

Septicemia in Farmed Atlantic and Chinook Salmon Due to a Rickettsia-like Agent

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Daily mortality rate increased from 0.01 to 0.06% in 2 netpen-reared Atlantic salmon cages at a fish farm site located in British Columbia coastal waters in November, 1991. Many dark, anorectic and lethargic slow-swimmers were at the surface along pen sides and in the corners. External lesions included bilateral exophthalmus, necrotic stomatitis, raised firm erythematous masses extending from the pseudobranch into the branchial cavity, and 1 cm diameter shallow dermal ulcers on the lateral caudal peduncle. Internal lesions included petechiation on serosa, fibrous adhesions in the abdominal cavity, mild spleno- and hepatomegaly with patchy areas of hepatic fibrosis, and multiple pale umbilicate hepatic capsular foci which extended into the parenchyma. Similar internal lesions were found in several Chinook salmon held in separate cages on site however the mortality rate remained unchanged.

a disease of farmed Chilean coho salmon associated with a Rickettsia-like organism (1,2,3). This condition has not been previously reported from British Columbia. A similar, probably identical condition, has been recognized in B.C. since 1970 in pink salmon cultured in seawater tanks for experimental purposes, and farmed coho and chinook salmon in the 1980's. Rickettsial infections have also been observed in crustacea (4) and Pacific coast molluscs (5). Chilean workers (2) suggest the organism may originate from a local marine source with severe stress or inadequate nutrition as necessary contributing factors.

In This Issue:

Rickettsia

Scallops

Pseudomonas

BKD
Confusion

Vibrio

IHNV Brook
Trout

Kudoa

Hexamita

1. Cvitanich JD, Garate O and Smith CE. The isolation of a Rickettsia-like organism causing disease and mortality in Chilean salmonids and its confirmation by Koch's postulate. *J. of Fish Dis* 1991; 14:121-145.
2. Branson EJ and Nieto Diaz-Munoz D. Description of a new disease condition occurring in farmed Coho salmon, *Oncorhynchus kisutch* (Walbaum), in South America. *J of Fish Dis* 1991; 14:147-156.
3. Fryer JL, Lannan CN, Garces LH, Larenas JJ and Smith PA. Isolation of a Rickettsiales-like organisms from diseased Coho salmon (*O. kisutch*) in Chile. *Fish path* 1990; 25(2): 107-114.
4. Sparks AK. Synopsis of invertebrate pathology exclusive of insects. Elsevier Science Publishers B.V., Amsterdam, Oxford, New York, 1985.
5. Figueras AJ. Mortalities, parasites and diseases of mussels (*Mytilus edulis* and *Mytilus galloprovincialis*) from natural and cultured populations. *Bull Aquacul Assoc Canada* 1991; 2: 54-62.

Morphologic histopathological changes included marked necrosis and pyogranulomatous inflammation in liver, kidney, spleen, heart, skeletal muscle, and meninges. Vasculitis and thromboses were found in association with multifocal hepatic infarctions. Small, slightly pleomorphic basophilic intracellular inclusions were found singly or in large numbers within macrophage cytoplasm. These inclusions were negative with Gram, acid-fast, PAS and Macchiavello's stain. The inclusions stained blue with Toluidine Blue and were equivocal on Giemsa stain.

Clinical signs and post mortem findings are consistent with

Epidemic of *Pseudoklossia* sp. (Apicomplexa) in Bay Scallops *Argopecten irradians* Maintained in Warm Water Recirculating Facility

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After transfer of 500 juvenile bay scallops *Argopecten irradians* (shell height 20 mm), from the Prince Edward Island Department of Fisheries and Aquaculture shellfish hatchery, to quarantine research facilities at the Atlantic Veterinary College, an epidemic of *Pseudoklossia* sp. (Apicomplexa) occurred. Bay scallops were divided equally among 13 pearl nets in a six tank warm-water recirculating system containing 2300 L of 28 ‰ artificial saltwater. Water was maintained at 15 °C, dissolved oxygen at 90% saturation, and ammonia-nitrogen at less than 0.2 mg/l. Bay scallops were batch fed algae. Mortalities within the bay scallop population occurred beginning two days post introduction (PI) and continued throughout the 45 day period PI. Mortalities totalled 226 individuals (45% of the population). Highest mortality was observed 5-11 days PI with 150 mortalities (66% of the total mortalities) occurring. Histological and ultrastructural examination of bay scallop tissues revealed developmental stages of *Pseudoklossia* sp. (Apicomplexa) in all organs of all scallops. The initiating cause of the epidemic was not apparent. The parasite persists at low intensity and high prevalence in our bay scallop population.

