Persistent Proliferative Gill Disease Problems and Taxonomic Chaos

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Proliferative gill disease (PGD) affecting farm raised channel catfish is a significant problem of production and economic impact, and of taxonomic classification. The farm raised catfish industry has experienced a significant increase in the incidence of this disease within the past several years and in Mississippi alone there has been over a 200% increase in reported cases over the past 3 years. Factors associated with the disease are poorly understood. The cause of PGD is controversial as are treatment and prevention methods. We have been conducting PGD research over the past 4 years and summarize our results in this report.

PGD is associated with severe branchial inflammation, epithelial cell hyperplasia and lamellar fusion (Fig. 1). Previous reports (e.g. Bowser and Conroy 1985, J. Wildlife Dis.; Kent et al. 1986, California Fish and Game) have well characterized the initial changes. We (MacMillan et al. in press. J. Aquatic Animal Health) have extended the observation period and have documented subsequent chondroplasia and branchial recovery. During the chronic inflammatory period, there is a significant neutrophilia with neutrophil percentage in circulating blood over 35% (normal 10%). Concomitantly there is a significant myeloid hyperplasia in hematopoietic tissue with almost complete loss of erythroid cell elements. Cortisol becomes elevated (3-6 times normal) as does serum aspartate aminotransferase (10 times normal). Blood pH, pO₂, HCO₃⁻, TCO₂₂, and O₂ saturation significantly decline compared to normal catfish controls. These clinical data indicate significant disturbances in acid-base balance, tissue oxygenation and cellular damage. Cause of death is attributed to asphyxiation. During recovery, PCV may become very low (5-15%) compared to normal levels (25-35%). The low PCV is attributed to hemorrhage associated with branchial fragility and necrosis, and impaired erythropoiesis.

Catfish mortality observed in individual ponds is variable. Mortality rates less than 1% up to 50% have been documented. There does not seem to be any correlation between the age of the fish and the mortality rate. There is great variation in the number of
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PGD parasites infecting gill tissues and individual gill filaments. Fish populations with few parasites seem to exhibit less mortality. The presence of external protozoan parasites particularly those that can also induce significant tissue destruction and inflammation (e.g. Ichthyobodo and Amphileptus) significantly increase mortality. Poor water quality also appears correlated with high mortality. The cause of PGD has not been clearly established. We have demonstrated a here-to-for unknown parasite capable of causing PGD in specific pathogen free (SPF) fish under laboratory conditions. A morphologically similar parasite is also observed in PGD from commercial catfish ponds. There are significant similarities of this parasite with the early developmental stages of a myxosporan parasite with formation of a pansporoblast. Ultrastructurally, a mother cell is detected with secondary cells. It is not clear however if the mother cell is of host or parasitic origin. Histologically the parasite has morphologic similarities to various coccidians. Further suggestion of its myxosporan affiliation is evident from the formation of so-called secondary cells which may become released into the gills and subsequently parasitize other organs (liver, kidney, spleen, and brain). However, histologically these organisms also resemble a coccidian parasite and with little inflammatory response. We have been unable to confirm the report of Hedrick et al. (1989, FHS Newsletter) who observed Sphaerospora-like spores in catfish with PGD. We have examined PGD affected fish for 3 months post-infection and do not find increased levels of Henneguya which has also been suspected as a cause of PGD (Bowser and Conroy 1985; Kent et al. 1987). Until specific diagnostic tests can be developed, the taxonomic classification of the PGD organism must remain obscure. Successful treatment for PGD seems correlated with various supportive measures. Maintenance of high dissolved oxygen, low un-ionized ammonia and elimination of external protozoan opportunists most frequently provides for lower mortality. Reports suggesting pumping across levees or potassium permanganate treatment have been successful remain controversial and must await further testing to establish their efficacy. There is currently no known therapeutant which can be successfully administered. Fish with PGD are frequently anorectic and thus chemotherapeutic delivery in feed does not seem feasible.

**Water-borne Transmission of Salmonid Gill-infecting Dermocystidium in the Laboratory**

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Dennocystidium is a gill parasite of Pacific salmonids in fresh water and has been associated with mortality in returning adult chinook salmon (Oncorhynchus tshawytscha) in the Columbia River system since the mid 1960's (Pauley, 1967, J. Fish. Res. Bd. Can. 24: 843-848). Although this pathogen has been observed in Oregon for a number of years, its association with mortality of returning adult chinook salmon was not noted until 1987. In 1988, large numbers of heavily infected chinook died in the Trask, Nestucca, Siletz and Applegate Rivers. Large losses of Dennocystidium-infected chinook smolts being reared at the Trask Hatchery also occurred in September, 1988.

