PREVALENCE AND EFFECTS OF INFECTION OF THE DORSAL AORTA IN YELLOWFIN TUNA, *THUNNUS ALBACARES*, BY THE LARVAL CESTODE, *DASYRHYNCHUS TALISMANI*

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ABSTRACT

Approximately 60% of small (<3 kg) yellowfin tuna, *Thunnus albacares*, caught near the Hawaiian Islands carry the plerocercoid (larval) stage of the cestode (tapeworm), *Dasyrhynchus talismani*, in their anterior dorsal aortas. Because the worms and the resultant host inflammation appear to occlude the vessel almost totally, we assumed that the parasite could increase natural mortality rates. Tuna could be limited in their ability to capture prey and therefore should show evidence of long- or short-term food deprivation. We measured body weight, fork length, liver weight, heart weight and, in fish captured from one school, RNA/DNA ratios (a measure of short-term growth rate), and otolith weight (a measure of long-term growth rate) from parasitized and unparasitized fish. We found no significant differences between infected and uninfected fish nor any evidence of starvation in infected fish. How small yellowfin tuna remain apparently unaffected by the parasitic occlusion of their dorsal aorta remains to be demonstrated.

We also examined changes in incidence of infection in small yellowfin tuna caught between February 1985 and March 1986 as well as the prevalence in large (>45 kg) fish. Large yellowfin tuna were rarely parasitized (5.2%) in the dorsal aorta, but showed a high rate (>80%) of infection within other major arteries. The prevalence in small fish varied dramatically with season, dropping suddenly from 66% in June-July 1985 to 11% in August-September 1985. Unparasitized fish caught during August-September 1985 showed significantly higher condition factors, relative heart weights, and relative liver weights than did unparasitized fish caught at other times of the year. We hypothesize that the sudden decrease in prevalence was due to influx of a separate group of small yellowfin tuna into the Hawaiian fishery. We believe that this parasite may therefore serve as a marker for tracing the movements of small yellowfin tuna into and out of specific fisheries or areas.

During a series of experiments that involved catheterization of the anterior dorsal aorta of small (1-3 kg) yellowfin tuna, *Thunnus albacares*, we discovered that approximately 60% of the experimental fish had this blood vessel infected with parasites. The parasites were white, round (2-4 mm in diameter), often more than 4 cm long, and usually folded repeatedly. As a result of the parasites and the tissue inflammation that develops as part of host immune response, the lumen of the infected aortas appeared almost totally occluded. Because all the blood to the internal organs and swimming muscles must flow past this occlusion, we assumed the parasite could be a major factor contributing to the natural mortality of small yellowfin tuna.

The first demonstration of a dorsal aorta para-site in yellowfin tuna was by Kishinouye (1923), who stated, "Often a species of nematod [sic] is found in the dorsal aorta of *Neothunnus macropterus* [now *Thunnus albacares*]; the parasite causes the tissues of the canal to become thick and tough, giving it at the same time a yellowish tint." Other investigators described intravascular parasites from the branchial vessels and arteries serving the stomach, liver, spleen, pyloric caecum, and gall bladder of this species (Baudin Laurencin 1971). The parasites have been described simply as the plerocercoids (larval cestodes) (Chen and Yang 1973), identified to the family Dasyrhynchidae (Ward 1962), or as the species *Dasyrhynchus talismani* (Baudin Laurencin 1971).

Intravascular infection by plerocercoids has been reported from yellowfin tuna caught in the western Pacific (Chen and Yang 1973), eastern Atlantic (Baudin Laurencin 1971), Gulf of Mexico (Ward 1962), and now central Pacific (this report). Infection rates have been reported to be as high as 100% (Baudin Laurencin 1971). If all re-

ports involve the single parasite species, *D. talis-mani*, this parasite is both common and ubiquitous.

Because of the apparent vessel blockage by parasites and tissue inflammation, we hypothesized that infected fish would be severely activity limited and not function well as predators. If so, infected fish should show evidence of short- and/or long-term food deprivation, including lower relative condition factors, smaller livers, and slower long- and short-term growth rates (Bulow 1970; Pollard 1971, 1973; Bulow et al. 1981). We also expected blockage of the dorsal aorta to cause increased blood pressures, increased cardiac work, and therefore cardiac hypertrophy (Poupa and Ostadal 1969).

