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EFFECT OF *LERNAEA CYPRINACEA* (CRUSTACEA: COPEPODA) ON STOCKED RAINBOW TROUT (*ONCORHYNCHUS MYKISS*)

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ABSTRACT: Prevalence, intensity and pathogenesis of *Lernaea cyprinacea* (anchorworm) in stocked rainbow trout (*Oncorhynchus mykiss*) fingerlings were monitored annually for 4 yr (1981 to 1984) in East Canyon Reservoir, Utah (USA). Anchorworms were first detected in midsummer each year and were most abundant in the fall. The mean parasite intensity was highest in October 1982 (19 anchorworms per fish); in other years, maximum density was 7 to 9. The dorsal and caudal areas of the fish were the most heavily parasitized. The histological response to parasite attachment included an infectious granuloma similar to that reported in other fish hosts. Bacteria were not found in the kidneys of fish before stocking, but afterward bacteria that were presumptively identified as belonging to the genus *Aeromonas*, were found in the kidneys of up to 45% of the parasitized fish. Most (94%) anglers noticed the anchorworms, but few (8%) discarded parasitized fish. Some 28% used special cleaning techniques to prepare fish but 49% did nothing special to clean them. Lernaeosis probably had little effect on the fishery management goals for the reservoir.

Key words: Anchorworm, *Lernaea cyprinacea*, rainbow trout, histopathology, parasite, recreational fishing, *Oncorhynchus mykiss*, copepoda.

INTRODUCTION

Lernaeosis is caused by parasitic copepods (*Lernaea* spp.) that infect many freshwater fish important to aquaculture and recreation. Trout in the United States are infected by *L. cyprinacea*, whereas *L. esocina* and *L. minima* are of concern in salmonid fisheries in Europe (McNeil, 1961; Kabata, 1970; Hoffman, 1973). Fish less than 20 mm long may be killed by the parasites if vital organs are penetrated by the anchors (Khalifa and Post, 1976). Uzmann and Rayner (1958) reported that "heavy" infections of *L. cyprinacea* caused death of yearling rainbow trout (*Oncorhynchus mykiss*). Copepods may open routes for secondary infection; however, quantitative data on these secondary effects are scant (Hoffman, 1976; Dempster et al., 1988). Moreover, the attached copepods may make the fish undesirable to anglers (Fig. 1), thus affecting fishery management goals.

The disease is enzootic in several Utah reservoirs (USA) where fingerling rainbow trout (7 cm long) are stocked, and the fishery is managed to maximize the return of stocked fish to the angler. On the premise that stocking the best-performing rainbow trout strain might be an economical means of improving the fishery, we conducted a 5-yr study to compare rainbow trout strains for post-stocking growth and survival (Shrader 1988; Babey and Berry 1989). As part of this study, we investigated the occurrence of lernaeosis, its potential for reducing survival of stocked fingerlings, and angler reaction to the appearance of the diseased fish.

METHODS AND MATERIALS

The study was conducted in East Canyon Reservoir, situated at 1,739 m above mean sea level, in Morgan County, Utah (USA; 40°55′N, 111°36′W). The reservoir is mesotrophic to slightly eutrophic as judged by primary productivity, but is classified as hypereutrophic as judged by phosphorus and nitrogen loadings (Merritt et al., 1980). It has a maximum depth of 46 m and a surface area that fluctuates seasonally from 200 ha in winter to 277 ha in spring.

TABLE 1. Dissolved oxygen and water temperature of a mid-reservoir water column in East Canyon Reservoir, Utah, during summer, 1982. Readings were taken at 1 m intervals with a portable dissolved oxygen meter.

Date	Temperature (C)	Dissolved oxygen (mg/l)
May 24	13.0 (9.5–20.0)*	7.0 (5.9–8.0)*
June 1	13.0 (9.4–23.0)	4.0 (3.7–8.5)
July 5	18.0 (10.6–23.0)	3.0 (2.1–7.7)
July 25	19.0 (10.0–22.0)	2.0 (1.8–7.4)
August 1	15.0 (9.0–21.0)	2.0 (1.2–7.0)
August 12	16.0 (12.0–17.0)	6.5 (1.1–6.9)
September 6	13.0 (12.0–13.0)	8.0 (7.0–9.0)
October 12	7.2 (7.1–7.3)	9.0 (9.0–10.0)

* Mean followed by range in parentheses.

