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Parasitology

ADULT CESTODE INFECTION OF THE MARINE TELEOST FISH *SAURIDA TUMBIL* (BLOCH)

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Nature of infestation of *Saurida tumbil* by the adult cestodes *Penetrocephalus ganapatii* and *Bothriocephalus indicus* is presented. A detailed study of *P.ganapatii* infection has been carried out and the results regarding histopathological studies, incidence and intensity of infection, effect of infection on body weight, liver weight, hepato-somatic index, peripheral blood and proximate composition of liver and white muscle are presented. The incidence and intensity of infection very significantly with season and with growht of the fish. Histopathological changes indicate that the infected fish become anaemic. The anaemia is of the macrocytic type. Infection causes depletion of the basic energy reserves in the liver and white muscle of the fish.

Since *B.indicus* exclusively occurs in fish infected with *P.ganapatii* and since the intensity of infection in very low, the actual damage caused by this worm is not clearly understood.

INTRODUCTION

Adult cestodes are not uncommon parasites in the digestive tract of fishes. However, compared to larval stages, adult helmint in general and cestodes in particular, are looked upon as of not having much adverse effect to their fish hosts. Nevertheless, quite a few reports on the pathogenicity of cestodes on fishes are available (Wiśniewski, 1932; Vik, 1957; Williams, 1960, 1967; Musselius at al., 1963; Chubb, 1964; Arme a.Owen, 1965; Reichenbach-Klinke a.Elkan,1965; Meyer a.Vik, 1968; Lien, 1970; Sindermann, 1970, Bylund, 1972; Bauer et al., 1973; Duijn, 1973; Korting, 1975; Ribelin a.Migaki,1975; Scott a. Grizle, 1979). But often the pathology of infection is reportedonly in very general terms and the exact nature of damage to fish hosts by adult cestodes is not fully examined. The present paper is an attempt to bring out the different aspects of pathology of infection of the edible marine teleost fish *Saurida tumbil* (Bloch) by the adult cestodes *Penetrocephalus ganapatii* Rao and *Bothriocephalus indicus* Ganapati et Rao.

MATERIALS AND METHODS

Specimens of *S. tumbil* were collected from shallow-water trawls operating off the South-west (Trivandrum) coast of India. During July to November 1976, during which period the fish were available along this coast, 270 specimens were examined for parasites and diseases. The standard length, body weight, liver weight and the sex of each fish were recorded and the fish were dissected, and their intestines cut open, to note the number and position of attachment of the worms. The data thus obstained were processed to determine the incidence and intensity of infection in relation to the months and the sex and length of the fish, and to determine the effect of infection on body weight, liver weight and hepato-somatic index. For histopathological studies, infected tissues with the parasite in situ and their normal counterparts were fixed in 10% neutral buffered formalin or in Zenker's fluid. Paraffin wax sections cut at 7 to 10μm were stained with Azan (Heidenhain) or Harris haematoxylin-aqueosus eosin.

Blood samples were collected directly from heart using 1 ml glass syringe and No. 22 needle, both previously rinsed with 3.8% sodium citrate solution and dried. Methods for haematological analyses were adopted from Hesser (1960) and Kolmer et al. (1969). Proximate analyses of liver and white muscle were carried out employing conventional methods.

RESULTS

Penetrocephalus ganapatii

The strobila of the parasite lies in the intestine while the (degenerate) scolex is embedded and encysted in the liver (Fig. 1). The neck, connecting the scolex with the strobila, is long and sturdy and its major part lies in the viscera; a part being incorporated in the liver. The neck comes out into the viscera at various levels on the wall of the anterior two thirds of the intestine. Occasionally, the neck seen passing through the intestinal wall to emerge into the viscera near the region of the pyloric caeca.

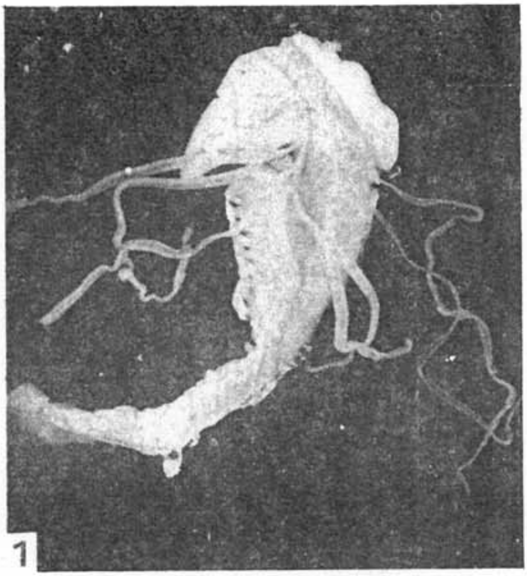

Fig. 1. Viscera of *S. tumbil* – intestine cut open to show heavy infection with *P. ganapatii*

Usually the neck of each specimen is seen running independently through the viscera and entering the liver separately. However, on a few occasions, parts of the neck of two or three specimens are ensheathed together in a dirty, yellowish tissus before entering the

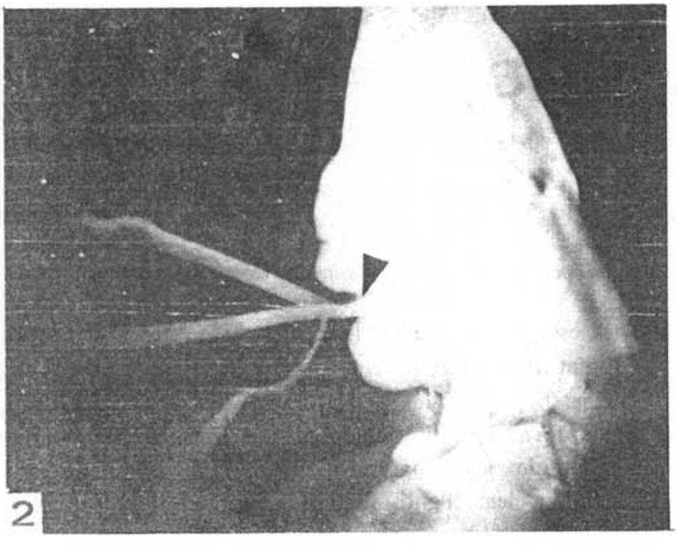

Fig. 2. Liver of *S. tumbil* infected with three specimens of *P. ganapatii.* Note that the necks of the worms entering the liver are encapsulated together (arrow head)