Observations made in Oregon during the fall of 1989 indicated that Dennocystidium infections were generally less severe and not associated with mortality of either adult or juvenile salmon. In an effort to develop an understanding of the conditions leading to epizootics, a study was conducted to determine the mode of Dennocystidium infection.

Gills containing moderate numbers of Dennocystidium cysts were collected from spawning adult chinook salmon at the ODFW Trask Hatchery in November, 1989. The gill tissue was held on ice for several hours and brought to the laboratory where cysts of the pathogen were removed and washed repeatedly in water until little extraneous material remained. The resulting accumulation of cysts was held in water at 4°C in the absence of antibiotics.

Over a period of 14 days, water from this culture was found to contain a variety of flagellate and ciliate protozoans. It was also noted that some spores within Dennocystidium cysts lost their characteristic eccentric vacuole and became granular in appearance. At this time, increasing numbers of small (2-3 um) biflagellate organisms began to accumulate in the culture. To determine whether or not these organisms were Dennocystidium zoospores, an experiment was set up to expose pink salmon (O. gorbuscha) fry to possible infection. Twenty-five fry were placed in each of three aquaria and water containing large numbers of the putative zoospores was added to two of the three, the third serving as an unexposed control. The fish were observed for 15 days while the temperature in the static aquaria varied between 12 and 15 °C. The fish were not fed during the observation period. After 11 days, a single pink salmon exhibited darkening and hyperventilation. Upon examination under a dissecting microscope, the gills were found to contain numerous small Dennocystidium cysts. During the next four days, all pink salmon fry that had been exposed died with massive Dennocystidium infections in the gills. No control fish died during the 15 day observation period and all were negative for Dennocystidium when examined several days later.

The zoospores continued to be active in the water containing Dennocystidium cysts for 30 days after they were first observed. Although the life span of an individual zoospore was not precisely determined, the presence of inactive individuals throughout the observation period suggested a span of several days. The extended period of zoospore activity observed (30 days) is likely due to varying stages of maturity of spores present in cysts on adult salmon gills. Preliminary observations of zoospores in the electron microscope have revealed no evidence of an apical complex of organelles that would indicate a relationship to Perkinsus marinus (formerly Dennocystidium), a well known pathogen of oysters.

This study is the result of research sponsored by the Oregon Department of Fish and Wildlife and U.S. Fish and Wildlife Service under PL89-304, the Anadromous Fish Act, project AFS-78-1.

Visceral and Systemic Infection With A Dennocystidium-Like Organism In Post Seawater Entry Smolts and Adults of Atlantic Salmon (Salmo salar)

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Chronic mortality in post seawater entry smolts of Atlantic Salmon (Salmo salar) reared in the Bay of Fundy, was associated with visceral and systemic infection with a Dennocystidium-like organism. Clinically, the fish were anorexic, lethargic, and black in color. The disease was evident in late summer and fall. Histologically, parasites were in macrophage-like cells, in various tissues including
cardiac muscle, liver, kidney, spleen, brain, and gills. Severe inflammation, including granuloma formation, was evident in the above tissues. Ultrastructurally, various parasitic stages, 3-7 μm in diameter, were present in melanin-containing macrophages. Recently in mid-winter, market weight fish have been severely affected by the parasite; microscopically visible granulomas were found in kidneys and livers of fish at processing. Histologically and ultrastructurally, granulomatous lesions and parasites were more advanced than in smolts. The short-term and long-term impacts of *Dermocystidium* spp. in farmed Atlantic salmon in Atlantic Canada are under investigation.

Myxobolus spp. in Two Species of Fish from the Tennessee-Tombigbee Waterway

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Several species of fish from the Tennessee-Tombigbee Waterway were examined to determine the prevalence of diseases. *Myxobolus* spp. have been observed in the gill cartilage of smallmouth buffalo (*Ictiobus bubalus*) and in the branchial blood vessels of largemouth bass (*Micropterus salmoides*). Extensive tissue reaction including severe inflammation with eosinophils and macrophages, chondroplasia, lytic changes in the cartilage, and thickened perichondrium were present in the gills of smallmouth buffalo. No tissue reaction was observed in largemouth bass. The significance of this parasite in the Tennessee-Tombigbee Waterway fishes is not known at this time.

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An Unusual Isolate of Vibrio anguillarum from British Columbia

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The purpose of this brief note is to alert fish health workers to the existence of a form of *Vibrio anguillarum* (Va) that, without adequate testing, could easily be mistaken for *Aeromonas salmonicida* (As), causative agent of furunculosis. The confusion might arise because of the ability of this particular Va isolate (RI:pigmented) to produce a brown diffusing pigment on tryptic soy agar (TSA) (or on NaCl-supplemented TSA).

