suggests that tubificid worms may be involved in the transmission of M. *cerebralis* (K. Wolf, USFWS, Kearneysville, WV, personal communication).

Susceptibility

Rainbow trout, brook trout, Atlantic salmon, kokanee salmon, and European grayling may become severely diseased. Brown trout, coho salmon, and lake trout are very resistant to this disease and, under experimental conditions, develop less than 1% of the average spore numbers found in rainbow trout (J. O'Grodnick, Pa. Fish and Game Commission, Bellefonte, PA, personal commu-

nication). According to O'Grodnick: *"since M. cerebralis* has already become established in certain geographical areas, policies of eradication are not practical and abandonment of existing facilities is not economically justifiable. An acceptable management alternative may be the rearing of proven resistant salmonids in contaminated hatcheries. State agencies could arrange production schedules so that resistant species which do not develop clinical whirling disease are substituted for susceptible rainbow trout or brook trout. Coho salmon, brown trout, and lake trout can be reared in contaminated hatcheries with no whirling disease development. The number of spores developed by these species is quite low and it would be unlikely that establishment of whirling disease in large water areas would be possible by stocking these fish. Also, hatchery effluent- receiving streams would receive fewer spores from very resistant production fish and contamination would be reduced. Since the value of the coho salmon and lake trout to the Great Lakes fishery is well documented, a careful evaluation of the policies regarding rearing these resistant species *in known M. cerebralis* contaminated waters should be made".

Fish are most susceptible to infection during the first 12 months of life. Sac fry at an age of 3 d are the youngest fish known to become infected (Putz and Hoffman 1966). Fish of 4.5 months of age or older do not develop acute clinical signs even though they may still become infected and serve as asymptomatic carriers (Hoffman and Byrne 1974; Hoffman 1976).

INCUBATION PERIOD

Infected fish may show signs of infection 28 wk after exposure (Hoffman 1976). Spore formation in infected fish takes 52 d at 17°C, 3 months at WC, and 4 months at 7°C (Halliday 1973). Acutely infected fish may not develop any overt signs of the disease.

METHODS OF CONTROL

PREVENTION

The only means of preventing infections of *M. cerebralis* is to keep the parasite away from any susceptible fish. The importation of infected fish or the use of contaminated water should be avoided.

THERAPY

No effective therapy is known.

KEY STEPS TO REMOVE THE DISEASE AND/OR AGENT FROM FISH POPULATIONS.

IMMEDIATE

There is no way known to eradicate the parasite from infected fish. Therefore, the only way that the parasite can be eradicated is to eliminate infected fish and to remove, seal off, or destroy all other infected material, such as mud and

contaminated water. All of the fish in ponds known to be infected must be eliminated. Incineration or deep burial with quicklime (CaO) is recommended but dressed carcasses may be used for food, provided they are cooked and that careful control is maintained over the disposal of the heads and offal. In certain cases, special landfill or burial permits may be necessary. Smoking also kills the parasite (Wolf and Markiw 1982). After the fish have been disposed of, disinfection of the contaminated facility can be considered if a water source free of contamination is available. Contaminated earthen ponds should be replaced with concrete or other impervious surfaces such as plastic or fiberglass, if possible. If not, the pond should be drained and thoroughly cleaned including removal of as much mud as practical. Quicklime at 380 g/m^2 (3,360 lb/acre) or calcium cyanamide at 500 g/m^2 (4,231 lb/acre) have been effective disinfectants when spread evenly on drained, wet pond bottoms and dikes, and if treatments are repeated several weeks or months later, preferably in the spring and autumn (Christensen and Bogdanova 1973; Hoffman 1976; Schaperclaus 1954).

Concrete surfaces or structures should be wet when disinfected with these chemicals.

Calcium cyanamide is no longer commercially available, thus quicklime is recommended (Schaperclaus, Kulow and Schreckenbach 1979). When using any chemicals, take adequate safety precautions and follow all label instructions. In addition, be sure that applications will have no adverse effects on downstream waters.

After a facility has been disinfected, small test plantings of susceptible rainbow trout should be used to see if the job was successful. The test fish should be young fingerlings less than 4 months of age and should be left in the test ponds at least 4 months.

The hatchery may be restocked the following season with uninfected fish. The young fish should be kept in fiberglass, metal, or concrete facilities as long as possible. Earthen ponds should not be used until the fish are at least 8 months old unless it is certain that the disease has been eliminated from the hatchery. Rearing tanks and ponds should be kept as clean as possible, and all dead fish should be removed daily. Although fish in these ponds may become lightly infected, they will usually show no signs of disease (Hoffman 1976). However, the population must be considered to be potential carriers and should not be planted into areas known to be free of whirling disease until at least 2 yr of rearing and repeated inspection work can certify that the population is free of the infection.

If the water supply is known to be contaminated with the parasite, it should be treated in an attempt to kill or remove the spores. Filtration of the water through pore sizes of less than 10 nm in diameter will remove the parasite. Irradiation of water with ultraviolet light at 35,000 microwatt s/cm2 after filtration through 25 nm filters is effective (Hoffman 1976). Sand gravel filters may be used as prefilters to ultraviolet irradiation. However, no examples of effective water supply decontamination for whirling disease on a hatchery scale are available.

Long Term

Prevention is the most effective control. If an outbreak occurs, eradication should be attemnted. In enzootic areas where the disease has spread tn natural

waters, and where there is no possibility of total fish stock destruction and disinfection, fish can be raised in the presence of the parasite. These fish will become infected, but if everything possible is done to lessen the degree of infection, most of the infected fish will be asymptomatic. These fish should be stocked under strict control and only into known infected areas, or be processed and used as table fish. It is important that the water supplies be made free of infection and that earthen ponds be replaced with concrete or completely disinfected. If fish must be raised in contaminated earthen ponds, the fish should be first raised to at least 7-13 cm (3-5 in) long and to at least 8 months of age in uncontaminated water before moving them to the contaminated earthen ponds. Covering the bottoms of contaminated earthen ponds with plastic and use of spore-free water has been effective in preventing clinical signs of whirling disease in Denmark (Hoffman 1976).

REFERENCES

Bogdanova, E.A. 1970. On the occurrence of whirling disease of salmonids in nature in U.S.S.R. Proc. 2nd Int. Congr. Parasitol. J. Parasitol. 56. Abstr. No. 719.

Christensen, N. 0. 1972. Some diseases of trout in Denmark, p. 83-88. In L. E. Mawdesley-Thomas (ed.) Diseases of fish. Symp. Zool. Soc. Lond. No. 30. Academic Press. 380 p.

Christensen, N.O., and E. A. Bogdanova. 1973. Whirling disease (Myxosomiasis) in salmonids, p. 217-223. In W.A. Dill (ed.) Symposium on the major communicable fish diseases in Europe and their control. EIFAC/T17 (Suppl. 2). FAO, Rome, Italy.

