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SUBSTANCE OF FISH DISEASE "LONG COURSE" AT LEETOWN, WEST VIRGINIA, 1972-73

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ABSTRACT

The writer was afforded the opportunity, on an inservice training basis, to attend the Fish Diseases "long course" put on by the United States Department of Interior, Bureau of Sport Fisheries and Wildlife, at their Eastern Fish Disease Laboratory, Leetown, West Virginia. The "school" was a full-time operation, from August 28, 1972 to January 12, 1973. A highly competent teaching staff of eight persons (six with PhD's in special fields) covered the subjects of fish anatomy, physiology, parasitology, bacteriology, immunology, virology, and histopathology. In addition, distinguished visitors put on seminars. This report consists of a summary of the lectures, plus an extensive list of references.

Introduction

The advanced course in fish diseases, often known as the "long course," given at the Eastern Fish Disease Laboratory (E.F.D.L.), Leetown, West Virginia, is perhaps the most comprehensive course in North America on such a special topic. The course is taught by the research staff of the laboratory. This laboratory is a research unit of the Division of Fisheries Research, Bureau of Sport Fisheries and Wildlife, United States Department of the Interior. The course is funded by the Division of Fish Hatcheries as a training session for Federal fish hatchery biologists. As space is available, biologists from state fisheries and commercial fish establishments are admitted. Students from Fordham

University (New York City) and from foreign countries are also admitted to the course. I attended the course, on a regular schedule, from August 28, 1972 to January 12, 1973.

The organization of the course reads as follows:

Subject	Instructor
Review of fish anatomy and physiology	Mrs. M. Landolt
Parasitology	Dr. G. Hoffman, Dr. F. Meyer
Bacteriology	Dr. G. Bullock, Dr. S. Snieszko
Immunology	Dr. G. Pauley
Virology	Dr. K. Wolf, Mr. D. Amend
Histopathology	Dr. S. Snieszko, Mrs. M. Landolt

In general, the daily schedule is 8-10 a.m. lecture, 10:30 a.m.-12:00 noon study or lecture; 1-5 p.m. laboratory, or study if time permits. The amount of time spent on additional study for the various subjects depends upon the background and interest of each trainee. Each subject section is concluded with a written exam covering the lecture portion of the course. Virology is concluded with both a lecture and a lab examination.

The E.F.D.L. is located approximately 70 miles from Washington, D.C. This proximity to the National Capitol, coupled with the world renown of the staff at E.F.D.L., creates a valuable meeting place for national and international visitors. Frequently, such visitors present seminars on their work for the staff and trainees (when the long course is in session). Distinguished visitors during the 1972-73 long course were:

Dr. Michael Fijan, Yugoslavia	Parasitic, bacterial, and viral problems in European carp culture.
Dr. Ian Papurna, Israel	Studies of parasites of fish in Africa.
Dr. Robert Rucker, W.F.D.L. Seattle, Wash.	General disease problems of west coast salmonids.

Dr. Edwin L. Cooper, School of Medicine, Univ. of California, Los Angeles	Lymphoid control of amphibian immunity, and evolution of cellular immunity.
Dr. John Cisar, Dep. of Microbiology, Columbia Univ., New York, N. Y.	Properties of anti-Aeromonas salmonicida antibodies in coho salmon.

The purpose of this report is to partially review significant lecture materials of use to fisheries biologists; it is not intended to be a textbook on fish diseases or a training manual. The list of references at the end should be helpful to readers with special interests.

Anatomy and physiology

A review of fish anatomy and physiology was given, with the fish text by Lagler et al. (1962) as a reference. Background knowledge of anatomy is important whether one searches for parasites, bacteria and viruses, or seeks to discern histopathological changes. The diagnostician needs to know what is "normal," before the "abnormal" condition can be recognized. The physiological processes are important for understanding disease conditions and immunity responses in fish.