This medium is in frequent use in the fish health field for primary isolation purposes and the Va pigment mentioned above is indistinguishable from that produced on TSA by isolates of "typical" As. In all other respects, however, the Va isolate is archetypical of Va. It is a short (1.0 X 3.0 μm), slightly curved, actively motile, non-sporeforming, Gram-negative rod; it is oxidase-positive, sensitive to novobiocin and compound 0/129, fermentative but anaerogenic with glucose, and reacts strongly in slide agglutination and immunodiffusion tests with rabbit anti-Va serum but not with rabbit anti-*V. ordalii* or anti-As serum. In immunodiffusion tests with rabbit anti-Va serum, it gives a precipitin line of identity with known isolates of Va. In API 20E tests, the isolate also proved identical to known Va isolates. It proved positive in 11 tests (ONPG, ADH, CIT, IND, GEL, GLU, MAN, SOH, SAC, AMY, ARA, and OXI) and negative in 9 tests (LDC, ODC, H2S, URE, TDA, VP, INO, RHA, and MEL). The pigment-producing Va isolate was obtained from a seawater-farmed chinook salmon in B.C. The chinook was one of a number of specimens that had died of a disease that was clinically indistinguishable from vibriosis. When the isolate was used to challenge 22 g coho salmon being held in fresh water at 13 °C (1 X 10⁶ Va cells per fish by intraperitoneal injection), 4 of 6 challenged fish died with clinical signs typical of vibriosis within 8 days. In all cases, the injected isolate was recovered in pure culture from the dead fish, its pigment producing ability intact. None of the mock-challenged (control) fish died during the 14-day experiment. Readers wishing to obtain a culture of the Va isolate may do so by writing to the authors.

An Epizootic of Infectious Hematopoietic Necrosis in Yearling Chinook Salmon

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In November 1987, mortality increased in yearling spring chinook salmon (*Oncorhynchus tshawytscha*) being reared at Clackamas River Hatchery in Northwestern Oregon. Moribund fish had dark body coloration, exophthalmos, lethargy, and an acute anemia. Internally these fish had
hemorrhaged air bladders and brain tissue, pale livers, and petechial hemorrhaging of the body musculature. Some moribund fish had acute fungal infections on the caudal region of their bodies. Bacteriological tests of fish showed a low prevalence of furunculosis and bacterial cold-water disease. Initial virological exams of kidney and spleen tissues were negative but when brain tissue was tested, infectious hematopoietic necrosis virus (IHNV) was found in high concentrations. Further examinations of brain tissue revealed IHNV in essentially all samples taken from fish with exophthalmia and/or fungused tails, and IHNV was present in a few samples from apparently healthy fish. The virus was subsequently found in juvenile winter steelhead (O. mykiss) and another stock of spring chinook salmon being reared at the hatchery. However, mortality in these groups remained very low and never increased. Chronic mortality continued in the infected group until the time of release. All fish being reared at the hatchery were approximately one year in age and ranged in size from 8-11 lb (41 - 57 g). The virus was also detected in wild coho salmon (O. kisutch) adults from the river and held at the hatchery until maturation.

Spawning chinook salmon present in the hatchery's water supply in September and October were suspected to be the source of IHNV. The virus was isolated in post-spawned winter and summer steelhead captured in the river above the hatchery. Record low river flow during the period when the epizootic was detected was probably an added factor. Virus was detected in the effluent water from the group undergoing the epizootic and this water was treated with chlorine to minimize the amount of virus discharged into the river. Total mortality in the infected group was approximately 19% (85,000 fish).

The other groups of fish suffered less than 1% loss. The stock of spring chinook not seriously affected was brought to the hatchery after the peak spawning period above the hatchery. This group was also being reared at a lower density as were the winter steelhead (3.3 - 5.7 versus 9.4 lb/gpm) which could account for why these fish were not severely affected. Furthermore, the detection of IHNV in all groups of fish being reared or held at the hatchery provides further evidence for the source of virus being infected fish in water supply. This IHNV epizootic had characteristics not observed in others that have occurred in the Columbia River basin in which primarily small steelhead (1-2 g) have had cumulative mortalities approaching 90% and routine detection of IHNV has been accomplished by examining kidney and spleen tissue, not brain tissue. Serological analysis of the new isolate showed it to be similar to other strains of IHNV isolated from infected fish in the lower Columbia River. Virulence studies indicated that rainbow fry were much more susceptible to this isolate than were spring chinook salmon fry, also consistent with the results of previous studies. Furthermore, in artificially induced infections, tropism for only nervous tissue was not detected. Results from these studies suggested that this was not a unique strain of IHNV but that the virus had manifested itself differentially in these larger and older chinook salmon.

No virus has subsequently been detected in any adults which have returned to the hatchery. In 1989, IHNV was detected in apparently healthy yearling spring chinook at Clackamas Hatchery but no other epizootics have occurred. Current management strategies consist of releasing chinook smolts earlier, bringing juvenile chinook to Clackamas Hatchery after the number of adults in the water supply has decreased and keeping fish rearing densities lower.

