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Plasmacytoid Leukemia (Marine Anemia): Viral or Microsporidian Etiology?

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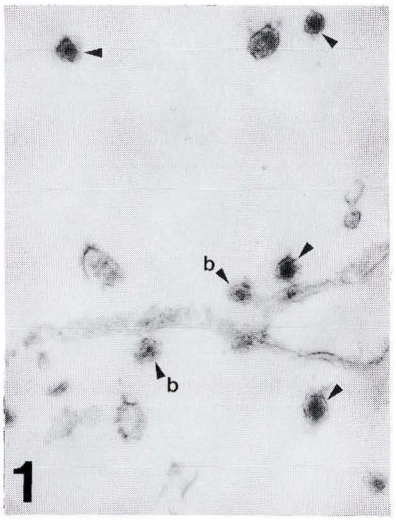
A plasmacytoid leukemia (PL) of salmon, referred to as marine anemia by fish farmers, has caused high mortality at many seawater netpen farms in British Columbia (Kent et al. 1990. Dis. Aquat. Org. 8:199-209; Newbound and Kent. 1991 Dis. Aquat. Org. in press). The disease is characterized by massive infiltration of immature plasma cells (plasmablasts) in the visceral organs and retrobulbar tissue.

The etiology of PL has not been determined, but field observations and laboratory transmission studies (Kent and Dawe. 1990. Cancer Res. (Suppl) 50: 5679-5681; Newbound and Kent. 1991. AFS/Fish Health Newsletter 19(1):1-2) clearly indicate that the disease is caused by an infectious agent. At present, there are two likely candidates for the cause of PL; a microsporidian parasite, *Enterocytozoon salmonis* (Chilmonczyk, Cox and Hedrick 1991), and an oncogenic virus (e.g. retrovirus).

Evidence for Microsporidian Etiology. The microsporidium has been observed in the nuclei of the plasmablasts from many fish with PL in BC. In addition, the parasite was consistently associated with a similar, if not identical, disease in freshwater-reared chinook in Washington and California (Morrison, et al. 1990. Dis Aquat. Org. 8: 99-104; Hedrick et al. 1990. Dis. Aquat. Org. 8: 189-197). Hedrick et al. (1991 Dis Aquat. Org. in press) showed that the disease in California could be controlled by oral treatment with Fumagillin DCH, a drug that is efficacious for microsporidian diseases of fish and other animals. Some aspects of our transmission studies using fish with PL support the microsporidium theory. Whereas transmission of PL readily occurred in fish injected with tissue homogenates, transmission with cell-free filtrates has been equivocal at best, which suggests that the agent is not smaller than 0.22 μm (e.g. a virus).

Evidence for a Viral Etiology. Probably the best evidence that *E. salmonis* is not the primary cause of PL also comes from our transmission experiments. We have transmitted PL by injection of tissue homogenates collected from affected chinook with severe PL that had no evidence of the *E. salmonis* infection. Essentially 100% of the recipient chinook developed PL, and the parasite was not detected in any of these fish. We have repeated this experiment 15 times, with groups of 15-30 fish/experiment. In addition, some of these transmission studies were conducted by second passage (using the recipient fish as donor fish). Some have contended that our experimentally PL-affected fish were infected with a cryptic stage of the parasite. However, this is unlikely considering the number of experimentally affected fish that showed no indication of the parasite. Furthermore, as observed in transmission studies conducted by Hedrick and



co-workers, when we used donor tissue that was infected with the microsporidium, the recipient fish exhibited the parasite infection as well as PL.

Some field observations also suggest that the parasite is not the primary cause for the disease. A disease histologically identical to PL was first described in freshwater-reared chinook in Washington (Harshbarger. 1984. Nat. Cancer Inst. Monogr. 65: 251-273), and after review of paraffin slides from these fish we found no evidence of the microsporidium. Conversely, *Enterocytozoon salmonis* was first reported from pen-reared chinook in Washington, where the parasite was associated with anemia but no signs of PL (Elston et al. 1987. J. Protozool. 24: 274-277).

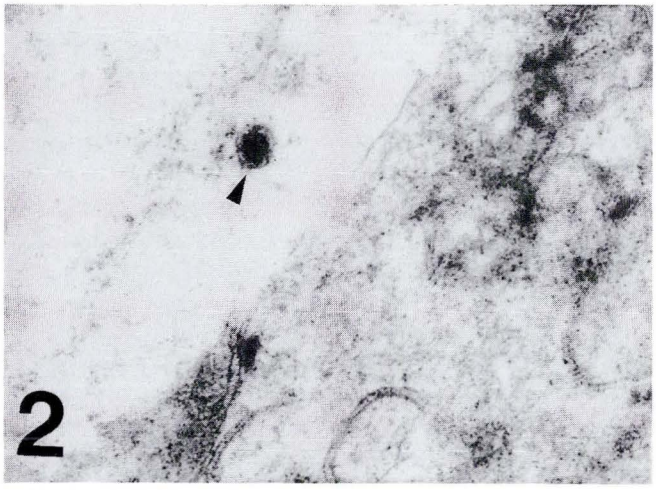


Figure 1, 2. Electron micrographs of virus-like particles (arrowheads) in intercellular spaces of retrobulbar tissue from a chinook salmon with plasmacytoid leukemia. B = possible budding viruses. x 57,000.