Halliday, M.M. 1973. Studies of Myxosoma *cerebralis,* a parasite of salmonids. II. The development and pathology of *Myxosoma cerebralis* in experimentally infected rainbow trout *(Salmo gairdneri)* fry reared at different water temperatures. Nord. Veterinaermed. 25: 349-358.

Halliday, M.M. 1976. The biology of *Myxosoma* cerebralis: the causative organism of whirling disease in salmonids. J. Fish Biol. 9: 339-357.

Hoffman, G. L. 1974. Disinfection of contaminated water by ultraviolet irradiation with emphasis on *whirling* disease *(Myxosoma cerebralis)* and its effect on fish. Trans. Am. Fish. Soc. 103: 541-550.

Hoffman, G. L. 1975. Whirling disease *(Myxosoma cerebralis):* control with ultraviolet irradiation and effect on fish. J. Wildl. Dis. 11: 505-507.

Hoffman, G. L. 1976. Whirling disease of trout. U.S. Fish Wildl. Serv. Fish Dis. Leafl. No. 47. Washington, DC. 10 p.

Hoffman, G.L., and C.J. Byrne. 1974. Fish age as related to susceptibility to *Myxosoma cerebralis,* cause of whirling disease. Prog. Fish-Cult. 36: 151.

Hoffman, G.L., and J. J. O'Grodnick. 1977. Control of whirling disease *(Myxosoma cerebralis):* effects of drying and disinfection with hydrated lime or chlorine. J. Fish Biol. 10: 175-179.

Hoffman, G.L., and R.E. Putz. 1969. Host susceptibility and the effect of aging, freezing, heat and chemicals on spores of *Myxosoma cerebralis.* Prog. Fish-Cult. 31: 35-37.

McDaniel, D. (ed.). 1979, p. 93-101. In Procedures for the detection and identification of certain fish pathogens. Am. Fish. Soc., Fish Health Sec., Bethesda, MD.

Putz, R. E., and G. L. Hoffman. 1966. Earliest susceptible age of rainbow trout to whirling disease. Prog. Fish-Cult. 28: 82.

Schaperclaus, W. 1954. Fischkrankheiten. Akademie - Verlag, Berlin. 708 p.

Schaperclaus, W., H. Kulow, and K. Schreckenbach. 1979. Fischkrankheiten. Teil 2. Akademie - Verlag, Berlin. 583 p.

Wolf, K., and M. E. Markiw. 1982. *Myxosoma cerebralis:* inactivation of spores by hot smoking of infected trout. Can. J. Fish. Aquat. Sci. 39: 926-928.

Yoder, W. G. 1972. The spread of *Myxosoma cerebralis* in native trout populations in Michigan. Prog. Fish-Cult. 34: 103-106.

28

PROLIFERATIVE KIDNEY DISEASE OF SALMONIDS

A. J. SIPPEL
Fisheries Branch
Ministry of Natural Resources
Toronto, Ont.

and

H. W. FERGUSON
Ontario Veterinary College
University of Guelph
Guelph, Ont.

Proliferative kidney disease (PKD) is a condition of salmonid fishes first recognized in North America following an outbreak of the disease in late 1981 at the Hagerman State Fish Hatchery in Idaho. The disease has been known in Europe and the British Isles for many years where it is recognized as a major problem affecting rainbow trout production, especially in France and Italy (Ferguson and Ball 1979). On severely-affected farms, 100% of the fish may be affected and mortality may approach 50-60%. Although the cause is unknown, it is believed that a protozoan parasite is involved, either an amoeba-like organism (trophozoite?) (Ferguson and Needham 1978) or possibly a haplosporidian protozoan (Seagrave et al. 1980). The potential impact of the disease on North American fish culture is, as yet, undetermined.

SIGNS OF INFECTION

The following description of external and internal signs has been adapted from Ferguson and Needham (1978).

External Signs

Affected fish typically have a distended abdomen with longitudinal swelling of the body wall at the level of the lateral line. Some fish may show dark body coloration with varying degrees of mono- or bilateral exophthalmia. Prior to death, respiratory distress is obvious, probably due to a pronounced anemia. In the final stages, there is also a marked nervous agitation with a loss of equilibrium.

Internal Signs

Internally, the most obvious change is a gross enlargement of the kidneys into swollen, greyish, bulbous ridges, This condition invariably involves the posterior portion of the kidney but, in very severe cases, it can extend along the whole length of the kidney and include the anterior hematopoietic tissue. The swim bladder may be laterally displaced and distorted and the abdominal swelling may be compounded by excess peritoneal fluid. The spleen may be smaller than normal or massively enlarged with patches of greyish mottling beneath the capsule and throughout the stroma. The liver may show a similar greyish mottling.

DIAGNOSIS

Diagnosis of the disease requires histopathological examination of kidney and other tissues for the presence of the distinctive protozoan-lie agent and associated tissue reaction. Gross lesions must be consistent with those described above and with the histopathological lesions described by Ferguson and Needham (1978). For the Great Lakes Basin, it is recommended that no diagnosis be made without consultation with a recognized authority who is familiar with the disease.

EPIZOOTIOLOGY

Geographic and Host RANges

PKD has only been reported in rainbow trout although it has been observed in brown trout and Atlantic salmon in Europe (Ferguson, unpublished observations). The disease occurs in Europe and the British Isles (Ferguson and Needham 1978). In North America, a single outbreak has occurred near Hagerman, Idaho.

Sources and Reservoirs of Infection

A possible secondary host has been suggested by Seagrave et al. (1980) but further studies on the causative agent and its life cycle are required.

Modes of Transmission

The mode of transmission is unknown, but Ferguson and Ball (1979) were unable to transmit the disease to rainbow trout reared for several months in water circulating through a tank of infected trout.

Susceptibility and Resistance Factors

The disease has usually been associated with soft, acidic water (Ferguson and Adair 1977). However, the outbreak in Idaho occurred in alkaline (pH 8.0) water similar to a minor outbreak of the disease on a chalk stream in southern England (Scott 1979). Fish that have recovered from an outbreak of PKD appear to be immune to further clinical disease in subsequent years.

Incubation Period

The incubation period is probably temperature dependent (Ferguson and Ball 1979; Ferguson 1981). Clinical disease may not develop if water temperatures are low (Ferguson 1981). At a northern Irish fish farm where summer water temperatures exceeded 15°C, the incubation period was approximately 2 months (Ferguson and Ball 1979).

Seasonal Incidence

In Northern Ireland and Scotland, the disease occurs during summer months (mid-July to early September) when water temperatures exceed 15°C (Ferguson and Ball 1979). The outbreak in Idaho occurred in November when water temperatures were approximately 15°C. Similarly, an outbreak was diagnosed in West Germany in mid-winter; again, the water temperature was 15-18°C (W. Korting, Tierarztliche Hochschule, personal communication).

