TR # 192

FEDERAL AID IN SPORT FISH RESTORATION

FINAL REPORT

June 1, 1973 - May 31, 1974

PROJECT F-48-R-1, 2, 3

IMPACT OF PARASITIC WORMS ON LAKE ERIE FISHES

THE OHIO STATE UNIVERSITY
CENTER FOR LAKE ERIE AREA RESEARCH

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FEDERAL AID IN SPORT FISH RESTORATION

FINAL REPORT

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PROJECT TITLE:	Impact of Parasitic Worms on Lake Erie Fishes
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STUDY TITLE:	Impact of Philometra sp. on Lake Erie Fishes
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DATE:	31 July 1975

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ABSTRACT

Small Aplodinotus grunniens, 0+ and 1+ age classes, from Lake Erie often exhibit a syndrome called "pop-eye" during June and July. This disease is caused by the presence of maturing female nematodes of the genus Philometra. These nematodes lodge in the eye orbit and its tissues. More than 64 percent of the freshwater-drum examined during 1972, 1973 and 1974 were infected with some stage of Philometra. Mature, gravid female nematodes penetrate the conjunctiva during July and August and extend outside for approximately four-fifths of their length. In the water the uterus prolapsis through the body wall, bursts and releases 70,000+ first stage larvae. The portion of the female which remains in the eye becomes encapsulated with connective tissue. Encapsulations may persist in the eye for 4 years or longer.

Cyclopoid copepods were utilized as experimental intermediate hosts and transmission vectors. Experimentally infected copepods ingest first stage larvae which penetrate the intestinal wall and enter the haemocoel. At 25°C the first cuticular molt occurs at 4 to 6 days post-infection and the second molt at 8 to 12 days. The morphology of these stages is described.

Freshwater-drum were administered infected copepods via stomach-tubes. Infective larvae penetrate the intestinal wall of the fish 6 to 10 days after infection and enter the coelom. They remain in the coelom from 9 to 25 days post-infection usually in films of liquid at the anterior end of the gas bladder. They migrate forward along the muscles of the paraspenoid bone and enter the orbit of the eyes by 28 days post-infection. Sexual differentiation occurs rapidly after reaching the eyes, males which were unknown for this species are characterized. After copulation the males die and the females continue to grow and develop as fertilization and zygote formation occurs in their uteri.

Juveniles and young adults were found in the eyes of naturally infected freshwater-drum during September and October. They invade and erode connective tissues, membranes, muscle, nerves and the sclera. They feed primarily on blood of the fish host and cause hemorrhages in the eyes. The fertilized females grow to a length of 3 to 5 cm by November and with the onset of winter temperatures they cease to grow and overwinter in the eyes. When the waters of the lake warm again in April and May growth continues and intra-uterine development of embryos to first stage larvae occurs, April to July. The females reach a length of 13

to 23 cm by July. They also feed on blood and tissues and cause hemorrhages, reddening and inflamation of the eye. Pressure created both by the size of worms and the damage inflicted causes the eye to swell and protrude from the socket.

The population biology, incidence and intensity of each stage and its seasonal occurrence in different sizes of naturally infected freshwater-drum from Lake Erie is presented in tabular form and graphically. Small freshwater-drum 5 to 20 cm in length, 0+, 1+, 2+ age classes, harbour the greatest burden of living philometrid worms. Double eye infections are more common than single eye infections.

The transmission cycle of Philometra sp. in Aplodinotus grunniens in Lake Erie is an annual cycle. The release of larvae is biosynchronized with the summer copepod blooms. There are slight fluctuations, probably related to water temperatures, but one generation occurs each year and it is seasonally timed and predictable.

All of the preceding factors were considered in making possible management recommendations.

BACKGROUND

Sport fishermen have been making inquiries for more than a decade about the cause of "pop-eye" in freshwater-drum in Lake Erie. A few were more observant and asked, "What is the long red worm which hangs from the eyes of sheepshead?" Requests from fishermen and various agencies prompted us to investigate this parasitic nematode. We found that it belonged to the genus Philometra, that only females were present in the summer and that it was undescribed. Preliminary experiments in 1971 and controlled experiments in 1972 and 1973 demonstrated that copepods could be infected with the free-lining larval stages and serve as intermediate hosts and vectors.