If the microsporidium is not the cause of PL, then the most likely cause is an oncogenic virus. Essentially all infectious leukemias and related disorders are caused by oncogenic viruses and retroviruses have been associated with some hemic neoplasms of fishes (Papas et al. 1976. Nature 261: 506-508; Gross. 1983. Oncogenic Viruses., 3rd Ed. Pergamon Press, Elmsford, New York). Our inability to transmit PL using cell-free filtrates does not preclude a viral etiology for the disease because it is well recognized that many retroviruses are cell associated and are difficult to transmit with this technique. Furthermore, we have recently detected virus-like particles associated with the plasmablasts in PL-affected fish (Fig. 1 & 2). The particles are suggestive of retroviruses by their size (105-120 nm), their intercellular location, and possible budding from cell membranes.

Is *E. salmonis* a co-factor in PL. Non-viral agents, including parasites, can be co-factors or promoters for virus-induced neoplasms. For example, malaria is an important co-factor in Burkitt's lymphoma in humans (Burkitt. 1969. J. Natl. Cancer Inst. 42: 19-28) and malaria promotes a viral-induced lymphoma of mice (Wedderburn. 1970. Lancet 2: 114-116.). A radiation-induced leukemia in mice is the result of activation, by radiation energy, of a latent retrovirus. Once activated, this virus, and the resultant leukemia, can be transmitted with cell-free filtrates in the absence of radiation (Gross. 1983. Oncogenic Viruses., 3rd Ed. Pergamon Press, Elmsford, New York). Similar results have been achieved with chemically induced leukemias in mice, which has lead Gross (1983) to speculate that the underlying cause of all hemic neoplasms may be oncogenic viruses. Possibly, *E. salmonis* plays the same role as a triggering cofactor in PL as radiation does in the mouse-leukemia model.

Assuming the disease of chinook in California is the same disease as PL, the control of the disease with Fumagillin DCH supports the theory that the parasite is an important co-factor, not just a secondary invader in fish that were immunocompromised by PL. Interestingly, the only other member of the genus *Enterocytozoon*, *E. bieneusi*, is associated with a retroviral disease, AIDS in humans (Desportes et al. 1985. J. Protozool. 32: 250-253).

There are many unanswered questions about PL of chinook in BC and the similar disease of freshwater-reared chinook in California and Washington. For example, it is not known if the disease in the USA is actually the same as PL in BC, the primary cause of PL is yet to be determined, and the role of the microsporidium in PL is unclear. Although, at present, we favor the oncogenic virus hypothesis, the mere presence of virus-like particles in PL-affected fish is insufficient evidence to indict the virus as the cause of PL. A major emphasis of our research on PL will continue to be directed toward elucidating the etiology of the disease. In addition, needed studies on the epizootiology of the disease in BC are planned. It is not known, for example, if PL occurs in freshwater-reared chinook in BC, nor is it known how the disease is transmitted (horizontally in netpens, through a carrier state in smolts and/or vertically through gametes?). Even if *E. salmonis* is not the primary cause of PL, recent work on the efficacy of Fumagillin for controlling the disease in California is encouraging, and we are testing this compound for the control of PL in BC. Research on PL has been supported in part by the Ministry of Agriculture and Fisheries Commercial Fisheries and Aquaculture Branch, Province of British Columbia.

Incidence of Birnavirus in Cultured Turbot (*Scophthalmus maximus* L.) in Northwest Spain

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During 1989 and 1990 a study was carried out in order to evaluate the health status of cultured turbot (*Scophthalmus maximus*) in several farms in Galicia (NW of Spain).

Samples of kidney, spleen and brain were pooled from fish of the same stock and processed for virus isolation following standard virological procedures. The CHSE-214 and EPC cell lines were used for virus assays.

A total of 9 viruses were isolated from 267 samples analyzed. They were identified as Birnaviruses according to their physical and chemical characteristics and their RNA patterns in SDS PAGE. Interestingly, the majority of the viruses were detected in routine surveys from fish without disease signs which indicates that they were asymptomatic carriers of these viruses (Table 1).

Table 1. Incidence of Birnavirus isolated from cultured turbot (1989-1990).

Type of Sampling	Weight	Year 1989	1990
Diseased fish	Juveniles[a]	0/7*	1/4
	Ongrowing[b]	1/52	0/32
Routine survey	Juveniles	0/2	2/31
	Ongrowing	2/40	3/99

*No. positive samples/No. samples tested
[a]weight less than 20 g
[b]weight greater than 20 g

One of two positive samples isolated in the routine survey was from imported juveniles which were analyzed before their transfer to the farm. This shows the importance of controlling the movements of fish among countries to avoid the spread of viral diseases.

