FISH HEALTH SECTION
AFS
NEWS LETTER

Volume 13, Number 2 April, 1985

Joint Fish Health Section and Western Fish Disease Workshop Date and Location Changed

The joint meeting of the FHS and 26th Annual Western Fish Disease Workshop will be held July 30-Aug. 1, 1985 in Seattle, WA. The first day of the meeting will be devoted to infectious fish diseases, the second day to non-infectious fish health matters, and the final half day to "open discussion". The format during the first two days will be one of contributed and invited papers, the latter focusing on two very important infectious diseases, BKD and IHN, in the Pacific Northwest and on fish health assessment and non-infectious fish health problems. The format on the last day originated in the round table discussions of 15 western fish health professionals in 1950 and has been retained because of its proven usefulnes, even with attendance exceeding 100. In addition, dozens of small round table discussions will occur over coffee, cocktails, meals, etc. All FHS members are urged to attend and contribute to this significant meeting.

Call For Papers

Each person invited to present a paper or interested in contributing a paper (limited to a total of 15 minutes for delivery and discussion for contributed papers) should submit an abstract, typed - single spaced on white bond paper (8½ x 11 with 1⅛ inch margins). Abstracts should not exceed one page in length and should be submitted no later than April 30, 1985 to: Dr. Trevor Evelyn, Pacific Biological Station, P.O. Dr. 100, Nanaimo, BC U9R5A6 Canada.

Registration Fee

The registration fee will be $15 to cover the cost of staging the workshop (hotel facilities, coffee and doughnut breaks, the workshop "proceedings" for the participants etc.).

BOARD OF CERTIFICATION ACTIVE

The Board of Certification has been very busy, especially during the last 12 months when the great majority of applications were received, processed, and reviewed. The following information summarizes activity of the Board since initiation of the Fish Health Inspector and Fish Pathologist certification program.

I. Fish Health Inspector Program
   1. Applications received: 45
   2. Applicants not approved: 13
   3. Applicants certified: 24
   4. Applications partially completed: 7
   5. Applications now being reviewed: 1

II. Fish Pathologist Program
   1. Applications received: 64
   2. Applicants not approved: 5
   3. Applicants certified: 42
   4. Applications partially completed: 7
   5. Applications now being reviewed: 10*

*Several of these applicants have been approved and notified by the Board but have not yet paid the final $40.00 certification fee.

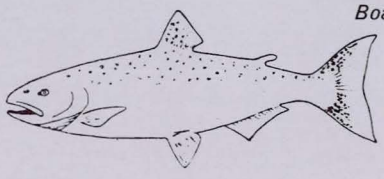
Certified Fish Health Inspector

1. James W. Warren
2. V. Charles Suppes
3. Douglas L. Mitchum
4. Paul W. Janeke
5. Dennis E. Anderson
6. John G. Hnath
7. John F. Conrad (retired)
8. Howard M. Jackson
9. Robert A. Busch
10. Roger S. Grischkowsky
11. Gary W. Camenisch
12. Ronald D. Major
13. Charles P. Carlson
14. Joseph R. Sullivan
15. A. Kent Hauck
16. Gerald R. Johnson
17. David O. Locke
18. Donald F. Amend
19. John A. Burke
20. Randy E. McLeary
21. Steven L. Leek
22. Joseph C. Lientz
23. John H. Schachte, Jr.
24. Warner G. Taylor

Certified Fish Pathologists

1. Stanislas F. Snieszko (Honorary)
2. John Grizzle
3. James W. Wood
4. Theodore R. Meyers
5. Ralph A. Elston
6. Douglas L. Mitchum
7. Paul W. Janeke
8. Paul R. Bowser
9. Donald F. Amend
10. John G. Hnath
11. Kevin H. Amos
12. Richard L. Westgard
13. James C. Carlisle
14. Andrew J. Mitchell
15. Warren J. Groberg
16. Joseph C. Lientz
17. Steven L. Leek
18. A. Kent Hauck
19. Howard M. Jackson
20. Thomas E. Schwedler
21. Thomas L. Wellborn, Jr.
22. Ronald L. Thune
23. John H. Schachte, Jr.
24. Roger Lee Herman
25. Ronald P. Hedrick
26. John K. Morrison
27. Mark G. DeCrew
28. Warner G. Taylor
29. John D. Cvitanich
30. Roger P. Dexter
31. Steven D. Roberts
32. Diane G. Elliot
33. Marshall H. Beleau
34. Robert E. Olson
35. Lee W. Harrell
36. John R. MacMillan
37. Ronald W. Goede
38. Dennis E. Anderson
39. Dorothee Kieser
40. Gary E. Hoskins
41. Richard A. Holt
42. Antonio Amandi

Doug Mitchum, Chairman
Board of Certification



FHS OFFICERS AND COMMITTEES 1984-85

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John Rohovec, President-Elect
Glenn Hoffman, Immediate Past President
Doug Anderson, Secretary-Treasurer
Diane Elliot, Chairman, Nominating Committee