Parasitology

One of the most diverse subject areas is parasitology; and certainly fish parasites are diverse, with some 6,000 species. We study parasites for a number of reasons. They can cause severe fish mortalities; they are often not aesthetic to sport and commercial fishermen; they are a part of the total ecology; and they serve as biological tags, especially in marine fish. A desirable pattern of study (applying also to any infectious agent) would be as follows:

1. Identify the parasite.
2. Learn its life history.
3. Learn ecological requirements.
4. Geographical range.
5. Immunological mechanisms.
6. Control and treatment methods.

In general, a parasite is an organism which lives on or in another animal at the expense of that animal. There are exceptions to this rule,

such as a parasite merely anchored to the gills but feeding on detritus in the water surrounding the gills. Some parasites may be temporary, living on the host, dropping off, then re-attaching to another host. Other parasites may be secondary invaders, as with aquatic fungi. Still other parasites are merely opportunists, attacking fish only under certain conditions.

The kinds of parasites are many, as stated previously. A few are in the plant kingdom; most are animals. Most are in the following taxonomic groups (see text by Hoffman, 1967):

Plant kingdom:

Viruses, bacteria, algae, and fungi.

Animal kingdom:

Protozoa: amoebae, flagellates, ciliates, sporozoans.

Platyhelminthes: trematodes, cestodes.

Nematoda

Acanthocephala

Hirudinia

Crustacea

Molluscs

Lampreys

Disease and parasites become established in fish in two ways: by inoculation, or by population growth. Inoculation is typical of bacteria and protozoa which multiply rapidly within the fish. Parasites of the population growth type attach or penetrate the fish one at a time, and accumulate over some time span. Damage is done in many ways. There can be smothering of fish gills, as parasites occupy respiratory space and cause additional mucous to form. Damage is done to cells and tissues, as parasites migrate through a fish. Blood vessels can become blocked. There is nutritional "robbing" of the fish by parasites in the gut. Some parasites are found in the body cavity where they cause adhesions in or on the viscera and reproductive organs. There is also some evidence of parasites producing toxins.

Fish can react to some parasites with an immunity response. Light infections with the protozoan Ichthyophthirius (Ich) can cause antibody response. Later infections of Ich are not as much of a problem. Immunity in such cases may be partial, and not total.

Treatment of fish parasites under cultural conditions has been a common practice for many years, albeit not always successfully. Treatments most commonly have been for external parasites. Chemical treatments involve dipping of fish in chemical, chemical flushes of short duration, continuous chemical flows for hours or days, or one-shot applications as in pond culture. Other treatment measures involve manipulation of water temperature and water flow.

There are six requirements that chemicals ideally should meet:

1. Federal Food and Drug Administration clearance.
2. Effective on parasite.
3. Non-toxic to fish.
4. Price not too expensive.
5. Available commercially.
6. Decompose or inactivate rapidly.

Many chemicals such as formalin, salt, malachite green, copper sulfate and potassium permanganate, have been in use for decades as controls for ectoparasites. FDA approval has not been given for these chemicals, except for copper sulfate. The reader is referred to the reference list for sources of treatments, application methods, and concentrations.

The best "treatment" for fish parasites is, after all, to avoid problems in the first place, as much as possible, by obtaining disease-free stock, and having disease-free water for the cultural establishment.

Bacteriology

Disease, per se, is not an entity or an end in itself. Disease is the end result of an interaction between a noxious stimulus and a biological system, and to understand disease is to understand all aspects of the biology of the species. A susceptible host, a virulent pathogen (parasite, bacterium,

virus) and an environmental stress--all are needed to produce disease. We cannot always blame the environment for creating a disease condition. A swollen gill condition may be due not only to myxobacteria, but also to a lack of pantothenic acid, or to poor water quality. At water temperatures other than optimum, enzymatic action is reduced or stopped. The epithelium of the intestine tends to slough, which allows bacteria to migrate freely between the lumen and other tissue layers of the intestine, with muscle tone decreasing.