Although in Norway heavy mortalities of marine fish and shellfish cultures were associated with the presence of a new serotype of IPNV (N_1) (Christie et al., Arch. Virol. 103: 167-177, 1988; Mortensen et al., Bull. Eur. Ass. Fish Pathol., 10: 42-43, 1990), no mortalities were detected in Spain in the stocks of turbot in which the isolations of Birnaviruses were made. We conclude that so far, the Birnaviruses can not be considered as a threatening problem for turbot culture in Spain.

Studies are currently being conducted to determine the serological relationship of turbot Birnaviruses with other IPN-like viruses, and the pathogenicity of the present isolates to turbot and other fish species cultured in our area.

Antibiotic Resistance in *Edwardsiella ictaluri*

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Since 1986, the catfish industry has had two antibiotics, Terramycin and Romet-30, cleared for the treatment of bacterial diseases. As with any antibiotic, bacterial resistance is always a major concern. Taylor (1987) documented resistance to Terramycin and Romet-30 in strains of *Aeromonas* and *Plesiomonas*. At that time there was no apparent resistance to either antibiotic in strains of *Edwardsiella ictaluri*, the causative agent of enteric septicemia of catfish (ESC).

In November, 1990, a strain of *E. ictaluri* resistant to both Terramycin and Romet-30 was isolated from an ESC epidemic by the MCES Fish Diagnostic Lab, Stoneville, MS (Durborow, personal communication). A comprehensive screening program for antibiotic resistance in *E. ictaluri* was initiated at both MCES Fish Diagnostic Labs (Belzoni and Stoneville, MS). During 1990 an increasing incidence of antibiotic resistance was noted in *E. ictaluri* isolates (Table 1). Some isolates (60 cases) were resistant to both Terramycin and Romet-30, others were resistant to only Terramycin (12 cases) or to only Romet-30 (43 cases). The resistant strains of *E. ictaluri* were isolated from 32 different fish farms.

Table 1. Antibiotic resistance of *E. ictaluri* isolates in Mississippi during 1990.

		Resistant Isolates			
Lab	ESC/Total	TM	Romet 30	Both	Total
Belzoni	698/1456	7	16	41	64
Stoneville	325/894	5	27	19	51
Totals	1023/2350	12	43	60	115

The initial screening project selected only one representative *E. ictaluri* isolate from each case (1 to 3 fish from any given pond). In August 1990, the Belzoni Lab began screening all isolates from from all fish from a given case (>1 fish necropsied per pond). In these cases, twelve showed a mixed resistance pattern among *E. ictaluri* isolates (Table 2). In thirteen cases, all *E. ictaluri* isolates were Romet-30 resistant and in 129 cases, all *E. ictaluri* isolates were TM and Romet-30 sensitive.

Table 2. *E. ictaluri* antibiotic (TM and Romet) sensitivity patterns where multiple fish were necropsied from a pond.

Antibiotic Sensitivity Pattern	Number of Cases
Mixed Romet resistant only and sensitive to both	3
Mixed TM resistant only and sensitive to both	2
Mixed Romet resistant only and resistant to both	3
Mixed resistant only and sensitive to both	4
All isolates Romet resistant but sensitive to TM	3
All isolates resistant to both	10
All isolates sensitive to both	129

Variations of antibiotic sensitivity in *E. ictaluri* have made proper treatment recommendation more difficult and time consuming. It is now imperative that fish farmers have every ESC outbreak properly diagnosed and each *E. ictaluri* isolate tested for antibiotic sensitivity prior to selection of the appropriate medicated feed. Mixed sensitivities from the same pond further complicates treatment recommendations. While it appears that resistance factors in *E. ictaluri* are increasing, there is not enough data available at present to determine the extent or eventual impact this will have on the catfish industry.

Durborow, R.M. 1989. Personal communication, MCES Fish Diagnostic Lab, Stoneville, MS.

Taylor, P.W. 1987. Antibiotic Resistance to Romet-30 in Bacterial Infections of Catfish. AFS/FHS Newsletter Vol. 15(2):4.