A review of the literature indicated that Philometra sp. from freshwater-drum was unique in that it was the only philometrid nematode known to infect the eyes of freshwater fishes. The transmission cycle was unknown, the times of host-parasite contact were unknown, the paths of migration and development within the host were unknown and histo-pathology was undescribed. There was no appraisal or evaluation of the impact of this nematode parasite on different year classes of natural populations of its fish hosts in different seasons of the year.

The objectives of this study were designed to fill these voids of knowledge concerning this parasite and its relationship with its fish hosts.

OBJECTIVES

Three research objectives were involved in this study. 1) An Experimental Investigation: Determination of the transmission cycle of Philometra sp. Demonstration of what intermediate hosts are involved in the processes of transmission and how and when the infective stages are passed from definitive host to another. Demonstration of the sequence and times of development of the stages in different hosts involved. 2) A Descriptive Investigation: Description of the parasitic adult and developmental stages occurring in the different hosts involved. Description and demonstration of routes of migration through the organs and tissues of the hosts and the resultant pathology. 3) A Population Investigation: Determination of the incidence and intensity of infection and the effects on natural populations of fish hosts in western Lake Erie.

PROCEDURES

Samples of freshwater-drum were taken from Lake Erie during the ice free periods of the year from the inception of the study, June through October, 1972, March through October, 1973, and April through October, 1974. Twenty-eight samples were taken in 1972, thirty-one in 1973, and twenty-two in 1974. The samples were collected from three sites in Lake Erie: 1) from a seine-haul at Cold Creek on Sandusky Bay; 2) from open-lake otter trawls taken with the motor vessel Bio-Lab, between Green and Rattlesnake Island; and 3) with a small 12 ft. otter-trawl off Locust Point. (Fig. 1.) Fish were iced immediately and brought to Stone Laboratory during the summer and fall months or to the Parasitology Laboratory at the Ohio State University, Columbus, Ohio, in April and May. The fish were autopsied and the eyes, body cavities and viscera were examined for the presence of philometrid worms and encapsulations of whole or partial worms. The data for each fish examined was recorded. Seven-hundred and ninety-nine Aplodinotus grunniens were visually autopsied, 42 were experimentally infected and 30 were studied by digestion techniques in the period of June through October 1972, a total of 871 fish examined in 1972. During the period March through October 1973, 1933 freshwater-drum were autopsied and 30 were experimentally infected. In 1974, 857 fish were autopsied. The total Aplodinotus grunniens examined for the 3 year period was 3,691.

A computer card was punched for each freshwater-drum autopsied throughout the entire study to date using FORTRAN computer language.

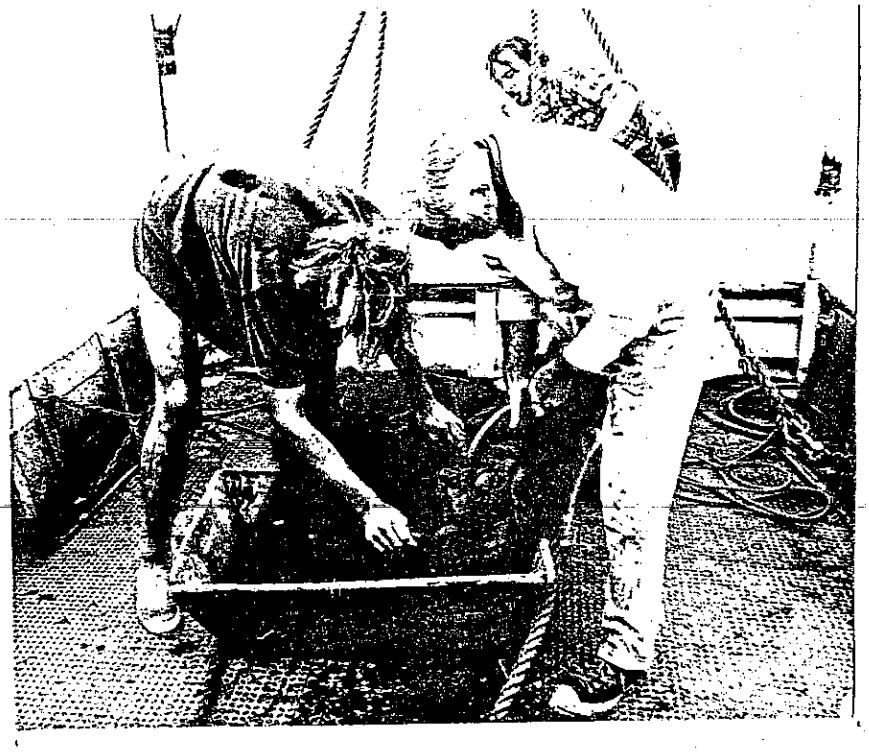


Figure 1.