To test our hypotheses, we measured fork length, body weight, liver weight, and ventricle weight from parasitized and unparasitized fish. We also determined RNA/DNA ratios as a measure of the parasite's effects on short-term growth rates (Bulow 1970; Bulow et al. 1981), and relative otolith weights as a measure of the parasite's effects on long-term growth rates. Since otolith weights are linearly related to a fish's chronological age (Boehlert 1985), fish that are older at a given body size should have relatively heavier otoliths. We also recorded the prevalence of infection in small (<3 kg) and large (>45 kg) yellowfin tuna, and in skipjack tuna, *Katsuwonus pelamis*, kawakawa, *Euthynnus affinis*, and bigeye tuna, *T. obesus*. Additionally, monthly prevalence in small yellowfin tuna was noted.

To measure directly the effect of the occlusion caused by the parasites and host inflammation, we used an in vitro perfusion test. We measured the pressure required to push various saline flow rates down the dorsal aorta in freshly dead infected and uninfected fish.

MATERIALS AND METHODS

Sampling Procedures

The small tunas (0.2-3 kg body weight) used in this study were captured at sea near Oahu, HI, returned alive to be held in shoreside tanks at the Kewalo Research Facility (National Marine Fisheries Service, Southwest Fisheries Center Honolulu Laboratory), or sacrificed at sea and held on ice. During necropsy, fork length and body weight were measured, ventricles and livers were removed, blotted dry, and weighed to the nearest 10 mg. Only those fish in captivity for 3 days or less

were included in the data for condition factor, and relative heart and liver weights. Large yellowfin tuna (>45 kg body weight) were examined at the Honolulu Fish Auction for the presence of dorsal aorta parasites while the fish were being prepared for sale. All fish were examined within 24 hours of death.

Thirty-five live yellowfin tuna, from one school, were caught 15 January 1986 and transported alive to the Kewalo Research Facility. Immediately upon arrival, the animals were sacrificed, weighed, and measured. Lateral white muscle samples were taken within 4 minutes of death and immediately frozen on dry ice. These samples were subsequently used for measurement of RNA/DNA ratios using the Schmidt-Thannhauser procedure as described in Murno and Fleck (1966). The ventricles and livers of the fish were also removed, blotted dry, and weighed to the nearest 10 mg. Sagittal otoliths were removed, cleaned, dried, and weighed to the nearest microgram.

Parasites for species identification were obtained most often from the major artery within the spleen of large (>45 kg) yellowfin tuna. After being removed from a surrounding capsule, parasites were placed in tap water until the holdfasts everted.

For histological examination, sections of dorsal aorta were fixed in 10% buffered formalin and processed by routine laboratory procedures. Tissue sections were stained with hematoxylin and eosin.

Direct Measurement of Pressure-Flow Relationships in the Anterior Dorsal Aorta

To quantify blockage, we choose to measure the pressures required to push various flow rates of saline through the anterior dorsal aorta of infected and uninfected fish. The dorsal aorta of freshly killed fish was exposed from the confluence of the efferent arteries of the first and second gill arches to the point where it enters the first hemal arch. All efferent and afferent vessels were tied off except for the confluence of the efferent arteries from either the left or right first and second gill arches. This portion of the vessel was cut and a short length of flared polyethylene tubing (PE160, 2.4 mm OD) inserted. The dorsal aorta was also transected at the point where it entered the first hemal arch to allow the saline perfusate to flow out. Parasites were never found poste-

rior to this point. Saline perfusion pressures, at various constant flow rates provided by an infusion pump, were recorded via a Uonix[4] pressure transducer.

Calculation of Relative Condition Factor and Relative Organ Weights

Use of relative condition factor, and relative organ and otolith weights allow groups of fish containing individuals of a range of body sizes to be directly compared (Pollard 1972). Using data from unparasitized fish, regressions of body weight (g) on fork length (cm), liver weight (g) on body weight (g), and heart weight (g) on body weight, were fitted by a least squares technique to the exponential equation:

$$Y = a \cdot X^b$$

using a log-log transformation of the data. Relative condition factor and relative organ weights for individual fish were calculated using the regression parameters (a, b) with the equation:

$$K = W/a \cdot X^b.$$

For relative condition factor, W = body weight and X = fork length. For relative organ weights, W = liver or heart weight, and X = body weight.