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John Rohovec, Newsletter and Publications Committee
Emmett Shotts, Awards Committee
Steve Leek, Membership and Ballotting Committee
Paul Janeke, Professional Standards Committee
Kevin Amos, Technical Procedures Committee

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Steve Leek, Chairman
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Doug Mitchum, Chrm. (2 years)
Jim Warren (1 year)
Gary Camenisch (1 year)
Kevin Amos (2 years)
Joe Lientz (3 years)

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(Appointed)

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Roger Herman
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Bylaws
Jim Warren, Chairman
Fred Meyer

Directory
Rowan Gould

Fund Raising
Dave McDaniel, Chairman
Doug Anderson
Roger Herman

International Meeting (1986)
Chairman to be selected
Bill Rogers
Leo Margolis
Barry Hill
Dave Conroy

Program (1985 meeting)
Trevor Evelyn
Ron Goede

Time and Place (86-89)
John Fryer, Chairman
Charles Suppes
Bill Rogers

The Fish Health Directory should be completed in the near future. Although response to a request for additional information was limited, the Directory will include all Fish Health Section members and their addresses. Rowen Gould, Directory Committee Chairman, Division of Fishery Research, USFWS, Washington, D.C. 20240.

FUTURE EVENTS

April 25-26, 1985 **PKD WORKSHOP,** University of California-Davis will host a two-day workshop on Proliferative Kidney Disease (PKD). There will be a technical session concerning the current knowledge of the disease and discussions on regulation and policy formulation by fisheries managers and pathologists. Speakers have been invited from British Columbia, Idaho and California. The meeting is designed to involve both fish pathologists and managers. For more information contact R.P. Hedrick, UC-Davis, School of Vet. Med., Davis, CA 95616 phone (916) 752-3411.

July 30-August 1, 1985. **Joint Meeting of the Western Fish Disease Workshop and Fish Health Section 1985 Annual Meeting.** This meeting will be held at the Seattle Sheraton Hotel. For registration materials contact Kevin Amos, Washington Dept. of Fisheries, Olympia, WA 98504 or John Majnarich, Biomed Research Lab., Seattle, WA 98122.

July 9-11, 1985, **Midwest Fish Disease Workshop.** The 16th Annual Midwest Fish Disease Workshop will be held in Peoria, IL at the Continental Regency Hotel. Registration and a no-host cocktail hour will occur on July 9. The emphasis this year will be in the area of non-infectious disease, with one segment slated to be a round table discussion of miscellaneous mysterious maladies.

A number of papers are slated on a variety of topics: John Hnath; N_2 Supersaturation in Coho Salmon. Gary Camenisch; Gas Supersaturation in Missouri. Andy Moore; Ozone Disinfection. Drew Mitchell; Water Quality in Warmwater Diagnostics. Terry Bradley; Epitheliocystis, Contagious, Pathogenic Cosmopolitan. Paul Bowser; Hamburger Gill in Catfish. Ron Thune; *Aeromonas.* Denise Desens; Microbiology at the La Crosse Lab. Vicki Blazer; Role of Nutrition in Fish Disease. A segment is reserved for contributed papers. If you have anything you wish to present, please contact Rod Horner at 309/968-7531, Sand Ridge Fish Hatchery.

A tour of Illinois' new Sand Ridge Fish Hatchery will highlight Wednesday afternoon, followed by a fish fry and beer bust at the hatchery. For more information contact: Rodney W. Horner, Program Chairman, Sand Ridge Fish Hatchery, Box 398, Manito, Il 61546, Telephone: 309/968-7531.

August 4-8, 1985, **Fish Parasite Symposium.** There will be an organized symposium pertaining to fish parasites at the 1985 meeting of the American Society of Parasitologists. The scientific meeting will be held at the University of Georgia (Athens). For more information pertaining to the symposium contact Dr. Richard Heckmann, Dept. of Zoology, Brigham Young University, Provo, Utah 84602.

PASSAGES

Paul R. Bowser has moved from Mississippi State University to Cornell University. His new address is: Department of Avian and Aquatic Animal Medicine, College of Veterinary Medicine, Cornell University, Ithaca, NY 14853.

Rowan W. Gould is departing Wellsboro, PA for Washington, D.C. where his new address is Division of Fishery Research, Fish and Wildlife Service, U.S. Department of Interior, Washington, D.C. 20240. His phone number is (202) 653-8772.

DELAYED APPEARANCE OF IHNV IN STORED SUMMER STEELHEAD EGGS

Scott La Patra, Department of Microbiology
and
W. J. Groberg, Oregon Department of Fish and Wildlife
Oregon State University, Corvallis, OR 97331

For the past two years, we have been conducting IHNV transmission studies at Round Butte hatchery in central Oregon. One of these studies employs delayed fertilization as part of the experimental protocol in an attempt to obtain high-titer/no-titer mating pair crosses.