The interaction of the three variables--host, pathogen, and the environment--are in a constant state of flux, capable of changing, in step with any variation in any of its components. We should speak in terms of epizootics, not of epidemics. Epidemic applies to people; "zootic" to animals, including fish. In considering epizootics, there is a threshold density factor. As the threshold density or number of susceptible individuals decreases, there is a corresponding decrease in the number of mortalities. This is because there are fewer individuals present and those which are left have at least some degree of immune response. The more severe the epizootic, the longer between epizootic occurrences. Bacteria are much less virulent at the beginning and at the end of an epizootic. Examination of a fish mortality curve (number of deaths versus time in days) is a presumptive diagnostic method. A very rapid increase in mortality over a short time span, followed by an equally rapid decrease in mortality, is suggestive of a chemical agent or an oxygen depletion. A slowly rising mortality rate, followed by slow decline, suggests a pathogen. A low, chronic mortality can indicate a chemical agent which borders on toxicity.

There are four types of infections. The first is acute; the fish die or recover, usually rapidly. The second, or latent infection, involves bacteria as present but not producing a disease. Bacteria will become acute, however, if a mutation occurs or if environmental conditions change. The third type is inapparent. This is an important form of infection encountered in the disease inspection and classification program. We may not find the bacterium, although we can demonstrate exposure at some time

or other through serological means. Stressing the fish is about the only means of demonstrating the presence of a pathogen. The fourth type is mixed infection. Both a virus and a bacterium may be present in the fish, and both must be tested for by diagnostic procedures.

Disease production may be in any one of three forms. There can be interference with the host, such as physical blockage of gill surfaces which in turn interferes with respiration and with elimination of nitrogenous wastes. Exotoxins are a second form. Gram-positive cells produce exotoxins as secretions. Kidney disease and acid-fast bacteria are two fish pathogens which produce exotoxins. Endotoxins are from gram-negative cells; the toxin is in the cell wall and is released on lysis of the cell. An endotoxin is not very toxic, when compared to an exotoxin.

Sanitation and the prevention of introduction of diseased fish are the most important factors in disease control.

Immunization methods have been and are under research at present. Trout injected with killed bacteria will develop antibodies; the exact response level depends upon the water temperature. Immunization by injection is not practical under culture conditions. Oral immunization has worked under laboratory conditions but not in field work. Selective breeding is not commonly done although, some eastern states have developed furunculosis-resistant brook trout.

Drug therapy has long been employed to control bacterial diseases of fish. The administration of a drug can cause an additional fish kill. This happens when bacteria, killed by the first treatment, release endotoxins which in turn cause fish mortalities. Blood concentrations of drugs are difficult to maintain in fish. Fluctuations in the concentration occur even though the same dosage is continuously administered. Bacteria are generally under control in spite of such fluctuations. Lowering the dosage during the treatment period leads to the risk that bacteria might survive and become resistant to the drug being used.

Methods of fish examination for bacteria and identification of bacteria are summarized in Bullock (1971). The utilization of proper autopsy techniques on moribund but not rotten fish are basic to arriving

at a diagnosis. The signs of bacterial fish diseases often overlap and are confusing. The determination of the exact fish pathogenic bacteria is necessary for disease diagnosis and effective control measures.

Bacteria range in size from a maximum of 1.5μ wide x 10-15μ long down to 0.25 μ x 0.5μ. They are the smallest organisms considered capable of independent existence. As to size (weight), it takes about 500 million cells to weigh one gram. Classified as to shape, spherical bacteria are the cocci; a grape-like cluster is a staphylococus, the configuration produced by division of cells in various planes; a bacillus is a rod-shaped bacteria and these are variable in shape and size; and spirilla are curved forms of bacilli in which the spiral may be loose or tight.

Bacteria have a cell wall which gives shape and rigidity to the cell, and physical protection of the cytoplasm. A bacterial capsule or slime layer protects the cell against drying. The capsule can be both discarded and replaced by the cell. Flagella on bacteria are slender, whip-like processes. The particular arrangement of flagella around the cell is diagnostic for species identification.