The relationship of otolith weight (mg) to body weight was found best fit with the simple linear regression:

$$Y = a + (b \cdot X).$$

Relative otolith weights were therefore calculated using the equation:

$$K = W/(a + b \cdot X)$$

where W = otolith weight and X = body weight.

A relative condition factor <1 indicates that an individual is lighter for its fork length than predicted based on data from unparasitized fish. Similarly, a relative liver or heart weight <1 indicates a smaller liver or heart for a given body size than that found for unparasitized fish. A relative otolith weight >1 means that an individual experienced a relatively slower long-term growth rate

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[4]Reference to trade names does not imply endorsement by the National Marine Fisheries Service, NOAA.

(i.e., is relatively older for a given body size and therefore has a larger otolith).

RESULTS

Identification of the Parasite

Tapeworms (class: Cestoda, order: Trypanorhyncha) can be identified to species based on scolex morphology and tentacular hooks (onchotaxy), mature segments are not required. The larval cestodes recovered from the yellowfin tuna during this study showed proboscis chainettes flanked by a single row of intercalary hooks, a characteristic that distinguishes *Dasyrhynchus talismani* from its congeners.

This parasite was originally described from five mature worms removed from the spiral valve of *Galeus glaucus* (= *Prionace glauca*, the blue shark) off Cape Verde, West Africa (Dollfus 1935). *Dasyrhynchus talismani* has also been reported in the Pacific from *Carcharinus longimanus* (Heinz and Dailey 1974). All other reports describe plerocercoids from the vascular systems of teleost fishes (Bussieras and Aldrin 1965; Baudin Laurencin 1971; Chen and Yang 1973).

Prevalence of Infection by Host Species, Fish Size, and Season

A total of 53 skipjack tuna, 27 kawakawa, 10 bigeye tuna, and 470 yellowfin tuna were examined for the presence of parasites. We found only yellowfin tuna to be infected.

Infection in yellowfin tuna varied with size class. We found a significantly lower incidence of dorsal aorta infection in large fish. Of 220 individuals weighing 0.21 to 2.7 kg, 48% were infected, while of 250 fish weighing more than 45 kg, only 5.2% carried the parasite in their dorsal aortas. Viscera of a small subsample ($N = 8$) of the larger fish were examined and indicated that in larger fish the parasite infects (in the order of prevalence) the major arteries of the spleen, intestinal caeca, liver, mesenteries, and lateral blood vessels. Fish in intermediate size classes were not available for this study. Fish >3 kg do not survive the trip from the fishing grounds to the Kewalo Research Facility and are therefore not normally captured by commercial fishermen for return to the laboratory. Fish <45 kg are not common at the Honolulu Fish Auction where they could be examined during normal processing. The

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purchase of intermediate-sized yellowfin tuna specifically for this study (yellowfin tuna intended for market cannot be necropsied and then sold) was prohibitively expensive.

Infection of yellowfin tuna appears to vary seasonally. Figure 1 shows changes in prevalence of dorsal aorta infection in small (<3 kg) yellowfin tuna captured between February 1985 through March 1986. Prevalence remained stable for approximately 6 months during the winter through early summer. Then in late summer of 1985, prevalence dropped dramatically from 66 to 11%. Beginning in October 1985, prevalence increased steadily, reaching 39% in February-March 1986, the last months for which data are available.
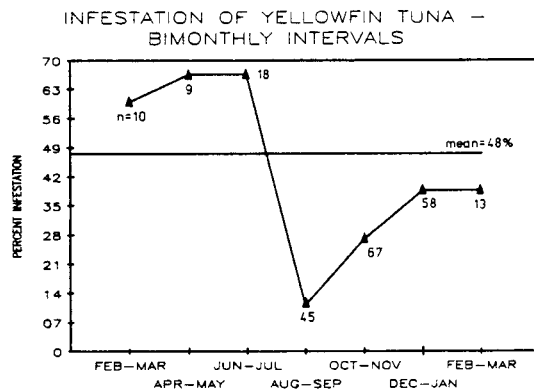

FIGURE 1.—Prevalence (percent infection) of the dorsal aorta by the plerocercoid stage of the cestode *Dasyrhynchus talismani* in small (0.3-3 kg) yellowfin tuna caught near the Hawaiian Islands from February 1985 to March 1986.