Gametes from individual fish are harvested into plastic bags. The bags are numbered, filled with oxygen, sealed and stored at 4°C. At the same time, correspondingly numbered individual spleen and sex fluid samples are taken. These samples are immediately plaque assayed and IHN virus titers are calculated in 7 days. On day 8, we return to the hatchery, the gametes are removed from storage, and specific mating pair crosses are made based on the results from the initial virus assay. The goal is to obtain a total of eight groups: four high-titer (IHNV positive) and four no-titer (IHNV negative), with each group composed of the eggs from eight females and the milt from three males. Egg incubation and rearing are carried out in UV treated water. Equipment and personnel are thoroughly disinfected to eliminate contamination. Through this experiment we hope to answer the question of whether or not vertical transmission of IHNV occurs.

At the 1984 Western Fish Disease Conference Informal Virology Session we reported an unexpected observation made during the first year of this experiment. Immediately prior to fertilization (eight days post-spawning) of the eggs in our planned mating pair crosses, we sampled the pooled gamets from each sex for all eight groups. We were concerned about possible loss of virus in the positive groups over the eight day storage period. Pooled ovarian fluid and milt samples were plaque assayed and virus titers calculated. Not only were the positive groups still positive, but all four of the eight-fish negative female gamete pools were now IHNV positive. The three-fish negative male gamete pools had all remained negative. Identical results were obtained two more times that year in the same stock of fish. Ovarian fluid from the gametes of females determined to be IHNV negative at spawning was virus positive after the gametes were stored and pooled after eight days.

This year the same experiment is in progress. In an effort to explain last year's results, all individual female gametes were resampled prior to pooling after the eight day storage period. The data indicate that the ovarian fluid from female gametes determined to be negative at spawning becomes IHNV positive when samples are again taken after the eight day storage period.

The data not only show some groups of stored gamets changing from negative to positive (e.g. 0 to 10^4 pfu/ml), but also indicate an increasing titer after the storage period (e.g. 10^2 pfu/ml to 10^4 pfu/ml) in some initially positive gametes. The gamete storage experiment has been repeated for the second time in 1985 and further results will be available. Additional studies are in progress to determine the mechanism of this phenomenon.

The IHNV transmission studies at Round Butte Hatchery are planned for subsequent years. This work is being funded by a grant from the Bonneville Power Administration. Groups cooperating in this project are Oregon State University, Oregon Department of Fish and Wildlife, USFWS, and Portland General Electric.

Brief reports are solicited from all members. Please submit to one of the editors.

SEARCH FOR A VIRUS-FREE SOCKEYE STOCK

Keith A. Johnson, SSRAA,
Ketchikan, AK 99901

Southern Southeast Regional Aquaculture Association (SSRAA) and ADFG/FRED are cooperating in a study to evaluate the pathogens in salmonid populations in the southern area of the Alaska panhandle. Sites to be evaluated are lakes with potential as hatchery water supplies. Many lakes were formed during periods of glaciation and, although currently inaccessible to anadromous fish, contain populations of native cutthroat and dolly varden trout and kokanee. The major emphasis of the pathology study is to determine if the kokanee populations carry IHNV. The implications for fish culture are obvious but it also may yield information about the origin of the disease in sockeye.

Numerous sockeye hatcheries were operated in Alaska by canneries and the Bureau of Fisheries between 1880 and 1930. Millions of sockeye eggs were incubated but results were disappointing. In many cases, transports of sockeye eggs from other drainages into hatcheries in Southeast can be documented. Other stocking activities using brook trout, rainbow trout, and grayling have also occurred in lakes and rivers in the area.

The goal of the evaluation program is to obtain sexually mature salmonids from candidate lakes. To date, about sixty kokanee and several cutthroat and dolly varden trout have been sampled from each of two lakes. No IHNV, BKD, or *Aeromonas salmonicida* have been found by ADF&G Fish Pathology Laboratory. Further sampling of these sites and of several additional lakes is planned for 1985.

IPNV FROM WILD LAKE TROUT - ALBERTA

Beverly Larson, Alberta Dept. of Agriculture
Edmonton, Alberta T6H 4P2

Infectious Pancreatic Necrosis Virus (IPNV) was isolated in the fall of 1983 from a spawning population of wild lake trout from a remote lake in northeastern Alberta. There is no history of artificial stocking into the region and the lake is accessible only by air. Spawners from this lake trout population were tested (lethal and reproductive fluid samples) from 1981 to 1983, because they were the source of eggs for provincial stocks. No virus was detected in the 1981 and 1982 test samples.

In 1983, 18 spawners were sacrificed and IPNV was isolated from all four pooled pyloric caecae and spleen tissue samples; pooled kidney samples were negative. The BF-2 and CHSE-214 cell lines were most sensitive, yielding greater than 50% positive test wells (pyloric caeca and spleen); RTG-2 assays yielded only one positive well (8%) within 13 days, with a second positive well developing at 19 days. The FHM cells were refractile.

Sixty (60) reproductive fluids were also assayed in 3 to 5-fish pools. All were negative at 21 days. Blind passages (performed randomly on half the pools) taken from BF-2 cells are tested in both BF-2 and CHSE-214 assays were also negative. Interestingly, at 6-7 weeks post-inoculation, **one** test well (CHSE-214 cells, original assay) became positive, confirmed by second passage.