An Intranuclear Microsporidium Infection in Chinook Salmon (Oncorhynchus tshawytscha) in California

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A low grade chronic mortality, approximately 0.9 to 1.5% each month, was observed among freshwater yearling chinook salmon (Oncorhynchus tshawytscha) during August, 1989 at Darrah Springs Hatchery, California. Affected fish were lethargic and easily captured for examination. Gross pathological signs were suggestive of proliferative kidney disease (PKD), caused by the PKX myxosporean, found at several other California State hatcheries. These signs consisted of pale gills and enlargement of the spleen and kidney. Exophthalmos and ascites were observed in some fish. PKX parasites however, were not observed by phase microscopy. Kidney imprints prepared and stained by the Leishman-Giemska method were examined by light microscopy. Kidney imprints stained by the Leishman-Giemsa method were examined by light microscopy. Kidney imprints prepared and stained by the Leishman-Giemska method were examined by light microscopy. Kidney imprints prepared and stained by the Leishman-Giemska method were examined by light microscopy. Kidney imprints prepared and stained by the Leishman-Giemska method were examined by light microscopy.

One to four prespore stages were observed within nuclei of infected cells. These stages stained poorly, being light blue to clear with Lieszman-Giemska, and could be mistaken for...
nucleoli or endosomes. Spores commonly existed in pairs or tetrads, however, any number from one to eight per nucleus were observed.

Sections from affected tissues stained with hematoxylin and cosin revealed a proliferation of lymphocytic cells in kidney, spleen, gut, liver, eye and brain. Parasites could be observed in many of these cells as lightly eosinophilic or non-staining spherical intranuclear bodies.

Blood smears, hematocrits, leukocrits and plasma protein levels were measured for 39 affected fish and 20 healthy fish from the same stock but held at a different facility. Hematocrits averaged 17.0 ± 8.5, leukocrits 5.04 ± 4.20 and plasma protein 2.03 ± 1.14 mg/dl. These values deviated significantly from those of healthy chinook salmon of the same stock held at a separate facility, having average hematocrits of 38.4 ± 1.7, leukocrits of 1.7 ± 0.77 and plasma proteins of 4.67 ± 0.46 mg/dl (n=20). A lymphoblastosis was observed in blood films, with lymphoblasts comprising from 9.3 to 58.7% (mean 32.6 ± 15.5%, n=20) of total blood cells. Both prespore stages and spores existed in blood films, either in smudge cells or in the nucleus of lymphoblasts. Numerous mitotic figures were observed among lymphoblasts in peripheral blood.

Due to the unknown pathogenicity and epizootiology of this parasite, and its first occurrence in California, all chinook salmon, approximately 93,000, at Darrah Springs Hatchery were destroyed in December, 1989. The question of host species susceptibility and pathogenicity is under investigation. Attempts to transmit the parasite from chinook salmon to rainbow trout, chinook salmon and coho salmon by injection, cohabitation and feeding infected tissues is being attempted.

In January of 1990 this microsporidian parasite was detected in fingerling rainbow trout (O. mykiss) at Hot Creek Hatchery, California. Histological slides dating back to 1968 indicate the presence of the parasite in chinook salmon, rainbow trout, brown trout (Salmo trutta), and golden trout (O. aquabonita) at Hot Creek Hatchery, California.

During a six month stay sponsored by the National Academy of Science in the USSR, Heckmann cooperated with Zhatakanbayeva on several fish parasite projects, one of which is reported below.

Trematodes of fish are potentially dangerous for fish farms in the USSR where they can cause high mortality, especially in the fingerling fish. However, therapeutics for such fish have been poorly developed in Russia. Our aim was to study the efficacy of Praziquantel injectable (56.8 mg/ml active ingredient) used as a water bath against two trematodes of fish. The two trematodes were Diplostomum psilium and Apatemon gracilis, commonly found in Nemachilus dorsalis, a native fish of Kazakhstan which acts as a parasite reservoir for commercially important fish. All of the host, N. dorsalis, checked contained metacercaria of the two trematodes and were used for the drug efficacy trials. Diplostomum psilium infects the posterior regions of the eye, both inside and outside the eye socket and A. gracilis is found in the brain, heart, liver, gonads, swim bladder, and intestine. Four series of experiments were completed during the six weeks the second author was in Kazakhstan. Twenty fish were used in each experiment (average total length 4.1 cm) which were exposed to various doses of Paraziquantel (0.1, 0.5, 1.0, and 2.0 ml per 50 liters of water). The drug was added directly to the water (water bath technique). Praziquantel penetrated through the parasite integument causing extensive muscle contraction and death due to necrosis of internal organs. At a dose of 0.1 to 0.5 ml the parasites died within 1 to 4 hours; at a dose of 1.0 ml the parasites died within 1 to 8 days; and at a dose of 2.0 ml, the parasites were dead within 20 to 60 minutes. The effectiveness of treatment in all the series was 100%. The preparation (treated water bath) did not cause fish death or deviation in fish behavior. We consider the use of Praziquantel against trematodes a valuable addition to the pharmaceuticals available for fish diseases, especially for valuable fish species in the USSR.