Pathology

In infected fish, the anterior dorsal aorta was partially to nearly completely occluded by a parasitic embolus which contained one to several larval cestodes. Figure 2a shows this vessel in a moderately infected yellowfin tuna. Parasites and a small amount of host inflammation are evident in the anterior end (to the right). A normal portion of vessel, with a smooth wall, is seen to the left. Figure 2b shows the anterior dorsal aorta from a very heavily infected fish.

Histological examination revealed that parasitic emboli were primarily composed of larval cestodes, mononuclear cells with eosinophilic granules (presumed to be eosinophils), epitheloid cells (histocytes), fibroblasts, and collagen fibers (Fig. 3). Larval cestodes within the dorsal aorta

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were associated with a chronic severe endarteritis and, to a lesser extent, mesoarteritis. In heavily infected fish, collapsed channels were commonly found within emboli. Undoubtedly these channels expanded in life with increases in intraluminal blood pressure to allow blood to flow through the vessel. Necrotic worms were also seen, suggesting that the host's defense system was at least partially capable of killing the larvae located in the dorsal aorta.

Infection of other arteries was usually by one, or at most two, larger parasites which were never seen to be folded. These parasites had a bulbous anterior end which always pointed downstream. No evidence of host immune response was observed when the parasites were in vessels other than the dorsal aorta.

Effect of Infection on Measures of Physiological Fitness

Mean (±1 SD) relative condition factor and mean (±1 SD) relative organ weight data are given in Table 1 for the fish sampled between May 1985 and September 1986. Table 2 lists the same parameters plus mean relative otolith weight, and mean RNA/DNA ratio for fish sampled from the single school captured on 15 January 1986. Means were compared with Student's two-tailed T test, with $P = 0.05$ taken as the minimum level for statistical significance.

Examination of data from all the fish caught May 1985-September 1986 reveals statistically significant differences in relative liver and heart weights for parasitized and unparasitized fish. On the average, parasitized fish, have 15% smaller livers, and 9% *smaller* hearts than unparasitized fish. Relative condition factors were not significantly different. When data from fish coming from the single school are examined, there are no statistically significant differences in these three parameters, relative otolith weight, or in RNA/

TABLE 1.—Relative condition factor, relative liver weight, and relative heart weight, of yellowfin tuna sampled between May 1985 and September 1986.

Fish	Mean (±SD) relative condition factor	Mean (±SD) relative liver weight	Mean (±SD) relative heart weight
Uninfected	1.00 (±0.10) N = 109	1.02 (±0.22) N = 82	1.01 (±0.18) N = 88
Infected	0.976 (±0.057) N = 35	0.867 (±0.098)* N = 22	0.919 (±0.085)* N = 32

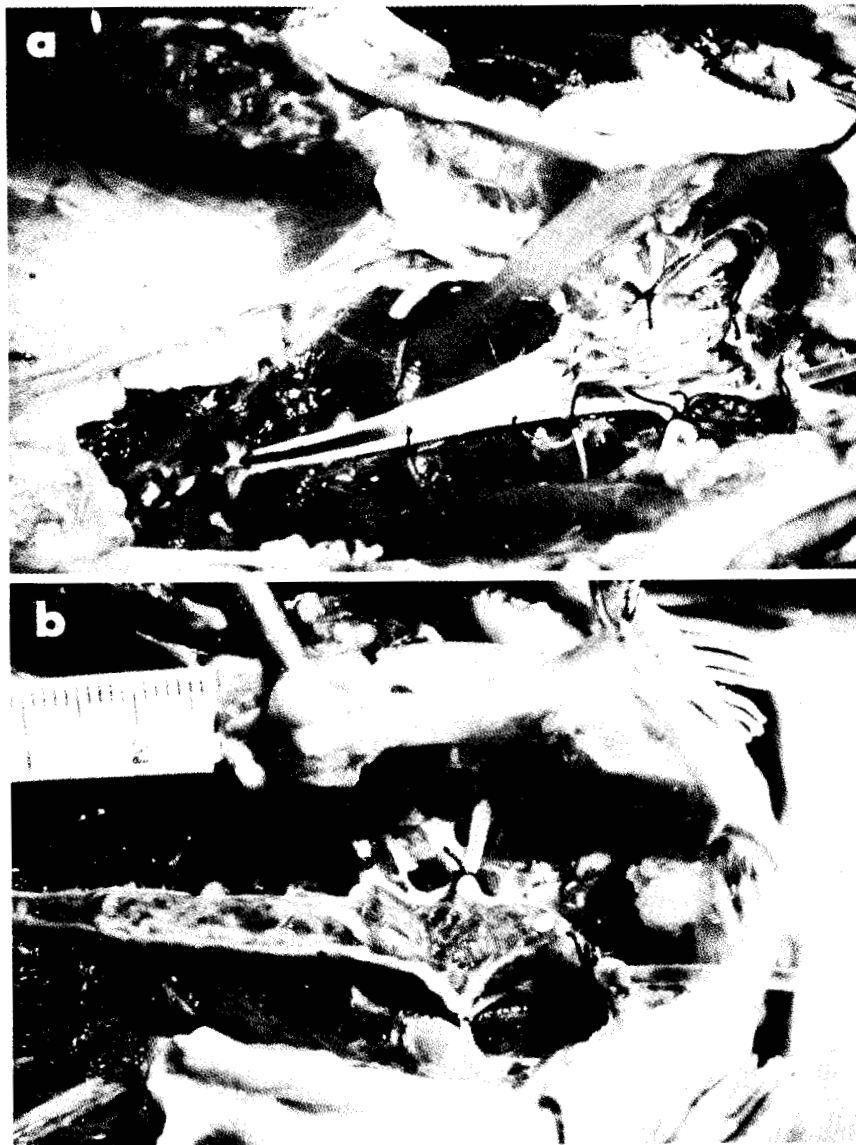*Uninfected and infected groups different at $P = 0.05$ level.