This isolate was only weakly neutralized by available IPN antisera (Jasper strain). With the cooperation of JoAnn Leong, Oregon State University, the isolate was confirmed as IPNV. Subsequent serological tests showed the isolate was more closely related to the Arctic Char IPNV than to the VR-299 strain, but further testing is required. Pathogenicity tests using this isolate have not been completed.

This is the first detection of IPNV in Alberta since T. Yamamoto's work with the Jasper IPNV isolate.

PROLIFERATIVE KIDNEY DISEASE IN CALIFORNIA: A SECOND LOOK

R.P. Hedrick, M.L. Kent, J.S. Foot, and R. Rosemark
Aquaculture Program, Department of Medicine, School of Veterinary Medicine, University of California, Davis, CA 95616
and
D. Manzer, California Department of Fish and Game, Fish Disease Laboratory, 211 Nimbus Road Rancho Cordova, CA 95670

In June of 1983 we reported on the first outbreaks of proliferative kidney disease (PKD) in Pacific salmonids. Since our initial observations, PKD has been detected in three new watersheds in which Pacific salmon, steelhead trout and rainbow trout are cultured. Four state hatcheries and one private farm are involved and all five sites use water from either a river or reservoir.

At one of these state hatcheries, an examination of histological sections made in 1966 from diseased rainbow trout showed the typical PKX protozoan that causes PKD. A condition of unknown etiology historically coined "lupus" by California Department of Fish and Game pathologists is now in retrospect known to have been PKD. Since descriptions of "lupus" date back to 1958, PKD presumably has been established in certain watersheds for years.

We have studied the disease among rainbow trout at one of the state hatcheries with a history of recurrent "lupus" by monthly or biweekly sampling of fish reared in the facility beginning in June 1984 through February 1985. Because the hatchery is a major producer of fish for stocking inland waters, numerous groups of fish of various sizes and ages are introduced throughout the year for subsequent stocking.

Unusually high mortality began in late August 1984 among the largest rainbow trout (ave. wt. 45g) that had been introduced in November 1983. All diseased fish examined showed the grossly swollen kidneys associated with PKD, but few of the typical PKX cells and intraluminal trophozoites thought to be later stages of the parasite. Concurrent infections with *Ichthyophthirius, Flexibacter columnaris* and gill bacteria, all contributed to a near 20% mortality in this group. Certainly, in these diseased fish the kidney pathology could have been a major cause of the observed mortality but causes of such an intensive reaction are unknown and may be due to factors such as size, age or prior exposure to PKX.

A second group of smaller rainbow trout (ave. wt. 7.5g) hatched on site in March also showed significant mortalities during the same period as the larger trout. Again however, complicating infections with *F. columnaris* were detected, but 100% of the 30 fish collected in a random sample showed some degree of kidney swelling (although much less severe than the 45g fish) and numerous PKX and intraluminal trophozoites were detected. The mortality in this group also subsided in November when water temperatures declined.

We have tried to separate the effects of water temperature from a possible seasonal nature of the infective stage in the water source by exposing two groups of 30 fish (ave. wt. 5-10g) on a monthly basis to hatchery water one group at ambient (fluctuating) and a second at a constant 17°C. After one month, both groups were transferred to the Fish Disease Laboratory at the University of California Davis, for further incubation in 18°C water. Eight weeks from initial exposure, fish were sacrificed and examined for gross pathology, by kidney wet mount and histological sections for the incidence of infection with PKX. From these studies we know that the infective stage is present as early as May, peaks in late June and steadily declines and then disappears in November even in fish exposed in warm water.

The water temperatures at the hatchery reach 16°C in May and by June are 18°C staying at this level until November when a steady decline to 7°C in December occurs. The last examination in which we were able to detect typical PKX cells was in 1 of 30 fish sampled in December of 1984. However, most fish had recovered from PKD and mortalities had declined substantially by late October when the water temperatures were still 18°C. A further reduction in mortality was correlated with declining temperatures in November and December. This may have been due more to the effects of reducing faculative pathogens and improving water quality than recovery from infections with PKX. We have observed the PKX parasite and associated pathology and mortality in one rainbow trout farm where water temperatures never exceeded 10°C. These observations indicate that recovery from PKD can occur at elevated water temperatures and the onset and course of infection are not prevented at temperatures between 13-16°C.

Ferguson has proposed that introductions of fish in September or October and harvesting prior to July or August the following year may be one method to minimize mortalities to PKD. Fish introduced in this manner probably receive an exposure to PKX at a low dosage, in the fall which can protect some but not all fish from infection upon re-exposure the following year. In contrast, fish recovering from a full exposure, as steelhead trout survivors from the 1983 Mad River epizootic, seem to have a more solid immunity. No clinical signs, PKX cells or intraluminal trophozoites were observed in 30 of these fish examined before, during, or after the peak period of infection experienced by hatchery fish exposed to the same water supply from October 1983 to November 1984 at our study site.

Preliminary studies suggest the basis of the immunity resides in the serum fraction. Passive transfer of serum from convalescing fish seems to speed the recovery and reduce the incidence of the parasite and pathology when administered to fish actively infected with PKX. Further studies are continuing to confirm that these serum components (presumably immunoglobulin) are important in recovery from PKD.