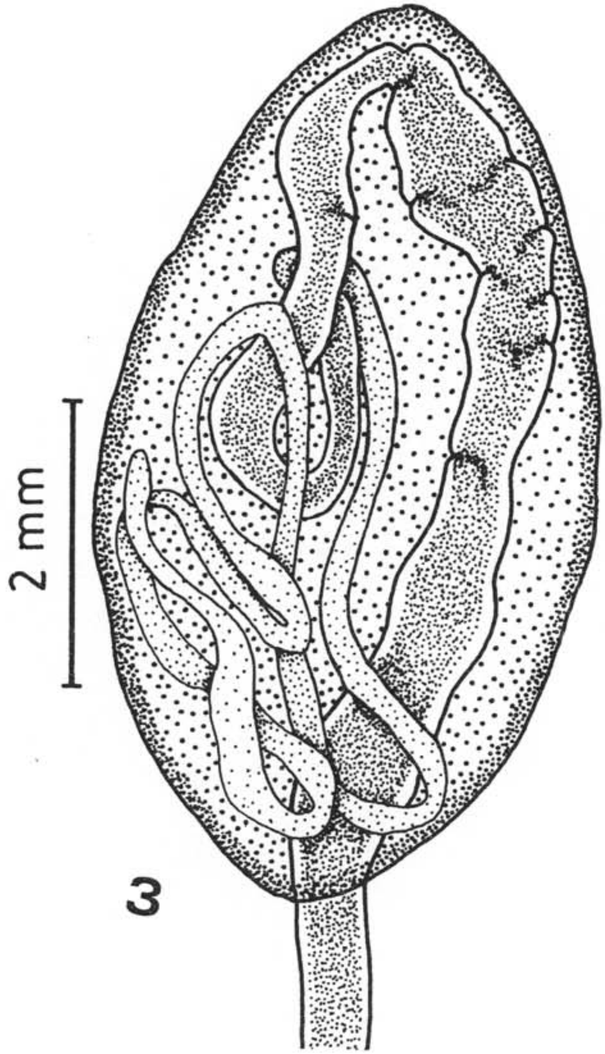

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Fig. 3. *P. ganapatii* cyst containing degenerate scolex and a part of the neck

liver (Fig. 2). Nevertheless, once inside the liver, the ensheathed necks separate and take independent paths so that the scolices are encysted separately inside the liver.

The cysts are oval in shape, translucent and are located at various levels in the liver. The wall of the cyst is formed of fibrous connective tissue of the host and each cyst contains the degenerate scolex and a part of the neck of the parasite (Fig. 3). The cysts are turgid being filled with a clear, slightly viscous fluid.

Bothriocephalus indicus

The worm localises in the intestine of the host. The scolex is usually embedded in the intestinal wall, or rarely in the pyloric caeca.

Specimens of *B. indicus* have been found only in those fish harbouring *P. ganapatii*. While one to seven *P. ganapatti* may infect a single fish, the number of *B. indicus* in a fish usually, is only one. In the nine cases of *B. indicus* infection, in seven one parasite each, and in two two parasites each were obtained from the infected fish.

Apparently *P. ganapatii* and *B. indicus* look alike. The latter may be tentatively set apart from the former by its whiter colour and the comparatively narrower body. However, when juvenile *P. ganapatii* is present, closer examination is needed to distinguish the worms.

INCIDENCE AND INTENSITY OF INFECTION

Of the 270 specimens of *S. tumbil* examined 56.7% were infected with *P. ganapatii*. Percentage incidence of infection in female with *P. ganapatii*. Percentage incidence of infection in female fish (62.6%) was considerably higher than that in the male (47.7%). Incidence of *B. indicus* infection was very low, 3.3%. Since *B. indicus* is always found along with *P. ganapatii*, percentage incidence of total cestode infection is equal to that of. *P. ganapatii* infection. The intensity of *P. ganapatii* infection was 1.9, that of *B. indicus* 1.2 and that of total adult cestode infection 1.96. As is clear from the data presented in Table 1, intensities of infection in male and female with were more or less comparable.

Data on the incidence and intensity of infection in different months and in different length groups are shown respectively in Figs. 4 and 5.

The results show that percentage incidence of *P. ganapatii* infection increased from July to November 76. Cent percent incidence was noted during October and November. *B. indicus* occured only during October and November 76. Cent percent incidence was noted during October and November. *B. indicus* occured only during October and November and its incidence was 3.7% during October and 26.7% during November. Intensity of *P. ganapatii* infection declined from July to September and then increased rapidly to reach the maximum in November. Intensity of *B. indicus* was 2.0 in October and 1.1 in November (Fig. 4).

The results show an apparent inverse relation between the incidence and intensity of *P. ganapatii* infection up to September, and thereafter a direct relation. For *B. indicus* an apparent inverse relation between the incidence and intensity of infection is noted.

It may be concluded from the results presented in Figure 5 that, in general, the incidence and in tensity of adult cestode infection in *S. tumbil* increased with growth of the fish.

Results of the analysis of variance tests shoved that the incidence and intensity of infection did not differ significantly between male and female fish. Differences in the incidence and intensity of infection observed among months and among the length groups of the fish were, however, highly significant.

Table 1

Incidence and intensity adult cestode infection of *Saurida tumbil*

	Male	Female	Total
Number of fish examined	107	163	270
Percentage incidence of cestode infection	47.66	62.58	56.67
Percentage incidence of *P. ganapatii* infection	47.66	62.58	56.67
Percentage incidence of *B. indicus* infection	2.80	3.66	3.33
Intensity of cestode infection	1.92	1.98	1.96
Intensity of *P. ganapatii* infection	1.84	1.91	1.89
Intensity of *B. indicus* infection	1.33	1.17	1.22
Maximum number of cestodes on a fish	6	8	–
Maximum number of *P. ganapatii* on a fish	4	7	–
Maximum number of *B. indicus* on a fish	2	2	–

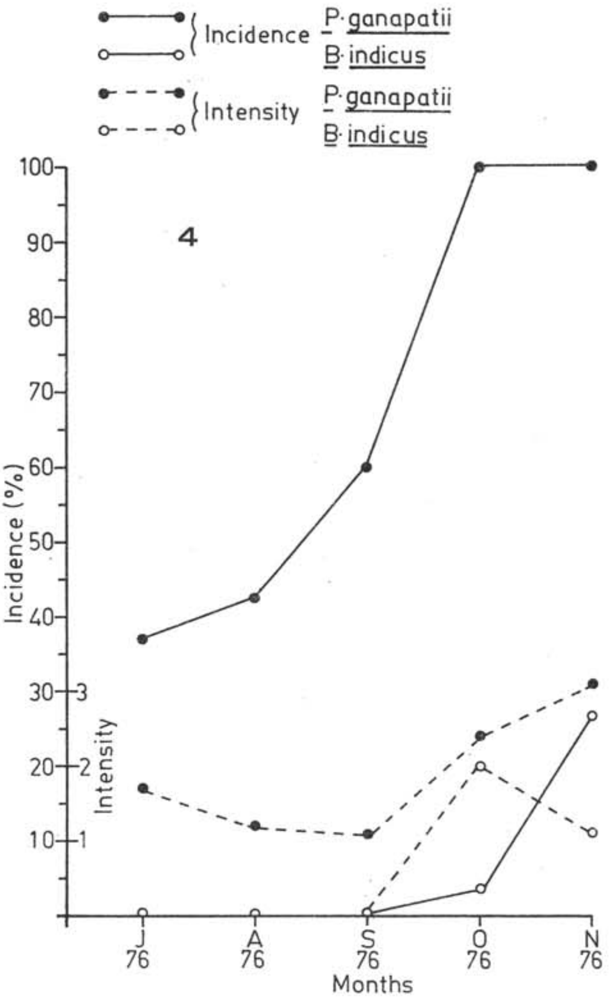


Fig. 4. Incidence and intensity of *P. ganapatii* and *R. indicus* infections of *S. tumbil* during different months

NATURE OF DAMAGE

Histopathology of *P. ganapatii* infection of the liver

The degenerate scolex of *P. ganapatii* is embedded in the liver, usually towards the centro-dorsal aspect of the organ. When many cysts are present, they are seen at different levels in the liver.