Subunit Vaccine to IHNV Provides Protection In Sockeye Salmon Fry in Alasaka

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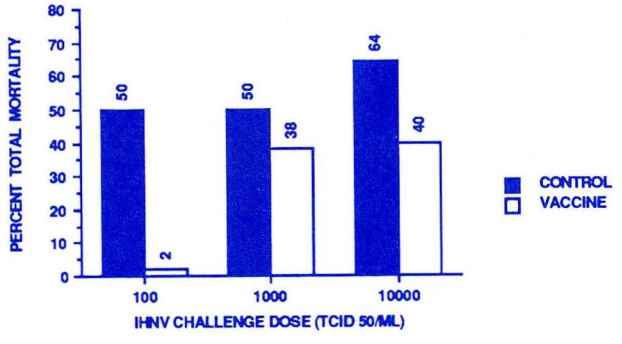
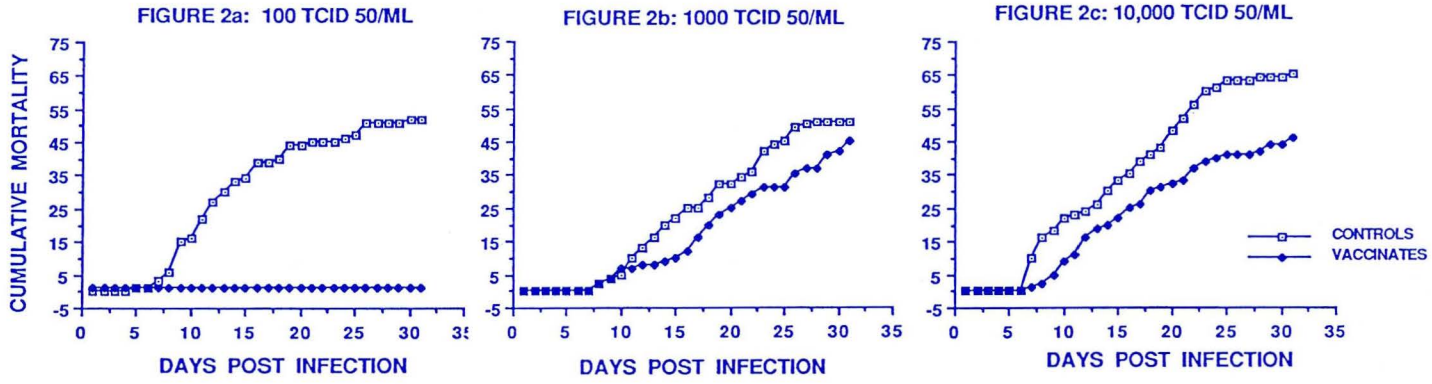
Infectious hematopoietic necrosis virus (IHNV) is endemic to all tested stocks of anadromous sockeye salmon (*Onchorhynchus nerka*) in Alaska and was a major cause of failure in the culture of this species (Meyers et al., 1990, J. Aq. Anim. Health 2:85-98). Since 1980, IHNV has been controlled by strict culture practices that has led to an IHNV induced mortality of 1 to 3.2% in 1990. Fish are transported and released very early in their life when the yolk is depleted or after approximately 3-6 weeks of rearing. If, however, the fish are reared for longer time periods, mortality is significant and the need for an IHNV vaccine is clear.

A prototype IHNV subunit vaccine was developed (Gilmore et al., 1988, Bio.Technology 6:295-300) and has been found to be effective against 3 different isolates of IHNV. The vaccine consisted of a cloned region of the IHNV glycoprotein gene which was expressed in *Escherichia coli*. A crude bacterial lysate containing the expressed viral protein was very effective as an immersion vaccine. Since that initial report, a larger portion of the IHNV glycoprotein gene was expressed in bacteria under the control of the *trp*E promoter in the plasmid, pXL3. This vaccine was found to protect rainbow trout (*O. mykiss*) fry against several different isolants of IHNV from Idaho and a summer steelhead trout (RB1) isolant from Round Butte, Oregon. Since the vaccine was very effective in rainbow trout laboratory trials, a laboratory test of the vaccine was made in sockeye salmon fry at the FRED Fish Pathology Lab in Anchorage, Alaska.

Crude bacterial lysates were prepared as described by Gilmore et al. (1990) and used to immunize fish by immersion. Approximately 300 sockeye salmon fry from Big

FIGURE 1: pXL3 VACCINE TRIAL





Lake Hatchery in Alaska were used for the control and immunized experimental groups. The average weight of each fish at the time of immunization was 0.5 g. Immunization by immersion was accomplished by bathing a group of 300 fry in 75 ml of the preparation (ca. 3 mg/ml total protein concentration) for 1 min. At that time, the immersion solution volume was increased to 750 ml with water and fish were kept in this diluted vaccine solution for an additional 4 min. These fish were then placed in aquaria of 5 gallons with a water flow rate of 0.25 gal/min at a constant water temperature of 10°C. Approximately 1 month after immunization, each fish group was subdivided into 50 fish groups and challenged with three doses of virulent IHNV. The IHNV in this case was the Packer's Creek, Alaska isolant of 1990. Each virus challenge level was done in replicate. The fish were maintained under static water conditions for 6 hours with light aeration of the water during the challenge.

The trial showed that the vaccine completely protected the sockeye salmon fry when these fish were challenged with a 0.0001 dilution of the Alaskan IHNV isolant. Pre-trail titration of the virus indicated that there were approximately 100 TCID50 units/ml of virus in 4 liters of water during the virus challenge period. When the virus challenge dose was raised ten- and hundred-fold, the vaccine was not as effective in protecting the fish (Figure 1). A daily comparison of the IHNV mortalities found in the vaccinated and control fish exhibited a longer incubation period before mortalities appeared and a lower final cumulative percent mortality (Figure 2).