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Pseudomonas Isolate Gives Positive Direct Fluorescent Antibody Test Using *Renibacterium salmoninarum* Antisera

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A bacterium, identified as *Pseudomonas fluorescens*, gave a positive reaction in a direct fluorescent antibody test (DFAT) using fluorescein isothiocyanate (FITC) conjugated goat anti-*Renibacterium salmoninarum* antiserum. The bacterium was isolated from coelomic fluid of wild broodstock rainbow trout (*Oncorhynchus mykiss*) obtained June 17, 1991 from Marlette Lake, Nevada. Cells reacted with conjugated antisera obtained from the Immunobiologics (IB) Section of the National Fish Health Research Laboratory, Kearneysville, West Virginia (Lot 181-022) and from a commercially available (CA) source. Both antisera were prepared using whole cell antigens; the CA antisera was affinity purified.

Coelomic fluid, in three fish pools, was collected from 150 fish. Slides for DFAT were prepared by centrifuging 150 µl of the pooled sample at 10,000 X g for 10 min; the supernatant decanted; and smears made of the pellet. Slides were air dried, heat fixed, immersed in xylene for 2 min, then air dried. Staining was done in a moist chamber for 35 min; slides were rinsed and washed for 1 min in phosphate buffered saline (PBS, pH 7.2) and counterstained for 1 min using 1:10,000 Eriochrome black T in PBS. Slides were then washed in PBS, air dried, and mounted using 1,4-diazabicyclo-(2,2,2)-octane in glycerol (pH 9.0). Fifty fields were examined using a Zeiss epifluorescent microscope (1,000 X).

Conjugates were diluted (CA: 1:30; IB: 1:40) and filter sterilized (0.2 µm). Control slides of *R. salmoninarum* cells were DFAT positive. Unstained *P. fluorescens* cells did not autofluoresce. Intensely fluorescent cells (ca. 0.5 x 1.5 µm) were observed in 4 of 50 pooled samples. Each pooled sample was cultured on brain heart infusion agar by dropping a 100 µl volume and incubating at 16 °C. Pure cultures of *P. fluorescens* were recovered from two of the four coelomic fluid pools that were DFAT positive. Biochemical identification of the *P. fluorescens* isolates was made with the following criteria: K/N on triple sugar iron agar; motile; oxidase positive and growth on MacConkey agar. Positive reactions were recorded with the following tests: nitrate reduction (no gas), Simmons citrate, gelatin liquefaction, arginine decarboxylase, pyoverdinin and growth at 4 °C. Negative tests were: indole, urea, phenylalanine deaminase, esculin, lysine and ornithine decarboxylase, malonate, pyocyanin, and growth on medium containing acetamide. The following sugars were oxidized: glucose, fructose, rhamnose, xylose, mannose, mannitol, trehalose, galactose, arabinose, and sorbitol. The following sugars were negative: maltose, melibiose, raffinose, dulcitol, sucrose, salacin, cellibiose, sorbose, and lactose.

Although *P. fluorescens* cells are generally larger than those of *R. salmoninarum*, false positives may arise due to

fragmentation of *P. fluorescens* or the plane at which the cell is observed. It may be possible that other serological tests such as those used for screening purposes or diagnostics may encounter the same problem if an antisera with similar activity is used.

Vibrio damsela Strain Virulence for Fish and Mammals

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Vibrio damsela is a common marine bacterium associated with wound infections in humans and occasionally this opportunistic pathogen has been reported to cause disease in fish. Reported fish infections include damselfish (Love, Fisher, Hose, Farmer, Hickman and Fanning, 1981. Science 214:1140-1141), brown shark, yellow tail, turbot (Fouz, Larsen and Toranzo, 1991. Bull. Eur. Assoc. Fish Pathol. 11:80-81) and seabream (Vera, Navas and Fouz, 1991. Bull. Eur. Assoc. Fish Pathol. 11:112-113).