The marked histopathology of *P. ganapatii* infection consists of hyperplasia of the connective tissue of the trabeculae. Proliferation of fibrous connective tissue leads to encapsulation of the scolex and a part of the neck of the parasite (Fig. 6). Necrosis of

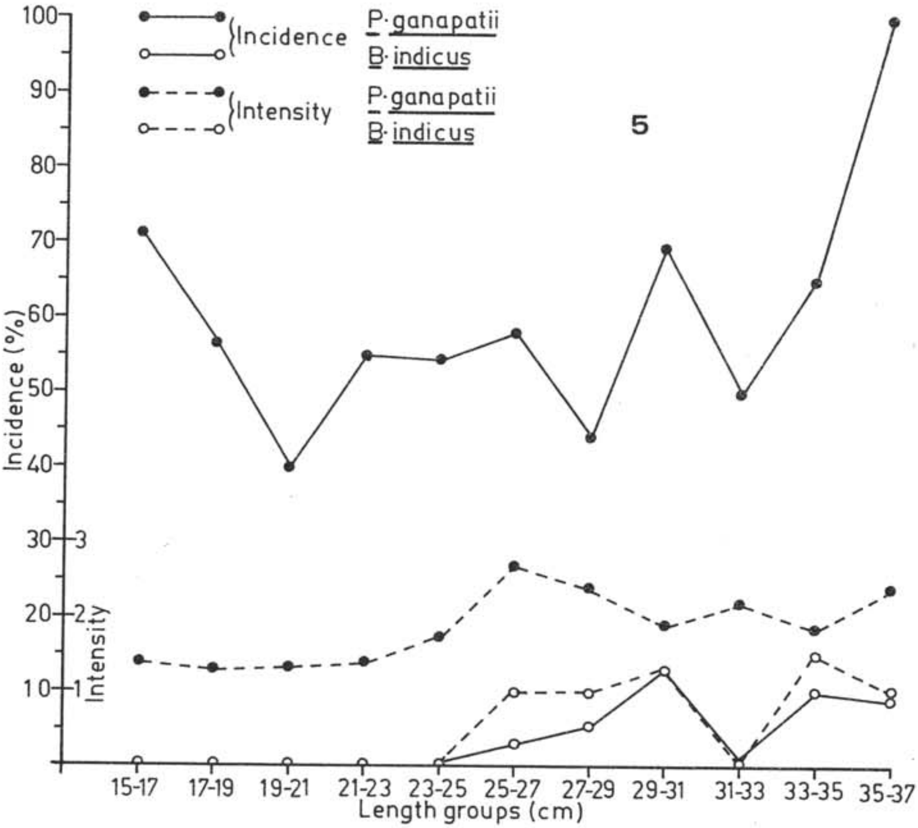


Fig. 5. Incidence and intensity of *P. ganapatii* and *B. indicus* infections of *S. tumbil* in different length groups of the fish

perenchyma cells immediately around the cysts is evident (Fig. 7). A large number of inflammatory cells is seen to accummulate in the parenchyma near the cyst. Similarly, the venules and arterioles also become infested by inflammatory cells; The predominant type being the lymphocyte. Besides, the staining reaction of the infected liver is weak and irregular.

Histopathology of *B. indicus* infection of the intestinal wall.

The scolex of the worm gets encysted in the intestinal wall, usually in the submucosa. A well formed fibrous connective tissue cyst is discernible around the scolex. The histopathological change, thus, is a typical inflammatory reaction in which only the submucosal layer of the intestine is involved, the mucosa and the muscularis being unaffected (Fig. 8).

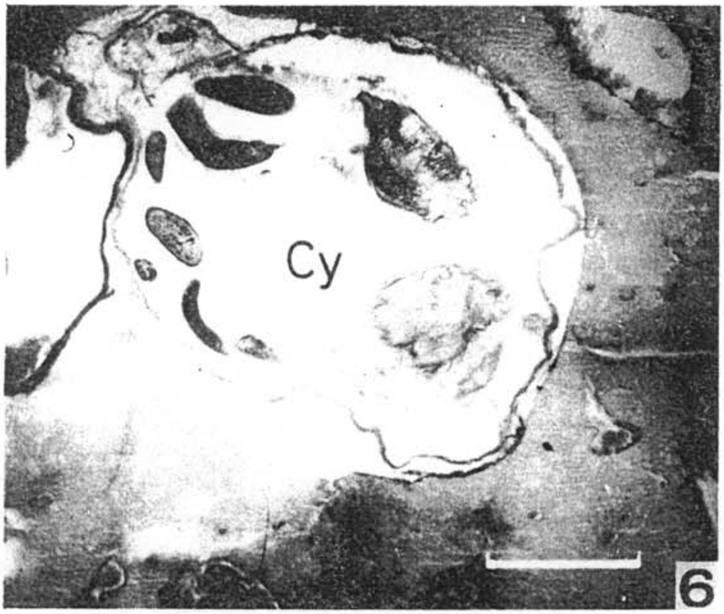


Fig. 6. Section of the liver of *S. tumbil* showing *P. ganapatii* cyst (Cy)

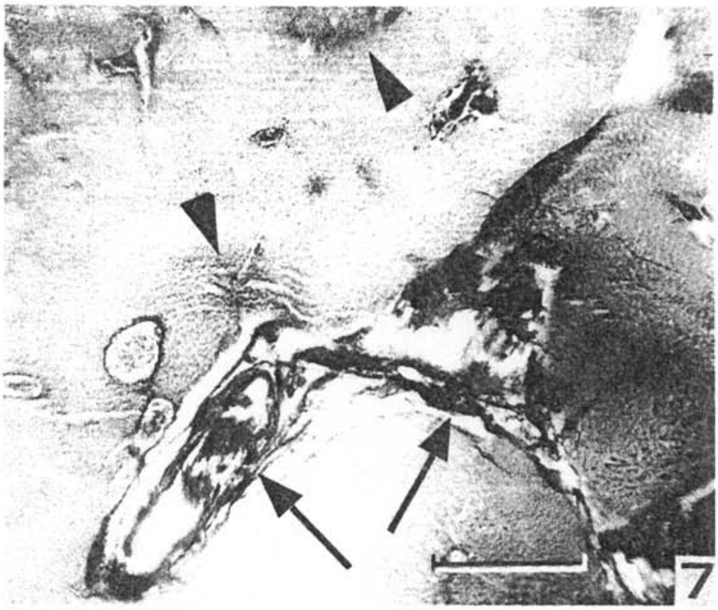


Fig. 7. Section of the liver of *S. tumbil* infected with *P. ganapatii*, showing extensive connective tissue reaction (arrows) and necrotic areas in the liver parenchyma (arrow heads)