L. cyprinacea is in the watershed and fish kills that were believed to be caused by a combination of lernaeosis and high water temperature have occurred in nearby reservoirs. Thermal stratification and hypolimnetic dissolved oxygen depletion in the summer (Table 1) retard fish growth and may force rainbow trout into thermoclinal areas (Babey, 1983, Shrader, 1988), thus promoting lernaeosis.

Angler use exceeds 300 angler-days/ha/yr; one half of which is in June. Stocked rainbow trout comprise most of the catch; native rainbow trout, brown trout (*S. trutta*), cutthroat trout (*O. clarki*) and kokanee salmon (*O. nerka*) are rarely caught. Also present are redside shiners (*Richardsonius balteatus*), Utah suckers (*Catostomus ardens*) and fathead minnows (*Pimephales promelas*).

Over a period of 4 yr (1981 to 1984), about 300,000 fingerling rainbow trout were stocked each year in May. In 1981 and 1982, equal numbers of fish from three strains (Shepherd of the Hills, Ten Sleep, and Sand Creek) were stocked; in 1983 and 1984, the strains stocked were Ten Sleep, Kamloops, and McConaughy. In 1981, fish were reared at hatcheries in Midway (Utah 84049, USA) and Loa (Utah 84747, USA); in other years all fish came from the Loa hatchery. The McConaughy strain was stocked nearly 2 mo after the other strains and, because of their small relative size when captured, are not addressed in this report. Rearing and stocking conditions were similar each year (Babey, 1983; Shrader, 1988). Stocked fish were marked with fluorescent pigment in order to distinguish them by strain and from the few native rainbow trout.

Gill nets, each with five mesh sizes (13, 19, 25, 32, and 38 mm; bar measure), were used to recover fish approximately monthly from May until November 1981 to 1984. Nets were set on the bottom (0.5 to 20 m), perpendicular to shore, and fished overnight. Individual captured fish were identified to strain with the aid of an ultraviolet light, weighed (g), measured (mm, total length), and their external surfaces were examined for anchorworms.

Anchorworms are 4 to 8 mm long and can be counted without the aid of magnification. Cutaneous lesions about 4 mm in diameter were assumed to be sites of parasite attachment and were included in the count because parasites were sometimes dislodged when the fish were being removed from nets. The gill chamber was also inspected, but anchorworms were rarely found in the gill chamber (Roberts, 1978). Anchorworms were counted by body region (Fig. 2) in 1982. The average area of four body regions of five rainbow trout was determined from gyotaku prints (McAllister, 1986) and used to determine the expected number of copepods attached to each body region for Chi-square analysis.

From fish collected in August, September and October 1982, we randomly selected 15 fish for histological examination of lesions and scars caused by anchorworms. The attachment site and surrounding tissue were excised and fixed in either 10% buffered neutral formalin or Bouin's fixative. Samples were dehydrated in alcohol, infiltrated with paraffin and sectioned with a rotary microtome. Sections that showed the maximum lesion depth and ulcer diameter were chosen for staining with hematoxylin and eosin. An ocular micrometer was used to measure ulcer diameter (amount of interrupted dermis) and depth of the inflammatory reaction.

In 1984, secondary bacterial infection was monitored four times during the lernaeosis outbreak: (1) at stocking, (2) when anchorworms began to appear on fish, (3) at the height of the epizootic and (4) after infection intensity had declined. Instruments were sterilized by flaming following their immersion in isopropyl alcohol. Fish were held in ice water for <3 hr after netting, after which the body surface was disinfected by swabbing it with a concentrated iodine/isopropyl alcohol solution. The posterior kidney was exposed by cutting dorso-ventrally just posterior to the dorsal fin. A tissue sample from the kidney was taken to inoculate media plates of trypticase soy agar. After incubation for 48 h at 20 C, the plates were examined for colony growth. If a single-colony morphology was observed, a sample from an isolated colony was Gram-stained; otherwise, the plate was restreaked until a pure culture was obtained. Presumptive identification was made by analysis of bacterial morphology, and reactions to Gram stain, cytochrome oxidase test and O/F glucose test (Bullock, 1971).

FIGURE 1. Rainbow trout infected with lernaeosis. Closed arrows indicate typical lesions caused by the parasitic copepod *Lernaea cyprinacea* (anchorworm); open arrows, scale loss from gill nets.