FIGURE 2a.—The anterior dorsal aorta of a moderately infected yellowfin tuna. In the anterior end of the vessel (to the right) are parasites and host inflammation.

FIGURE 2b.—The anterior dorsal aorta of a heavily infected yellowfin tuna. The vessel's lumen is all but occluded by parasites and host inflammation. Ruler divisions are in mm.

TABLE 2.—Relative condition factor, relative liver weight, relative heart weight, relative otolith weight, and RNA/DNA ratio of yellowfin tuna sampled 15 January 1986.

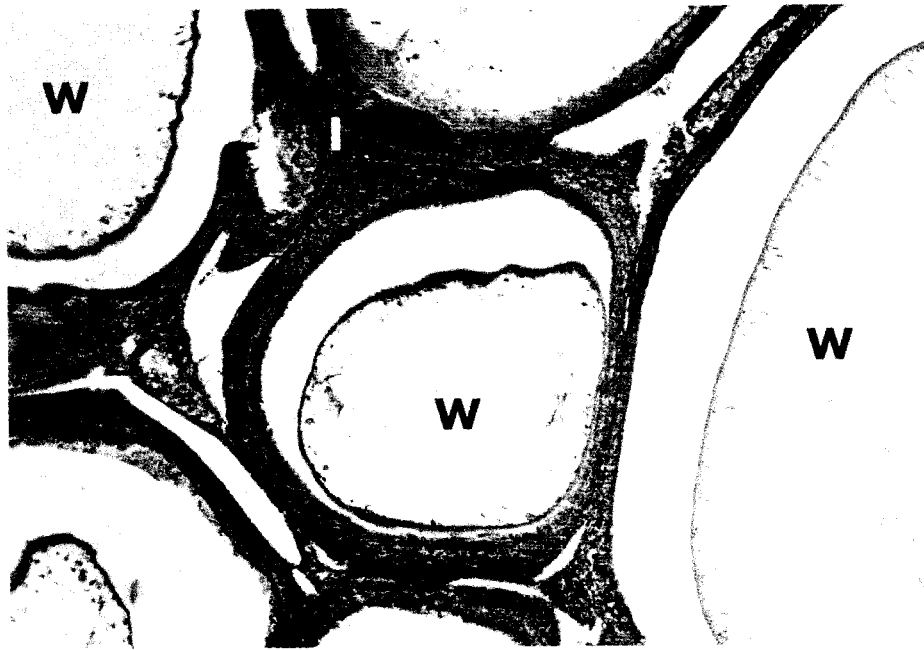| Fish	Mean (±SD) relative condition factor	Mean (±SD) relative liver weight	Mean (±SD) relative heart weight	Mean (±SD) relative otolith weight	Mean (±SD) RNA/DNA ratios
Uninfected	1.00 (±0.04) N = 23	1.00 (±0.08) N = 23	1.00 (±0.06) N = 23	1.00 (±0.04) N = 12	28.9 (±8.9) N = 21
Infected	0.995 (±0.035) N = 12	1.00 (±0.07) N = 12	0.975 (±0.074) N = 12	0.980 (±0.058) N = 8	33.1 (±11.1) N = 10