TRANSMISSION OF PROLIFERATIVE KIDNEY DISEASE (PKD); FURTHER EVIDENCE THAT THE PKX PARASITE BELONGS TO THE PHYLUM MYXOZOA

M.L. Kent and R.P. Hedrick, School of Veterinary Medicine, University of California, Davis, California 95616

Proliferative kidney disease was first reported in California at the Mad River Hatchery in 1983 and was subsequently identified at the American River and Nimbus State hatcheries in the summer of 1984. Myxosporidan trophozoites and developing spores were observed in infected fish and were suspected to be later stages of the PKX parasite. Numerous developing spores were observed in the kidney tubule lumens of several infected fish by squash preparations from September 1984 through January 1985 as shown in Figures 1-3. They were all monosporoblastic and enclosed in enveloping cells with prominent refractile granules that may be analogous to the electron dense multilaminated bodies described in electron micrographs. We have yet to observe valves in these spores and possibly development is incomplete in rainbow trout.

Although epidemiological and morphological data indicate that the myxosporidan forms are later stages of PKX, the possibility of a mixed infection must be considered. To examine this in more depth we inoculated uninfected fish with the blood and spleen of PKD infected fish. In our experience, the myxosporidan trophozoites and spores that occur in PKD infected fish are found only in the kidney tubule lumen and never in the spleen and blood where typical PKX is observed. If fish receiving blood and spleen tissues via intraperitoneal injection develop typical PKX cells and the intraluminal myxosporidan stages, it would support our hypothesis that all are different stages of the same organism.

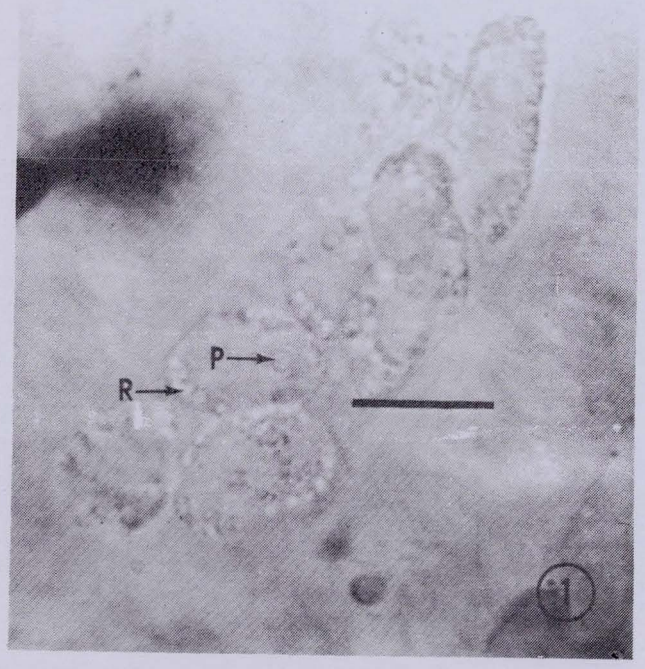
Steelhead trout from both the blood and spleen injected groups became infected with the intraluminal myxosporidan stages as well as the typical PKX parasite in the kidney interstitium as shown in Table 1. Control fish remained normal throughout the study.

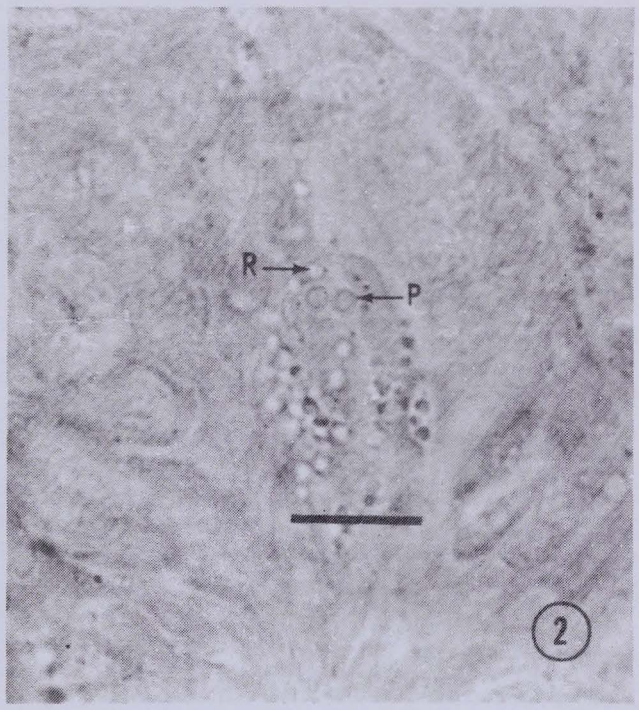
Since only the typical PKX form is observed in the blood and spleen during PKD infections, these experiments provide strong evidence that the intraluminal trophozoites and spores we have observed are later stages of PKX. This further supports the hypothesis that PKX belongs with the phylum Myxozoa. However, a more precise taxonomic placement of PKX will depend on observations of mature spores. We have observed what are apparently immature spores without valves throughout infection. Steelhead and rainbow trout may not be the primary hosts and other species of fish should be examined as a possible host in which the parasite completes its development.