We determined anglers' awareness of the disease and their acceptance of diseased fish by having them circle responses to two written questions. The questions and possible responses were: (1) Do you notice sores on fish? (responses were yes or no), and (2) What do you do with the fish that have sores? (responses were release the fish, clean and eat as usual, special cleaning and eat, keep and discard, other). We surveyed all anglers (n = 67) fishing at East Canyon Reservoir on 6 and 11 September 1982 and on 11 and 27 November 1982.

RESULTS

Anchorworm intensity varied significantly among strains on only four occasions, so data were pooled for all strains to show trends (Babey and Berry, 1989). Anchorworms were first seen on stocked rainbow trout in July of three years, and in September in 1983 (Fig. 3). The highest intensity occurred in 1982 when rainbow trout were infected with a mean of 19 anchorworms each (range 4 to 81, n = 102).

Dorsal and caudal areas had signifi-

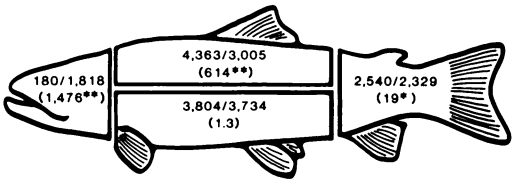

FIGURE 2. Rainbow trout body outline showing four body areas in which the number of anchorworms were counted. Anchorworm densities on each region are shown as: observed/expected (Chi-square value); *, significant at $P \leq 0.05$; **, significant at $P \leq 0.01$.

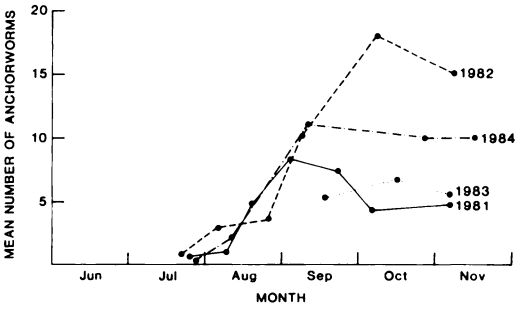

FIGURE 3. Mean number of anchorworms on rainbow trout stocked in May of four years in East Canyon Reservoir, Utah. Sample size on each date ranged from 10 to 102 fish. Data are shown only for dates when anchorworms were first detected and thereafter; prior dates when no anchorworms were undetected were omitted for clarity.