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FIGURE 3.—Photomicrograph of a cross section of the dorsal aorta containing parasites and host inflammation of a heavily infected yellowfin tuna. The host inflammation (I) and cross sections of the parasites (W) can be easily differentiated. Hematoxylin and eosin stain.

DNA ratios. RNA/DNA ratios were compared directly (rather than by calculating relative RNA/DNA ratios) because they were found not to be correlated with body weight (correlation coefficient = 0.04).

Data from *unparasitized* fish collected in August and September 1985 were analyzed separately from all remaining *unparasitized* fish. This was done because we found dramatically lower rates of infection (11%) during those 2 months than during the preceding 2 months (67% infected) and assumed this to be due to an influx

of a new group of small yellowfin tuna into the Hawaiian fishery. New regression parameters for body weight on fork length, heart weight on body weight, and liver weight on body weight were calculated using data from unparasitized fish excluding those caught during August-September 1985. Mean relative condition factors and relative organ weights were then recalculated.

Table 3 shows mean relative condition factors, mean relative liver weights, and mean relative heart weights for unparasitized fish captured during August and September 1985, unparasitized

TABLE 3.—Relative condition factor, relative liver weight, and relative heart weight from uninfected yellowfin tuna sampled during August and September 1985, from all other uninfected yellowfin tuna, and all infected fish.

Group	Mean (±SD) relative condition factor	Mean (±SD) relative liver weight	Mean (±SD) relative heart weight
August-September 1985 uninfected fish	1.11 (±0.08)* $N = 36$	1.24 (±0.28)* $N = 35$	1.22 (±0.18)* $N = 36$
All other uninfected fish	1.00 (±0.09) $N = 73$	1.01 (±0.17) $N = 47$	1.00 (±0.09) $N = 52$
Infected fish	1.01 (±0.06) $N = 35$	0.937 (±0.102) $N = 22$	1.00 (±0.09) $N = 32$

*Uninfected groups different at $P = 0.01$ level.

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fish captured at all other times of the year, and all parasitized fish, based on the new regression parameters. The two groups of *unparasitized* fish show statistically significant differences in all three. Fish caught during August and September were on the average 11% heavier at a given body size (i.e., relative condition factor = 1.11), had livers an average of 23% heavier, and had hearts an average of 22% heavier than those unparasitized fish captured at other times of the year. When unparasitized fish, excluding those caught during August and September, are compared with parasitized fish, there are now no statistically significant differences in mean relative condition factors, mean relative liver weights, or mean relative heart weights. The data from unparasitized fish captured in August and September, when included in the complete data set, are therefore responsible for the observed differences in relative heart and liver weights between infected and uninfected fish seen in Table 1.

In Vitro Perfusion of the Dorsal Aorta

Three parasitized and five unparasitized fish, ranging in weight from 0.915 to 2.666 kg, were used in this series of experiments. The intensity of infection was subjectively classified as slight, moderate, or heavy. Perfusion pressures were normalized to a 1 kg fish weight by dividing the observed pressures by the reciprocal of body
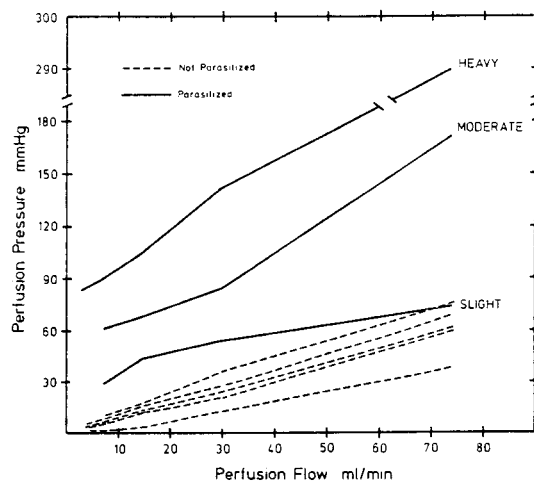