METHODS OF CONTROL

Prevention

Until the cause of PKD disease is better understood, restricting the transfer of fish (and water) from areas where the disease is known to occur appears to be the best preventive measure.

Therapy

Chemotherapy has proved unsuccessful although work continues in Europe (Ferguson and Ball 1979). Changes in management have been successful in reducing losses from the disease but not in eliminating infections (Ferguson and Ball 1979). Avoiding prolonged exposure (greater than 2 months) to water temperatures in excess of 15°C during the fingerling stage appears to be the key to controlling clinical disease (Ferguson 1981).

KEY STEPS TO REMOVE THE DISEASE AND/OR AGENT FROM FISH POPULATIONS

As indicated above, PKD can be controlled in certain circumstances by controlled management practices. Methods to eliminate the disease after it has become established are unknown and await a better understanding of the causative agent's life history. The success of the eradication procedures (stock destruction and chlorination) employed in Idaho are not yet known. The Great Lakes Fishery Commission recommends that emergency disease eradication procedures be applied if PKD is diagnosed.

REFERENCES

Ferguson, H. W. 1981. The effects of water temperature on the development of proliferative kidney disease in rainbow trout, *Salmo gairdneri* Richardson. J. Fish Dis. 4: 175-177.

Ferguson, H. W., and B. McC. Adair. 1977. Protozoa associated with proliferative kidney disease in rainbow trout *(Salmo gairdneri)*. Vet. Rec. 100: 158-159.

Ferguson, H. W., and H. J. Ball. 1979. Epidemiological aspects of proliferative kidney disease amongst rainbow trout, *Salmo gairdneri* Richardson, in Northern Ireland. J. Fish Dis. 2: 219-225.

Ferguson, H.W., and E.A. Needham. 1978. Proliferative kidney disease in rainbow trout, *Salmo gairdneri* Richardson. J. Fish Dis. 1: 91-108.

Seagrave, C.P., D. Bucke, and D.J. Alderman. 1980. Ultrastructure of a Haplosporean-like organism: the possible causative agent of proliferative kidney disease in rainbow trout. J. Fish Biol. 16: 453-459.

Scott, P.W. 1979. The occurrence of proliferative kidney disease on a chalk stream. Vet. Rec. 105: 330-331.

PART VI

APPENDICES AND GLOSSARY

APPENDIX I. TRAINING AND INFORMATION

APPENDIX II. COMMON AND SCIENTIFIC NAMES OF FISH SPECIES

APPENDIX III. GREAT LAKES FISH DISEASE CONTROL PROGRAM

GLOSSARY OF FISH HEALTH TERMS

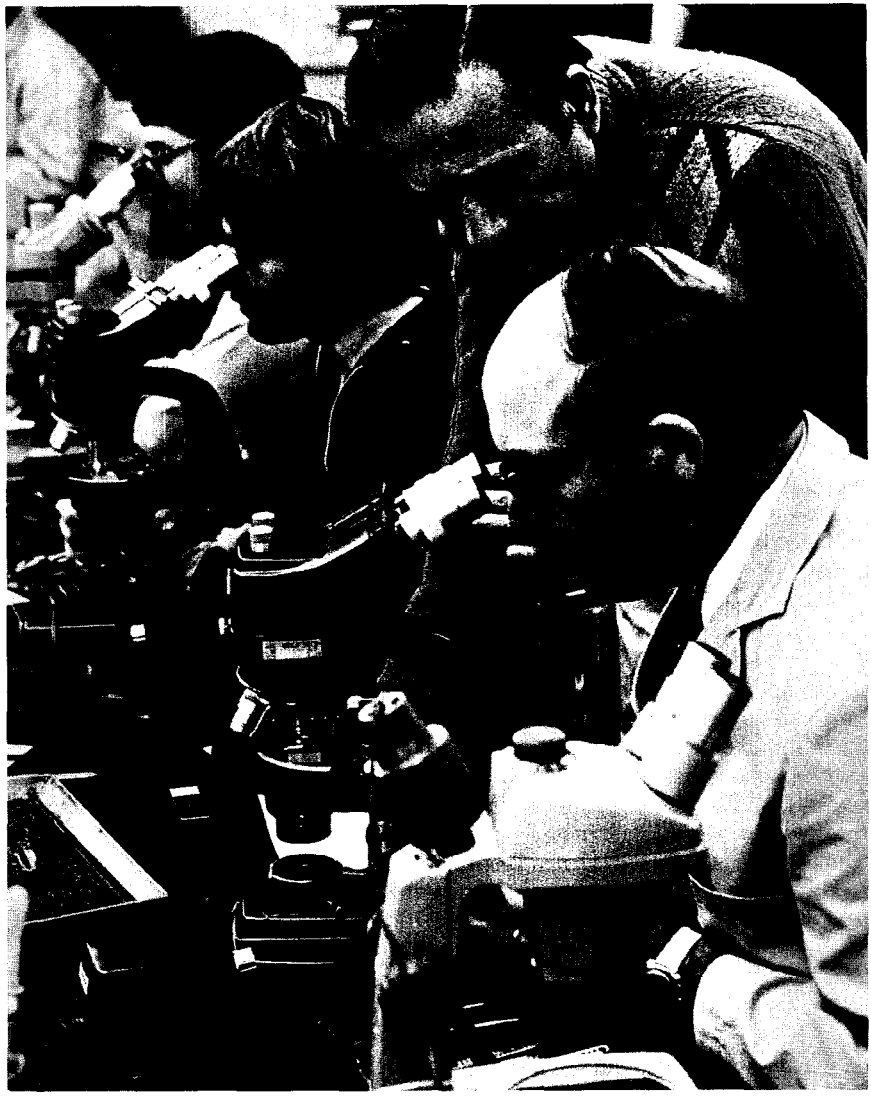
APPENDIX I: TRAINING AND INFORMATION NEEDS

R. W. RITZERT
Owens-Illinois
Corporate Technology
Toledo, OH

Proper and appropriate methods of fish health management require continual updating and revision as new developments in our knowledge of diseases and their control occur. The practicing fish culturist is dependent upon the transfer of information from researchers to assist him in making the best possible decisions in managing to prevent or treat fish diseases. That information is usually available in the form of reports or publications, but such information is often not widely disseminated or available to all fish culturists. Therefore, the most effective transfer of information is often transmitted through short courses, trade associations, or word of mouth. Such sources also help to serve the needs of the novice fish culturist, for whom training and information is especially important, in making appropriate management decisions.

CONTINUING EDUCATION OPPORTUNITIES

Several short courses (less than one week duration) and other more intensive training opportunities in relation to fish diseases in the Great Lakes region are available to fish culturists from both the public and private sectors. In general, the short courses are intended to assist individuals in recognizing signs of disease, treating those diseases, and in managing to prevent their recurrence. Intense courses of study are available for those persons whose position or interests may require disease diagnostic capabilities in addition to appropriate therapeutic knowledge. Individual needs can best be satisfied by contacting the appropriate laboratory or institution for a course outline, specific dates of offering, and application forms. The courses which should best assist hatchery personnel in the Great Lakes area are listed in Table 1.



