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Broodstock Erythromycin Injection Prevents Renibacterium Vertical Transmission

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Bacterial kidney disease, caused by *Renibacterium salmoninarum* (Rs), is the most important infectious disease affecting farmed salmon in British Columbia (B.C.), Canada. Chemotherapeutant control of the disease is unsatisfactory, and vaccination is still not feasible. The bacterium is transmitted from parent to progeny (vertically transmitted) via the egg, and Rs-infected eggs are a major source of Rs for the B.C. salmon farming industry. The purpose of this experiment was to determine if egg infection could be avoided by using broodstock erythromycin injections. Using this technique, we demonstrate that therapeutic levels of erythromycin can be obtained in intraovum eggs.

Five weeks before spawning, 10 wild, healthy, adult coho salmon females were intraperitoneally injected with Rs (10^8 Rs cells per fish). Five days later, 5 of the fish were injected with erythromycin (20 mg per kg fish via the dorsal sinus) and 5 were left as untreated controls. Following spawning ca. 4 weeks later, the eggs from each female were fertilized with milt pooled from three healthy males and incubated in trays (eggs from 1 female per tray) until hatching. Yolk samples from eyed eggs and alevins from each of the treated and control females were tested for their antibiotic content (microbiological assay) and for the presence of Rs using culture (eyed eggs) or the millipore filtration-fluorescent antibody technique (alevins).

Our results (Table 1) indicate that prespawning injection of Rs into non-antibiotic treated female coho salmon resulted in egg infections (8 to 22%) in all 5 females tested. These egg infections could all be eliminated by injecting the Rs-infected females with erythromycin approximately 4 weeks prior to spawning. The end result was that the progeny from the treated fish proved to be Rs-free. We conclude that a prespawning injection of erythromycin into mature female salmon is a promising procedure for preventing the vertical transmission of Rs and that closely monitored field trials using this technique are warranted.

Table 1. Erythromycin (E) levels and *Renibacterium salmoninarum* (Rs) prevalence in the yolk of eggs and alevins derived from E-treated and untreated Rs-infected females.

Pre-spawning Treatment	Spawner Number	E level [1]		Rs prevalence[2]	
		Eggs	Alevins	Eggs	Alevins
E injected 5 days after Rs injection	1	1.6	0.7	0	0
	2	1.0	0.7	0	0
	3	1.1	0.7	0	0
	4	1.3	0.6	0	0
	5	1.1	0.7	0	0
Untreated; Rs injected (Controls)	6	0	0	15	2
	7	0	0	19	3
	8	0	0	11	1
	9	0	0	22	2
	10	0	0	8	3

[1] Level = μg E per ml yolk.
[2] Yolk from 100 eggs and 100 alevins tested per spawner; yolk from alevins was tested in 10 alevin pools.

Systemic Diplomonad Flagellate Infection in Chinook Salmon from British Columbia Seawater Netpens

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Severe systemic infections by a diplomonad flagellate (family Hexamitidae) were associated with high mortality in chinook salmon (*Oncorhynchus tshawytscha*) reared at a seawater netpen site in Sechlt, British Columbia. The fish were introduced to sea water in the spring of 1990 and showed unusual mortality starting in September 1991. Over the next six weeks these fish suffered approximately 20% mortality, with one pen of fish suffering mortality of about 70%. The infection was also detected in a few moribund chinook introduced to the site in the spring of 1991, but was not detected in coho salmon (*O. kisutch*) reared at the same site.

Infected fish appeared normal externally except that some fish exhibited abdominal swelling. The gills of most of the fish were pale, indicating anemia. The hallmark gross pathologic changes were extreme liver hypertrophy, ascites, and visceral cavity blood clots. The liver was mottled and often exhibited petechiae. Spleen and kidney were moderately enlarged, and petechiae were evident throughout skeletal muscle.