Little information exists on the host-specificity of *V. damsela*. Our objective was to examine the different susceptibility of cold and warm water fish species as well as of mammals to infection with this pathogen. For this purpose, several reference strains and isolates of *V. damsela* implicated in disease problems in mariculture were tested (Table 1).

To assess the degree of virulence for fish, strains were tested for pathogenicity in rainbow trout (8 g), turbot (10 g), eel (12 g) and striped bass (8 g). Furthermore, a mouse virulence assay with BALB/C mice (4 to 5 weeks old) was performed to evaluate bacterial virulence to a homeothermic animal.

Pathogenicity trials were conducted by intraperitoneal inoculation of 10^2 to 10^8 bacterial cells per animal (6 to 10 animals per dose) as previously described (Toranzo, Barja, Potter, Colwell, Hetrick and Crosa, 1983. Infect. Immun. 39:1220-1227). Fish mortalities were recorded daily for 14 days and the 50% mean lethal dose (LD_{50}) was calculated by the Reed & Muench method (1938). In the case of mammals, strains displaying an $LD_{50} < 10^7$ in a 7-day period were considered virulent (Daily et al, 1981. J. Clin. Microbiol. 13:769-777).

The virulence assays demonstrated that *V. damsela* strains isolated both from turbot and humans were highly pathogenic for all species challenged displaying an LD_{50} ranging from 1×10^3 to 7×10^6 live cells. In addition, the inoculated strains were recovered from all the surviving fish, which suggested that the bacteria can establish a carrier state

Table 1. Origin and degree of virulence for different host species of *Vibrio damsela* strains.

Strain	Origin		LD_{50}^a for fish				LD_{50} for Mice
	Host	Country	Turbot	Rainbow Trout	Eel	Striped Bass	
RG-91	Turbot	Spain	1×10^3	8×10^3	5×10^4	3×10^4	1×10^6
RG-191	Turbot	Spain	5×10^3	9×10^3	7×10^4	4.1×10^4	1.3×10^6
RG-214	Turbot	Spain	8×10^3	1×10^4	9×10^4	8×10^4	3×10^6
RM-71	Turbot	Spain	8×10^4	1×10^4	7×10^6	9×10^4	2.8×10^6
RI-162	Turbot	Spain	9×10^4	1×10^4	NT	NT	1.1×10^6
LD-07	Seabream	Spain	1×10^4	5×10^4	$> 10^8$	7×10^6	$> 2 \times 10^7$
ATCC 33539	Blacksmith	USA	6×10^3	1×10^4	3×10^4	2.2×10^4	$> 3.3 \times 10^7$
ATCC 35083	Brown shark	USA	$> 10^8$	$> 10^8$	NT	$> 10^8$	$> 3 \times 10^7$
CDC-2227-81	Humans	USA	3×10^5	1×10^4	4×10^6	4.1×10^6	2.5×10^6

^a, 50% mean lethal dose; NT, not tested.

in fish. Other isolates presented some differences in virulence: strain ATCC 33539 was pathogenic for fish but not for mice and strain LD-07 was not pathogenic either for eel or mice. *V. damsela* ATCC 35083 (isolated from brown shark) was the only strain non-virulent for the poikilothermic and homeothermic animals challenged (Table 1).

Interestingly, rainbow trout proved to be highly susceptible to vibriosis caused by *V. damsela* isolated from different sources under laboratory conditions with LD₅₀ ranging from 8×10^3 to 8×10^4 live cells.

Although some strains seem to possess host specificity, such as *V. damsela* ATCC 33539 (virulent only for poikilothermic animals), most of them were highly pathogenic for cold and warm water fishes, and mammals.

All these findings suggest that *V. damsela* has a host range broader than that observed by Love et al. (1981). Furthermore, although *V. damsela* has been more frequently associated with human wound infections than with fish diseases, its potential importance in salt water aquaculture should be recognized.