Students on a fish disease short course at the National Fisheries Center, Leetown, WV (U.S. Fish and Wildl. Serv.)

TECHNICAL EXCHANGE

As knowledge and techniques for fish health management are developed, it is necessary that such information be transmitted to fish culturists on a timely basis. The exchange of scientific information traditionally occurs through the publication of results in technical journals, oral presentation of papers at scientific meetings, and workshops or seminars dedicated to a specific topic. A list of

periodicals with technical and non-technical information on the subjects of fish diseases and fish health management is provided below.

Aquaculture Magazine
Fish Disease Leaflets
Fish Health News
Journal of Fish Biology
Canadian Journal of Fisheries and Aquatic Sciences
Journal of Ichthyology
Progressive Fish Culturist
Salmonid
Transactions of the American Fisheries Society

Annual meetings of the American Fisheries Society (5410 Grosvernor Lane, Bethesda, MD 20014), and the U.S. Trout Farmers Association (PO. Box 171, Lake Ozark, MO 65049) and Ontario Trout Farmers Association (c/o Trillium Springs Fish Farm, R.R. 2, Holland Centre, Ontario N0H 1R0) offer opportunities to hear presentations of recent scientific findings. The AFS meetings can be expected to be technical in scope, while the USTFA and OTFA meetings will provide information directed toward applications of new knowledge.

Workshops offer excellent opportunities for the exchange of ideas among scientists and fish culturists. Regional fish disease workshops are held on an annual basis exactly for this purpose. Detailed information on dates and program are available from the U.S. Fish and Wildlife Service, Fish Disease Control Center, I? 0. Box 1595 La Crosse, WI 54601-0146.

CONCLUSIONS

Adequate opportunities for training and updating fish culturists on fish health management techniques exist. The real concern then becomes the need for greater participation by fish culturists in these activities. Within the public sector, budgetary restrictions and priorities at the hatchery often prohibit the attendance of the hatchery manager or biologist at conferences, workshops, or short courses. Fish culturists in the private sector suffer the same problems, in addition to a lack of awareness of what can be gained.

The need for fish health management must be impressed on all fish culturists. Public agencies should insure that all facilities, both public and private are promptly made aware of any new activities or techniques related to fish health management as they are developed. The present flow of information on a timely basis all too often excludes fish culturists in the private sector, and even then, the reports are not on a level that is useful or understandable to them. Their interests in fish health management exist but are not always fertilized. Primary contact with other fish culturists is through regional or national trade associations. Agencies, as a group or singly, need to include all of the fish culture community in the flow of information on this subject. Communication, and not regulation, will establish a positive approach to educating fish culturists at all levels.

Table 1. Continuing Education in Fish Health Management

Sponsoring Institution/Agency	Course Title	Instructor	Location	Frequency	Contact
Fish and Wildlife Service, U.S. Dept. of Interior: and Univ. of Wisconsin La Crosse	Introduction to Fish Health	J.W. Warren H.M. Jackson	La Crosse, WI	Annual (one-week course)	J. W. Warren: Fish Disease Control Center; P.O. Box 1595; La Crosse, WI 54601-0146 AC 6081783-6451
Fisheries Academy, Fish and Wildlife Service U.S. Dept. of Interior	Fish Health Management	Staff of National Fish Health Research Laboratory	Leetown, WV	Annual (Nine-month course)	Superintendent Fisheries Academy; National Fisheries Center; Leetown; Route 3; Box 700; Kearneysville, WV 25430 AC304/725-8461
	Fish Diseases	"	"	Annual (one-week courses)	"
University of Idaho	Fish Health Management	G.W. Klontz	At Hatchery	As requested by host agency	Dr. G.W. Klontz; Dept. of Fish and Wildlife Resources; Univ. of Idaho: Moscow, Idaho 83843; AC 280/885-6336
Haywood Technical College	Fish Health	Invited Experts	Clyde, NC	Annual	Charles W. Johnson: Fishery Training Specialist; Haywood Technical College: Freedlander Drive; Clyde, NC 28721-9454 AC 704/627-2821

APPENDIX II: COMMON AND SCIENTIFIC NAMES OF SELECTED FISHES

COMMON NAME	SCIENTIFIC NAME
Salmonidae	
Altantic Salmon	
Atlantic salmon (anadromous form)	*Salmo salar*
Landlocked salmon (freshwater form)	*Salmo salar*
Grayling (Arctic)	*Thymallus arcticus*
Grayling (European)	*Thymallus thymallus*
Pacific Salmon	
Amago salmon	*Oncorhynchus rhodus*
Chinook salmon	*Oncorhynchus tshawytscha*
Chum salmon	*Oncorhynchus keta*
Coho salmon	*Oncorhynchus kisutch*
Pink salmon	*Oncorhynchus gorbuscha*
Himemasu salmon (Japan)	*Oncorhynchus nerka*
Sockeye salmon (North American)	*Oncorhynchus nerka*
Kokanee (land-locked sockeye salmon)	*Oncorhynchus nerka*
Trout and Char	
Brook trout	*Salvelinus fontinalis*
Brown trout	*Salmo trutta*
Char (Arctic)	*Salvelinus alpinus*
Cutthroat trout	*Salmo clarki*
Lake trout	*Salvelinus namaycush*
Rainbow trout (freshwater form)	*Salmo gairdneri*
Steelhead trout (anadromous form)	*Salmo gairdneri*

COMMON AND SCIENTIFIC NAMES OF SELECTED FISHES (Continued)

COMMON NAME	SCIENTIFIC NAME
Other species	
Bream	*Abramis brama*
Carp	*Cyprinus carpio*
Catfish	*Ictalurus spp.*
Eel	*Anguilla rostrata*
Goldfish	*Carassius auratus*
Muskellunge	*Esox masquinongy*
Perch	*Perca fluviatilis*
Pike	*Esox lucius*
Roach	*Rutilus rutilus*
Sea Lamprey	*Petromyzon marinus*
Walleye	*Stizostedion vitreum vitreum*
White sucker	*Catastomus commersoni*

Reference: Robins, C.R., R.M. Bailey, C.E. Bond, J.R. Booker, E.A. Lachner, R.N. Lea, and W.B. Scott. 1980. A list of common and scientific names of fishes from the United States and Canada. Am. Fish. Soc., Spec. Publ. No. 12. Bethesda, MD. 174 p.