Bacterial "growth" is a poor word. What is meant is multiplication of bacteria in numbers, not strictly an increase in size as with mammalian growth. There are four phases of multiplication of bacteria:

1. Cell enlargement or lag phase.
2. Logarithmic or log phase. Cells rapidly dividing.
3. Stationary phase. Death of cells equals multiplication.
4. Logarithmic death phase. Death of cells is greater than multiplication.

Bacteria have minimum, optimum, and maximum temperatures for replication. Cold-replicating bacteria prefer 15-20 C (Psychrophiles). Mesophiles normally grow at 30 C, but will replicate at 37 C. Thermophiles grow at over 37 C; however, none of these are fish pathogens.

Bacteria may be destroyed by either physical or chemical methods. Physical includes heat (autoclave at 121 C, 15 lb pressure, 15 minutes; oven heat, 170 C, 3-4 hours), freezing, drying, radiation (rays at lower

end of spectrum; ultra violet, etc.). Chemical methods are usually by disinfectant which will kill vegetative stages. Bacteriostatic chemicals inhibit replication of bacteria while bacteriocidal chemicals kill bacteria directly. Depending upon the dilution used, a chemical may be either bacteriostatic or bacteriocidal. The types of disinfectants range from soaps, detergents, acid-alkalies, alcohols, heavy metals and oxidizing agents to antibiotics. Antibiotics are products of micro-organisms such as molds and bacteria, and they act on bacteria by inhibiting cell-wall synthesis.

Following are the more common bacterial diseases of fish:

Disease	Etiological Agent
Bacterial hemorrhagic septicemia	Aeromonas liquefaciens
Furunculosis	Aeromonas salmonicida
Columnaris	Chondrococcus columnaris
Coldwater disease	Cytophaga psychrophila
Bacterial gill disease	Myxobacteria
Kidney disease	Corynebacterium
Redmouth	Enteric bacteria

Immunology

Immunology is the study of immunity in animals, of how the body responds to infectious agents which can be any foreign body, living and non-living. Immunity as such, is a specific form of resistance that depends on the presence of immunocytes or antibodies. It varies in degree from one individual to another and is directed against specific diseases. Acquired immunity occurs when specific barriers are invoked by foreign bodies entering the host. Natural immunity can be actively acquired when bacteria enter the host and evoke a response. Passively acquired immunity occurs when antibodies are passed in reproduction (female to offspring). Artificial immunity can be active as when animals are injected with a denatured form of the bacteria, which then evokes an antibody response

without the disease. Passive artificial immunity also is accomplished when bacteria are first injected into one host, then globulin fraction is withdrawn after response is evoked, and finally the globulin is injected into a second host which thereby acquires the immunity.

The cells of immunity are of two types--non-granular, and granular. The non-granular series is composed of cells with a large nucleus and clear cytoplasm. Cells of this series are the lymphocytes (antibody production), monocytes (phagocytic), and plasma cells (antibody production). Plasma cells are found in the spleen and lymph nodes following antigenic stimulation. The granular series is characterized by a kidney-shaped or lobed nucleus, and by granules in the cytoplasm. These are the neutrophiles, eosinophiles, and basophiles, which function in inflammation and wound repair.

Antigen intake by cells is by pinocytosis or phagocytosis. Pinocytosis is "cell drinking," whereby the cell takes in soluble antigens. In phagocytosis, the cell takes in particulate matter and forms phagosomes. Lysosomes in the cell enzymatically destroy the invading microorganism. All cells, except red blood cells, contain lysosomes.