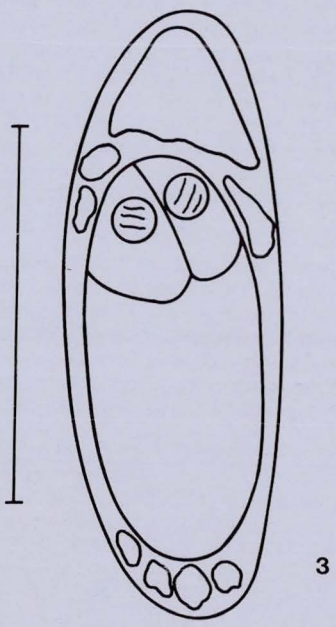
Why the intraluminal myxosporidans reported by Kent and Hedrick are not frequently observed in fish with clinical PKD in Europe remains to be determined. In our experience small fish, usually less than 10 to 20 grams, often become heavily infected with PKX parasites, but do not exhibit the severe kidney hypertrophy that is often reported with the disease. These fish also exhibit a high prevalence of the intraluminal stages. Possibly, the severe inflammatory response seen in larger fish may inhibit the further development of the parasite. The disease also causes tubular atrophy, which would decrease the available sites for the parasite to sporulate. Most descriptions of the parasite have been from clinically affected fish and close examination of small fish without clinical disease from enzootic waters should reveal sporulating stages similar to those we have described.

Table 1. Incidence of intraluminal myxosporidan and typical interstitial forms of PKX in the kidney of steelhead trout injected with the blood or spleen from trout with PKD.

	Spleen injected	Blood injected
No. with PKX and intraluminal forms	2	3
No. with intraluminal forms only	1	3
No. with PKX only	0	1
Total examined	17	16







Figures 1 and 2. Squash preparations of kidney tissue from PKX exposed rainbow trout showing intraluminal myxosporidan sporo blast with developing spores. P = polar capsule, R = retractile granules.

Figure 3. Intraluminal myxosporidan from PKX exposed rainbow trout. Bars = 20 um.

PKD IN IDAHO

Robert A. Busch, Clear Springs Trout Company
Buhl, Idaho 83316

Proliferative kidney disease (PKD) is an infectious disease of trout and salmon believed to be caused by a parasitic protozoan or sporozoan organism (PKX). It was first discovered in North America in the summer of 1981 in rainbow trout at Hagerman State Fish Hatchery on the Riley Creek drainage in southern Idaho. Since that time it has been shown to be endemic to the drainage and recurrent every spring, resulting in significant mortality due to the parasitic infection itself and the exacerbation of other infectious and non-infectious or environmental diseases or conditions.

All Clear Springs Trout Company (CST) production operations and particularly Riley Creek stocks are routinely monitored for the presence and severity of PKX infection by means of microscopic examination of stained kidney smears. Since PKD is relative slow to develop as a clinical condition (i.e. 6 to 8 weeks at 58°F and below 56°F) and because the Riley Creek hatchery demonstrates a wide seasonal variation in temperature (above 56°F in the summer and below 56°F in the winter), a "farm pond" management plan for stocking and harvesting the Riley Creek facility, coordinated with seasonal temperature changes and the incidence and occurrence of PKD, is being tested. The hatchery waters appear to contain infective PKX organisms from approximately April to September. Stocks present in the Riley Creek hatchery in April are harvested before the disease reaches its full clinical potential in June and July. Replacement stocks ponded into the facility during the summer begin to recover from the infection as waters cool in the fall before the disease again reaches its full clinical potential.

Stocks of fish present in the Riley Creek facility in the spring of 1984 were generally harvested during the summer months prior to the appearance of PKD related losses. However, the replacement stocks ponded back into the facility during the summer months developed clinically apparent cases of the disease quite rapidly in the seasonally elevated water temperatures.

Starting approximately September 1, 1984, mortality in these fish began to increase. Clinical examination for external parasites showed a light incidence of *Trichodina, Ichthyophthirius* and *Gyrodactylus.* Gross internal signs included swollen or extended posterior kidneys symptomatic of PKD. Microscopic examination of stained kidney smears confirmed the presence of PKX. On September 10, 1984, a random sample of fish from 3 ponds was taken for hematological analysis and stained kidney smears. Both the blood hemoglobin and hematocrit were **below** normal ranges in 15/15 fish. The same 15 fish were also shown to be positive for PKX in stained smears. On September 14, 1984, another sample was taken for stained kidney smears and found to be 100% positive for PKD.

On September 25, 1984, the same ponds in which fish were suffering a continuing mortality, were sampled again. Laboratory results indicated no external bacterial involvement, 100% incidence of PKX infection, pyknosis and cell debris in the spleen and kidney, and clinically active IHN virus. Mortality appeared to be caused by a dual infection of IHN virus and PKD.