Wet mount preparations of liver, kidney, gastrointestinal tract, and ascitic fluid revealed massive numbers of diplomonad flagellates. The parasites were indistinguishable from *Hexamita salmonis* in Giemsa and protargol stained imprints. Histological examination revealed massive numbers of parasites in blood vessels in all organs, with concentrations particularly high in the liver and lower intestine. The liver was edematous and showed diffuse infiltration of inflammatory cells resembling lymphoblasts and plasmablasts in the sinusoids. The renal interstitium was hyperplastic. Although many parasites were observed in blood vessels of the lamina propria and submucosa of the pyloric caeca and intestine, the epithelium was usually intact. Bacterial culture of kidney and spleen on Tryptic Soy Agar with 1% NaCl revealed no bacteria, while cultures of ascites from some fish grew mixed bacteria types, including *Vibrio* spp.

Hexamita salmonis is a common parasite of the intestinal tract of salmonids reared in fresh water. Most infections do not cause disease, but some reports have attributed anorexia, emaciation, poor growth, and mortality in salmon fry to the infection. (Becker, C.D. 1977. in J.P. Kreier [ed.] Parasitic Protozoa. vol. I. Academic Press. 357-416). Other diplomonads (family Hexamitidae) infect strictly marine fishes (Poynton, S.L. and C.M. Morrison. 1990. J. Protozool. 37:369-383), and it has been suggested that *H. salmonis* may persist in salmonids after they migrate to sea water (Lom, J. 1984. in Kinne, O. [ed.]. Diseases of Marine Animals Vol. IV. Part I. Biologische Anstalt Helgoland, Hamburg, German, pp. 114-168). We have yet to determine if the infection reported herein was contracted in sea water or if fish were subclinically infected before they were transferred to netpens. Systemic infections by diplomonad parasites in fish are rare and we are aware of only two other reports of such infections. Mo, T.A., T.T. Poppe, and L. Iversen (1990. Bull. Eurp. Asso. Fish. Pathol. 10[3]:69-70) reported a systemic hexamitosis in pen-reared Atlantic salmon (*Salmo salar*) from Norway, and Ferguson, H.W. and R.D. Moccia (1977. J. Am. Vet. Med. Asso. 177:854-857) reported a similar disease in Siamese fighting fish (*Betta splendens*).

An understanding of the source and mode of transmission of the infection would be helpful for implementing effective control strategies or prophylactic treatments. Studies underway include an ultrastructural examination of the parasite to determine its precise identity, and determination of the transmissibility and survival of the parasite in fresh and sea water.

Rhodococcus - Associated Granulomatous Nephritis in Atlantic Salmon Smolt

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Differential diagnosis of *Rhodococcus*-associated granulomatous nephritis (Claveau, R. 1991. Med. Vet. Quebec 21(4): 160-161), BKD, and perkinsiosis (Cawthorn, R.J. et al, 1991. FHS/AFS Newsletter 19 (1):5.) has become important to the commercial salmon industry located in eastern Canada.
Each of these diseases can be associated with similar nephric

lesions. Perkinsiosis and *Rhodococcus*- associated granulomatous nephritis have only recently been detected in this region.

Rhodococcus-associated granulomatous nephritis, as first described by Claveau (1991), involved a chronic episode of low level mortality (approx. 0.1% per week) affecting salmon smolt housed in 10 m diameter plastic-lined galvanized circular tanks supplied with 3° C brackish water pumped from a seepage well. Affected fish had markedly enlarged kidneys, which contained randomly distributed, 1 to 4 mm diameter round, white, soft foci that on cut section had a necrotic core. Histologically, these foci were poorly encapsulated granulomas that contained large numbers of Gram-positive pleomorphic rod-shaped bacteria.

We tested the pathogenicity of the dominant strain of bacteria (identified as *Rhodococcus* sp. by conventional biochemical and gas-liquid chromatographic analysis of the cellular fatty acids) isolated from kidneys by intraperitoneal (IP) injections of bacteria (10^6 or 10^9 bacteria per fish) into Atlantic salmon smolt. A severe granulomatous peritonitis developed within 3 weeks. Despite this, the mortality rate was low (less than 10% of fish in the 10^9 group), and morbidity was rarely apparent despite the development of severe internal lesions in most of the injected fish.