NOTE: Dr. Zhatakanbayeva will continue the study of drug efficacy using Praziquantel after the departure of Dr. Heckmann from the USSR. Twelve additional studies have been planned and started.

The Efficacy of Praziquantel Against Helminths of Fish in the USSR

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Detection of the Cutthroat Trout Virus in Oregon

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A picorna-like virus that was isolated from numerous trout broodstocks in California (Yun, Hedrick and Wingfield, Fish Health Newsletter 17(2), 5, 1989) has recently been detected in Oregon. The virus has been designated the cutthroat trout virus (CTV) because it was first found in cutthroat trout (Oncorhynchus clarki). Isolation of the virus was from ovarian fluids collected from Oregon’s two major rainbow trout (O. mykiss) broodstocks. Prolonged incubation (21-30d) with blind passes of samples on CHSE-214 cells at 15 C produced cytopathic effect (CPE) previously described as diffuse necrosis. Positive controls were utilized to aid in the identification of CPE because of its subtle nature. The virus was subsequently confirmed by electron microscopy and shown to exhibit similar morphological characteristics as CTV. Definitive identification will rely on the development of appropriate serological tests. There was no evidence of disease in any of the adult fish that were tested or their resulting progeny.

Initial reports of the virus caused a moratorium on egg and fish shipments from California rainbow trout producers into Oregon. However, because of the recent isolations of CTV these restrictions have been removed. Possibly CTV has been present in many trout broodstocks on the west coast of the United States but because of the prolonged incubation period in cell culture, the subtle CPE, and it’s nonpathogenic nature it was not detected. Examinations of feral and wild trout broodstocks are continuing in Oregon and recently a presumptive isolation of CTV was made from ovarian fluids obtained from brown trout (Salmo trutta). Furthermore, there have been preliminary reports of a CTV isolation from adult trout in Utah. The virus could be much more widespread than previously thought.

**Sphaerospora - Like Organisms in Three Species of Tennessee Tombigbee Waterway Fishes**

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A survey was conducted to determine the prevalence of fish diseases in Tennessee-Tombigbee Waterway fish. *Sphaerospora*, a myxosporean parasite hypothesized to cause proliferative gill disease in cultured channel catfish (*Ictalurus punctatus*), was found in the gills of channel catfish examined from the system. The severity of tissue reactions varied from mild hyperplasia to extreme proliferation of epithelial cells and chondrocytes. Heavy infiltration of neutrophils and mononuclear cells were associated with these proliferative lesions. *Sphaerospora* - like organisms were also found in the gills of bluegill (*Lepomis macrochirus*) and largemouth bass (*M. salmoides*). Little or no tissue response was present in these fish species. Cysts were often found in the sinusoids of gill lamellae. This organism was not seen in other tissues of these fishes. This report documents the occurrence of this organism in three species of fish in the system. The significance of the occurrence of this organism in non-ictalurid species is not known at this time.

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**Emergency Information Request**

**Black Sea Urchin Plague:** A mass mortality of *Diadema antillarum* sea urchins has occurred in one Caribbean island. Investigators **urgently** need to know if this event is occurring elsewhere. They need fresh, moribund specimens to determine the cause of the problem. During a mass mortality in 1983-1984, and a secondary outbreak in 1985, mortalities occurred at different times in different localities. Please be on the lookout for this problem, and call or FAX immediately with details. They are looking for preserved and moribund samples. Inquiries by others are welcome.

Also, reports of new moderate, but widespread bleaching of coral reefs in a northern Caribbean site, and unknown amounts in southern Caribbean and eastern Pacific sites have been received. Authorities are attempting to determine the severity and extent of bleaching. Please contact M. Hernandez Avila, Caribbean Aquatic Animal Health Project, Department of Marine Sciences, University of Puerto Rico, P.O. Box 908, Luquillo, PR 00667-0908. Telephone (809) 899-2048; FAX 809-899-5500; Telex UPR 354204.

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**Honors**

**Award of Excellence** - At their annual meeting, the Japanese Society of Fish Pathologists honored Dr. John L. Fryer with their Award of Excellence. John is the first foreign recipient of this society’s highest honor. John was also recognized by the Institute of Fisheries, Oregon State University when he received the Distinguished Professor Award, the highest honor the institute can bestow on its faculty. -submitted by John Rohovec.