On October 17, 1984, water temperatures had declined below 56°F and ponds were again sampled. On gross clinical examination, fish showed signs of swollen posterior kidneys typical of PKD. However, stained smears demonstrated what appeared to be PKX organisms but in an obviously degenerating form. No external bacteria or parasites were found. Cell debris and pyknosis was still noted in the spleen and kidney. Samples were confirmed to be positive for clinically apparent IHN virus.

On the basis of random sampling and periodic laboratory analysis, it appears that all populations of fish at Riley Creek are most likely infected with PKX. The earliest summer restock date was July 10, 1984, at which time the water temperature averaged 60°F. Approximately eight weeks after restocking, typical gross and microscopic signs and evidence of clinical PKX infection were present and water temperature averaged 60°F. At the present time it appears that the PKX infection is in a degenerative stage most likely attributable to the declining water temperature which now averages 50 to 52°F daily.

It is difficult to tell exactly what percent of the mortality loss can be attributed to PKD - a guess would be 50%.

At the present time our knowledge and understanding of proliferative kidney disease, based upon the literature, our own experience and the experience of others, seems to indicate that the disease may not pose a serious threat to major production operations with direct spring water supplies or coldwater broodstock operations. However, at any site with an extensive water supply and constant or seasonal temperatures of 58°F or greater, such as Riley Creek and Idaho Springs, PKD can be expected to be an endemic and non-treatable cause or contributor to mortality. At the present time, the only recognized means of control available is to coordinate stocking and harvesting with temperature fluctuations remembering that the clinically apparent disease condition takes 6 to 8 weeks to develop. Cold water (below 56°F) appears to cause the parasites to rapidly degenerate, and once recovered, surviving fish appear to be immune to future infection.

Other than seasonal coordination of stocking and harvesting, the use of induced cold shock and immunization by intentional exposure followed by cold shock could be investigated. Some investigators are screening systemic drugs and chemicals for potential therapeutic value and the use of ultraviolet light to disinfect water supplies appears to have some beneficial effect on limiting transmission.

IPN AT THE JOCKO RIVER SFH, MONTANA

Jim Peterson, Montana Dept. FWP
Box 423, Bridger, MT 59014

In November 1983 IPN virus was discovered in brook and brown trout in the spring water supply of Montana's Jocko River Trout Hatchery. The Jocko River hatchery is the home of Montana's primary rainbow trout broodstock and supplies eggs to Montana's four largest production hatcheries for distribution statewide. All rainbow brood lots are routinely examined for pathogens, including IPN, and during 1984 all age classes were extensively tested for IPN virus. IPN has never been detected in any rainbow trout at the hatchery even though they were reared in water containing IPN-infected brook and brown trout. The water supply was chlorinated in Feb. 1984 and again in Feb. 1985 to remove the fish from the water supply, and a considerable effort has been initiated to bury the open spring and enclose flowing water in pipelines.

SURVIVAL OF *EDWARDSIELLA ICTALURI* IN POND WATER AND MUD

John A. Plumb, Department of Fisheries & Allied Aquaculture, Auburn University, Auburn, AL 36849

Evidence indicates that *Edwardsiella ictaluri* (enteric septicemia of catfish) is a relatively host specific and obligate pathogen. Previous work by John Hawke demonstrated that *E. ictaluri* did not survive well in water at 25°C and we recently confirmed his data. However, after seeding sterile bottom mud with *E. ictaluri* we recovered high numbers of the bacterium ($1 \times 10^{6.3}$ cells/ml) for 95 days when the mud was incubated at 5°C. The fact that *E. ictaluri* survives in mud at 25°C for so long may indicate that it is not a total obligate pathogen and the mud may serve as a reservoir for *E. ictaluri.*

FISH PASTEURELLOSIS IN CULTURED STRIPED BASS (*MORONE SAXATILIS*) IN COASTAL ALABAMA

John P. Hawke and R. Vernon Minton
Alabama Department of Conservation and Natural Resources
Marine Resources Division, Claude Peteet Mariculture
Center, Gulf Shores, AL 36542

Fish pasteurellosis in cultured populations of striped bass, *Morone saxatilis*, caused by the bacterium *Pasteurella piscicida* is reported for the first time. In an outbreak of the disease in twelve .08 ha brackish water ponds at the Claude Peteet Mariculture Center approximately 80% of the stocks or 49,000 fingerlings were killed before the disease was controlled.

Feed was medicated with Oxytetracycline HCl at 150 mg/kg biomass/day and fed at 3% body weight per day for a total of 17 days. Medicated feed was first administered 24 hours after initial isolation of the organism following antibiotic susceptibility testing. Muscle tissues taken at weekly intervals post-treatment were assayed for antibiotic residues.