Based on the course of the natural disease, and from experimental challenge studies reported herein, *Rhodococcus* sp. is pathogenic to Atlantic salmon. Although the mortality rate was low, the sub-clinical effects on growth could be significant. Most importantly, since *Renibacterium salmoninarum* and *Rhodococcus* sp. are Gram positive, intracellular bacteria, they could be easily misdiagnosed unless confirmatory tests were conducted.

Production of Dermonecrotic Factors by Gram-negative Fish Pathogens

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Virulence associated dermatotoxic activity has only been associated with the fish pathogens *Edwardsiella tarda* (Ullah and Arai, Fish Pathol. 18: 65-70, 1983) and *Aeromonas hydrophila* (Olivier et al. Can. J. Microbiol. 27:330-333, 1981). In the present work a total of 46 pathogenic strains of different origin (*A. hydrophila*, 13 isolates; *Vibrio anguillarum*, 11; *Pasteurella piscicida*, 6; and *Yersinia ruckeri*, 16) were evaluated for the presence of dermatotoxins in their extracellular products (ECP). Skin permeability assays were performed in adult New Zealand rabbits by intradermal injection.

The extracellular products from all *Y. ruckeri* isolates produced strong hemorrhagic and/or edematous zones at the rabbit inoculation site. However, within the other bacterial species, strains positive and negative for this activity were found (Table 2). Interestingly, differences in the expression of dermatotoxic activity were observed in all the isolates depending on the growth media and ECP extraction procedure. The detection was significantly favored when the cellophane plate technique of Liu (J. Bacteriol. 74: 718-727, 1957) was employed. Preliminary thermolability trials (inactivation at 80° C for 10 min) suggest that proteolytic enzymes could be responsible for this activity.

Although no correlation has been established between bacterial virulence and dermonecrotic activity for these isolates, we cannot rule out the contribution of this factor to the pathogenicity of a particular strain.

Table 2. Presence of Dermonecrotic factor in 47 strains of Gram-negative bacterial fish pathogens.

Bacterial Species	No. positive strains/ No. strains tested
A. hydrophila	*10/13*
V. anguillarum	*7/11*
P. piscicida	*5/6*
Y. ruckeri	*16/16*

Standardization of Infectious Hematopoietic Necrosis Virus Challenge Procedures

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Different experimental procedures have been used to infect fish with infectious hematopoietic necrosis virus (IHNV). Variable experimental parameters include established fish cell lines used to replicate IHNV and number of fish tested in each treatment group. It has been postulated the cell line used to produce virus stocks, of either salmonid or non-salmonid origin, may influence virulence. Additionally, researchers have speculated that amplification of the original infectious dose in a group of fish and subsequent horizontal transmission of virus among fish may influence results. In an attempt to provide some information for standardization of waterborne infection procedures, we determined the affect on virulence when duplicate 25 or 50 fish groups were exposed to one IHNV isolate, replicated in three different established fish cell lines. We also developed an experimental waterborne IHNV-infection method that provides reproducible results and may allow better comparison of experimental data.

An IHNV isolate (220-90), was obtained from clinically infected rainbow trout after routine virological procedures using the CHSE-214 cell line (1x pass). The isolate was passed two more times on established cell lines that originated from rainbow trout *Oncorhynchus mykiss* (RTG-2), chinook salmon *Oncorhynchus tschawytscha* (CHSE-214), and common carp *Cyprinus carpio* (EPC) to produce stock virus for waterborne exposures. At each subculture, 2 mL of a 1:1,000 dilution of virus was inoculated into a 150 cm^2 flask of a particular cell line. After each passage the virus concentration of the lysate was determined by EPC cell plaque assay using a methyl-cellulose overlay.