Thus, we have shown that a recombinant DNA-based subunit vaccine to IHNV will protect sockeye salmon fry against a lethal challenge of IHNV-Packer's Creek isolant. The glycoprotein gene which was used in the construction of the vaccine was cloned from IHNV (RB1) isolated from a summer steelhead at Round Butte, Oregon in 1975. It is apparent that the cloned RB1 glycoprotein gene contains immunogenic epitopes which are conserved in the Packer's Creek, Alaska IHNV glycoprotein. The potential for using an IHNV subunit vaccine was demonstrated in these studies.

This report is the result of research sponsored by the Oregon Resource and Technology Development Corporation; Bonneville Power Administration Contract DE-A179-84BP16479, Project 84-43 (G.R. Bouck and R. Morinaka served as the Contracting Office Technical Representatives on the project); the United States Department of Agriculture to the Western Regional Aquaculture Consortium under grant nos. 87-CRSR-2-2319 and 88-38500-4027; and Oregon Sea Grant with funds from the National Oceanic and Atmospheric Administration, Office of Sea Grant, Department of Commerce, under grant no. NA85AA-D-SG095 (project no. R/FSD-11).

Virulence Comparison of IHNV from Spawning Salmonids

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Virulence differences among strains and isolates of infectious hematopoietic necrosis virus (IHNV) have been examined, but little work has been done to determine the variation among isolations made within a population of fish. Typically, a single viral isolate is assumed to be representative of all that are found in its host population. It was our objective to determine if virulence varied among isolates obtained from a population on the same day and if isolates collected from a single population at intervals throughout a spawning season differed.

Virulence is perhaps the least sensitive, but most important indicator of difference among strains of IHNV and we used it as an indicator of variance among the isolates examined. A more sensitive assay for measuring potential intra-strain differences is comparison by monoclonal antibody panels; however, when this study was undertaken an extensive panel did not exist. A future study will use monoclonal antibodies to examine the isolates described in this report.

Virus was collected from the ovarian fluid of seven chinook salmon on six distinct spawning days over the course of one month -- approximating early, mid and late spawnings -- during the winter 1989-1990 spawning cycle at the Oregon Department of Fish and Wildlife's, Elk River Hatchery. These viral isolates were used to measure the extent pathogenicity varied among isolates collected throughout a spawning season. Virus was also collected on the same day in the fall of 1988 from the ovarian fluid of five kokanee post-spawning on the headwaters of the Metolius River in central Oregon. These viral isolates were used to determine if isolates collected from a population of fish on the same day demonstrated a range of virulence. Stocks were made of each isolate and stored at -70°C until tested.

Juvenile kokanee (mean weight 1.2g) were challenged with the Metolius isolates and juvenile chinook (mean weight 1.6g) were challenged with Elk River isolates. Dilutions of the virus stocks were made in two liters of static water to give final concentrations of 10^5, 10^4, 10^3, and 10^2 plaque forming units (PFU)/mL. Each 12 hour exposure was done in triplicate with twenty-five fish. Water flow was resumed and mortality was monitored daily. The challenges were terminated when all tanks had not experienced any mortality for at least one week. Virus from dead fish was reisolated in tissue culture and confirmed by fluorescent antibody assay.

Table 1. Cumulative percent mortality in kokanne salmon produced by IHNV isolates collected on the same day.

	Exposure Concentration (PFU/mL)			
Isolate	10^5	10^4	10^3	10^2
1	100	87	72	59
2	86	65	58	50
3	96	95	77	43
4	100	94	82	53
5	96	81	74	44

Although lethal dose fifty-percent values could not be calculated for each isolate, in general, mortalities produced were equivalent and differences observed could be explained by standard error and titration effects.

Average cumulative percent mortality for the same day isolates (Table 1) showed a dose response relationship. Mortality was similar for each exposure concentration and were within one standard deviation of each other. Mortality produced by the isolates collected at intervals during a spawning season was low (Table 2). Again, a clustering of mortality was observed and differences were approximately within one standard deviation of each other. Isolates collected approximatley one month apart, December 15, 1989 and January 16, 1990 produced equivalent mortalities. Our observations suggest significant differences in virulence do not exist in isolates collected from separate fish at the same point in time nor among viral isolates collected during the course of a spawning season.

Table 2. Cumulative percent mortality produced in chinook salmon by IHNV isolates collected during a spawinging season.

	Exposure Concentration (PFU/mL)			
Isolate	10^5	10^4	10^3	10^2
12/15/89	19	21	4	11
12/20/89	10	7	7	1
1/ 3/90	15	9	4	0
1/ 8/90A	11	12	3	1
1/ 8/90B	10	17	1	3
1/12/90	19	12	11	1
1/16/90	12	5	0	2

We would like to thank John Kaufman of the Oregon Department of Fish and Wildlife for supplying data on the Elk River's IHNV history and the Bonneville Power Administration for funding this study.