FIGURE 4.—In vitro pressure required to pump various rates of saline down the dorsal aorta of parasitized and unparasitized yellowfin tuna. The fish ranged in size from 0.915 to 2.666 kg. Data have been normalized to a body weight of 1 kg.

weight, in kilograms. Moderately and heavily infected fish showed higher perfusion pressures at a given flow rate than did unparasitized animals (Fig. 4). While no data are available on the normal cardiac outputs and blood pressures in swimming yellowfin tuna, restrained and lightly anesthetized yellowfin tuna have cardiac outputs of approximately 40-60 mL/kg and dorsal aorta blood pressures of 50-70 mm Hg (D. R. Jones and R. W. Brill, unpubl. obs.). Figure 4 shows that at normal cardiac outputs, the apparent occlusion of the dorsal aorta caused by the parasites and host inflammation is indeed real and should cause moderately and heavily infected fish to have excessively high blood pressures, high cardiac energy demands and presumably reduced fitness.

DISCUSSION

Prevalence of Infection by Species, Size, and Season

Our data indicate that in Hawaiian waters *D. talismani* is limited to yellowfin tuna. However, *D. talismani* has been reported to occur in Atlantic bigeye tuna (Bussieras and Aldrin 1965). Since we examined relatively few individuals of this species, we cannot rule out the occurrence of this parasite in bigeye tuna in the central Pacific.

Skipjack tuna, bigeye tuna, kawakawa, and yellowfin tuna often occur simultaneously in the same areas and show a great overlap in prey species (King and Ikehara 1956; Waldron and King 1963). It is therefore unlikely that host specificity is attributable to only yellowfin tuna ingesting the intermediate host, which is not known but is most likely a small crustacean (Deardorff et al. 1984). Host specificity could arise if the procercoid of *D. talismani* is not stimulated or is unable to penetrate the gut wall of tuna species other than yellowfin tuna, or that species of tuna other than yellowfin are capable of immune rejection (Orr et al. 1969).

The reasons for the dramatic decrease in incidence of dorsal aorta infection in large (>45 kg) yellowfin tuna are unknown. Possible reasons include procercoids ingested less frequently by larger animals, host destruction of the parasite, increased mortality of parasitized fish, or movement of the parasite out of the dorsal aorta into other major arterial vessels. Of these alternatives, increased mortality of infected fish seems unlikely.

While we did not demonstrate directly the pre-

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ence of antibodies against the parasite, several histological cross sections show worms in stages of degeneration, and host destruction of larval and adult cestodes has been shown in other teleosts (Kennedy and Walker 1969; Smith 1973; MacKenzie 1975). Examination of a small number of pyloric caeca, liver, spleen, lateral arteries, and stomach vasculature of large yellowfin tuna (>45 kg) showed >85% infection, suggesting 1) that the parasite may move out of the dorsal aorta into other large arteries as yellowfin tuna grow, 2) that the host response to the parasite may be less vigorous in vessels other than the dorsal aorta, and/or 3) that the parasite may preferentially select other vessels in larger fish. Baudin Laurencin (1971) found a decrease in branchial artery infection of yellowfin tuna with increasing body size, but no change in rates of infection of abdominal vessels.

The change in the rate of infection seen in August and September 1985 in small yellowfin tuna (Fig. 1) is, we believe, due to a large influx of uninfected fish into the Hawaiian fishery. Although we have no direct corroborating evidence, such as increased catch per unit effort for this size yellowfin tuna at that time, Tester and Nakamura (1957) have shown that there are repeated influxes of small yellowfin tuna into areas near the main Hawaiian Islands during late summer and early fall. Furthermore, the dramatic differences seen in relative condition factors, relative liver weights, and relative heart weights between unparasitized fish caught during August-September 1985, and the remaining unparasitized fish, clearly imply that the former group had a different history.

We have no evidence nor are we hypothesizing that these groups come from genetically isolated subpopulations. We do believe, however, that the two groups were separate most likely since hatching. We do not know the maximum lifespan of the dorsal aorta parasite, but one yellowfin tuna killed after 172 days in captivity at the Kewalo Research Facility was parasitized. Since fish in captivity are fed only frozen food and their tanks are supplied with filtered seawater (Queenth and Brill[5]), it is unlikely this fish became infected after capture. Yellowfin tuna of the 1-3 kg size range are about 270-360 days old (Uchiyama and

Struhsaker 1981) and therefore could have carried the parasite most of their lives.