Virulence comparisons were done, using virus that had been passed twice on each cell line, in rainbow trout obtained from Clear Springs Trout Company Broodstock operations. Duplicate specific pathogen-free groups of 25 (1.6 g mean weight) or 50 fish (1.7 g mean weight) were exposed to 10,000 plaque forming units (PFU)/mL in a volume of water that was 10x the total weight (in grams) of the fish. Virus dilutions, based on the stock virus concentration determined before freezing at -80 °C, were made in 10 mL of buffered saline solution so that each group was exposed to the same number of PFU's on a weight to volume basis. Waterborne virus exposures were conducted in closed systems for 1 h with aeration. Each group was placed into a separate 19 L aquaria receiving constant temperature (15 °C), ultra-violet disinfected, single-pass spring water at a flow rate of about 2 L/min. Fish were monitored 28 d for mortality and daily (4x) fed *ad libitum* a dry pelleted feed (Clear Springs Trout feed). A minimum of 20 % of each days mortality was individually examined for virus. Quantitation of virus used in fish exposures or isolated from dead fish was accomplished by plaque assay. Virus concentrations of kidney-spleen-liver homogenates were determined for some of the dead fish examined in each test. Cumulative percent mortality (CPM) and mean number of days to death (MDD) of the replicates were analyzed by chi-square analysis and analysis of variance.

Concentrations of IHNV determined after each passage on each of the cell lines tested and after freeze-thawing for each of the waterborne infection experiments are summarized in Table 3. Virus concentrations appeared to remain constant when replicated on a particular cell line and after a freeze-thaw after different durations of storage at -80 °C. This provided a basis for consistent determination of exposure concentrations which aids in reproducibility of results.

Table 3. Infectious hematopoietic necrosis virus concentrations after one or two passages on established cell lines that originated from rainbow trout *Oncorhynchus mykiss* (RTG-2), chinook salmon *Oncorhynchus tschawytscha* (CHSE-214), and common carp *Cyprinus carpio* (EPC) and after storage at -80 °C.

	IHNV Concentration[a]			
	Passages in Cell Culture		Storage at -80°C	
Cell Line	One	Two	9 d	82 d
RTG-2	7.08	7.23	7.29	7.35
CHSE-214	7.79	7.92	8.05	8.00
EPC	7.96	7.62	7.62	7.73

[a]Virus concentrations are reported as $\log_{10}$ plaque forming units/mL.

There was no significant difference in CPM (chi-square analysis; $p>0.1$) or MDD (analysis of variance; $p>0.1$) between any of the groups tested in the infection experiments (Table 4). Virus was isolated from 84% (115/137) of the dead fish examined. Virus concentrations ranged from $10^{2.3}$ to $10^{7.3}$ PFU/g of tissue examined from randomly selected dead fish at each exposure with a mean of $10^{5.6}$ PFU/g.

The results from this study suggest that cell line used to produce stock IHNV, after two passes, does not effect viral virulence in this stock of rainbow trout. Furthermore, the number of fish (25 or 50) in our system did not effect CPM or MDD suggesting that horizontal transmission of virus does not influence results. We have routinely used this IHNV-waterborne infection procedure and maintain

consistent reproducibility. Further confidence can accrue by using the same lot of fish between trials. Even still, the two challenges reported here were conducted about 70 d apart with different lots of fish and the results were still consistent. We attribute this reproducibility to the standardized waterborne infection procedure employed. Additional parameters that need to be maintained for consistency include fish stock, size, age, and nutritional status and use of an uncontaminated, constant temperature water supply.

Table 4. Cumulative percent mortality (CPM) and mean number of days to death (MDD) in rainbow trout *Oncorhynchus mykiss* infected with one isolate of infectious hematopoietic necrosis virus replicated on three different cell lines.

	Cell Line Used for Virus Replication							
	EPC		CHSE-214		RTG-2		Control	
Number[a] of fish	CPM	MDD	CPM	MDD	CPM	MDD	CPM	MDD
25	59 (29/49)	12	56 (25/45)	12	53 (26/49)	12	0 (0/24)	nm[b]
50	58 (56/97)	10	56 (54/97)	12	64 (62/97)	12	0 (0/50)	nm

[a] Duplicate 25 or 50 fish (1.6-1.7 g mean weight) were challenged with 10^4 PFU/mL of virus.