Figure 2.

The OMNITAB, CROSS-TABS and specially developed programs in FORTRAN language were utilized for analysis of population data as well as standard statistical test. The basic data was stored on computer tapes.

First stage larvae were released from gravid, mature female philometrids by placing the entire female in distilled water or lake water until the uterus prolapsed through the body wall. Free-living, first stage larvae were tested for longevity at 20 and 25 degrees centigrade.

Cyclopoid copepods were collected with a plankton net and cultured at 22 to 26 degrees centigrade until needed for infection experiments. More than 3,000 cyclopoid copepods were experimentally infected in 1972, 2,000 more in 1973 and 1,500 in 1974 by exposing them to first stage larvae in petri dishes, cyclopoid copepods readily ingest the larvae. Infected copepods were maintained in controlled temperature cabinets at 20 and 25 degrees centigrade. Experimentally infected copepods were examined daily, they were crushed under a cover slip to release the larval nematodes and the larvae were studied microscopically to determine the extent of development and molting. All developmental stages were measured and photographed.

Living Aplodinotus grunniens and Perca flavescens to be used for infection experiments were collected before the release of larvae from gravid female philometrids in the eyes. The live fish were collected using a small 12 ft. otter-trawl, pulled for 10 minutes, in 10 to 15 feet of water. The fish were kept alive by placing them in a cascade system of freshly pumped lake water aboard the motor vessel Bio-Lab. Upon reaching Stone Laboratory the fish were transferred to large isolation tanks of running lake water and allowed to stabilize for at least four days. Those fish which showed no signs of disease or abnormal behavior after four days were transferred to 15 gallon aquaria and maintained at ambient temperatures. Each aquarium contained well water which had been treated with chemicals for Ichthyoptherius and fungi, not more than 4 small freshwater-drum, less than 18 cm in length, were placed in an aquarium. Each aquarium was equiped with an air pump and filter system. All experimental fish were fed only commercially grown earthworms. Fish were allowed to stabilize for one week before being utilized in infection experiments. Each fish used in infection experiments was anesthetized by placing it in a solution of quinaldine (0.25 ml of quinaldine per 2.5 gallons of water.) Each fish was administered at 15 cyclopoid copepods infected with at least 4 third stage philometrid larvae. Experimentally infected

fishes were autopsied at pertinent intervals of time after infection to determine the migration, resultant pathology and the extent of development in the fish host.

The intestines of some experimentally infected fish were examined by digestion in artificial gastric solutions to determine the migration of larvae through these tissues. Parallel examinations of fish from natural populations were conducted to confirm that the same processes were occurring at the same time in fish in Lake Erie. During June and July of 1972 sixteen heads of small "pop-eyed" sheepshead were excised immediately upon removal from the trawl while still on the Bio-Lab and fixed in alcoholic Bouin's solution for later sectioning. This process was repeated for "young of the year" freshwater-drum in September and October of 1972. These heads were later double-imbedded in celloidin and paraplast, sectioned and stained with Mallory's Triple stain and hematoxylin and eosin stains. Sections through the eyes of a normal, uninfected sheepshead were prepared for comparative purposes. During 1973 and 1974, this process was repeated and particular attention was given to sections of the gas bladder and the muscles of the parasphenoid bone to determine the migration route from the coelom to the orbit of the eye.

All nematode stages were studied alive utilizing both ordinary and phase microscopy. Nematodes from specific samples and of each stage of development were fixed with alcoholic Bouin's solution for later sectioning or Alcohol-Formalin Acetic Acid (AFA) solutions for preparation of whole mounts. These nematodes fixed with AFA were later cleared in glycerin-alcohol and mounted in pure glycerine on microscope slides for study, in some cases, nematode stages were stained with Semichon's Carmine stain before clearing to enhance the study of certain morphological features.