**Snieszko Student Travel Awards** - This is the first year that travel funds have been awarded from money willed to the Fish Health Section by Dr. S.F. Snieszko. Three students were granted $400 each for travel to the FHS Annual Meeting in Minneapolis this year. The winners were Stewart Alcorn, University of Maryland for "The immune response
of wild brown bullhead catfish to 5 known bacterial pathogens is not affected by pollutants found in the Chesapeake Bay; Larry Hanson, Louisiana State University, for "Biochemical characterization and gene-mapping of the channel catfish virus thymidine kinase"; and Jiraporn Kasornchandra, Oregon State University, for "Characterization of a rhabdovirus isolated from Snakehead fish." Candidates for next year's awards to the 1991 meeting are now being solicited. Proposed abstracts and recommendations should be sent to Dr. Alec G. Maule, Oregon Cooperative Fishery Research Unit, Dept. of Fisheries and Wildlife, Oregon State University, Corvallis, Oregon 97331-3804. -submitted by Doug Anderson (Chairperson), Alec Maule and Pete Walker.

passes

Dr. Graham (Pete) L. Bullock, Scientific Director of the National Fish Health Research Laboratory, quietly retired March 30, 1990 from the Fish and Wildlife Service. Pete is one of the founding fathers of the Fish Health Section, a past president and was deeply involved in the development of the first 'Blue Book'. Pete served the Fish and Wildlife Service well for 31 years and the Fish Health Section from its inception. We will miss him and we hope he will think of all of us as he basks in the sunshine of retirement.- submitted by Ed Noga

Dr. Scott E. LaPatra has left the Oregon Department of Fish and Wildlife. His new address is Clear Springs Trout Co., Research Department, P.O. Box 712, Buhl, Idaho 83316. Phone (208) 543-4316.

Dr. Randy MacMillan has left the College of Veterinary Medicine at Mississippi State University. His new address is Clear Springs Trout Co., Director of Research and Development, P.O. Box 712, Buhl, Idaho 83316. Phone (208) 543-4316. Effective Aug. 1, 1990.

Dr. Paul Reno has accepted a position at Oregon State University in the Coastal Oregon Marine Experiment Station and the Department of Microbiology. Paul's new address is Oregon State University Hatfield Marine Science Center, Newport, OR 97365. The phone is (503) 867-3011.

FISH PATHOLOGIST

Applications are invited from established fish pathologists for a faculty position in a new fish health laboratory in the Virginia-Maryland Regional College of Veterinary Medicine. The position is to serve a rapidly expanding warm-water fish farming industry in Virginia. There will be close integration of these activities with the College of Agriculture's aquaculture program, the fish producers of the State, and other interested groups. The fish pathologist will be responsible for establishing and supervising a laboratory for the diagnosis of fish health problems, leading research in aspects of fish health and therapy, for teaching graduate and veterinary students and for supervising technical work, and for integrating this aspect of the work with fish production research in the College of Agriculture.

The appointee should have several years of practical experience and proven ability in working with fish health problems in warm-water, high density, high volume fish production systems. In addition to technical competence, the fish pathologist should be skilled at communicating with other professionals and producers and laboratory management. The appointee should have post-graduate qualifications in fish pathology and may have a D.V.M. degree.

This is a tenure-track position.

Applications will be accepted through August 1, 1990 or until position is filled. The rank, salary and starting date will be negotiable.

Applications should be addressed to:
Dr. Donald O. Cordes
Professor and Head, Pathobiology
College of Veterinary Medicine
Virginia Polytechnic Institute and State University
Blacksburg, VA 24061

VPI& is an EO/AA Employer
"Women and minorities are encouraged to apply."

Courses

Haywood Community College and the N.Y. Dept. of Environmental Conservation will sponsor a course in Coolwater Alternatives held September 10-13, 1990, at the Regional High Technology Training Center near Clyde, NC. Instruction will focus on the intensive commercial production of walleye. Some attention will also be given to yellow perch and tiger muskies.

Included in the lecture and slide presentations will be care of fish at all stages of growth, feeds and feeding, maintaining water quality, and marketing. Production in ponds, cages and recirculation systems will be covered.

The instructor will be Dr. Richard Colesante, Senior Aquatic Biologist at the Oneida Hatchery Research Unit in Constantia, NY. He has conducted extensive research on the intensive culture of walleye and other coolwater fish for over 17 years.