APPENDIX III: GREAT LAKES FISH DISEASE CONTROL PROGRAM

GREAT LAKES FISHERY COMMISSION

1451 GREEN ROAD

ANN ARBOR, MI 48105

The following recommendations of the Great Lakes Fish Disease Control Committee of the Great Lakes Fishery Commission were approved by the Commission at its 1975 Annual Meeting. These recommendations are hereby transmitted to the member agencies.

(1) The Commission recommends agency adoption of the Great Lakes Fish Disease Control Policy and implementation of appropriate elements of the Model Fish Disease Control Program. Both are attached. The Fish Disease Control Policy is the established policy of the Commission. The Model Fish Disease Control Program is hereby presented to the member agencies as a Commission guide to the coordinated development of fish disease controls in the Great Lakes basin.

(2) The Commission recommends agency endorsement and active support of legislation similar to H.R. 1083 "The Fish Disease Control Act of 1975".

(3) The Commission recognizes the threat of certain infectious diseases and recommends their inclusion in the Great Lakes Fish Disease Control Program according to the following three major categories:

a. *EMERGENCY DISEASES*
Whirling Disease caused by *Myxosoma cerebralis; Ceratomyxa shasta* infections of salmonids; Viral Hemorrhagic Septicemia (VHS); Proliferative Kidney Disease.

b. *CERTIFIABLE DISEASES*
Whirling Disease caused by *Myxosoma cerebralis; Ceratomyxa shasta* infections of salmonids; Infectious Hematopoietic Necrosis (IHN); Viral Hemorrhagic Septicemia (VHS); Enteric Redmouth (ERM) caused by *Yersinia ruckeri.*

In addition, the following diseases shall be monitored for observational and hatchery disease classification purposes:

Infectious Pancreatic Necrosis (IPN)
Bacterial Kidney Disease (BKD)
Furunculosis

c. *REPORTABLE DISEASES*
In addition to the diseases listed in 2 above, the following diseases shall be reported should they be detected:

Protozoans -*Ichthyophthirius*
Copepods - *Lernaea, Salmincola*

Drug resistant or velogenic strains of - Motile Aeromonads
Pseudomonads
Columnaris

Other diseases, as determined by the Great Lakes Fish Disease Control Committee.

(4) The Commission recommends the prompt and effective eradication of "Emergency Diseases", wherever practicable, which are detected in the Great Lakes basin.

(5) The Commission recommends that each donor agency furnish a report of the disease history of all eggs and fish to the receiving agency prior to the transfer of such stocks.

The Great Lakes Fishery Commission also expresses its appreciation to the Great Lakes Fish Disease Control Committee members and Chairman, Mr. James W. Warren, who spent numberless hours in cooperative toil developing the policy and recommendations.

W.M. Laurence, Chairman
GREAT LAKES FISHERY COMMISSION

Updated June, 1982

GREAT LAKES FISH DISEASE CONTROL POLICY

Efficient propagation of fish may be severely affected by the occurence of fish diseases. Major disease outbreaks have caused serious losses in fish hatcheries and in Great Lakes fish populations as well. Disease problems have resulted in reduced survival of stocked fish, production cost increases of 20 to 30 percent, significant losses of fish to the public, and diminished economic returns to Great Lakes communities.

To work toward the attainment of fish disease control in the Great Lakes basin, it shall be the policy of the Great Lakes Fishery Commission to encourage each member agency to:

- develop, by 1980, legislative authority and regulations to allow control and possible eradication of fish diseases,
- prevent the release of seriously diseased fish,
- discourage the rearing of diseased fish,
- prevent the importation, into the Great Lakes basin, of fish infected with certain certifiable diseases,
- prevent the transfer, within the Great Lakes basin, of fish infected with certain certifiable diseases, and
- eradicate fish diseases wherever practicable.

The Great Lakes Fishery Commission will strive to coordinate the fish disease control program of the member agencies. To this end the Commission endorses and supports the following Fish Disease Control Program as a guide for member agency program development.

GREAT LAKES FISH DISEASE CONTROL PROGRAM*

INTRODUCTION

Fish disease control in the Great Lakes basin is the responsibility of the natural resources agencies managing the fisheries resources. The Fish Disease Control Committee of the Great Lakes Fishery Commission has developed this model program designed to unify and coordinate the fish disease control efforts of the member agencies. This program sets forth the essential requirements for the prevention and control of serious fish diseases. These include a system for inspecting and certifying fish hatcheries and the technical procedures to be used.

The major elements of this program and its annexes have been liberally adapted from the "Draft International Convention for the Control of Major Communicable Fish Diseases" of the European Inland Fisheries Advisory Commission (EIFAC) developed at Aviemore, Scotland, April 30 - May 1, 1974. In addition, guidance was also provided by the "Resolution of the Colorado River Drainage System" and from written policies of the United States Fish and Wildlife Service.

* This is an edited version of the Model Fish Disease Control Program. Detailed copies may be obtained by writing to:
Great Lakes Fishery Commission,
1451 Green Road,
Ann Arbor, MI 48105

The Committee wishes to make it abundantly clear that it is in no way seeking fish disease control authority. The recommendations advanced by this program are provided as an aid to the member agencies in the development of legislation, regulations, and the Committee seeks the advice and counsel of the member agencies in the continuing development of fish disease control programs to assure they serve the best interests of all Great Lakes fishery resources.

Section A. Definitions

For the purposes of this program the term:

(1) "Commission" means the Great Lakes Fishery Commission;

(2) "member agency" means the fishery management or conservation agency of each Federal, Provincial, or State government normally participating in the activities of the Commission;

(3) "Great Lakes basin' means that geographical area encompassing Lake Ontario (including the St. Lawrence River from Lake Ontario to the forty-fifth parallel of latitude), Lake Erie, Lake Huron (including Lake St. Clair), Lake Michigan, Lake Superior, their interconnecting waters, and all tributaries to said lakes and waters;

(4) "fish" means live fish, viable fish eggs, viable sperm, or fish products used for fish foods which have not been so processed as to render them incapable of transmitting a certifiable fish disease;

(5) "fish hatchery" means any facility which holds, rears, or releases fish of the species listed in Annex I in the waters of the Great Lakes basin or whose effluent waters drain into said basin;

(6) "certifiable fish disease" means certain infectious diseases of fish caused by viral, bacterial, or parasitic agents which are transmissible, directly or indirectly, from one fish to another;

(7) "certificate" means Fish Disease Inspection Certificate as referred to in Section F. and exhibited in Annex II; and

(8) "certifying official" means those fish health specialists who meet the requirements set forth in Section G.

Section B. *Basic Obligation*

The member agencies shall take all appropriate measures including the development of legislative authority and regulations, where necessary, to restrict the spread of certifiable fish diseases, to contain them within their known geographic ranges, and to strive for their elimination in accordance with the provisions of this program.

Section C. *Application*

(1) The provisions of this program apply to:
(a) fish of the species identified in Annex I;
(b) certifiable fish diseases as listed in Annex IV;
(c) fish disease research on fish infected with, or exposed to, certifiable fish diseases and/or the possession of the infectious agents causing these diseases.