Susceptibility of Brook Trout *Salvelinus fontinalis* to Infectious Hematopoietic Necrosis Virus

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In January 1991, an epizootic of infectious hematopoietic necrosis (IHN) occurred at Summerland Trout Hatchery, British Columbia. The epizootic was detected in F1 native rainbow trout held in outdoor rearing ponds. At that time six week old brook trout fry were also on-hand inside the hatchery, however no unusual mortalities occurred and IHNV was not isolated from these production fish either during random sampling or repeated testing of mortalities. The questions then arose as to the status of the brook trout. Had they been infected, but showed no mortalities due to inherent resistance? Since there was no information on the susceptibility of brook trout to IHNV in the literature, a rudimentary bath-challenge was run. If found susceptible, it would be reasonable to assume that the production brook trout had not been infected and could be released.

IHNV isolated from the Summerland January 1991 epizootic and serotyped by Dr. M. Engelking and Dr. J. Leong as type 1, was used for the challenge. A single group of 300 two month old brook trout from Summerland Trout Hatchery were exposed to 4×10^4 PFU/ml for two hours at 4 °C and then transferred to freshwater holding at the Pacific Biological Station quarantine unit. Fish were maintained on chlorinated/dechlorinated city water for 61 days post-challenge. Water temperature on day one was 5 °C and rose gradually to 10.5 °C at termination of the experiment. Mortalities were frozen at -70 °C and later tested individually for IHNV. Moribund fish were fixed, sectioned and examined for evidence of histopathology characteristic of IHN. On termination, a random sample of 60 survivors were tested for IHNV (tested in pools of 5 fish).

Combined mortality and morbidity post-challenge was 6.7%. Losses started 18 days post-challenge and continued sporadically until the 45th day. No mortalities occurred between day 46 and 62. IHNV was confirmed via DFAT (monoclonal antisera, Dr. S. Ristow) in 93% of mortalities assayed (n=14). Marked necrosis of renal interstitial tissue was found in histological sections of moribund fish. IHNV was also confirmed in 1/12 pools of survivors tested at the termination of the experiment.

These results indicate that brook trout are susceptible to IHNV. Unfortunately due to the somewhat advanced age of the fish (two months) and the low water temperatures at the beginning at the holding period (5 °C), the degree of susceptibility under optimum experimental conditions was not determined. The fact that brook trout did succumb under such unfavorable circumstances (particularly given the low water temperatures) may indicate a fair degree of susceptibility. Had the water temperature been optimum for IHNV (10-12 °C) and the fish younger, it is likely that losses would have been higher. The detection of IHNV in asymptomatic (four month old) survivors was unexpected.

Kudoa thyrssites (Myxosporea) and Soft Flesh in Pen-reared Coho Salmon

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The muscle-infecting myxosporean parasite *Kudoa thyrssites* has a wide range of fish hosts on Canada's west coast (Z. Kabata and D.J. Whitaker, Can. J. Zool. 59:2085-2091, 1981; Z. Kabata and D.J. Whitaker, Can. Tech. Rep. Fish. Aq. Sci., No. 1303, 1984). The parasite infects seawater pen-reared Atlantic salmon *Salmo salar*, and has been associated with

morbidity (L.W. Harrell and T.M. Scott, J. Fish Dis., 8:329-332, 1985) and soft flesh (D.J. Whitaker and Michael L. Kent, J. Aquat. Anim. Health, in press). In the first cases of *K. thyrssites* in Pacific salmon the parasite was detected in the cardiac muscle of several *Oncorhynchus* spp., including coho, (*O. kisutch*), that were returning from the sea to spawn (Z. Kabata and D.J. Whitaker, Can. J. Zool., 67:341-342, 1989).

In September, 1991 a sample of pen-reared coho (2-3 kg), that had soft, mushy flesh, was brought to the Pacific Biological Station for examination. Spores of *K. thyrssites* were found in the musculature. A sample of post-smolt coho (approx. 150 g) from the same farm was also examined in September. These fish had been put into sea pens about four months earlier. Two of the 50 fish sampled were infected. In January, 1992, a further sample of these fish was examined and 60 % (31/51) were found to be infected. It has yet to be determined whether this infection of coho salmon was an isolated, unusual occurrence or whether it is an indication that *K. thyrssites* has the potential to become a problem in pen-reared Pacific salmon. At present, there have been no reports of *Kudoa* associated morbidity or soft flesh in wild Pacific salmon.