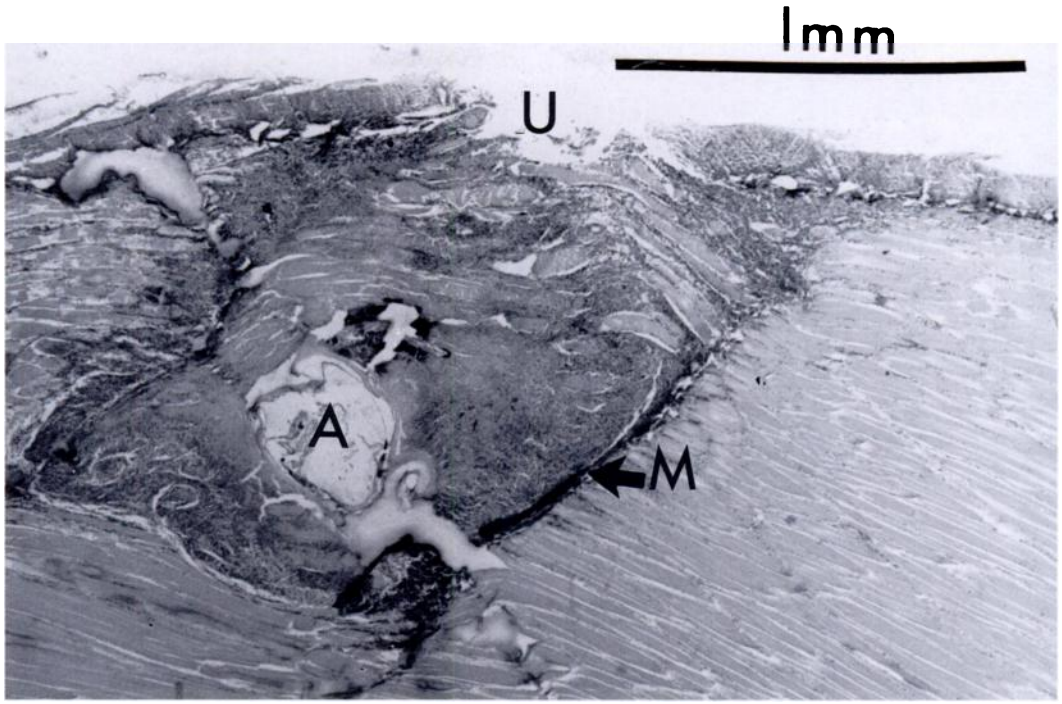

FIGURE 4. Histological section of rainbow trout skin and underlying muscle showing a lesion and ulcer (U) caused by the anchorworm parasite. Inflammation around a horn of the anchor process (A) is limited by the myosepta (M). H&E.

cantly more parasites than was expected if anchorworms had attached randomly (Fig. 2). The bases of the dorsal and anal fins were common attachment sites, whereas attachment to the head was less frequent than expected.

Ulcerations were not observed around some parasites that we presumed had recently attached, but ulcers were commonly observed with and without an attached parasite. The loss of scales and skin resulted in circular ulcers that averaged about 1.4 mm (diameter) in August, 2.8 mm in September and 3.8 mm in October. Gross and microscopical examination of lesions did not reveal evidence of fungi.

TABLE 2. Prevalence and intensity of anchorworms on the skin, and bacteria in the kidney of rainbow trout that were stocked in East Canyon Reservoir, Utah, in May 1984.

Date	Number of fish examined	Anchorworm		Number of fish with bacteria (%)
		Prevalence	Intensity (SD)	
May 16	30	0	0	0 (0)
July 25	30	30	0.5 (0.6)	11 (37)
September 11	25	25	8.24 (21.4)	6 (24)
November 20	20	20	4.5 (22.0)	9 (45)

The embedded anchors were surrounded by fibrous granulation tissue, and there was considerable leucocytic response below the dermis (Fig. 4). Myofibril degeneration and hemorrhage were noted in most sections, but these reactions were not extensive. Bacteria were occasionally seen in histological sections. Fish stocked in 1984 were initially free of bacteria in the kidney but bacteria appeared in the kidney when parasites appeared on the skin (Table 2). Bacteria were presumptively identified as a species of the genus *Aeromonas*.

The number of ulcers declined and the number of scars increased in the fall. Scars were whitish, scaleless pockets that could not be penetrated by gentle probing with

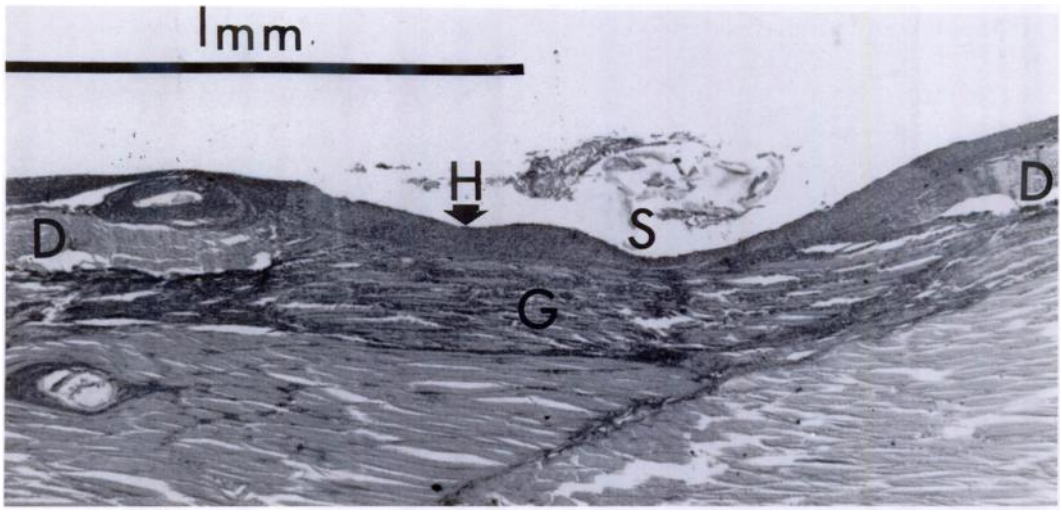

FIGURE 5. Histological section of rainbow trout skin and underlying muscle showing a scar (S) left by the anchorworm parasite. Note the hyperplastic epitheliam (H) and partially repaired dermal collagen layer (D) of the skin over the remaining granuloma (G). H&E.