(2) The provisions of this program shall not apply to:
(a) fish in transit through the Great Lakes basin which are not released from their original shipping containers;
(b) specimens of fish imported or exported for purposes of diagnostic or inspection services and related laboratory tests provided that all necessary biological containment measures are taken to avoid any dissemination of fish pathogens.

(3) Nothing in this program shall derogate from the right of the member agencies to apply additional measures of inspection, quarantine, and disease eradication for the control of fish diseases.

Section D. *Traffic in Fish*

Except as provided in Section C, paragraph (2), no fish may be imported into the Great Lakes basin, transferred between fish hatcheries within the basin, or released into basin waters unless:

(1) In the case of fish imported into the basin or transferred between hatcheries, the source hatchery possesses a valid certificate issued by a certifying official in accordance with Section F.

(2) In the case of fish imported from outside the jurisdiction of a member agency, they are accompanied by a certificate or other document giving equivalent assurance as to the state of health of the fish which is prepared and signed by a certifying official in accordance with Section F.

Section E. *Release of Fish*

(1) No fish hatchery may release fish in the Great Lakes basin until a fish disease inspection certificate has been issued.

(2) No fish exhibiting clinical signs of any infectious disease may be released into the waters of the Great Lakes basin.

(3) Hatchery certification shall be immediately revoked upon the confirmation of any certifiable disease and releases prohibited until until the hatchery once again meets the requirements for certification.

Section F. *Fish Disease Certificates*

(1) Fish disease inspection certificates, listing the certifiable fish diseases, shall be in the form prescribed in Annex II.

(2) Certificates may only be issued by the certifying official performing the on-site fish hatchery inspection.

(3) Certificates, valid for a period not to exceed one year subject to the provision of Section E, paragraph (3), may be issued only after inspection, by means of the procedures set forth in Annex III, fails to reveal evidence of certifiable fish diseases.

(4) Fish disease inspection certificates and on-site hatchery inspections shall be used to support a hatchery classification plan such as described in Annex V for the purpose of fish disease control.

Section *G. Certifying Officials*

(1) Each member agency shall identify by name those individuals whom the agency desires to be responsible for carrying out inspections and issuing certificates in accordance with this policy.

(2) Competence of certifying officials shall be based upon standards set forth by the Fish Health Section of the American Fisheries Society and/or by the Great Lakes Fish Disease Control Committee.

(3) All certifying officials shall have, or have access to, adequate laboratory facilities and qualified personnel to assure the prompt and accurate conduct of inspections and diagnoses under the procedures set forth in Annex III.

(4) Each member agency shall inform the Chairman of the Great Lakes Fish Disease Control Committee of the identity of certifying officials for the compilation and distribution of a list of certifying officials.

(5) All certifying officials shall submit copies of all certification forms to the appropriate member agency under whose jurisdiction the inspected hatchery lies.

Section H. *Reports by Member Agencies*

(1) Member agencies shall present to each periodic meeting of the Great Lakes Fish Disease Control Committee a report covering the status of fish diseases, the measures adopted for their control, the activities and problems of their certifying officials, and such other information as may be requested to enhance the effectiveness of this program.

(2) The Chairman of the Great Lakes Fish Disease Control Committee shall maintain records of the reports submitted to him in an appropriate form.

Section I. *Amendment of the Model Program and the Annexes*

Amendment to this model program or its annexes may be proposed by any member agency or by the Great Lakes Fish Disease Control Committee. Any such proposal made by a member agency shall be submitted to the Committee for its comments and recommendations thereon. The proposed amendment, together with the comments and recommendations of the Committee, shall be communicated to the Commission for consideration.

ANNEX I

SPECIES COVERED BY THE PROGRAM

All species and hybrids of the family Salmonidae are subject to provisions of the Fish Disease Control Program for the Great Lakes basin.

ANNEX II

(FISH DISEASE INSPECTION FORM - NOT SHOWN)

ANNEX III

INSPECTION PROCEDURES AND METHODS OF DIAGNOSIS

A. *INSPECTION PROCEDURES*

The data obtained from the inspection program is an essential part of our program to control and improve the quality of fish that are produced at fish hatcheries. Therefore, it is essential that all hatchery inspections be conducted in accordance with the following procedures:

1. *Sample Population.* - The following definitions will apply to the designation of populations for sampling purposes:

   a. For all fish except those being inspected for whirling disease, the sample population is determined on the basis of the lot and production environment. For our purposes the lot is defined as those fish which originated from the same broodstock during the same year, and are being raised on the same water source.

   b. When conducting a whirling disease inspection, the sample population is defined as all fish in the hatchery five (5) months of age or

older held in the same water supply. Samples should be weighted toward the most susceptible species and ages of fish available. Whirling disease spores are difficult to detect in lake trout and coho salmon and in fish larger than 12 inches in length.

c. Wild broodstocks must be inspected at least once during the time that eggs destined for a Great Lakes basin hatchery are being obtained. All brookstock present at the time of inspection will constitute the sample population. The sample size should be large enough to detect diseases at an assumed incidence of infection of 2 percent. Where it is not feasible to sample wild brookstocks at the 2 percent assumed incidence level a smaller sample may be taken at the discretion of the inspecting pathologist after all risks are considered.

2. *Sample Size*

a. For viral and parasitic diseases the number of samples to be collected from a given lot is based upon stratified random sampling which provides 95 percent confidence of detecting a disease with an assumed minimum incidence of detectable infection of two or five percent depending upon conditions outlined as follows:

Minimum sample sizes for populations varying from 50 to infinity are as follows:

Population or lot size	Assumed Incidence 2%	5%
	Size of Sample	
50	50	35
100	75	45
250	110	50
500	130	55
1,000	140	55
1,500	140	55
2,000	145	60
4,000	145	60
10,000	145	60
100,000 and any larger	145	60

The above sample sizes are minimum, and in situations where disease is suspected, larger samples may be necessary and should be taken at the discretion of the pathologist.

The method of collecting subsamples from rearing units to obtain a representative sample is left to the discretion of the pathologist.

b. For bacterial diseases - Sampling of broodstock populations and production fish should be accomplished on a continuing basis throughout the year using the moribund and/or dying fish whenever possible. Samples of fixed material for the detection of the gram positive *Renibacterium* can be sent to agency laboratories by hatchery managers on a periodic basis. Training should be provided to hatchery managers in preparing culture material for diagnosis of the Gram-negative bacterial pathogens. Cultures also can be sent to agency laboratories for confirmatory diagnosis. The annual case history of each designated lot should be compiled by the pathologist using this accumulated sampling data. The minimum number of samples is left to the discretion of the pathologist. Symptomatic and moribund fish should be sampled during any inspection.