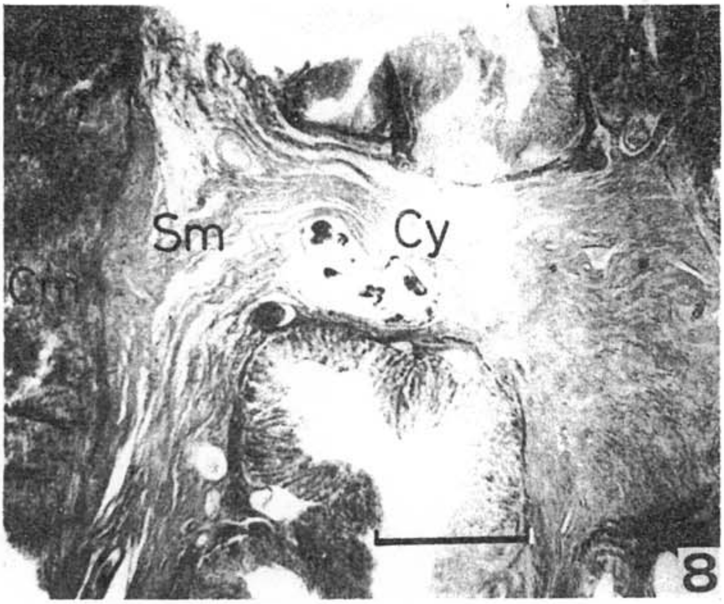
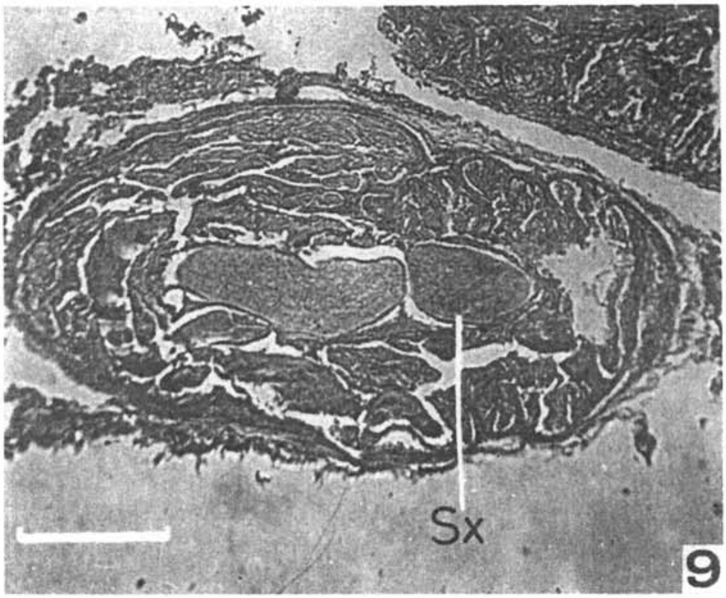
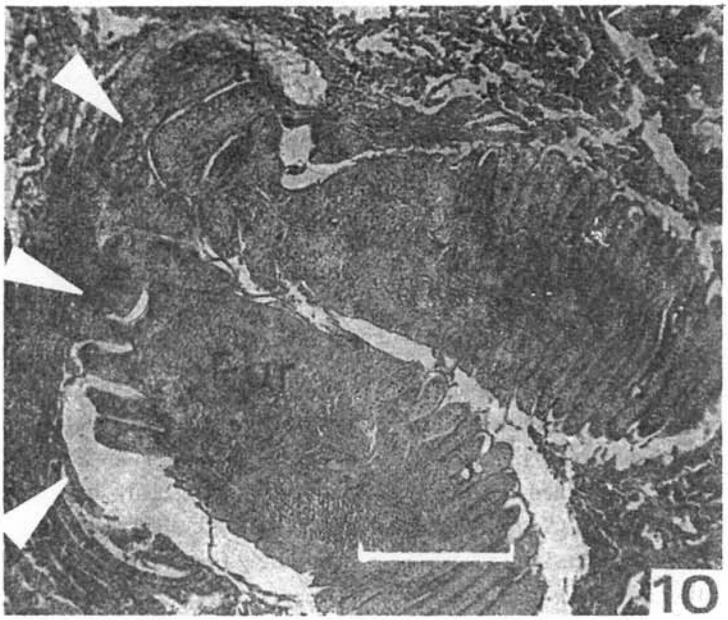
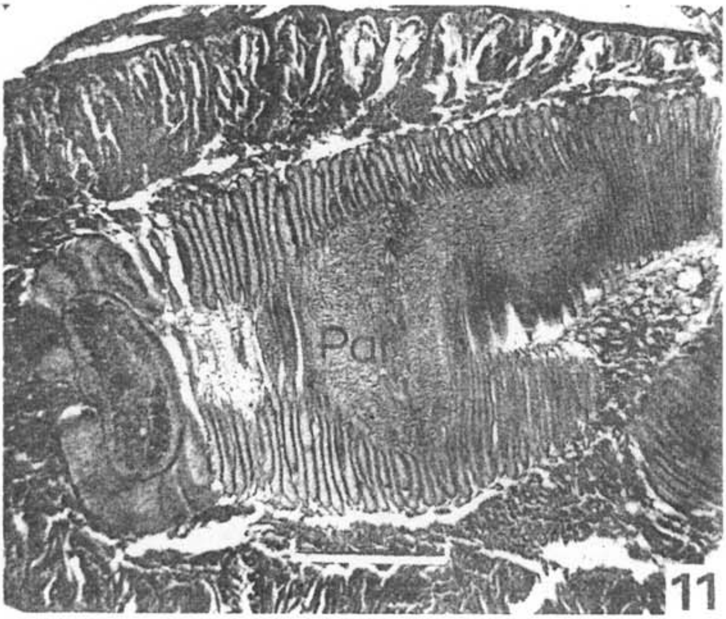


Fig. 8. T.S. of intestinal wall of *S. tumbil* infected with *B. indicus,* showing scolex of the worm encysted in the submucosa (Cm – Circular muscle; Cy – parasitic cyst; Sm – submucosa)







Figs. 9–11. T.S. of pyloric caeca of *S. tumbil* infected with *B. indicus* to show scolex of worm in the caecum (9), erosion of mucosal ridges (arrow heads) (10) and occlusion of lumen by the strobila of the worm (11) (Par – parasite, Sx – scolex)
(Scale: Figs. 6, 8 & 11 – 600 μm; Figs. 7, 9 & 10 – 400 μm)

Histopathology of *B. indicus*, infection of the pyloric caeca

As in the case of intestine, the main histopathological change in the pyloric caeca is inflamantory reaction. Even though the infection induces proliferation of connective tissue, unlike in the case of intestine a well formed cyst is not discernible (Fig. 9). A rich assortment of inflammatory cells infiltrates the caecal matrix.

Even though the primary and secondary mucosal ridges are often completely eroded away as a result of *B. indicus* infection, the caecal wall preserves its integrity (Fig. 10). Often the caecal lumen is almost completely occluded by parts of the strobila of the worm (Fig. 11).

Changes in the haematology and proximate composition

For haematological and biochemical studies, fish ranging from 25 to 31 cm in standard length were selected. Fish infected with *P. ganapatii* were categorised into two groups – those with 1 to 3 parasites (light infection) and those with 4 to 7 parasites (haevy infection). Thirty uninfected, 20 lightly infected and 14 heavily infected fish were subjected to the analyses. (*S. tumbil* harbours about 14 different species of parasites. Of the 270 specimens examined only one fish was absolutely free of parasitic infections. The intensity of other infections, and the variety of other parasites, other than adult cestodes, were very low in fishes ranging from 25 to 31 cm in standard length. Therefore, fishes of this group were selected for the present study. 15 uninfected fish, 10 lightly infected and 7 heavily infected of each sex were analysed for haematological and biochemical changes. The comparative low number of infected fish is due the fact that only this much were available in the collection which had very low intensities of infection by other parasites, that would not mask the actual effect of adult cestode infection.