FINDINGS AND ANALYSES

Descriptive and Experimental Investigations of Philometra sp. Prior to this project only large, gravid or larvigerous females of Philometra had been reported from the eyes of naturally infected Aplodinotus grunniens and no males were known. In this study, only large, gravid females were present in the eyes of freshwater-drum during the periods, April through the first two weeks of August 1972 and March through August 1973 and 1974. No philometrid males were present at those times (Figures 67, 68, and 69). The females grow in length from 4 to 6 cm in April to 12 to 20 cm in late July. Concomitant intrauterine development of the embryos occurs during this same period of time. Table 1 shows the progress of intrauterine development as it occurred in 1972 and 1974. This development began a few days later in 1973 but was slightly more rapid. During April and May the embryos undergo cleavage stages and develop to blastula and gastrula stages. In June the embryos develop through tadpole and early vermiform stages to become inactive coiled larvae maintained within an egg membrane which surrounds all of the embryonic stages. The coiled larvae become active during late June and July, break through the egg membrane and move actively within the lumen of the uterus and are now ready for release. The first stage larvae from 1 cm of uterus were counted using a Sedgewick-rafter cell and we estimate that each mature, adult female contains at least 70,000+ larvae in her uterus.

The intrauterine embryos increased in size as they underwent cleavage and developed through vermiform stages to fully formed first stage larvae. Table 2 presents the average measurement of each stage in microns, in each case at least ten individuals were measured. Not only did these embryos increase in size but the number of refringent granules present in each individual which eventually were encorporated into the intestine of the first stage larva increased in number. These granules were positive when stained with the Periodic-Acid-Schiff reagent, indicating stored glycogen and they stained with Sudan B-Black and other Sudan stains for the presence of lipids. This indicates that as these embryos grew in size they also stored materials. There may be an exogenous source of energy in the liquids which fill the uterus of the gravid female worm, liquids which surround the embryos during their development.

TABLE 1

TIMES OF INTRA-UTERINE DEVELOPMENT-1972.

Date	Stages of Inra-uterine Embryonic Development
May 2	zygote, 2 cell, 4 cell, 8 cell stage
May 16	4 cell stage through early blastula
May 30	blastula and early gastrula
April 19	blastula, late gastrula through early tadpole stage.
June 21	late gastrula thru early vermiform stages
June 26	vermiform and coiled larvae
June 28	coiled larvae and inactive first-stage larvae
June 29	coiled larvae through active first-stage larvae
July 5	coiled larvae through active first-stage larvae
July 11	coiled larvae through active first-stage larvae and release-streaming
July 18	active first-stage larvae-streaming
July 20	active first stage larvae-streaming
July 26	Active first-stage larvae-peak of streaming, encapsulations began

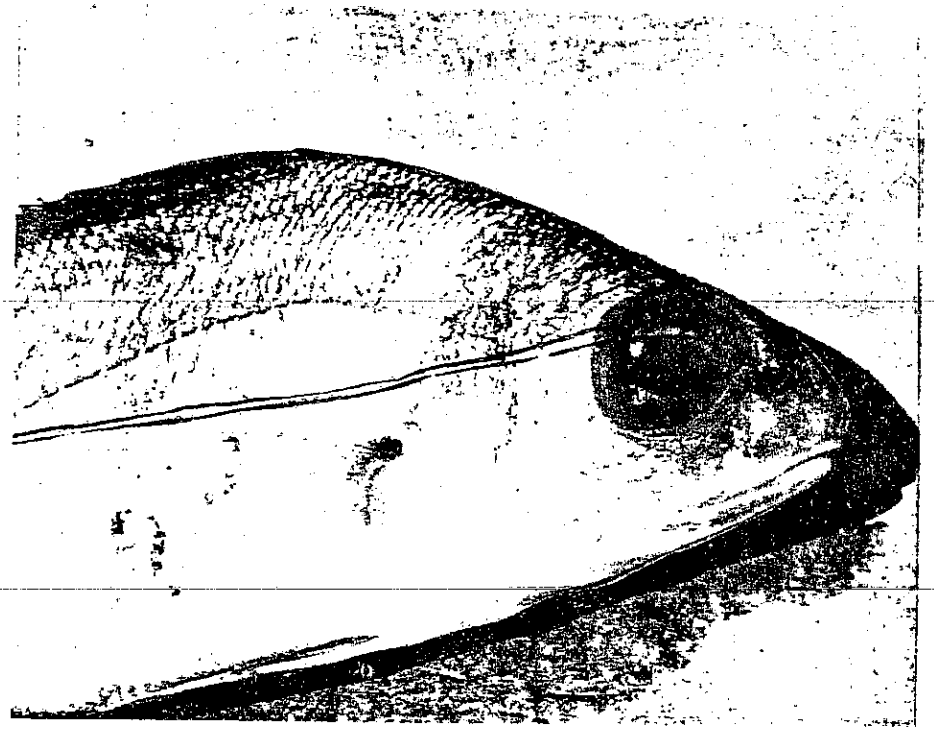


Figure 5. Two adult, gravid philometrid females streaming from the eyes in late July.