The slow increase in prevalence from October 1985 through March 1986 remains to be explained, but could be due to emigration of the new group of yellowfin tuna out of the Hawaiian fishery or slowly increasing infection of the new group. This latter explanation implies higher prevalence of the parasite around islands which could be due either to a greater number of final (shark) or intermediate hosts around islands.

Pathology

The severe enarteritis associated with *D. talismani* infection suggests that the parasite is recognized by the fish's defense system as foreign material in the dorsal aorta. Dead worms within the inflammatory tissue imply that the parasite is not well adapted for survival in this location. Presumably, *D. talismani* would elicit, and be attacked by, a similar inflammatory response irrespective of its location within the vasculature. This response was not observed in other vessels and additional work is needed to clarify the site specificity of the immune response. Our findings also suggest that cellular elements are responsible for the destruction of the larval cestodes when located in the dorsal aorta.

Effect of the Infection of the Dorsal Aorta on Natural Mortality

We found no evidence to support our original hypothesis that infected yellowfin tuna are activity limited and therefore less able to secure food. When the data from the unparasitized fish caught August-September 1985 are excluded, there are no differences in relative condition factors, relative liver weights, or relative heart weights between parasitized and unparasitized fish. When parasitized and unparasitized fish from the single school caught 15 January 1985 are compared, no significant differences in these parameters, mean short-term (i.e., RNA/DNA ratios), or mean long-term (i.e., relative otolith weights) growth are evident.

Because parasite emboli appear to cause almost complete occlusion of the anterior dorsal aorta (the only blood vessel supplying the viscera and swimming muscles), the lack of differences between infected and uninfected fish was not expected. Overstreet (1977), investigating the effects of plerocercoid infection on sciaenid fishes in

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[5]Queenth, M. K. K., and R. W. Brill. 1983. Operations and procedures manual for visiting scientists at the Kewalo Research Facility. Southwest Fish. Cent. Admin. Rep. H-83-7, 16 p. Natl. Mar. Fish. Serv., NOAA, Honolulu, HI.

the Gulf of Mexico, found no apparent detrimental effect on the host, but these parasites were found encysted in the muscle, not the vasculature. Although the ability of parasitized yellowfin tuna to function as predators are apparently not affected, their ability to escape predation remains to be tested. It is possible that infected yellowfin tuna are subjected to differential predation, as has been shown to be for roach, *Rutilus rutilus*, infected in the coelomic cavity by the plerocercoid of *Ligula* sp. (Van Dobben 1952).

Pressure Flow Relationships in the Dorsal Aorta

The unphysiologically high pressures required to pump saline through the dorsal aortas of moderately and heavily infected fish remain to be explained in light of apparent lack of effects of the parasite on other measures of the fish's condition, including the absence of cardiac hypertrophy. It is possible that the dorsal aorta, because of its thick muscular wall (J. Brock, unpubl. obs.), becomes significantly less compliant postmortem. Such changes could require that higher pressures be generated to create a given degree of expansion. Therefore higher pressures would be required postmortum to push a given flow rate of saline through the vessel.

In summary, *D. talismani* appears to have no significant adverse effects on the physiological fitness and natural mortality of small yellowfin tuna in spite of apparent vascular blockage. How these fish are able to cope with dorsal aorta infection requires further investigation.

Use of *Dasyrhynchus talismani* as a Natural Tag for Tracing Movements of Small Yellowfin Tuna

We feel that this parasite offers excellent potential as a natural marker for tracking the movements of separate groups of small yellowfin tuna between or into specific fisheries. (For a review of the use of parasites to delineate stocks for management purposes, see MacKenzie 1983.) *Dasyrhynchus talismani* does not fulfill all seven requirements for an ideal natural tag listed by Sindermann (1983), but it does appear to meet the requirements of 1) having significant differences of geographic prevalence, 2) being easily detected, 3) being able to be definitively identified, 4) having minimum effect on host survival, and 5) surviving in the host for long periods. Data on preva-

lence of *D. talismani* could be combined with data on prevalence of other parasites, as has been shown by Lester et al. (1985) for skipjack tuna, or combined with data on relative condition factor, relative heart weight, and relative liver weight to provide information on fish movements.

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