a blunt instrument. Microscopic examination revealed epidermal regeneration (Fig. 5). Dermal collagen bridged the wound below the basal epidermal tissue, but was thinner than that in the adjacent stratum compactum layer. The amount of granulation tissue under scars was less than that observed in lesions. Melanocytes were scattered throughout the fibrotic tissue but were not abundant. Muscle bundles under most scars appeared normal.

Most anglers (94%) noticed sores on rainbow trout when infection with copepods was highest; about 9% released fish. Most anglers released fish because the fish were unsightly. Of the anglers that kept fish, 28% used special cleaning techniques to remove the parasites, but 49% used no special cleaning techniques. Only 8% of the anglers kept and later discarded infected fish; 15% checked "other uses" but we did not inspect the survey forms in the presence of the angler and therefore we do not know what these other uses were.

DISCUSSION

The importance of lernaeosis to managers of the East Canyon fishery appears to be negligible, although the external appearance of fish in late summer and autumn sometimes causes concern among managers and some anglers. The disease was probably not an important cause of mortality in fingerlings stocked in the spring because fish had grown to at least 160 mm in length when first parasitized (Babey, 1983; Shrader, 1988). At this size, fish were probably too large for the parasite to penetrate vital organs.

Year-to-year differences in water temperature may influence the onset and intensity of copepod attachment. For example, the highest intensity occurred in 1982 when water temperatures averaged about 2 C warmer during the summer than in other years. Also, the latest onset of the disease occurred in 1983 when reservoir warming was delayed by runoff from snow packs that were 186% of normal.

Anchorworm attachment on rainbow trout was not random, as also reported by others (McNeil, 1961; Damaree, 1967; Eisen, 1978; Bulow et al., 1979). There is less attachment in the head region than in other regions because copepodids mature and mate on the gills, and females move to other body areas for permanent attachment. The ectosteal bones just beenath the skin of the head probably limit attachment.

Pamphlets and articles designed to help anglers identify fish parasites and to answer questions about fish acceptability for human consumption are frequently produced for public education and information (e.g., Rogers and Plumb, 1979; MSG, 1982; Hoffman and Mitchell, 1986). One goal of such information is to reduce the waste of fish that are caught. We concluded that the need for such a publication about the East Canyon fishery should be a low priority because anglers wasted only a few fish, even when parasite intensity was greatest. Fortunately, this period occurred when fishing pressure was low, further minimizing waste. Most of the fishing pressure at East Canyon Reservoir was in June, before fish appearance was noticeably affected. During numerous creel censuses in June, we observed that almost all caught fish were kept.

Tissue response of rainbow trout to attachment by anchorworm was similar to that reported for other fish (Haley and Winn, 1959; Khalifa and Post, 1976; Lester and Daniels, 1976; Shields and Goode, 1978). The granulomatous response was also similar to that caused by *L. crucinata* in white bass (*Morone chrysops*) (Joy and Jones, 1973), and to that caused by adults of the family Lernaeoceridae (Radhakrishnan and Nair, 1981). The reports differ concerning the extent of graulocytic response to parasitism, and the extent of the damage to nearby muscles.

Few authors have reported on the healing of parasite-caused wounds. We could not determine the age of scars and thus could not definitely describe the sequence of histological events in the healing process. However, the healing that we observed was similar to that of experimentally wounded rainbow trout (Ashley et al., 1975) and channel catfish (*Ictalurus punctatus*) (Marty and Summerfelt, 1990). Typically, wounds are plugged in hours by epithelialization, followed by pro-collagen development of the dermis within days (Roberts, 1975; Roberts and Bullock, 1976). Most scars that we served were covered or almost covered by repair tissue.

Bacteria of the genus *Aeromonas* were present in the kidney of some fish, but their connection with lernaeosis could not be clearly established. Bacteria from the environment may continuously enter tissues of healthy fish (Bullock and Snieszko, 1969), and external parasites probably facilitate entry (Cusack and Cone, 1986). Aeromonads are among the most common bacteria in freshwater and are frequently associated with septicemias in fishes (Cipriano et al., 1984).