Transmission of a Systemic Diplomonad Flagellate of Chinook Salmon Reared in Sea Water

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A systemic infection by a diplomonad flagellate protozoan that is morphologically indistinguishable from *Hexamita salmonis* has caused high mortality in seawater pen-reared chinook salmon (*Oncorhynchus tshawytscha*) in British Columbia (Kent, M.L. and J. Ellis. 1991. AFS/FHS Newslett. 19(4):2). A similar infection has been observed in pen-reared Atlantic salmon (*Salmo salar*) in Norway (Mo, T.A., T.T. Poppe, and L. Iversen. 1990. Bull. Europ. Asso. Fish. Pathol. 10(3):69-70).

Transmissibility of the flagellate from British Columbia was assessed by waterborne exposure of chinook salmon (avg. wt 150 g) to infected tissue and ascites (henceforth referred to as waterborne exposure), and by cohabitation with infected

fish. These experiments were conducted in seawater and freshwater tanks at 10 and 8 °C, respectively. In addition, the susceptibility of Atlantic (avg. wt. 75 g) and chinook salmon was assessed by the intubation of infected ascites. The intubated fishes were held in sea water as described above.

The systemic flagellate was transmitted to chinook by intubation in sea water, and by waterborne exposure and cohabitation in both sea water and fresh water. Experimentally infected chinook in all groups exhibited intense infections in the blood, and identical gross and histological changes as those seen in chinook from the netpen site (anemia, bloody ascites, and enlargement of the liver, kidney, and spleen). The infection was first observed in the waterborne-exposure group in fresh water by 10 days, in the waterborne-exposure group in sea water by 14 days, and in the intubated group by 21 days. In contrast, none of the Atlantic salmon exhibited the infection 35 days after exposure.

Our transmission experiments demonstrated that the parasite and the associated disease is transmitted directly from fish to fish in fresh water and sea water. In these experiments, previously healthy (and presumably immunocompetent) chinook developed the systemic form of the infection, exhibited the typical pathological changes observed in the field, and ultimately died from the infection. This suggests that the parasite is either a new, highly invasive strain of *H. salmonis* or a different species because *H. salmonis* is normally confined to the gastrointestinal tract of salmonids reared in fresh water. This diplomonad parasite may also represent a different strain or species from the *Hexamita* like parasite that causes systemic infections in pen-reared Atlantic salmon in Norway because we were unable to establish experimental infections in Atlantic salmon using the chinook parasite.

Report on Furunculosis Workshop January 30-31, 1992

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B.C. Ministry of Agriculture, Fisheries and Food

Furunculosis has had severe impacts on the Norwegian and Scottish salmon farming industries. The causative bacterium, *Aeromonas salmonicida*, is global in distribution, and occurs throughout Canada. This disease has at times been a problem in Canada, primarily in freshwater farming or government hatchery operations. In Scotland and Norway it has "become" a significant problem in saltwater.

In British Columbia, cases of furunculosis have fortunately been relatively few. Marine occurrence is not common, but

furunculosis could become a major factor in B.C. salmon farming if it is ignored and steps are not taken to understand and manage the disease.

The British Columbia Ministry of Agriculture, Fisheries and Food sponsored and organized an international furunculosis workshop held in Richmond, B.C. on January 30 and 31, 1992. It was designed to familiarize salmon farmers with the bacterium, the disease, possible preventative measures (vaccines, husbandry practices) and drug therapy. Over half of 130 attendants were employees of fish farms. The remainder represented the academic community, vaccine and pharmaceutical suppliers and government scientists and regulators. Visitors came from eight Canadian provinces, four American states, France and Scotland.

Aeromonas salmonicida is an efficient bacterial invader with an assortment of strategies to infect the fish and resist its defenses. This bacterium readily exists in a carrier state, although the exact location is unknown. Several speakers postulated that it is a common resident among the intestinal flora.