Haematological changes

From the results presented in Table 2 the following changes are noticeable:

(1) Significant reduction in RBC count ($p < 0.01$), haemoglobin content ($p < 0.001$) and haematocrit ($p < 0.05$) in heavy infection.

(2) Significant increase in WBC count ($p < 0.001$) and E.S.R. ($p < 0.001$) in light and heavy infections.

(3) Significant increase in haemoglobin content ($p < 0.001$) in light infection.

Even though M.C.V. is higher in light and heavy infections than in the uninfected fish, the observed increase is not statistically significant. M.C.H. shows significant increase ($p < 0.01$) in lightly infected fish, but in heavily infected fish it is more or less comparable to that of the uninfected. As with M.C.V., even though M.C.H.C. shows some changes – increase in light infection and decrease in heavy infection are not statistically significant.

Considering erthrocyte indices, V.I. increase to 1.03 in light infection ($p < 0.05$), and then decreases slightly to 1.02 in heavy infection. However, the difference in V.I. between uninfected and heavily infected fish is not significant. C.I. in light infection is

Table 2

P. ganapatii infection of *S. tumbil* – Haematological parameters (mean ± S.E.) of uninfected, lightly infected (1 to 3 worms) and heavily infected (4 to 7 worms) fish

Haematological parameters		Uninfected fish	Infected fish	
			Light	Heavy
Total erythrocyte count – RBC (x $10^6/mm^3$)		2.63 ±0.0538	2.60 ±0.0310	2.37 ±0.0428
Total leucocyte count – WBC (x 10^4/mm)		1.60 ±0.0338	3.85 ±0.0333	3.20 ±0.0316
Haemoglobin content – Hb (g%)		11.25 ±0.17	12.08 ±0.12	10.00 ±0.18
Haematocrit – Ht (%)		28.66 ±0.78	29.13 ±0.53	26.11 ±0.40
Erythrocyte sedimentation rate – E.S.R. (mm/h)		3.87 ±0.12	5.50 ±0.17	5.43 ±0.46
WBC/RBC ratio		0.0062 ±0.0002	0.0149 ±0.0014	0.0193 ±0.0055
Erythrocyte constants	Mean Corpuscular Volume – M.C.V. (μ^3)	108.68 ±1.75	112.06 ±1.69	110.84 ±2.80
	Mean Corpuscular Haemoglobin – M.C.H. ($\mu\mu$g)	43.04 ±0.76	46.59 ±0.73	42.43 ±1.12
	Mean Corpuscular Haemoglobin Concentration – M.C.H.C. (%)	39.96 ±1.03	41.68 ±0.68	38.42 ±0.91
Erythrocyte indices	Volume Index (V.I.)	1.00	1.03 ±0.02	1.02 ±0.03
	Colour Index (C.I.)	1.00	1.09 ±0.02	0.99 ±0.03
	Saturation Index (S.I.)	1.00	1.04 ±0.02	0.96 ±0.02

1.09, compared to 1.0 in the uninfected fish. It is 0.99 in heavy infection. The difference in C.I. between the uninfected and heavily infected fish not statistically significant: but that between uninfected and lightly infected is highly significant ($p < 0.001$). The difference in S.I. between uninfected and lightly infected fish is significant at 1% level ($p < 0.01$) and that between uninfected and heavily infected fish at 5% level ($p < 0.05$).

Regarding differential count of WBC, the results presented in Table 3 show that there is progressive reduction in the percentage of lymphocyte with increasing severity of infection, concurrent with progressive increase in the percentage of neutrophil, basophilic granulocyte and monocyte. While the percentages of plasmocyte and macrophage increase considerably in lightly infected fish, they slightly decrease (compared to that in light infection) in heavily infected fish. Eosinophil increases (4.22%) in light infection and decreases in heavy infection, but the percentage in heavily infected fish (2.38) is lower than that in the uninfected (3.30).

Table 3

P. ganapatii infection of *S. tumbil* – Leucocyte differential count (%) of uninfected, lightly infected (1 to 3 worms) and heavily infected (4 to 7 worms) fish

Cell type	Uninfected fish	Infected fish	
		Light	Heavy
Lymphocyte	58.25	43.18	34.20
Neutrophil	19.89	23.80	26.14
Basophilic granulocyte	6.35	9.60	18.28
Plasmacyte	5.88	8.92	8.00
Monocyte	5.49	8.28	9.36
Eosinophil	3.30	4.22	2.38
Macrophage	0.84	2.00	1.64

BIOCHEMICAL CHANGES

The results of the analyses of the proximate composition of liver and white muscle of uninfected and infected fish are presented in Table 4.

The changes noticeable for the liver are increase in water content and decrease in fat, glycogen and protein contents in heavy infection. While the differences in water and protein contents are not significant, those of fat and glycogen are highly significant ($p < 0.001$). It is also noticeable that both fat and glycogen slightly increase, thought not significantly, in lightly infected fish.

In the case of muscle, water content progressively increases with increasing severity of infection (uninfected VS lightly infected $p < 0.05$; uninfected VS heavily infected $p < 0.01$. Fat contend shows negligible decrease with increasing severity of infection. Glycogen content slightly increases in light infection and significantly decreases

Table 4

P. ganapatii infection of *S. tumbil* – Proximate composition (mean ± S.E.) of liver and white muscle of uninfected, lightly infected (1 to 3 worms) and heavily infected (4 to 7 worms) fish

Biochemical parameters	LIVER			WHITE MUSCLE		
	Uninfected fish	Infected fish		Uninfected fish	Infected fish	
		Light	Heavy		Light	Heavy
Water*	73.25 ±1.05	73.31 ±1.10	74.37 ±5.00	76.34 ±0.77	78.72 ±0.88	80.82 ±1.35
Fat*	20.00 ±1.01	20.91 ±1.13	13.44 ±0.93	5.05 ±0.60	3.71 ±0.71	3.62 ±0.81
Glycogen**	19.49 ±0.99	22.13 ±1.19	12.43 ±1.74	18.20 ±0.78	19.82 ±1.17	11.58 ±1.02
Protein**	65.18 ±0.99	63.60 ±0.77	63.03 ±1.89	76.84 ±1.01	74.22 ±1.24	70.63 ±0.81

* Percentage of wet weight of tissue

** Percentage of dry weight of tissue

($p < 0.001$) in heavy infection. Protein content shows a continuously decreasing trend with increasing severity of infection and the difference in protein content between uninfected and haevily infected fish in significant at 1% level ($p < 0.01$).