[b] nm = no mortality.

Whirling Disease Discovered in Utah

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In June 1991, the spores of *Myxobolus cerebralis* were discovered in rainbow trout (*Oncorhynchus mykiss*) during a routine fish health inspection of a private aquaculture facility in the Colorado River drainage in southern Utah. The spores were first detected by the plankton centrifuge technique and found to be morphometrically consistent with *M. cerebralis*. Definitive diagnosis was made by identification of spores in cerebral cartilage by histopathologic examination. This represents the first known presence of this parasite in Utah.

Subsequent testing of the watershed has shown the parasite to be more widespread than earlier thought. Salmonids located in 2 upstream reservoirs and several miles of river downstream from the affected hatchery have also tested positive. In one section of river below an affected reservoir, rotenone sampling showed approximately 8% of the wild rainbow trout with gross deformities of the head or spine. In addition, spores of *M. cerebralis* have been detected at 3 other aquaculture facilities in the drainage and 2 other sites in the Great Basin drainage. The finding of spores at these locations suggest transfer of infected live fish from affected facilities may have occurred. The origin of the parasite into the state remains a mystery.

Parasite eradication plans are continuing to develop as the scope of fish infection is determined. Efforts so far have involved chemical eradication and removal of all wild/feral fish from the watershed to eliminate the fish host. Fish at the affected facilities will be grown to marketable size prior to removal. Efforts are being made to protect unaffected state and private facilities located downstream from contamination.

Update on ESC Study

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A previous report from our laboratory in the FHS/AFS Newsletter (Chen and Kumlin, 1989) suggested that *E. ictaluri* may exist in the lower intestine of asymptomatic channel catfish. Rectal smears of channel catfish were stained by an indirect fluorescent antibody technique (IFAT) using an anti-ESC antiserum prepared in rabbits. Positive staining rods were observed in smears from fish which had high antibody titer to *E. ictaluri*. However, further tests with this polyclonal antiserum indicate that it has some reactivity with another bacteria species known to exist in the digestive tract of channel catfish (MacMillan and Santucci, JAAH, 1990). Cross-absorption experiments suggest that this reactivity is not due to a common antigen between *E. ictaluri* and *Plesiomonas shigelloides*, but that the rabbits used for antiserum production had a pre-existing titer to *P. shigelloides*, as well as titer to other bacteria which both rabbits and channel catfish carry as normal flora. The antiserum was used at 1:50 in the IFAT, whereas we have recently found a 1:100 dilution necessary to avoid this undesirable cross-reactivity. The questionable results of the 1989 IFAT rectal smears, and negative results of ourselves and others (MacMillan and Santucci, 1990) in attempting to culture *E. ictaluri* from the lower intestine of asymptomatic catfish, suggested that a carrier state did not exist.

The formulation of a selective media for isolation of *E. ictaluri* (Shotts and Waltman, JWD, 1990) encouraged us to again attempt isolation from the lower intestine. We recently isolated *E. ictaluri* from the lower intestine (rectal swab), but not the kidney or brain of two (thirty fish tested) apparently healthy yearling channel catfish. Fish from this pond had an antibody titer against *E. ictaluri* exceeding 4096 by microtiter agglutination, with 30 of 60 fish having a titer of 16 or greater. A total of 120 fish from this pond were grossly examined for external signs of ESC, but none were found, and no mortality was noted. These isolations further confuse the ESC carrier state picture. We are continuing field sampling and other studies to improve diagnostic methods, especially nonlethal methods, and to determine the distribution of ESC in California.

Iodophor Toxicity to Grayling Eggs?