Fish coexist readily with *A. salmonicida* and do not develop furunculosis, but overcrowding, poor water quality, rough handling, poor grading, and inappropriate feeding strategies will bring on disease. Vaccination will help to carry the fish through these difficult times, although we were surprised to learn that much of the protective effect may come from stimulation of non-specific immunity by vaccine adjuvants. Treatments are restricted because of a lack of approved drugs for use on food fish. In fact, access to a most promising drug, oxolinic Acid, is not permitted even for experimental use by the Canadian Bureau of Veterinary Drugs. Canadian regulators require submissions from pharmaceutical companies proving the safety and efficacy of their products, while companies note that the Canadian aquaculture market is too small for sales to cover the costs of these studies.

Once again, the emphasis returns to "management" as the most effective means of disease control. Yet this term is particularly nebulous, and farmers ask "what in particular needs to be changed about a site's management to prevent furunculosis?" Certainly management will not control an outbreak of furunculosis already in progress. There was considerable support for the Cooperative Assessment of Salmon Health (CASH) program coordinated by Dr. Grace Karreman. This is one project that will allow us to understand site management as a series of production goals, and help to identify factors that may indicate increased disease susceptibility before an outbreak occurs.

Meeting proceedings will be produced as the Bulletin of the Aquaculture Association of Canada, March issue, to make the presentations available to a wide audience. Interested readers should contact the Aquaculture Association of Canada at the following address to obtain a copy.

Aquaculture Association
P.O. Box 1987
St. Andrews N.B.
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New Journal "Ecological Parasitology"

The first volume of "Ecological Parasitology" has been published through the efforts of Drs. O. N. Bauer, E.P. Ieshko and O.N. Pugachev editors for the journal. All articles are in English. Articles are accepted from scientists from all countries.

Contents for volume 1, Number 1 are as follows:

O.N. Bauer and Yu I. Polyanski. Ecological Parasitology: present state and perspectives.

I.D. Trombitsky, A. Ja. Moshu and A. V. Bordenjuk. *Ambiphrya ameirui* (Ciliophora, Scyphidiidae), its hosts specificity and ecology in Europe.

E.P. Ieshko and L.V. Anikieva. Life tables of fish helminths and their analysis with *Proteocephalus percae* (Cestoda, Proteocephalidae), a specific parasite of the perch, *Perca fluviatilis*, taken as an example.

J.F. Sovenyi and K. Molnar. Studies on the survival of *Sinoichthyonema amuri* (Garkavia, 1972) transplanted into homologous and heterologous fish hosts.

A.N. Alekseev. Ecology of tick-borne encephalitis virus: part of Ixodidae ticks males in its circulation.

A.N. Ryss. Plant parasitic nematodes found in permafrost and in plant communities above it.

A.A. Philipchenko. Ecological conception of parasitism and independency of parasitology as a branch of science.

O.N. Bauer. Ecology and morphology of helminths of Kazakhstan animals. (Ed. E.G.Sidorov). A review

Yu. A. Strelkov. Parasites and diseases of hydrobionths of the Arctic province. (Ed. O.N. Bauer and N. M. Pronin). A review.

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"Typed manuscripts to be published in--*Ecological Parasitology*--should be written in English with a Russian summary (or prepared for Russian translation) of not more than two typed pages. Two copies should be sent to the Secretary of the Journal, Dr. A.K. Galkin using the address: 199034, USSR, Leningrad, Zoological Institute, Department of Parasitic Worms."

-submitted by Richard A. Heckman, Dept. Zoology, Brigham Young University, Provo, Utah 84602

Translation Available

Schaperclaus, W., editor. 1991. *Fish Diseases*. Published for the U.S. Department of Interior and the National Science Foundation by Amerind Publishing Co., Pvt. Ltd., New Delhi, India. 2 Vols.

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-submitted by Drew Mitchell, Fish Farming Experimental Lab., P.O. Box 860, Stuttgart, AR 72160-0860

Third International Symposium for Ichthyoparasitology "Problems of Fish Parasitology" Petrozavodsk, USSR

The third International Symposium, "Problems of Fish Parasitology", for Ichthyoparasitology was held at Petrozavodsk, Karelia, USSR (Commonwealth) during August 14-21, 1991. Scientists from several countries were present to listen to research results and conduct informal conversations with colleagues. The compendium of abstracts contains 102 titles from presentations. There were a wealth of ecological studies among the printed abstracts as well as host - parasite interactions. Anyone interested in the abstracts should write to: Dr. Richard A. Heckmann, Dept. of Zoology, Brigham Young University, Provo, Utah 84602