Effect of infection on body and liver weights and on hepato-somatic index

From the results shown in Table 5 it is noticeable that liver weight is adversely affected by the infection. In heavily infected fish the reduction in liver weight in maximum ($p < 0.001$). The difference in liver weight between uninfected and lightly

Table 5

P. ganapatii infection of *S. tumbil* – Standard length, body weight, liver weight, and H.S.I. (mean ± S.E.) of unifected, lightly infected (1- to 3 worms) and heavily infected (4 to 7 worms) fish

Parameters	Unifected fish	Unifected fish	
		Light	Heavy
Standard length (mm)	29.35 ±0.38	29.46 ±0.48	29.32 ±0.46
Body weight (g)	289.95 ±9.52	287.23 ±17.11	275.34 ±11.49
Liver weight (g)	5.78 ±0.24	4.66 ±0.26	3.27 ±0.18
H.S.I. (%)	2.06 ±0.11	1.60 ±0.09	1.22 ±0.09

infected fish is significant at 1% level ($p < 0.01$). Even though body weight also decreases with increasing severity of infection, the differences are not statistically significant. On the other hand, reduction in the H.S.I. in light infection ($p < 0.01$) and in heavy infection ($p < 0.001$) is highly significant.

DISCUSSION

Saurida tumbil, the common greater lizard fish, contributes to a valuble fishery along the south-west coast of India during July-November period. The fish is known to harbour as many as 14 different species of parasites (Radhakrishnan, 1979).

Reports on the occurrence of adult cestodes in marine teleost are rare. *Penetrocephalus ganapatii* was first described as *Bothriocephalus ganapatii* by Rao (1954), from *S. tumbil* collected from the Bay of Bengal. Subhapradha (1955) identified this cestode as *Bohriocephalus penetrans* (from the Madras coast) wich was later redescribed by

Table 6

P. ganapatii infection of *S. tumbil* – Results of Student's 't' test comparing different parameters of uninfected and infected fish (* – $p < 0.05$; ** – $p < 0.01$; *** – $p < 0.001$)

Parameters		Uninfected vs lightly infected	Uninfected vs heavily infected	Lightly infected vs heavily infected
Standard length		0.1843	0.0389	0.1950
Body weight		0.1466	0.8922	0.5103
Liver weight		2.9923**	6.5621***	3.8731***
H.S.I.		2.8969**	4.6938***	2.7793**
RBC		0.4761	3.0921**	4.3579***
WBC		7.9945***	7.0412***	1.3223
Hb		3.5134***	4.4164***	9.6071***
Ht		0.4311	2.1219*	4.0605***
E.S.R.		7.9250***	4.2209***	0.1601
WBC/RBC ratio		7.5164***	3.4356**	0.8787
M.C.V.		1.2990	0.6578	0.3857
M.C.H.		3.1477**	0.4417	3.1588**
M.C.H.C.		1.2271	0.9240	2.8392**
V.I.		2.4345*	1.1081	0.4201
C.I.		5.3235***	0.3373	0.6528
S.I.		3.0994**	2.5064*	2.9194**
Liver	Water	0.0412	0.2941	0.2348
	Fat	0.5746	3.9710***	4.6401***
	Glycogen	1.6693	3.6838***	4.6247***
	Protein	1.1350	1.0845	0.3037
White muscle	Water	2.2051*	3.2018**	1.3135
	Fat	0.4604	0.4992	0.0805
	Glycogen	0.7949	5.2477***	4.8867***
	Protein	0.8782	3.1134**	2.1306*

Rao (1960a) and, on the basis of many unique characters of this worm he proposed a new genus, *Penetrocephalus* incorporating the only species, *P. ganapatii*. Rao (1960b) studied the histochemistry and egg formation of this cestode and briefly analysed its incidence and intensity of infection in *S. tumbil*.

Ganapati and Rao (1954) first described *Bothriocephalus indicus* from *S. tumbil* from the Bay of Bengal. Unlike their observation that ,,...we have not come across more than a single worm in each infected host...", during the present study two fishes with two worms in each were obtained. In both cases the scolices were embedded close by in the intestinal wall their strobilae twisted together.

Both *P. ganapatii* and *B. indicus* are highly host specific. The present record of the worms extend their distribution along the west coast of India also.

Even though the results of the analysis of the incidence and intensity of *P. ganapatii* and *B. indicus* infections do not provide a complete picture of the seasonal variation in infection, they point to the fact that the infection rate and severity tend to increase in colder months. Similarly, the incidence and intensity of infection apparently increase with icreasing length (age) of the host. Changes in the feeding habit and physiology of the fish with growth and possibly changes in the defence mechanisms of the host, as postulated by Polyanski (1958), might favour high incidence and intensity of infection in older fish.

It is known that a vertebrate host s immediate reaction to a metazoan parasite penetrating its tissues is typically inflammaion. Proliferation of fibrous connective tissue around the invading elements, encapsulating them in well formed cysts, is an affective defence mechanism to wall off the parasite from the host tissue there by minimising tissue damage. The same mechanism is as work both in *P. ganapatii* and *B. indicus* infections. Ganapati and Rao,(1954) reported that the scolex of *B. indicus* attaches itself to the intestinal wall and gets embedded in ,,...a specially formed intestinal pouch". This pouch, evidently, is the fibruous connective tissue cyst formed around the scolex, elaborated by the submucosa of of the intestinal wall.

Even though encapsulation of the scolex is effective in proventing further damage due to penetratio and attachment of the parasite, since the strobila lies freely in the intestine, the worms still may adversely affect the host by interfering with its nutrition. That this is true is evident from the present observations on the haematological and biochemical changes in the infected fish.

The sum total of the haematological changes shows that the lightly infected fish become anaemic. As is evident from the erythrocyte constatnts and indices, the anaemia is of the macrocytichyperchromic type, characterised by increased M.C.V. and M.C.H. (see Wintrobe, 1961). In man, pernicious anaemia, a typical case of macrocytic anaemia, occurs as a result of liver disease of the deficiency of vitamin B_{12} and folic acid, as well as due to *Diphyllobothrium latum* infection (Kassiroski a. Alexeev, 1972); (Davidson a. Henry, 1977). Typical hyperchromia and macrocytosis and marked eosinophilic leucocytosis are characteristic of *Diphyllobothrium* anaemia in man. The present case is, evidently, similar to the foregoing anaemic condition in man.

In man, vitamin B_{12} deficiency in cases of *Diphyllobothrium latum* infection, is atributet to absorption of this factor by the worm; this leads to perniciosus anaemia (see Baker and Douglas, 1966). The combined effect of absorption of vitamin B_{12} (And perhaps of other antianaemic factors also) by the worms and the inability of the fish to replenish the factoer(s) by way of food because of the distrupted absorptive capacity of the intestine (occluded by the strobilae of the worms) must be the reasons for the occurence of macrocystic-hyperchromic anaemia in lightly infected fish.

In heavily infected fish, the erythrocyte constants and indices indicate persistence of macrocytosis and occurrence of hypochromia. In man, anaemic conditions seen in chronic infections and chronic systemic diseases are usually mild and are ,,overshadowed by the basic disease" (Davidsohn and Henry, 1977). The erythrocytes in such conditions are ,,usually normocytic and normochromic but often normocytic and hypochromic". Perhaps the condition observed in heavily infected *S. tumbil* is similar to the foregoing.

A few authors who ventured to study the haematological changes in helminth infection of fishes have shown that cestode infections cause reduction in RBC count and haemoglobin content and increase in the relative percentage of circulating phagocytic (inflamatory cells such as neutrophil and monocyte, concurrent with a decrease in that of the lymphocyte (Sadkovskaya, 1953; Shpolyanskaya, 1953, 1953; Kosheva, 1956; Bauer, 1958; Bylund, 1972). The results of the present study on *P. ganapatii* infection of *S. tumbil* are in conformity with the foregoing.