Transmission of IHN Virus to Sockeye (*Oncorhynchus nerka*) and Atlantic Salmon (*Salmo salar*) in Sea Water

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Infectious hematopoietic necrosis (IHN) is a serious disease of Pacific salmon and steelhead trout. The susceptibility of sockeye salmon to IHN virus has been repeatedly demonstrated (Pilcher and Fryer, 1980. CRC Critical Reviews in Microbiology). Atlantic salmon fry have also been shown to be susceptible to IHN virus infection, which can result in high losses (Mulcahy and Wood, 1986. J. Fish Dis. 9:173-175). Extreme losses due to IHN tend to occur during the fresh water life cycyle phase of the of the host. Generally, as fish become older, resistance to infection by IHN virus increases. However, there have been documented IHNV infection occurrences among sockeye smolts and 2-year-old kokanee (Traxler, 1986. J. Fish Dis. 9:545-549), both in freshwater. Little information is available on the susceptibility of salmonids to IHN virus in sea water. In British Columbia, there is widespread netpen culture of Atlantic salmon in sea water, and sockeye salmon are now being farmed on an experimental basis. Sea water transmission experiments were conducted because of the concern that sockeye might serve as a source of IHN virus for other cultured

salmonids in netpens. To determine whether the virus is transmitted in sea water, Atlantic and sockeye salmon post-smolts were exposed to IHN virus while in sea water. Exposure was accomplished by bath and cohabitation methods, as outlined below.

Bath exposures of Atlantic and sockeye salmon were conducted by immersing 20 fish of each species in 9°C sea water containing IHN virus for 3 hr. The initial viral titer of the bath suspension was 8.98×10^3 pfu/mL; after 3 hr the titer had dropped to 2.70×10^3 pfu/mL. Each species was then placed in a separate tank supplied with flowing sea water at 9°C. Fish were fed and losses monitored daily for 8 weeks.

Transmission by cohabition in sea water was accomplished by holding Atlantic and sockeye salmon with fish that had been injected intraperitoneally with 1.6×10^3 pfu of IHN virus (47 uninfected fish / 10 infected fish). Fish were fin-clipped to denote the treatment received. They were held and fed for 8 weeks, and losses monitored daily.

Table 1. Susceptibility of Atlantic and sockeye salmon to IHN virus in sea water. All experiments were conducted in sea water at 9°C.

Challenge protocol	Cumulative mortality due to IHN-8 weeks	Mean viral titer pfu/g kidney tissue*
Bath		
Sockeye	0/20	0
Atlantic	2/20	7.1×10^4
Injection		
Sockeye	4/10	6.9×10^5
Atlantic	8/10	3.3×10^6
Cohabitation with injected sockeye		
Sockeye	0/25	0
Atlantic	2/22	1.2×10^6
Cohabitation with injected Atlantics		
Sockeye	1/25	1.6×10^3
Atlantic	6/25	1.2×10^6

*Dertermined for moribund and dead fish.

Atlantic salmon, maintained in sea water, were susceptible to IHN by the bath and cohabitation methods (see Table). Atlantic salmon proved to be more susceptible than sockeye salmon. The bath exposure of sockeye and Atlantic salmon to IHN virus resulted in no losses among the sockeye and a 10% loss in the Atlantic salmon. Interestingly, intraperitoneal injection of IHN virus also resulted in higher losses in the Atlantic salmon, thus supporting the results on susceptibility obtained by bath exposure. Mean viral titers measured by titration of kidney tissue were nearly 1 log higher in the Atlantics than in the sockeye.

Injected Atlantic salmon infected both Atlantic and sockeye salmon cohabiting with them, while injected sockeye transmitted the disease only to Atlantic salmon. This is the first reported case of transmission of IHN virus in sea water. The cohabitation results bear out the greater susceptibility of Atlantic salmon to IHN noted in the bath-exposed and injected fish. They also suggest that Atlantics produce, and presumably release, higher titers of the virus.

Affected fish were dark and lethargic, but showed no external hemorrhages or erythema. Infected Atlantics exhibited severe petechial and ecchymotic hemorrhaging in the viscera, pale livers, bloody ascites, and anemia. In contrast, internal signs in sockeye were restricted to anemia.

This study demonstrates that both Atlantic and sockeye salmon are susceptible to IHN virus in sea water by waterborne exposure, and indicates how important it is to avoid introducing fish infected with IHN virus to netpens. The marked susceptibility of Atlantic salmon to the virus, noted in this study, is of particular importance to the netpen aquaculture industry in B.C. because the industry is relying increasingly heavily on this species. To date, epizootics of IHN have not been reported in pen-reared salmon. However, because the gross signs observed were similar to those seen in Gram-negative septicemias (e.g. vibriosis), viral assays should be considered when conducting diagnostics on pen-reared fish.

Chinook salmon are also susceptible to IHN, and are presently the most widely farmed species in B.C. Additional exposure studies are planned to determine the susceptibility of chinook salmon to IHN virus in sea water.

1991 Mid-year Report of the Fish Health Section to the Executive Committee of the American Fisheries Society

During the 1990-1991 year the Section has continued its efforts in many established areas as well as undertaken some new initiatives. The Professional Standards Committee has now a formal set of criteria and guidelines as the basis for writing questions for the Fish Pathologists examination. Additionally, there are now chairpersons within each discipline who, along with other recognized authorities in the discipline, are preparing quotas of questions and 35-mm slides based on the new guidelines. The efforts are leading to a well-balanced, comprehensive examination that will test academic knowledge, problem-solving abilities, and proficiency.