Editorial

Seafood safety issues continue to be a significant concern to various consumer groups, federal agencies and congressional committees. Divergent views about the safety of seafoods and the effectiveness of various government agencies to ensure seafood safety have been widely reported, and distorted. This has created some confusion among consumers. Concerns center around microbial contaminants and violative or otherwise unsafe chemical residues in consumed seafoods. Considerable evidence suggests raw seafood consumption, particularly of bivalve molluscs, accounts for the bulk of reported seafood borne illnesses. While there is considerable debate as to the safety of chemical residues (e.g. pesticide and heavy metal), certain fishes may indeed contain various chemical residues obtained from the environment.

There is no reported evidence that violative or otherwise unsafe therapeutic residues have occurred in aquacultured products produced in North America. It is widely assumed that this has occurred because the compounds used by aquaculturists are safe and used properly according to label instructions. Another possibility is that little regulatory attention has been placed on aquacultured products. It is clear that regulatory neglect is no longer an option. The FDA has instituted a thorough examination of aquaculture, particularly the use of fishery compounds as therapeutants. FDA is working with the Joint Subcommittee on Aquaculture (JSA) to address various therapeutic issues. The JSA Working Group on Quality Assurance for Aquaculture is the primary aquaculture force addressing these issues.

It is clear that Quality Assurance Programs (QAP) can be a valuable method to ensure safe therapeutic and chemical use in public and commercial aquaculture. A QAP helps ensure that compounds are used according to label instructions, that chemical cross-contamination does not occur, that appropriate withdrawal times are always followed and that the environment is well protected. The Working Group on Aquaculture Quality Assurance may provide some future guidance in therapeutic QAPs. As fish health professionals, we in the FHS have a major responsibility to participate in QAP development, implementation and monitoring. Fish health professionals must use compounds according to label. When we become AFS/FHS certified Fish Pathologists we agree to use therapeutants "within legal constraints..." FDA and EPA clarification of fishery compound use will help delineate what is legal. As fish health professionals we are most familiar with fish diseases, the effect of various compounds on fish and usually have the greatest influence over production practices. This places great responsibility on us but also creates great opportunity. We must be proactive in the aquaculture community. If we do not actively participate, as a section and as individual fish health professionals, we may be excluded from the process.- Randy MacMillan, editor.

Skinner Memorial Award- 1992

The John E. Skinner Memorial Fund was established in memory of John Skinner, former Chapter and Western Division American Fisheries Society president. The Fund provides monetary travel awards for deserving graduate students or exceptional undergraduate students to attend the annual American Fisheries Society meeting. The 1992 meeting will be held in Rapid City, South Dakota, 13-17 September.

Awardees are chosen by a committee of the AFS Education Section. Selection is based on academic qualifications, professional services and promise, and reasons for wishing to attend the annual meeting. We are making a special effort to distribute announcements for this award widely, to attract eligible women and minorities to apply.

Travel support not to exceed \$650 per award will be made available to successful applicants. We anticipate selecting at least six recipients from among the pool of applicants.

Please request the revised 1992 application form for the award from: Christine M. Moffitt, Chair Skinner Awards, Dept. Fish and Wildlife Resources, University of Idaho, Moscow, ID 83843

EEO Section Established

The AFS has formed the Equal Opportunities Section to help promote women and minorities in fisheries. Interested individuals should contact: Dr. Mary Fabrizio, U.S. FWS/NFRC-Great Lakes, 1451 Green Road, Ann Arbor, MI 48105.

Meetings

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.

2nd International Colloquium: Microbiology in Poikilotherms. Budapest, Hungary, June 15-17, 1992. Contact: Intercongress LTD, Budapest, Doza Gyorgy ut 84/a H-1068, Hungary.

1992 World Congress on Cell and Tissue Culture. Washington, DC, June 20-25, 1992. "Genetic Applications of Tissue Culture." For information contact Aaron Rosenfield, NOAA/NMFS, Oxford Laboratory, 904 S. Morris Street, Oxford, Maryland 21654. Phone 301-226-5193.

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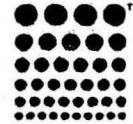
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