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In the spring of 1991 the Montana Department of Fish, Wildlife and Parks used an iodophor (Betadine) solution to water-harden grayling eggs. On two occasions at two different locations attempts were made to water-harden grayling eggs in 75 mg/l buffered iodophor for 30 minutes. Immediately after water-hardening, eggs began to die and within a few hours all eggs were dead. Treated eggs appeared to swell and burst resulting in a soupy mess. Even the slightest touch caused the eggs to break. Another group of eggs was collected during the second egg take which was not exposed to the iodine. These eggs were collected from the same spawning population on the same day as the second iodophor-treated group. They were treated in exactly the same way as the iodophor-treated group, except they were water-hardened in plain lake water. These eggs developed normally and had a very successful hatch.

Several possible causes of the egg loss are being considered, including possible trauma to eggs while pouring off the iodophor after 30 minutes and the possibility that iodine is more toxic to grayling eggs than other fish species. Anyone water-hardening grayling eggs in iodine should use extreme caution.

Fish Mortality Investigations in Kentucky

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Ninety-five (95) fish mortality investigations were performed in the first 11 months of 1991 at the Kentucky State University Fish Disease Diagnostic Laboratory in Frankfort. Species and number of cases examined were as follows: channel catfish (46 cases), hybrid striped bass (32 cases), black bass (6 cases), rainbow trout (3 cases), tilapia (3 cases), fathead minnows (2 cases), hybrid bluegills (2 cases), and bluegills (1 case).

Internal bacteria were involved in 60 cases. Of these cases, 18 were caused by *Edwardsiella ictaluri*, and 2 were caused by internal *Flexibacter* sp. External *Flexibacter columnaris* was a contributing cause of mortality in 19 cases. Parasites were responsible for mortalities in 23 cases. These cases involved *Trichodina* (5 cases), *Ichthyophthirius multifiliis* (4 cases), *Ambiphrya* sp. (4 cases), *Henneguya* sp. (3 cases), *Chilodonella* sp. (2 cases), gill monogenes (2 cases), *Trichophrya* sp. (1 case), *Lernaea* sp. (1 case), and *Posthodiplostomum mimimum* (1 case).

One case of channel catfish virus (CCV) was identified among 2 to 3 inch fingerlings crowded heavily at over a half million per acre. They also had many gill worms brought on by the crowding.

The remaining cases were as follows:

Cause or Contributing Factor	Number of Cases
Ammonia toxicosis	2
Nitrite toxicosis	1
Nutritional (starvation)	2
Gas Bubble Trauma	5
High Temperature (greater than 70° F for trout)	1
Crowding	1
Handling stress	1
Routine check	6
Inadequate sample	3
Unknown	10

Private aquaculture businesses (including fish farmers and pay lakes) submitted 61 cases, state hatcheries and laboratories submitted 25 cases, and universities had 9 cases examined.

President's Message

The past few months have been a busy time and there are some areas the section has become involved in which the membership should be aware of. First, the FHS is a

co-sponsor of Aquaculture'92 which will be held May 21-25, 1992 in Orlando, Florida. This is an excellent opportunity for us to interact with the Fish Culture Section, World Aquaculture Society and other aquaculture associations. I hope we will be well represented at this meeting.

I was asked to represent the Fish Health Section as a member of the Joint Subcommittee on Aquaculture's Working Group on Quality Assurance in Aquaculture Production. This is a group composed of representatives from FDA, USDA, EPA, FWS, the major aquaculture producer groups, AFS (FHS, Fish Culture and Fisheries Administrators Sections) and some state organizations. A number of very important subjects were discussed. First, members of the group are involved in developing and reviewing producer guides on FDA/food safety issues. Second, both commercial producer and public hatchery quality assurance programs were discussed. It is agreed that these are necessary to attempt to assure a healthy and safe product and hopefully to avoid any incidences or consumer perceptions that would adversely impact the aquaculture industry.