The fourth edition of the Blue Book "Suggested Procedures for the Detection and Identification of Certain Finfish and Shellfish Pathogens," is nearing completion. In addition to finfish, this new edition includes a section on shellfish. Editor John Thoeson informed me that December 1, 1991 is a realistic date for publication.

The Sections Fourteenth Annual American Fisheries Society/Fish Health Section Meeting will be held in conjunction with the thirty second annual Western Fish Disease Conference in Newport, Oregon from August 1 through August 3, 1991. It will be coordinated with two other relevant meetings: the Second International Symposium on Viruses of Lower Vertebrates at Oregon State University in Corvallis, Oregon on July 28-31, 1991 and the Fifth International Congress for the Society of Developmental and Comparative Immunology at Reed College in Portland, Oregon from August 5-9, 1991. This should insure good attendance from our members as well as those from other countries.

A new Special Achievement Award has been recognized by the Fish Health Section beginning this year. This award will be given in addition to the S.F. Snieszko award and is designed to 1) identify outstanding candidates who have made a unique contribution to the fish health field, or 2) provided a significant research accomplishment, or 3) provided outstanding leadership in resolving a major fish health problem. The purpose of the award is to provide timely recognition for one-time accomplishments that have a significant impact on the management or control of fish health problems. The achievement must meet high standards of science and survive peer review. Anyone, not just the researcher, is eligible to receive such an award. The award will be presented at the Annual FHS meeting in Newport in August.

There are 606 members in the Fish Health Section. The membership and balloting committee is in the process of revising the Section's membership directory. Unfortunately, there was a poor response to a questionnaire requesting pertinent information from each member for the directory. However, names of the most active members and their expertise and affiliation, as well as updated bylaws and laboratories involved in fish health will appear in the new directory. The directory should be completed by July, 1991. As with the AFS directory, this document will provide a valuable resource to our membership as well as to other fishery professionals.

In general, things are going quite well with the Fish Health Section to date. Participation among members has been outstanding and I anticipate continued active participation by the membership during the remainder of the year.

Sincerely yours, *Charlie Smith, President FHS*

A New Russian Journal of Parasitology

Russian parasitologists are rather disappointed that their publications, especially on general and ecological parasitology, are poorly known in the English speaking countries. This is attributed to language barriers and secondly by poor exchange of publications.

Because of this, a group of Russian speaking parasitologists including Prof. Yu. I. Polyanski (Leningrad), Prof. V.L. Kontrimavichus (Vilnus), Prof. E.V. Gvozdev (Alma-Ata), Prof. O.N. Bauer (Leningrad) and others have proposed to publish a new journal "Ecological Parasitology" in English to inform parasitologists of other countries on parasitological news in the USSR. English speaking specialists are welcomed to send papers to this journal for publication. Several parasitologists of other countries have accepted an invitation to participate on the editorial board of "Ecological Parasitology". These include Prof. C.R. Kennedy (Exeter, U.K.), Dr. J.C. Chubb (Liverpool, U.K.), Prof. C. Combes (Perpignan, France), Dr. K. Molnar (Budapest, Hungary), and Dr. J. Thulin (Oregrund, Sweden).

"Ecological Parasitology" will contain several parts: 1. survey papers on different problems of ecological parasitology; 2. original papers not more than 24 typed pages; 3. short communications up to 6-7 typed pages; 4. discussions and reviews of books on ecological parasitology (especially published in Russian); 5. information on activities of departments and institutes in ecological parasitology; 6. chronicle on the parasitological life of scientific bodies (especially in the Soviet Union); 7. history of parasitology. The Journal will be distributed abroad and within the USSR. It is proposed to annually publish one volume with 4 issues. The first issue is to be published at the end of 1990. The price is not yet settled but it will be about $160 annually. Manuscripts should be submitted in duplicate with an abstract (0.5 pages) and summary (1.0-1.5 pages) for translation to Russian. Information requests sholud be sent to Dr. A.K. Galkin, secretary (199034, USSR, Leningrad, Zoological Institute Ac. Sc. USSR, Department of Parasitic Worms).

Notice to contributors will be published in the first issue.

O.N. Bauer, E.P. Ieshko, O.N. Pugachev

For more information contact Dr. Richard Heckmann, Department of Zoology, Brigham Young University, Provo UT 84602

Letters to the Editor

I recently received a copy of Vol. 18(4) of the FHS/AFS Newsletter and noted, with interest, your President's Message encouraging development of non-lethal sampling methods for

fish health inspections. The Northeastern Regional Aquaculture Center (NRAC) will be undertaking a project in this area coordinated by Dr. Pei Chang of the University of Rhode Island, and involving a number of researchers in the northeast. I have enclosed a copy of the cover page and objectives of the proposal which is currently being reviewed by USDA. A copy of the full proposal can be provided if desired. We have communicated with Mr. Angelo Incerpi about this project (letter enclosed) in the hope of coordinating our interests with those of NEHSC.