We did not observe moribund fish or fish with symptoms that can be caused by gram-negative, motile, aquatic bacteria (Snieszko and Bullock, 1976) and concluded that disease was not an important mortality factor in stocked rainbow trout in East Canyon Reservoir. However, if water quality or temperature conditions degrade, environmental stresses may combine with the disease complex to cause fish mortalities (Wedemeyer et al., 1976; Sinderman, 1979). This situation has occurred in fish infected with *Epistylis* sp. and *Aeromonas hydrophila* (Esch et al., 1976).

Lernaeosis in this rainbow trout population was similar to that described in populations of other fish species. The disease had little effect on angler acceptance of creeled fish, and should be of only minor concern to managers because it did not affect survival of stocked fish. Management attention should be given to preserving environmental quality.

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LITERATURE CITED

ASHLEY, L., M. J. E. HALVER, AND R. R. SMITH. 1975. Ascorbic acid deficiency in rainbow trout and coho salmon and effects on wound healing. *In*

The pathology of fishes, W. E. Ribeli and G. Migaki (eds.). The University of Wisconsin Press, Madison, Wisconsin, pp. 769–786.

BABEY, G. J. 1983. Evaluation of three strains of rainbow trout stocked in a reservoir where the ectoparasite *Lernaea cyprinacea* is endemic. M.S. Thesis. Utah State University, Logan, Utah, 83 pp.

———, AND C. R. BERRY. 1989. Post-stocking performance of three strains of rainbow trout in a reservoir. North American Journal of Fisheries Management 9: 309–315.

BULLOCK, G. L. 1971. Diseases of fish, Book 2B: Identification of fish pathogenic bacteria. TFH Publications, Jersey City, New Jersey, 41 pp.

———, AND S. F. SNIESZKO. 1969. Bacteria in blood and kidney of apparently healthy hatchery trout. Transactions of the American Fisheries Society 98: 268–271.

BULOW, F. J., J. R. WINNINGHAM, AND R. C. HOOPER. 1979. Occurrence of the copepod parasite *Lernaea cyprinacea* in a stream fish population. Transactions of the American Fisheries Society 108: 100–102.

CIPRIANO, R. C., G. L. BULLOCK, AND S. W. PYLE. 1984. *Aeromonas hydrophila* and motile aeromonad septicemias of fish. Fish Disease Leaflet 68, Division of Fishery Research, U.S. Fish and Wildlife Service, Washington, D.C., 23 pp.

CUSACK, R., AND D. K. CONE. 1986. A review of parasites as vectors of viral and bacterial diseases of fish. Journal of Fish Diseases 9: 169–171.

DAMAREE, R. S., JR. 1967. Ecology and external morphology of *Lernaea cyprinacea*. American Midland Naturalist 78: 416–427.

DEMPSTER, R. P., P. MORALES, AND F. X. GLENNON. 1988. Use of sodium chlorite to combat anchorworm infestations of fish. Progressive Fish-Culturist 50: 51–55.

EISEN, S. 1978. Ecology of *Lernaea cyprinacea*. Ph.D. Dissertation. Indiana University, Bloomington, Indiana, 120 pp.

ESCH, G. W., T. C. HAZEN, R. V. DIMOCK, AND J. GIBBONS. 1976. Thermal effluent and the epizootiology of the ciliate *Epistylis* and the bacterium *Aeromonas* in association with centrarchid fish. Transactions of the American Microscopical Society 95: 687–693.

HALEY, A. J., AND H. E. WINN. 1959. Observations on a lernaean parasite of freshwater fishes. Transactions of the American Fisheries Society 88: 128–129.

HOFFMAN, G. L. 1973. The effect of certain parasites on North America freshwater fishes. Verhandlungen-internationale Vereiigung Fuer Theoretische und Angewandte Liminologie 18: 1622--1627.

———. 1976. Parasites of freshwater fishes. IV. Miscellaneous. The anchor parasite (*Lernaea elegans*) and related species. Fish Disease Leaflet 46, U.S. Fish and Wildlife Service, Washington, D.C., 8 pp.

———, AND A. J. MITCHELL. 1986. Some parasites and diseases of warmwater fishes. Fish and Wildlife Leaflet 6, U.S. Fish and Wildlife Service, Washington, D.C., 22 pp.