B. *METHODS OF DIAGNOSIS*

The "Procedures for the Detection and Identification of Certain Fish Pathogens", developed by the Fish Health Section (FHS) of the American Fisheries Society or the "Fish Health Protection Manual of Compliance" of the Department of Fisheries and Oceans, Canada provide the basis for the work supporting fish hatchery inspections and certifications. If more sensitive or more definitive procedures are available, they may be used but any departures from the basic procedures set forth by the FHS must be noted on all associated inspection certificates. The Fish Disease Control Committee, in an effort to encourage the use of the best possible methodology, should be notified of technical advances enhancing the implementation of the program. Procedural changes issued by the FHS will be incorporated into the program by the Committee as appropriate.

ANNEX IV

LIST OF DISEASE AGENTS COVERED BY THE PROGRAM

1. Inspections of fish populations shall be conducted so as to detect evidence of the following CERTIFIABLE fish disease agents.
   a. Viral Hemorrhagic Septicemia (VHS) Virus
   b. Infectious Hematopoietic Necrosis (IHN) Virus
   c. Whirling Disease caused by *Myxosoma cerebralis*
   d. *Ceratomyxa shasta* for all salmonids reared and/or shipped from west of the North American Continental Divide.
   e. Enteric Red Mouth (ERM) caused by *Yersinia ruckeri*

2. In addition to the above certifiable disease agents, inspections shall also be conducted to include the following disease agents for observational purposes in order to support hatchery classification programs:
   a. Infectious Pancreatic Necrosis (IPN) Virus
   b. Furunculosis caused by *Aeromonas salmonicida*
   c. Bacterial Kidney Disease (BKD) caused by *Renibacterium*

If these disease agents are detected these findings shall be noted on the inspection certificate and incorporated in the hatchery classification.

3. The causative agents of Infectious Pancreatic Necrosis (IPN), furunculosis, and Bacterial Kidney Disease (BKD) shall be added to the list of certifiable fish disease agents at a date and time deemed appropriate by the Great Lakes Fish Disease Control Committee.

ANNEX V

HATCHERY DISEASE CLASSIFICATION PROGRAM

A. *DISEASES*

Each hatchery rearing salmonids and each spawning population, whether wild or domesticated, will be inspected and classified for the following:

DISEASE OR AGENT	*ABBREVIATION*
Viral hemorrhagic septicemia (VHS) virus	VE
Infectious hematopoietic necrosis (IHN) virus	VH
Infectious pancreatic necrosis (IPN) virus	VP
Bacterial kidney disease (BKD) *(R. salmoninarum)*	BK
Furunculosis *(A. salmonicida)*	BF
Enteric redmouth (ERM) (Y *ruckeri*)	BR
Whirling Disease (M. *cerebralis)*	SW
Ceratomyxosis (C. *shasta)*	SC

B. *CLASSIFICATION*

1. Class *A-l*

The A-l classification is assigned to those fish hatcheries meeting the following criteria:
(1) All fish cultural water must be obtained from enclosed sources such as springs or wells which are free of resident fish.
(2) All fish reared on the station must have been inspected for all diseases listed above, at least annually. Three successive negative inspections over a continuous two year period are required.
(3) To maintain A-l status hatcheries must assure that all fish or eggs have been obtained only from properly inspected Class A-l or Class A-2 sources.

2. Class A-2

The A-2 classification differs from A-l only to the extent that the hatchery has an open water supply such as a stream or lake with resident fish. The A-2 classification is also assigned to discrete spawning populations of free-ranging fish which have met all other class A-l inspection requirements.

3. Class B

Hatcheries and free-ranging spawning populations are assigned a B classification when one or more of the diseases or disease agents listed in (l)(a) above have been detected within the past two years.

4. Class C

Hatcheries and free-ranging spawning populations having an unknown disease history, have not been inspected for all diseases listed, or have undergone only one or two complete annual inspections, will be assigned a C classification.

C. RESTRICTIONS

No shipments of fish or eggs will be made without prior approval of the receiving authorities whenever that shipment will knowingly downgrade the classification of the receiving hatchery. Shipments of fish or eggs between hatcheries will be governed by the disease status of the hatcheries involved. At least one inspection for each designated disease, except as noted above for unnecessary or unavailable samples, will be conducted on all lots of salmonids, regardless of age, prior to the transfer of eggs or the transfer or stocking of fish.

GLOSSARY OF FISH HEALTH TERMS

Abrasion:	a localized area denuded of skin, mucous membranes, or superficial epithelium caused by rubbing or scraping.
Abscess:	a localized inflammation and swelling, frequently filled with necrotic debris and white blood cells.
Acclimation:	the process through which fish become fully adapted to new environmental circumstances; such as being placed into water of different quality, temperature, or different holding situations.
Acid fast:	bacteria that retain red phenolic fuchsin stain after being treated with acid alcohol solution.
Acute:	severe or crucial, often progressing rapidly; i.e., acute inflammation.
Adhesion:	the abnormal fibrous union of an organ or part to another.
Adjuvant:	a material administered with and enhancing the action of a drug or antigenic substance.
Adipose (tissue):	fatty animal tissue.
Aerobic:	said of an organism or life process that utilizes or can only exist in the presence of oxygen.
Anadromous:	fish that leave the sea and migrate to fresh water to spawn.
Anaerobic:	said of an organism or life process that flourishes in the absence of oxygen.
Anemia:	a condition characterized by a deficiency of hemoglobin or red blood cells (erythrocytes).
Aneurysm:	a sac formed by the dilation of the walls of an artery or a vein and filled with blood.
Anthelmintic:	an agent that destroys or expels parasitic worms in the gut.

Antibiotic:	a chemical substance produced by living organisms, usually mold or bacteria, that is capable of inhibiting other organisms.
Antibody:	a specific immunoglobulin molecule produced by an organism in response to an antigen.
Antigen:	a high molecular weight protein or polysaccharide which stimulates the formation of specific antibody with which it will react. Examples include killed bacterial cells or flagella.
Bacteremia:	the presence of living bacteria in the blood, with or without significant response on the part of the host; usually refers to a generalized bacterial infection in the blood.
Bacterin:	a vaccine prepared from bacteria that have been inactivated by heat or chemicals without altering the cell antigens.
Bacteriocidal:	having the ability to kill bacteria.
Bacteriostatic:	having the ability to inhibit or retard the growth or reproduction of bacteria.
Benign:	not endangering life or health.
Boil:	a furuncle; a localized infection or abscess within subcutaneous tissue that drains externally.
Carcinogen:	any agent or substance which produces cancer or accelerates the development of cancer.
Carrier:	an individual harboring the specific organism(s) which can cause a disease, without indication of signs of the disease.
Catadromous:	fish that leave fresh water and migrate to the sea to spawn.
Cataract:	partial or complete opacity of the crystalline lens of the eye or its capsule.
Chemotherapeutic:	a chemical agent used for the prevention or treatment of disease.
Cilia:	short hair-like processes on protozoans by which they move or produce currents.