The other major subject of discussion was, of course, the lack of approved drugs for aquaculture, the lack of clarification on the use of "gray area" compounds and what needs to be done about the problem. FDA recognizes the problem and is willing to do what they can to work with us in trying to find some solutions to the problems. In the next few months they will be issuing a list of compounds which can be used without approval. This list will include some (or possibly all) of the following: carbon dioxide, sodium chloride, acetic acid, iodine, sodium sulfite, sodium bicarbonate. There may also be additional compounds which we can petition them to consider in this category and the FHS is taking a role in identifying these compounds and then petitioning FDA for clarification on their status.

Other areas which still need to be pursued are approvals on crop groupings rather than one species at a time; exempting various life stages--eggs and fry from the food fish category; extra label use of some compounds and a prioritized list of compounds for which approval will be sought. FDA does seem to be more open to the use of INAD's (investigational new animal drug) permits to generate useful data in commercial situations.

These are areas which are of prime importance to all of us in the fish health area. Both Randy MacMillan and I are members of this Committee and if anyone would like more information on what is going on or has any input they wish to offer please do not hesitate to contact us.

New Fish Disease Bulletin

A new fish disease bulletin has been published by the Australian NSW Agriculture Division. Fisheries Bulletin No. 4 "Disease of Australian native freshwater fishes with particular emphasis on the ectoparasitic and fungal disease of Murray cod (*Maccullochella peeli*), golden perch (*Macquaria ambigua*) and silver perch (*Bidyanus bidyanus*)" was written by Stuart J. Rowland and Brett A. Ingram. This publication is the first major report on the disease of Australian native freshwater fishes and is based primarily on research conducted at the NSW Fisheries' Inland Fisheries Research Station, Narrandera and the Eastern Freshwater Fish Research Hatchery, Grafton. This Fisheries Bulletin was written to serve as a review, and as a practical guide to native fish hatchery operators, fish farmers in the emerging native fish aquaculture industry, and biologists, other fisheries staff and veterinarians involved in the monitoring of diseases and fish kills on farms and in the wild.

Further information can be obtained from Dr. Stuart Rowland, Fisheries Biologist, Eastern Freshwater Fish Research Hatchery, Grafton, N.S.W. 2460, Australia. Telephone 066-420420; FAX 066-447251.

Award Solicitation

The Awards Committee is soliciting nominations from the Fish Health Section membership for both the S.F. Snieszko Distinguished Service Award and the new Special Achievement Award.

The S.F. Snieszko Award is the highest award given by the Fish Health Section and is to recognize fish health scientists for continued outstanding contributions to the field of fish health. Individuals to be considered for this award must be nominated by a current member of the Fish Health Section. The person making the nomination should obtain six letters of recommendation from fish health scientists that address the candidate's dedication to research, teaching and/or service to the field of fish health. These six letters, along with a current curriculum vitae for the candidate and a letter of nomination that clearly states the qualities of the candidate and the specific reasons for the nomination should be sent to the chairman of the Awards Committee prior to February 1, 1992.

The Special Achievement Award is to provide timely recognition for one-time accomplishments that have a significant impact on the management or control of fish health problems. This award may be given for 1) a unique contribution to the fish health field (such as a new diagnostic tool, a new technique to control the spread of a serious disease, etc.), 2) a significant research accomplishment, or

3) outstanding leadership in resolving a major fish health problem. The achievement must meet high standards of science and survive peer review. Individuals to be considered for this award must be nominated by a current member of the Fish Health Section. The nomination letter should clearly state 1) the accomplishment; 2) the significance of the accomplishment to fish health; 3) the implications of the accomplishment to aquaculture (local, regional, national or world-wide). Copies of any publications, or other documents relating to the work should be included. Nominations for the Special Achievement Award should be made within one year after the work has been completed and may be submitted to the Chairman of the Awards Committee at anytime.

For further information contact:

David O. Locke, Chairman
Awards Committee, FHS
Department of Inland Fisheries and Wildlife
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Passages

Dr. Alec G. Maule is now a research physiologist with the U.S. Fish and Wildlife Service. His new address: U.S. Fish and Wildlife Service, National Fishery Research Center, Columbia River Field Station, MP 5.48L, Cook/Underwood Rd., Cook, Washington 98605-5701. Phone 509-538-2299.