A $15.00 registration fee will be charged on September 10. For more information or to preregister, contact: Charles W. Johnson, Fishery Training Specialist, Haywood Community College, Freedlander Dr., Clyde, NC 28721. (704) 627-2821. FAX No. 704-627-3606.
Call for Presentations

Problems of Chemotherapy in Aquaculture: Theory and Reality. March 12-15 1991, Paris, France. Sponsored by the Office International des Epizooties (OIE). This international conference aims to collate up-to-date information on the use of chemotherapy in farmed aquatic animals, and to prompt active discussion on the perceived problems of concern to regulatory authorities and whether these are based more on theory than on the reality of the situation. Subjects will be presented and discussed in 5 main theme sessions, with a final discussion and summing up session at which the main conclusions will be identified. Selected experts will present keynote papers in each of the sessions, followed by offered oral communications to provide specific detail to exemplify or complete the overview. In addition, there will be a limited number of poster presentations. Language of the conference will be English or French with simultaneous interpretation.

The program of sessions will be:

Session 1: Chemotherapy in Aquaculture Today
Session 2: Poster Session
Session 3: Toxicity and Side Effects of Chemotherapeutants
Session 4: Problems of Resistance
Session 5: Residues
Session 6: Discussion and Conclusion

The selection of presentations to be included in the program will be made by the program Committee on the basis of abstracts submitted by prospective speakers and received before July 15, 1990. Notification of acceptance will be issued soon after September 15, 1990. Submitted papers not selected for inclusion in the oral presentations may be offered the alternative of space for a poster presentation. All contributors will be required to submit a full typescript of their presentation to the OIE before January 15, 1991, so that a working document can be provided to all registered participants at the start of the conference. The final document containing the texts of the Conference and of the discussions and recommendations will be issue by the OIE by the end of 1991.

Limitation of space at the OIE necessitates restricting the number of participants to 250. The registration fee will be 1,800 French franc.

Notification of interest in participating and requests for further details should be addressed to:

International Conference on Problems of Chemotherapy in Aquaculture
Office International des Epizooties
12 rue de Prony
75017 Paris
France
Telephone: 33 1 42.27.45.74

FAX : 33 1 42.67.09.87
Telex : EPIZOTI 642 285 F

Note for Contributors

A printed set of all papers to be presented will be prepared before the meeting is held. Documents will be transferred to a PC system and it will be possible to send them directly as diskette files. The technical specifications will be as follows:

diskette format 5 1/4
capacity 364 Kb or 1,2 Mb
double sided/double density or double sided/high density, MS-DOS formatted
document converted and saved into an ASCII file.

Use the command "COPY" and not "BACK UP" when you copy your file on the diskette.

There are no format specifications for submitted abstracts which must merely be detailed enough to provide a clear idea of the content of the communication.

Manuscripts may be submitted in English or French, but they must be accompanied by a detailed summary in the other language.

Editors Page

Erratum- The newsletter item published in the Fish Health Section Newsletter Vol. 18 (1): 4-5 entitled "Infectious Hematopoietic Necrosis Virus (IHNV) Transmission Studies in Oregon" by Scot LaPatra contained information which was not approved by the author. Specifically, the absolute statements regarding no vertical transmission of IHNV were inserted by an unknown individual who had also submitted this item to the editors. Dr. LaPatra believes it is premature to make such an absolute statement. We (the editors) regret this occurrence.

Readers Comments

Congratulations on your first issue of the Fish Health Newsletter. I found the format clean, crisp and easily readable and the articles pertinent and informative.

I took exception to the inclusion of the editor's note following my article on isolation of an oxidase-negative Aeromonas salmonicida, however, and would appreciate an explanation in the next newsletter of your objective in publishing it and a statement of your editorial policies for the newsletter.

The back page of the newsletter indicates that material
published in it is done so with the understanding that it is not peer reviewed. If this is truly the case, then I believe your actions were indefensible, particularly in light of the fact that you neither contacted me to discuss the points you addressed nor requested a culture of the bacterial isolate in question to confirm my findings or support your claims. The validity of the points you raised in the note notwithstanding, these, I believe, have no place in a newsletter designed to be an informal vehicle for the quick dissemination of information in the field of fish health. Your comments would have been more appropriate had they come from an anonymous reviewer of an article submitted to a peer reviewed journal. Even in that case, they would not have been published, but rather sent to the author to consider in revising a manuscript.

While as editor you must necessarily make decisions on format and content of the newsletter, I believe you also have the responsibility to use your position in a fair and impartial manner. The fact that comment was limited to my article when other articles also contained controversial data and statements (e.g. LaPatra’s IHNV article) leads me to conclude that your positions are not being used fairly or impartially. I must admit that this conclusion does not encourage me to submit material to the newsletter in the future.

While I believe in and defend open and frank discussion and review of all scientific findings, I only do so provided that these are done appropriately. The statement in your note that further testing “should” (emphasis added) indicate the presence of an oxidase enzyme is scientifically and editorially irresponsible, in my opinion, because it is made without knowing the exact methods I used, it is an opinion of the editors that is not supported by any data and is presented without an opportunity for me to refute it. Science is filled with findings that, when reported, were in conflict with accepted beliefs of the time. These were confirmed or refuted only by further research, not by making unsupported statements.