An increase in E.S.R., which is characteristic in pathologic situations in higher vertebrates and man, has also been observed in *P. ganapatii* infection. Increased concentration of immunoglobulins in blood plasma is said to cause an increase in E.S.R. (see Frankel et al. 1963; Davidson and Henry, 1977). Perhaps, in *S. tumbil* some toxic metabolic waste produced by the worm (see Rogers and Sommerville, 1963; Fletcher, 1978) might have caused cellular immune response in blood resulting in an increased rate of immunoglobubin synthesis and consequent increase in E.S.R. Change in the specific gravity of erythrocytes also is likely to have contributed towards this end.

Most of the pathologic (infections) conditions in higher animals are accompanied by an increase in the number of leucocytes (leucocytosis) in the peripheral blood and this type of reactive leucocytosis disappears as the subject recovers from the pathologic conditions (Kassirsky and Alexeev, 1972). In *P. ganapatii* infection of *S. tumbil* also leucocytosis has been noted. However, the fact that it was concurrent with increase in the relative percentage of inflamatory/phagocytic elements leads to the conclusion that the cellular defence machinery of the host was activated by the infection.

The observed changes in the proximate composition of the liver and white muscle of the infected fish, simulating the picture of depletion of energy reserves during starvation (Love, 1970), are obviously the result of the mobolisation of the energy reserves to meet the energy reqiurements of both the host and the parasite, and because of the inability of the host to replenish the reserves by way of food owing to the diminished absorptive power of the intestine caused by the worms, especially in heavy infection. It is noteworthy that heavy infection causes reduction in the muscle protein value of the fish.

An interesting observation in regard to the haematological and biochemical changes is that in lightly infected fish, haemoglobin content, haematocrit and fat and glycogen contents of the liver and glycogen content of muscle had a slight quantitative gain. In the case of *Melanogrammus aeglefinus* infested by the copepod *Lernaeocera obtusa* and of *Hemiramphus xanthopterus* infested by *Lernaeonicus hemirhamphi* Kabata (1958) and Natarajan (1975) respectively showed that the initial stage of the infestation was characterised by an increase in haemoglobin content. Kabata described this feature as the result of a „compensatory reaction" on the part of the host to compensate for the loss of blood resulting from the feeding activity of the parasite. In *S. tumbil* also the onset of the infection (or light infection) induced a sort of compensatory reaction to combat the adverse effects of the infection which resulted in the observed quantitative gain in the above said parameters.

Since fish infected with only *Bothriocephalus indicus* were not available, whether this parasite is also as injurious as *Penetrocephalus ganapatii* remins to be determined. However, preliminary observations on fishes with combined infection by the two worms, point to the fact that *B. indicus* does not substantially contribute to pathogenesis. Since the intensity of infection by *B. indicus* is very low, it is quite unlikely that this parasite, unlike *P. ganapatii*, produces gross pathological changes.

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REFERENCES CITED

Arme, C., Owen R.W., 1965: Ligulosis in cyprinid fishes: a unique form of parasite inference with a vertebrate reproductive cycle (Abstract). – Parasitology **25**: 7 pp.

Baker, N.F., Douglas J.R., 1966: Blood alterations in helminth infection. In: E.J.L. Soulsby (ed.) – Biology of Parasites, Academic Press, New York.

Bauer, O.N., 1958: Relation between host fishes and their parasites. In: V.A. Dogiel, G.K. Petrushevski and Yu.I. Polyanski (eds.). Parasitology of fishes. Leningrad Univ. Press, Leningrad. Z. Kabata (eng. transl.). T.F.H. Publications, Hong Kong.

Bauer, O.N., Musselius, V.A., Strelkov, Ya.A., 1973: Diseases of Pond Fishes. Israel Program for Scientific Translations. Jeruzalem.

Bylund, G., 1972: Pathogenic effects of a diphyllobothrid plerocercoid on its host fishes. – Comment. Biol., **58**: 1–11.

Chubb, J.C., 1964: Observations on the occurence of the plerocercoids of Triaenophorus nodulosus (Pallas, 1781) (Cestoda: Pseudophyllidea) in the perch, Perca fluviatilis L. of Llyn tegid (Bala Lake), Merionethshire. – Parasitology, **54**: 481–491.

Davidsohn, I., Henry, J.B. (eds.), 1977: Todd-Sanford Clinical Diagnosis by Laboratory Methods. The Macmillan Co of India Ltd., Delhi: 1443 pp.

Duijn C., van, 1973: Diseases of fishes. Ilffe Books, London: 372 pp.

Fletcher, T.C., 1978: Defence mechanisms in fish. In: D.C. Malins and J.R. Sargent (eds.) Biochemical and biophysical perspectives in marine biology. Academic Press, London.

Frankel, S., Reitman, S., Sonnewirth, A.C., 1963: Gradwohls' Clinical Laboratory methods and diagnosis – A textbook on Laboratory Procedures and interpretation. The C.V. Mosby Company, U.S.A.: 1036 pp.

Ganapati, P.N., Rao, K.H., 1954: On two species of Bothriocephalus Rudolphi (1808), (Cestoda) from the gut of Saurida tumbil (Bloch). – Proc. Indian Sci. Congr. (Abstract), Part III: 1972.

Hesser, E.F., 1960: Methods for routine fish haematology. – The Progr. Fish-Cult., **22**: 164–171.

Kabata, Z., 1958: Lernaeocera obtusa n.sp.- Its biology and its effects on the haddock. – Mar. Res., **3**: 1–26.

Kassirsky, I., Alexeev, G., 1972: Clinical haematology (Eng. Transl. M.C. Creighton). – Mir Publishers, Moscow: 775 pp.

Kolmer, J.A., Spaulding, E.H., Robinson, H.W., 1969: Approved laboratory technic. Scientific Book Agency, Calcutta: 1180 pp.

Kosheva, A.F., 1956: Effect of Ligula intestinalis and Digramma interrupta (Cestoda) on fish. (in Russian) Zool. Zeit. **35**: 1629–1632.

Korting W., 1975: Larval development of Bothriocephalus sp. (Cestoda: Pseudophyllidea) from carp (Cyprinus carpio L.) in Germany. – J. Fish. Biol. **7**: 732–733.

Lien, L., 1970: Studies on the helminth fauna of Norway. XIV: Triaenophorus nodulosus (Pallas, 1760 [1781]) (Cestoda) in Bogstad Lake.II. Development and life span of the plerocercoids in perch (Perca fluviatilis L., 1758). – Nytt Mag. Zool. **18**: 85–96.

Love, R.M., 1970: The chemical biology of fishes. Academic Press, London: 547 pp.

Meyer, M.C., Vik, R., 1968: Observations on Diphyllobothrium sebago plerocercoids in the fish hosts. – Proc. Helminth. Soc. Wash. **35**: 92–96.

Musselius, V.A., Ivanova N., Laptev, V., Apazidi, L., 1968: Khawia sisnesis Hsu, 1935 of carp. – Ribov. i Ribol., **3**: 25–27.

Natarajan, P., 1975: Studies on parasitic copepods with special reference to host-parasite relationship. Ph: D. Thesis, University of Kerala, India: 262 pp.