JOY, J. E., AND L. P. JONES. 1973. Observation on the inflammatory response within the dermis of a white base, *Morone chrysops* (Rafinesque), infected with *Lernaea cruciata* (Copepoda: Caligidea). Journal of Fish Biology 5: 21–23.

KABATA, Z. 1970. Diseases of fishes, Book I: Crustacea as enemies of fishes. TFH Publications, Neptune City, New Jersey, 171 pp.

KHALIFA, A. K., AND G. POST. 1976. Histopathological effect of *Lernaea cyprinacea* (a copepod parasite) on fish. Progressive Fish-Culturist 38: 110–113.

LESTER, R. J. G., AND B. A. DANIELS. 1976. The eosinophilic cell of the white sucker, *Catostomus commersoni*. Journal of Fisheries Research Board of Canada 33: 139–144.

MARTY, G. D., AND R. C. SUMMERFELT. 1990. Wound healing in channel catfish by epithelialization and contraction of granulation tissue. Transactions of the American Fisheries Society 119: 145–150.

MCALLISTER, D. E. 1986. Technique of making fish illustrations, 16: Gyotaku, direct prints from fish specimens. Environmental Biology of Fishes 15: 90.

MCNEIL, P. O., JR. 1961. The use of benzene hexachloride as a copepodicide and some observations on lernaean parasites in trout rearing units. Progressive Fish-Culturist 23: 127–133.

MERRITT, L. B., A. W. MILLER, R. N. WINGET, S. R. RUSHFORTH, AND W. H. BRIMHALL. 1980. East Canyon Reservoir-water quality assessment. Mountainland Association of Governments, Provo, Utah, 193 pp.

MSG (MINNESOTA SEA GRANT) 1982. Parasites, are the fish good enough to eat? Sea Grant Extension Program, University of Minnesota, Duluth, Minnesota, 11 pp. (pamphlet).

RADHAKRISHNAN, S., AND N. B. NAIR. 1981. Histopathology of the infestation of *Diodon hystrix* L. by *Peniculisa wilsoni* Radhakrishnan (Copepoda: Lernaeoceridae). Journal of Fish Diseases 4: 83–87.

ROBERTS, R. J. 1975. The effects of temperature on diseases and their histological manifestations in fish. *In* The pathology of fishes, W. E. Ribelin and G. Migaki (eds.). The University of Wisconsin Press, Madison, Wisconsin, pp. 477–497.

———. (editor). 1978. Fish pathology. Lea and Febiger, Philadelphia, Pennsylvania, 318 pp.

———, AND A. M. BULLOCK. 1976. The dermatology of marine teleost fish. II. Dermatopathology of the integument. Oceanography and Marine Biology Annual Review 14: 227–246.

ROGERS, W. A., AND J. A. PLUMB. 1979. Principal diseases of sportfish. A fisherman's guide to fish parasites and diseases. Agricultural Experiment Station, Auburn University, Auburn, Alabama, 16 pp. (pamphlet).

SHIELDS, R. J., AND R. P. GOODE. 1978. Host rejection of *Lernaea cyprinacea* L. (Copepoda). Crustaceana 35: 301–307.

SHRADER, T. M. 1988. Performance of three strains of rainbow trout in East Canyon Reservoir. M.S. Thesis. Utah State University, Logan, Utah, 75 pp.

SINDERMAN, C. 1979. Pollution associated diseases and abnormalities of fish and shellfish: a review. U.S. Fishery Bulletin 76: 717–749.

SNIESZKO, S. F., AND G. L. BULLOCK. 1976. Diseases of freshwater fishes caused by bacteria of the genera *Aeromonas*, *Pseudomonas*, and *Vibrio*. Fish Disease Leaflet 40, Division of Cultural Methods Research, U.S. Fish and Wildlife Service, Washington, D.C., 10 pp.

UZMANN, J. R., AND H. J. RAYNER. 1958. Record of the parasitic copepod *Lernaea cyprinacea* L. in Oregon and Washington fishes. The Journal of Parasitology 44: 452–453.

WEDEMEYER, G. A., F. P. MEYER, AND L. SMITH. 1976. Diseases of fishes. Book 5: Environmental stress and fish diseases, S. F. Snieszko and H. R. Axelrod (eds.). TFH Publications, Inc., Jersey City, New Jersey, 192 pp.

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