Due to space limitations I did not explain specific methods used to test the oxidase reaction. These methods are completely outlined in a manuscript I have submitted for publication in a peer reviewed journal. Both dimethyl-p-phenylenediamine and tetramethyl phenylenediamine di-HCl were used to test multiple isolates grown on various media. Contrary to the implication in the editor’s note, oxidase reaction was consistently positive or negative for each particular isolate regardless of which reagent was used.

Subsequent to submitting my article to the newsletter, the American Type Culture Collection has included the isolate in the collection and has confirmed its identification as an oxidase-negative *A. salmonicida* (ATCC #49385). I encourage interested colleagues to obtain a culture for further testing and to contact me for more information.

*Sincerely, Patrick F. Chapman.*

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**Editor’s reply**

*We apologize to Mr. Chapman and any of his co-workers who took exception to our comments regarding the presence of an oxidase negative *A. salmonicida*. Our intent was to provide educational information and not to question the validity of their results nor to peer review the submission. A consulting microbial physiologist (who reviewed our comments) provided the factual basis on which our comments were based. Subsequent review of the literature substantiate the possibility of bacteria losing cytochrome oxidase enzymes or at least the possibility of the bacterial electron transport chain losing the ability to convert certain aromatic amines into colored products which is the basis of the oxidase test. Obviously, aerobic bacteria do exist which are oxidase negative.*

Mr. Chapman and the problems we have encountered regarding Scott LaPatra’s newsletter items raise important issues regarding the editorial policies of the editors. We have consequently examined these policies and propose the following guidelines:

1. All materials submitted to the Fish Health Section Newsletter must be accompanied by written release authorization from one of the authors. These will be kept on file.

2. Items submitted to the Newsletter will not be peer reviewed nor will we provide editors comments. It is the responsibility of the persons submitting the newsletter items to ensure scientific accuracy. Discussion of controversial issues or scientific findings will be reserved for editorial.

3. We reserve the right to edit materials for clarity or to shorten material as appropriate because of space limitations. We will strive to give authors an opportunity to approve of our editing prior to publishing.

4. The newsletter is intended to be a means for rapid dispersal of information. We will strive to publish the newsletter in a timely fashion.

Once again we apologize to Mr. Chapman and to Dr. LaPatra for the difficulties encountered by our zealous efforts or for the lack of clearly defined editorial policies. We encourage their continued support of the newsletter and the Fish Health Section. - Editors.

**Meetings**

Joint Meeting of Fish Health Section of the American Fisheries Society and the Midwest Fish Disease Workshop, July 16-19, 1990. Minneapolis, MN. Contact Dr. Paul R. Bowser, Department of Avian and Aquatic Animal Medicine, N.Y. State College of Veterinary Medicine, Cornell University, Ithaca, New York 14853 for further information. Phone (607) 253-3365.
Symposium on Diseases of Asian Aquaculture, November 26-29, 1990. Sawer Beach, Bali, Indonesia. Information may be obtained from Dr. Mohd. Shariff, Asian Fisheries Society, Fish Health Section, c/o Faculty of Fisheries and Marine Science, Universiti Pertanian Malaysia 43400 Serelang, Selangor, Malaysia.


Notes Added in Proof

FHS Membership Directory is still accepting submissions. Please relay information to Kathy Hopper, Dept. of Fisheries, 115 General Administration Bldg., Olympia, WA 98504. Phone (206) 753-6600.

Additional Members of FHS Membership Committee- Beth MacConnell and Pat Chapman.-submitted Kathy Hopper.

Dr. Richard A. Heckmann announces the availability of a 3 volume series edited by Dr. Oleg Bauer pertaining to the Parasites of Fishes in the USSR (1987). The series is to be translated into English by Drs. Pugachov and Bauer. Dr. Bauer would like to know how many fish parasitologists in the USA and Canada would need copies. Contact Dr. R.A. Heckmann, Dept. of Zoology, Brigham Young University, Provo, Utah 84602 who will in turn contact Dr. Bauer.

Deadline For Fall Edition is August 15, 1990. Address to Dr. Randy MacMillan, Director of Research, Clear Springs Trout Company, P.O. Box 712, Buhl, Idaho 83316 or any member of Newsletter Committee.
Fish Health Newsletter

The Fish Health Newsletter is a quarterly publication of the Fish Health Section of the American Fisheries Society. Submissions of any length on a topic of interest to fish health specialists are encouraged with the understanding that material is not peer reviewed. Submissions should be addressed to one of the editorial staff or to a member of the publication committee.

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