Polyanski, Yu.I., 1958: Ecology of parasites of marine fishes. In: V.A. Dogiel, G.K. Petrushevski, Yu.I. Polyanski (eds.) – Parasitology of Fishes. Leningrad Univ. Press. Leningrad Z. Kabata (engl. transl.). T.F.H. Publications, Hong Kong.

Radhakrishnan, S., 1979: Studies on certain fish parasites and the nature of infestation along the South-west coast of India. Ph. D. Thesis. University of Kerala, India: 830 pp.

Rao, K.H., 1954: A new bothriocephalid parasite (Cestoda) from the gut of the fish Saurida tumbil (Bloch). – Curr. Sci. **23**: 333–334.

Rao, K.H., 1960a: Studies on Penetrocephalus ganapatii, a new genus (Cestoda: Pseudophyllidea) from the marine teleost Saurida tumbil (Bloch). – Parasitology, **50**: 155–163.

Rao, K.H., 1960b: The problem of Mehli's gland in helminths with special reference to Penetrocephalus ganapatii (Cestoda: Pseudophyllidea). – Parasitology, **50**: 349–350.

Reichenbach-Klinke, H.H., Elkan, E., 1965: The principal diseases of lower vertebrates. Akademic Press, London: 600 pp.

Ribelin, W.E., Migaki G., (eds.), 1975: Pathology of fishes. The Univ. Wisconsin Press, Wisconsin: 1004 pp.

Rogers, W.F., Sommerville, R.I., 1963: The infective stage of nematode parasites and its significance in parasitism. In: Ben Dawes (ed.) Advances in Parasitology. Academic Press, London.

Sadkovskaya, O.D., 1953: Changes in the leucocyte index of the blood of gudgeon upon invasion with ligulidae. (In Russian). K.I. Skryabins' 75 year's Jubilee Volume. Academy of Science USSR Press, Moskov.

Scott, A.L., Grizzle , J.M., 1979: Pathology of cyprinoid fishes caused by Bothriocephalus gowkongensis Yeh, 1955 (Cestoda: Pseudophyllidea) – Fish Dis., **2**: 69–73.

Shpolyanskaya, A.Yu., 1953: Influence of Ligula on changes occurring in leucocytic formula for the blood of fishes. (In Russian) Dokl. Akad. Nauk SSSR, **90**: 319–320.

Sindermann, C.J., 1970: Principal dissases of marine fish and shellfish. Academic Press, New York: 369 pp.

Subhapradha, C.K., 1955: Two new bothriocephalids from the marine fish Saurida tumbil (Bloch). – Proc. Indian Acad. Sci. **41**, B:20–30.

Vik, R., 1957: Studies on the helminth fauna of Norway. I. Taxonomy and ecology of Diphyllobothrium norvegicum n.sp. and the plerocercoid of Diphyllobothrium latum (L.). – Nytt Mag. Zool. **5**: 25–93.

Williams, H.H., 1960: Some observations on Parabothrium gadi pollachii (Rudolphi, 1810) and Abothrium gadi van Beneden, 1870 (Cestoda: Pseudophyllidea) including an account of their mode of attachment and of variation in the two species. – Parasitology, **50**: 303–332.

Williams, H.H., 1967: Helminth diseas of fish. Helminth. Abstr., **36**:261–295.

Wintrobe, M.M., 1961: Clinical haematology. Lea and Fibiger. Philadelphia: 472 pp.

Wiśniewski, L.W., 1932: Cyathocephalus truncatus Pallas, ein Fishparasit aus dem Vrele Bosne. – Rib. List. Fischerb., **7**: 4 pp.

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ZARAŻENIE MORSKIEJ RYBY *SAURIDA TUMBIL* (BLOCH) PRZEZ TASIEMCE *PENETROCEPHALUS GANAPATII* i *BOTHRIOCEPHALUS INDICUS*

STRESZCZENIE

Autorzy przebadali 270 ryb z gatunku *Saurida tumbil* (Bloch), występującego powszechnie w połowach przybrzeżnych u południowo-zachodnich wybrzeży Indii (*Trivandrum*), w okresie od lipca do listopada. Badany materiał pochodzi z połowów 1976 r. Przebadano szczegółowo stosunek płci, wielkości jak i pozostałe parametry, jak stosunek ciężaru wątroby do ciężaru ciała, obraz krwi, zmiany histopatologiczne jak i niektóre biochemiczne dane. Celem badań było prześledzenie częstości występowania i wpływu pasożytów na zarażone ryby przez tasiemce. W grę wchodziły dwa gatunki tasiemców występujące w jamie ciała między trzewiami – *Penetrocephalus ganapatii* Rao, 1954 i *Bothriocephalus indicus* Ganapati et Rao, 1954. (Fig. 1 i 2):

Pasożyty były uczepione skoleksami do miąższu wątroby nieraz po kilka, choć *B. indicus* występował wyłącznie w przypadku zarażenia uprzednio *P. ganapatii* i wyłącznie pojedynczo.

Oprócz tego w miąższu wątroby spotykano cysty zawierające skoleksy *P. ganapatii*. (Fig. 3) Cysty występowały na różnej głębokości w tkance wątrobowej. Wokół miejsca uczepu pasożytów powstawało ognisko zapalne tkanki wątrobowej.

U ryb porażonych pasożytem stwierdzano zdecydowany spadek hemoglobiny, białych ciałek krwi jak też spadek tłuszczu, glikogenu i białek.

Ogólnie zmiany dotyczyły głównie w przypadku *P. ganapatii*, a minimalnie przy *B. indicus*.

С. Радгакрисхнан, Н.В. Наир, Н.К. Балясубраманиан

ЗАРАЖЕНИЕ МОРСКОЙ РЫБЫ SAURIDA TUMBIL (BLOCH) ЦЕСТОДАМИ PENETROCEPHALUS GANAPATII И BOTHRIOCEPHALUS INDICUS

Резюме

Авторами были проведены исследования 270 штук рыбы сорта Saurida tumbil (Bloch), **повсеместно присутствующего в ловах проводившихся в прибережной**

полосе северо-западных берегов Индии (Trivandrum) в период с июля по ноябрь. Исследовали материал полученный из ловов проведенных в 1976 г. Подробным исследованиям подвергались: соотношения полов и величины, а также остальных параметров, в том числе: соотношение веса печени и веса рыбы, картина крови, гистопатологические изменения и некоторые биохимические данные. Целью исследований являлось проследить частоту присутствия и влияние паразитов на рыбу зараженную цестодами. Учитывались два вида цестод, присутствующих в полости тела, между внутренностями: Penetrocephalus ganapatii Rao, 1954 и Bothriocephalus indicus Ganapati et Rao, 1954) рис. 1 и 2).

Паразиты, иногда несколько, ухватываются печени сколексами. B. indicus присутствовал исключительно в случае раньшего заражения P. ganapatii и всегда поодиночке.

Кроме того в мякоти печени встречались цисты содержащие сколексы P.ganapatii . Цисты присутствовали на разной глубине по тканям печени. Вокруг места зацепления паразитов образовался воспалительный очаг ткани печени.

У рыбы зараженной паразитами установлено решительное снижение гемоглобина и лейкоцитов крови, а также жира, гликогена и белков.

В общем изменения наблюдались главным образом при заражении P.ganapatii и в минимальной степени - B. indicus.

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