Short Course

Disease Diagnosis and Control in Marine Shrimp Culture. June 1-12, 1992. The University of Arizona, Tucson, Arizona. The course, to be taught in english, will consist of comprehensive lectures and practical laboratory training on current diagnosis, prevention, and treatment methods for the principal diseases of cultured penaeid shrimp. Participants should have at least a bachelor's degree in fisheries biology, microbiology, or a related field. Registration is limited to 40, with preference given to those working in shrimp disease diagnostic labs or shrimp culture research programs. For information contact Dr. Donald V. Lightner, Department of Veterinary Science, The University of Arizona, Building 90, Room 202, Tucson, Arizona, 85721. FAX: 602-621-6366; Tel: 602-621-2355.

Meetings

Aquaculture Expo V. Jan. 12-16, 1992. New Orleans, La. For information: Aquaculture Productions, Inc. 4640 S. Carrollton Ave., New Orleans, La. 70119; Phone 1-800-527-7631 or 504-486-9868; FAX 504-488-4135.

Furunculosis Workshop. Richmond, British Columbia, Jan. 30-31, 1992. Contact Mrs. Mona Jane, Aquaculture and Commercial Fisheries Branch, Ministry of Agriculture, Fisheries and Food, 808 Douglas St., Victoria, BC, V8W 2Z7; 604/356-1604; FAX 604/356-7280.

Annual Meeting Catfish Farmers of America. Feb. 26-28, 1992. Perdido Hilton, Orange Beach, Alabama. Contact Bill Glasscock, Little Rock, Arkansas; 501-225-6102; FAX 501-223-8230.

5th International Colloquium on Pathology in Marine Aquaculture. April 2-4, 1992. Montpellier, France. Information: PAMAQ 5, BIOCIM/IMIM, Mini Parc, Bat.2, Rue de la Croix Verte, 34090 Montpellier, France. Tel. (33) 67.61.95.62; FAX (33) 67.61.15.10.

World Fisheries Congress. May 3-8, 1992. Athens, Greece. For information: World Fisheries Congress c/o AFS, 5410 Grovesnor Lane, Bethesda, Maryland 20814. USA.

Aquaculture '92. May 21-25, 1992 in Orlando, FL. There will be a technical session on disease co-sponsored by the Fish Culture and Fish Health Sections. Information: C/O The Crest Organization, 940 Emmett Ave, Suite #14, Belmont, CA 94002. Phone 415-595-2704 inside California or 800-222-8882 outside California.

Pathological Conditions of Wild Salmonids, An Atlantic Salmon Trust Symposium. May 6-8, 1992 at the Marine Laboratory in Aberdeen, Scotland. Information: Drs. D.W. Bruno or K. MacKenzie, SOAFSD, Marine Laboratory, P.O. Box 101, Victoria Rd., Torry, Aberdeen AB99DB, Scotland. FAX: (0224) 879156.

1992 World Congress on Cell and Tissue Culture. Washington, DC, June 20-25, 1992. "Genetic Applications of Tissue Culture." The Invertebrate Division of the Tissue Culture Association and the Society for Invertebrate Pathology will co-sponsor several workshops, contributed paper, and poster sessions as part of the World Congress on Cell and Tissue Culture. Among the joint proceedings will be a workshop-contributed paper session on "Invertebrate Cellular Immunity: In-vitro Aspects." A workshop-contributed paper session is also being organized that will address the topic of "Invertebrate Neoplasia: Initiation and Promotion Mechanisms." A special evening workshop covering "Marine Plant and Animal Cell, Tissue and Organ Culture: Applications to Biotechnology" is in the planning stages. For information contact Aaron Rosenfield, NOAA/NMFS, Oxford Laboratory, 904 S. Morris Street, Oxford, Maryland 21654. Phone 301-226-5193.

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Deadline for Spring Newsletter

March 1, 1992

Fish Health Section Newsletter

The Fish Health Section 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 the editor or to a member of the publications committee.

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