Antibody formation is initiated when a foreign body enters an animal, or when non-self material is introduced. The body produces specific antibody as a result of this introduction. The phagocytes pick up the antigen, "break" it down, and then pass on to plasma cells and lymphocytes the stimulus to produce antibody. The "break" down is of large polypeptide chains to their component amino acids (or large polysaccharides to small sugars). What is passed on is a portion large enough for "memory" stimulation of antibodies. Antibodies are synthesized in the ribosomes of cells. Light and heavy chains are produced independently. The cell keeps a pool of light chains, and makes heavy chains after the antigen enters the cell. The chains are hooked together and secreted out of the cell.

There are a number of factors which affect antibody metabolism. The age of the organism must be considered. Sexually mature animals are immunologically mature, while neither young animals nor older ones are

capable of competent immune response. There is, in essence, an age period of immunological competence. Animal metabolism is a factor. Faster metabolic rates create antibody more rapidly, but also degrade them more rapidly. The type of antibody also affects the response. Once antibodies are formed, they are removed; the larger ones are removed more quickly than smaller ones. In fish, temperature has a dominant role, as at lower water temperatures the immune response is slower to build up. If temperatures are very low, the response will be suppressed or will build up very slowly.

Teleost fishes have specific antibody sites in the spleen, anterior kidney and the thymus. Oral immunization appears to work, although it is difficult to immunize fish. The problem is that immunization would have to be constant, as one or two injections cannot be given and have a lasting antibody titer. Fish have mucoid antibody response and they, like mammals, can discriminate against soluble antigen.

Virology

Virology is based upon cell and tissue culture (TC), and a background should be discussed on TC work before discussing viruses and their detection. First of all, tissue culture is a group of techniques in which cells or tissues from plants or animals are removed and kept alive and growing in vitro. Biologically, this culture method is a new art. Tissue culture is a uniform methodology for making viruses "visible" in culture. By watching for changes in the cultures, we can see the effect of the virus on the cell. The use of a living host animal itself is not a constant, uniform method of virus examination. Theuse of cells from a host was not a usable method until cell cultures were developed for constant, reproducible results. Cell lines are manipulative by temperature, pH, and media composition for optimum growth under management conditions. The cells are "naked" and have no innate protection or immune response. Therefore, all protection must come from the culture conditions, the container and the medium. Tissue culture is easy when a careful, methodical approach is employed by the virologist.

The first fish cell line was established in 1960 with the rainbow trout gonad or RTG-2 line. The cell lines developed to date employ either fibroblast-like or epithelial-like cells. Fibroblast cells are spindle-shaped and ellongate, tending to look quite irregular. Epithelial cells are polygonal and closely adherent. Cell growth is by mitotic division (prophase, metaphase, anaphase, and telophase). The RTG-2 cell line will double its population in 2.5 days. Other cell lines will vary as to time. The lower the temperature, the longer the doubling time. At 5 C, RTG-2 will take 13.2 days to double.

Cell requirements in tissue culture are critical. The respiration needs are those of the atmosphere of the culture system, or of the room when an open dish is used. Cells will grow in a pH range of 6.8 to 8.6; the optimum range is 7.3 to 8.0 for cell growth and replication of viruses. Maintenance of pH is by a buffering system, using sodium bicarbonate or organic buffers. Normal incubation temperature range for cells is 15-30 C. The nutrient media used is Minimum Essential Medium (MEM) as developed by Dr. Eagle. It consists of 13 amino acids, 8 vitamins, glucose and a basal salt solution. The MEM is supplemented with Fetal Bovine Serum (FBS) which stimulates cell growth in vitro. There are no antibodies in the serum; therefore, no antibodies against fish viruses. Contamination by bacteria is an ever present problem in TC work. The addition of antibacterial compounds like streptomycin and penicillin to the MEM provides bacterial control, while nystatin (mycostatin) works against yeast and fungus. We must remember that the glassware or plasticware, and the medium are the only protection that the cells have.