Clinical:	when applied to a disease or signs of disease, a term that indicates a condition is readily apparent, overt, or obvious by gross inspection.
Coagulation:	the process of clotting.
Communicable disease:	a disease that is naturally transmitted directly or indirectly from one individual to another.
Complement:	factors present in the serum of normal animals which enter into various immunologic reactions.
Culture:	population of bacteria grown on artificial medium.
Culture media:	material (solid or liquid) on which bacteria are grown.
Disease:	a pathological condition of the body that presents a group of of signs indicating the existence of an abnormal histological or physiological entity.
Disinfectant:	an agent which will destroy infective agents.
Ectoparasite:	a parasite that lives on the external surface of the host.
Edema:	excessive accumulation of fluid in the tissue space or body cavities.
Embolus:	undissolved material carried in the bloodstream, such as a blood clot, air bubbles, cancerous or other tissue cells, fat, clumps of bacteria, or a foreign body.
Endogenous:	originating in the cells or tissues of the body.
Endoparasite:	a parasite that lives within the host.
Enteritis:	any inflammation of the intestinal tract.
Enzootic:	a disease which is present in an animal population at all times.
Epizootic:	outbreak of disease attacking many animals in a population at the same time and rapidly spreading.
Etiology:	the study of the causes of a disease.

Exophthalmos:	abnormal protrusion of the eyeball from the socket.
Facultative fish pathogens:	occurring naturally as non-pathogens in the environment but capable of causing disease outbreaks under conditions of stress.
Flagella:	whip-like organelles of locomotion on protozoans.
Free-Living:	not requiring a host to survive.
Furuncle:	a localized infection of skin or subcutaneous tissue which develops a solitary abscess that may or may not dram externally.
Gram-negative:	bacteria which lose the purple crystal violet stain when treated with alcohol solution in the Gram-staining process.
Gram-positive:	bacteria which retain the purple crystal violet stain when treated with alcohol solution in the Gram-staining process.
Gross pathology:	pathology that deals with the superficial or overt appearance of organs and tissues.
Hematocrit:	volumetric relationship of the cellular elements of blood to the total blood volume; sometimes referred to as the packed cell volume.
Hemoglobin:	the respiratory pigment of erythrocytes capable of taking up and giving off oxygen.
Hemolysis:	destruction of erythrocytes.
Hemorrhage:	an escape of blood from the vessels, either through intact blood vessel walls or through ruptured vessels.
Histopathology:	the study of microscopic changes in diseased tissue.
Host:	an animal or plant which harbors or nourishes another organism.
Hyper-:	a prefix denoting excessive, above normal or situated above.

Hyperplasia:	abnormal increase in the number of cells in a tissue or organ accompanied by enlargement or an increase in the size of the tissue or organ.
Hypertrophy:	enlargement of an organ due to an increase in the size of cells rather than in the number of cells.
Hypo-:	a prefix denoting a deficiency, less than normal, below or beneath.
Immunity:	resistance to disease; lack of susceptibility
Immunization:	the act or process of rendering immune by the introduction or administration of an antigen.
Incubation:	period of time between exposure or introduction of pathogens into the host and development of typical signs of disease.
Inflammation:	the reaction of the tissues to infection or injury characterized clinically by swelling and redness.
Inoculation:	the introduction of a pathogenic organism into the tissues of a living organism or culture medium.
Intra-:	within or between layers of same tissue.
In Vitro:	used in reference to tests or experiments conducted in vessels or in an artificial environment.
In Vivo:	used in reference to tests or experiments conducted in or on living organisms.
-itis:	a suffix indicating inflammation.
Lesion:	any visible alteration in the normal structure of organs, tissues, or cells.
Lordosis:	dorso-ventral curvature of the spine.
Lysozyme:	an enzyme which is capable of destroying certain bacterial cell walls.
Melanin:	a dark pigment responsible for the yellow to black coloration of fishes.
Moribund:	obviously progressing towards death, nearly dead.

Morphology:	the study of the form and structure of animals and plants.
Mortality:	the death rate, also the ratio of dead to living individuals in a population.
Mucus:	the slime produced by mucous membranes or by special cells in fish shin.
Necropsy:	a medical examination of animals to ascertain the cause of death.
Necrosis:	the process of death of cells or tissues within the living body.
Non-pathogenic:	refers to an organism which does not cause disease.
Obligate fish pathogens:	disease-causing organisms that cannot survive in nature unless susceptible or carrier fish are present.
-oma:	a suffix used to denote tumours, i.e. fibroma.
Overt disease:	a disease, not necessarily infectious, that is apparent or obvious by gross inspection; a disease exhibiting obvious clinical signs.
Parasite:	an organism that lives in or on another organism (the host), that depends on the host for its food, and that is suspected of harming the host when present in large numbers.
Pathogen, opportunistic:	an organism capable of causing disease only when the host's resistance is lowered or when unusual circumstances favor its growth and development.
Pathogenesis:	the origin and process of development of any disease or morbid process.
Pathogenic:	causing disease.
Petechia:	a minute hemorrhage on a surface.
Predisposing factors:	physical, chemical or biological factors which increase the susceptibility of an organism (host) to disease; sometimes called stressors.

Prophylaxis:	actions taken to prevent disease or measures taken to prevent the development or spread of disease.
Putrefaction:	the enzymatic decomposition of organic matter, especially proteins, by anaerobic micro-organisms.
Resistance:	a natural ability of an organism to withstand the effects of various physical, chemical, and biological agents which might otherwise cause disease in the organism.
Sensitive, drug:	susceptibility of a micro-organism, usually a bacterium, to be controlled (inhibited or killed by use of a drug).
Septicemia:	generally involving the significant invasion of the bloodstream by micro-organisms; a severe bacterial infection in the blood.
Sign:	any manifestation of disease, such as an aberration in structure, physiology, appearance or behaviour, as interpreted by an observer.
Specificity, host:	extreme host requirements that limit a parasite to one host species only. Loose host specificity indicates a parasite can infect many hosts.
Subcutaneous:	beneath the skin.
Synergism:	refers to an interaction wherein two agents produce a greater effect than would be predicted from the sum of their individual effects.
Scoliosis:	lateral curvature of the spine.
Therapeutic:	serving to heal or cure.
Toxicity:	ability of a substance to kill or cause an adverse effect.
Ubiquitous:	universally or widely distributed.
Ulcer:	an open sore or a break in the skin or a mucous membrane with loss of surface tissue; localized disintegration and necrosis of epithelial tissue.

Vaccine:	a preparation of nonvirulent or killed disease organisms administered into the body to stimulate the production of antibodies against them.
Vector:	a living organism which carries an infectious agent from one infected individual to another, directly or indirectly.
Virulence:	the relative capability of a pathogen to produce disease.