SUSCEPTIBILITY OF EUROPEAN CATFISH (*SILURUS GLANIS*) TO CHANNEL CATFISH VIRUS

John A. Plumb, Department of Fisheries and
Allied Aquaculture, Auburn University, Auburn, AL 36849

In recent years there have been increased exports of channel catfish fry and fingerlings to Europe. Because there is no accurate method of certifying these fish free of channel catfish virus (CCV), European fish patholgoists are concerned about the possibility of introducing CCV to Europe and what effect the virus may have on European catfish (*Silurus glanis*). In cooperation with V. Hilge in Germany we imported European catfish fingerlings to Auburn University to evaluate their susceptibility to CCV. Although the answer is not totally clear, our work indicates that CCV has no detrimental effect on *S. glanis*. High doses (1×10^5 $TCID_{50}$ per fish) failed to produce clinical signs of CCVD or death. However, CCV was isolated for 4 days only after intraperitoneal injection, but after dipping the fish in a bath of CCV only one fish yielded a low level of virus (<10 $TCID_{50}$ per 0.1 g of tissue) 5 days after exposure. We concluded that it is unlikely that CCV would seriously harm European catfish fingerlings, but we plan to do more work on the problem. After experimentation, all *S. glanis* fingerlings were destroyed to prevent accidental introduction into North American waters.

POSITION ANNOUNCEMENT

Area Extension Fisheries Specialist
Extension Wildlife and Fisheries Department, Stoneville, Mississippi

Qualifications Required:

Minimum: Master's Degree from an accredited institution of higher learning in Fisheries Management with academic training needed for AFS/Fish Health Section certification as a Fish Pathologist and some experience in commercial aquaculture systems.

Preferred: Ph.D. from an accredited institution of higher learning in Fisheries Management with academic training needed for AFS/Fish Health Section certification as a Fish Pathologist; two or more years' experience in disease diagnosis and control in commercial aquaculture. production systems.

Inquiries should be directed to Milburn Gardner, Personnel Officer, P.O. Box 5446, Mississippi State, Mississippi 39762, telephone (601) 325-3462.

PATHOGENESIS OF *EDWARDSIELLA ICTALURI* IN CHANNEL CATFISH

Vicki Blazer, Cooperative Fish and Wildlife Unit
School of Forest Resources
and
E.B. Shotts, and W.D. Waltman, College of Veterinary
Medicine, University of Georgia, Athens, GA 30602

Channel catfish (*Ictalurus punctatus*) fingerlings were exposed via the gut and by water contact to *Edwardsiella ictaluri*. The pathogenesis of the disease was studied histopathologically over a thirty day period. Catfish exposed via the gut developed a systemic infection within two weeks. Enteritis, hepatitis, interstitial nephritis and myositis of the skeletal muscle around the head and jaws were observed. These lesions began as acute inflammation (primarily granulocytes) and progressed to chronic, diffuse granulomatous reactions. By two weeks post exposure macrophages packed with bacteria were observed in all affected organs. None of the gut exposed fish exhibited the classical "hole in the head" lesion or had involvement of the brain.

Contact fish, placed in tanks with gut exposed fish, did not show signs of infection until 16 days after contact. From 16 to 35 days after contact, fish were sampled which: 1) showed signs of a systemic infection (gut and liver lesions) with no brain involvement; 2) had olfactory and brain lesions with no involvement of other organs and 3) had olfactory and brain lesions as well as systemic infections. A total of eight contact fish exhibited the "hole in the head" lesion with the first observed 27 days post exposure.

We believe in natural infections, *E. ictaluri* enters the fish both through the nares and the gut. Infection via the nares proceeds from the olfactory epithelium via the olfactory nerve into the olfactory bulb and telencephalon of the brain. The external lesion is actually an erosion of the connective tissue, muscle and skin, extending from the meninges and areas around the brain to the outside. Lesions in the brain ranged from primarily perivascular cuffing and meningeal involvement to fish in which large areas of the telencephalon were replaced by a diffuse granulomatous reaction.

BRIEF REPORTS

Epitheliocystis-like organisms have been observed infesting Mississippi farm-raised channel catfish. They were observed histologically (not grossly) in gill epithelial cells of catfish affected by "Hamburger gill disease". The relationship, if any, between epitheliocystis and hamburger gill disease is unknown. J.R. MacMillan, Mississippi Cooperative Extension Service, P.O. Box 142, Stoneville, MS 38776.

Chills Syndrome (Chinook Lateral Line Syndrome), a disease which has been sporadically detected was recently seen in chinook salmon at the Washington Department of Fisheries, Lyons Ferry Hatchery. Clinical signs are primarily hemorrhage in the musculature below the lateral line. Jim Wood, WDF, M-2 Fisheries Center (WH-10), University of Washington, Seattle, WA 98195, phone (206) 543-4278.

Cutthroat trout broodstock, Atlantic and coho salmon in seawater net pens at the National Marine Fisheries Service Marine Experimental Station at Manchester, Washington are periodically infested with a marine gyrodactylus. Wayne Brunson (WDG) has devised an effective treatment of immersion for two minutes in 10% salt (100 ppt). Lee Harrell, National Marine Fisheries Service, Manchester Marine Experimental Station, P.O. Box 38, Manchester, WA 98353.

FISH HEALTH NEWSLETTER

The Fish Health Newsletter is a quarterly publication of the Fish Health Section of the American Fisheries Society. Submissions of any length on a topic of interest to fish health specialists are encouraged and should be addressed to one of the editorial staff or to a member of the publication committee.

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