The Cytopathic Effects (CPE) on tissue culture are the visible effects of the virus on cells. This is an in vitro test for virus. Viruses have no metabolism of their own, and are therefore dependent on the host cell for survival. Some cell lines are known to replicate virus without CPE. In critical work, cultures showing no CPE at the conclusion of the test are superinfected with virus. Cell line susceptibility should also be confirmed by using known virus inoculants. The appearance of CPE due to virus is presumptive diagnosis, and definitive diagnosis requires further testing.

Definitive tests may include part or all of the following:

1. Lipid sensitivity. These tests are based on the fact that there are essential lipids in the virus. If these lipids are dissolved, the virus loses its infectivity.
2. Heat sensitivity. Heating of a suspect virus for 30 minutes at 50 C. The loss of one or more logs of infectivity is diagnostic that the virus is heat labile.
3. pH. The pH of the virus medium is lowered to 3.0 for 30 minutes. If acidity reduces infectivity by one or more logs, the virus is pH labile.
4. Differential filtration. The size of viruses can be estimated from data obtained by filtering through various filter sizes.
5. Histology. There are specific changes, useful in diagnosis, which can be seen in prepared fish tissues.

Rivers' postulates (Wolf, 1970) must be fulfilled to establish firmly the etiological role of a viral agent as the cause of a fish mortality.

1. Virus must be found in all clinical cases of the disease in question.
2. Agent must be isolated and grown in another system; cell or tissue culture is preferred, but an alternate host may be used.
3. Infect test animals and reproduce in them the essential clinical features of the original disease.
4. After having reproduced the disease, the virus must be re-isolated and identified as the same as that used to start the experimental infection.

A virus (virion) is a single, submicroscopic, intracellular, obligate parasite. During its replication it goes through an eclipse phase when it cannot be "seen." This phenomenon is a period when the virus' nucleic acid (RNA or DNA) is beginning to serve as a map for replication

by the host cell. Only the nucleic acid of the virion is present, as the virion has been uncoated. This nucleic acid is either RNA or DNA, not both, and the virion's RNA or DNA is different from that of the host cell.

Viruses range in size from 10 nm (nanometers) to 300 nm. There is no variability in size of viruses. They have no regulated membrane although at times they have envelopes. Viruses have no metabolism of their own, and no movement, nor do they respond to a stimulus. They have a protein structural coating and contain various enzymes within their structure.

Viruses are replicated at great rates. A single virion may replicate to thousands or more virions. The generation time is short, from minutes for virions in bacteria to a few hours for those in fishes. The generation time for some viruses may take days as in fish lymphocystis. Viruses depend totally on living cells but can survive for years in a dry state, maintaining infectivity.

Viruses are subject to damage by biophysical means, just like other organisms. Oxidizing agents (chlorine, etc.) attack viruses as do organic solvents (chloroform, ether). One exception to organic solvent lability is IPN virus, which has no lipid content in the outer envelope or coat. All other viruses have envelopes with lipid content and are sensitive to organic (lipid) solvents. No fish virus is known to cause hemaglutination, the clumping of red blood cells.

Most viral infections fail to produce disease, but we are confronted with exceptions where disease or a clinical entity is produced. Agents can thus be infectious, taken in by cells and replicated, without causing disease. Antimicrobials attack and interfere with bacteria, but not with the host. The threshold of viral attack by antimicrobials is too near that of the host when the virus is in the host cell. The future of finding an anti-viral agent is not good and we must look elsewhere for control measures. The problem is one of damage to the virus so as to render it non-infective while not damaging the host cell.

Fish viruses may enter through the gut or by the mouth. Entrance via the gills is also known. Bluegill sunfish have been experimentally infected by spraying lymphocystis virus on the gills. Abrasions of the skin are also an entry method for lymphocystis. Vertical transmission--i.e., parent to progeny--is suspected for IPN, IHN and CCVD (see below). There are no known vectors of fish viruses, but neither has there been much work in this area. There are cases where the virus appears to be in balance with its host fish. The application of genetic selection of fish as resistant to virus may be applicable. Dr. Wolf's opinion is that, in Europe, Egtved virus is in balance with the brown trout. The introduced rainbow trout constituted a new host, readily susceptible to the disease. There can be two viruses in a single fish. Egtved virus has been produced in fish which already had IPN. In tissue culture, the same cell can produce both viruses. Thus, viral isolation can involve more than one virus type. No problem viruses have as yet been isolated in dual in epizootics.