NRAC would welcome the opportunity to collaborate with other agencies in work related to non-lethal sampling techniques and would appreciate your encouraging other interested parties to communicate with us.

Henry S. Parker
Executive Director
Northeastern Regional Aquaculture Center
Southeastern Massachesetts University
North Dartmouth, MA 02747

Positions Open

U.S. Department of the Interior, Fish and Wildlife Service-Microbiologist/Histopathologist (GS-403-11/12) (Longview, WA)- The U.S. Fish and Wildlife Service seeks a microbiologist/histopathologist to research the effects of nutrition, disease organisms, and fish culture practices on the health and histology of anadromous fishes. Salary range $31,116 - $48,481. Call Julie Swanberg at 503-231-6136 for job description, qualifications required, and applications forms. Deadline: 6/30/91.

Meetings

World Aquaculture Conference and Exposition. June 16-20, 1991. Caribe Hilton, San Juan, Puerto Rico. For information: Conference Headquarters, The Crest Organization, 940 Emmett Avenue, Suite 14, Belmont, CA 94002, 415/593-2704.

4th International Symposium on Reproductive Physiology of Fish.
July 7-12, 1991. University of East Anglia, Norwich, United Kingdom. For information: Dr. A.P. Scott, 4th ISRPF, Fisheries Lab, Lowestoft, Suffolk NR33 0HT, UK.

AFS Western Division Annual Meeting. July 15-19, 1991. Boseman Montana. Student participation is especially encouraged. Students should contact Mike Moberly, Chairman, Student Concerns Committee, Coordinator, Student Papers Session, 4704 N.E. 55th, Seattle, Wa. 98105; Phone 206-522-6402.

Second International Symposium on Viruses of Lower Vertebrates. July 29-31, 1991. LaSells Stewart Center, Oregon State University, Corvallis, Oregon, USA. Contact J.L. Fryer, Department of Microbiology, Oregon State University, Corvallis, Oregon 97331-3804. Telephone 503-737-4441. Fax 503-737-0496.

14 th Annual AFS/FHS Annual Meeting. Mark O. Hatfield Marine Science Center, Oregon State University, Newport, Oregon. Aug. 1-3, 1991. Contact Paul Reno at HMSC 2030 S. Marine Science Dr., Newport, OR 97365; phone 503-867-0147, FAX 503-867-0105.

European Association for Veterinary Pharmacology and Toxicology, 5th Congress. August 18-22, 1991. Copenhagen, Denmark. Contact Folke Rasmussen, Chairman of the Congress, Karlegogard, 91 Karlegovej, DK-3400 Hillerod, Denmark.

3rd International Congress of Comparative Physiology and Biochemistry. August 25-30, 1991. Tokyo, Japan. Contact Congress Secretariat, 3rd International Congress of Comparative Physiology and Biochemistry, Zoological Institute, Faculty of Science, University of Tokyo, Hongo, Tokyo 113, Japan. FAX 81-3-816-1965.

European Association of Fish Pathologists Fifth International Conference: Diseases of Fish and Shellfish. August 25-29, 1991. University for Horticulture, Budapest, Hungary. Contact EAFP Meeting Secretary, Institute for Veterinary Medicine, Federal Health Office, Alt-Marienfelde 17-21, D-1000 Berlin 48, Germany.

121st Annual Meeting of AFS. September 8-12, 1991. San Antonio Marriott Riverwalk, San Antonio, Texas. For information: Paul Brouha, AFS, 5410 Grosvenor Lane, Suite 110, Bethesda, MD 20814-2199, 301/897-8616; FAX 301/897-8096.

Annual Meeting of the Southern Division of AFS. November 3-6, 1991. Greenbriar Hotel, White Sulphur Springs, West Virginia. For information: Bert Pierce, West Virginia Dept. Natural Resources, P.O. Box 697, Sutton, WV 26601, 304/364-5695.

12th Annual Meeting of the Society of Environmental Toxicology and Chemistry. Seattle Convention Center, Seattle, Washington. For information: Bill Williams, Program Chair, USEPA-ERL, 200 SW 35th Street, Corvallis, OR 97333, 503/757-4679; FAX 503/757-4799.

Annual Meeting of the North Central Division of AFS. November 30-December 4, 1991. Marriott Hotel, Des Moines, Iowa. For information: Al Farris, Iowa Department of Natural Resources, Wallace State Office Building, Des Moines, Iowa 50319, 515/281-5145.

FHS Officers and Committees 1990-91

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*Robert Durborow (Warmwater parasites)
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*Jack Ganzhorn (*Vibrio*)

AFS Distinguished Service Award

Phyllis Barney, Chair
Rosalie Schnick
Dennis Anderson
Toni Amandi

Criteria for Best Paper Award, JAAH

Bruce Barton, Chair
Phyllis Barney
Carl Schreck
Dave Groman
Doug Anderson

*Designates Disease Committee Network Chair

Deadline for Fall Newsletter:

August 15, 1991

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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