The following list of viruses with their diseases have been identified from fish:

Salmonid
Infectious hematopoietic necrosis, IHN
Infectious Pancreatic Necrosis, IPN
Viral Hemorrhagic Septicemia (VHS), Egtved virus

Non-salmonid virus diseases
Channel Catfish Virus Disease, CCVD
Lymphocystis disease
Epitheleocystis

Histopathology

Disease development may be due to degenerative changes or to necrosis. Degenerative changes are generally reversible. Such changes as cloudy swelling or vascular degeneration, fatty metamorphosis, hyaline degeneration, calcification and mucoid degeneration are known. Frank

necrosis is irreversible. Necrosis is cell death, as when an agent is not removed or overwhelms the cell. There are various effects on the cell nucleus which are diagnostic. The types of necrosis include coagulative, liquefactive, caseation, fibrinoid, and fat necrosis. Postmortem necrosis is autolysis or tissue breakdown after the death of the organism.

Disease is any disturbance of the structure or function of any part of the body. Pathogenesis is the sequence of events in the development of the disease. Many types of diseases are recognized:

1. Inherited or familial. Chromosomal disease is passed from parent to offspring.
2. Congenital. During embryogenesis, a defect is caused as development goes awry.
3. Toxic. Ingestion or exposure to a toxic chemical.
4. Infectious. Caused by a living organism.
5. Traumatic. Result of direct physical injury.
6. Degenerative. Aging, process of growing older.
7. Allergic diseases. Result of foreign antigen in body of host.
8. Auto-immune. Fish builds antibody against self for some reason.
9. Neoplastic. Tumor forming diseases, esocid lymphosarcoma.
10. Nutritional. Due to deficiency, excess or failure to absorb nutrients.
11. Metabolic. Derangement in the physiologic process, as in thyroid goiter.
12. Molecular. Cell produces abnormal products.
13. Iatrogenic. Doctor induced.

Vascular disturbances are encountered in histopathology. The circulatory system is constantly in motion, usually under tight control which can break down under some conditions. Types of conditions are plethora, generalized increase in blood volume; hyperemia, abnormally increased amount of blood in smaller vessels; edema, presence of excessive amount of fluid outside of vascular spaces; hemorrhage,

escape of blood from vessel or heart due to mechanical defect in the wall; and aneurism, localized dilation of the lumen of a blood vessel.

Growth disturbances are commonly seen in fish and they are grouped as follows:

1. Physiologic. The degree of disturbance is proportional to the degree of stress. Is reversible.
2. Congenital. Absence of structure.
3. Hypoplasia. Structure fails to grow up to normal size.
4. Atrophy. Decrease in size and function due to disuse, destruction of nerves or to pressure.
5. Hypertrophy. Increase in size of individual cells.
6. Hyperplasia. Increase in the number of cells.
7. Metaplasia. Replacement of one type of normal cell or tissue with a different normal adult tissue.
8. Dysplasia. Dis-organization in layering or patterning of tissues.
9. Anaplasia. Increase in mitotic figures.
10. Neoplasia. Uncontrolled growth of new tissues, benign or malignant.

The effects of a tumor vary. It can obstruct a vessel. There can be perforation as of the gut. Secretions can be stimulated. An embolus with cells in circulation can occur. Compression of adjacent organs or collapsible structures may occur. Finally, there can be loss of secretion from organs.

Conclusion

The Advanced Course in Fish Diseases is very valuable practical training. The lecture information is well balanced with laboratory experiences. As a result of this specialized training, trainees are more capable of understanding and diagnosing pathogenic problems in fishes.

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