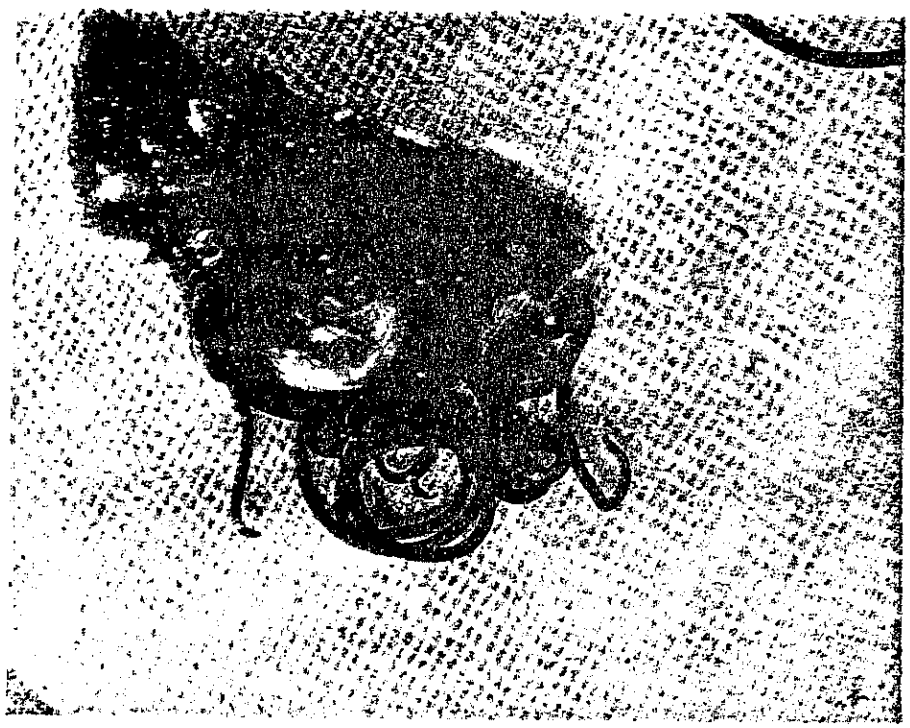


Figure 6. An excised eye containing 3 adult, gravid female worms.

When the female streams from the eye approximately one-fifth of her body remains anchored in the tissues of the eye (Fig. 5.) This portion of the female dies and almost immediately tissue reactions begin. After a period of time the remains of the partial worm are encapsulated with connective tissue. I have studied the formation of these encapsulations both grossly and in sections. Encapsulations can be seen in gross examinations, they appear first as soft membranes surrounding the disintegrating worm during the late fall months. By the next spring these encapsulations have been invaded by fibrous connective tissue and have a tan appearance. As they become older they become dark brown in color and finally have an almost blackened appearance and they become very brittle. Sections of encapsulations demonstrated that the membrane surrounding the worm in the early stages was composed primarily of leucocytes, fibrocytes and histiocytes. In older and tanned encapsulations one could observe the inner core of disintegrating worm which was surrounded by a layer of fibrous connective tissue. Staining reactions with Mallory's Triple Stain indicated that the fibers were primarily callogen. The outer layer of leucocytes could still be observed. In the oldest encapsulations the tissue of the worm was completely sclerotized and the connective tissue capsule may be invaded by salts. The encapsulations exist in the eyes for several years and they serve as markers of generations of philometrid worms from previous infections.

When gravid females containing active first stage larvae were removed from infected eyes and placed in petri dishes of distilled water uterine prolapsis occurred. First stage larvae released by uterine prolapsis were quite active and moved vigorously but they soon settled to the substrate and continued moving or attached by their tails and waved rapidly back and forth. These larvae were used for experiments and for morphological observations. The free-living first stage larvae were photographed alive and their morphology studied. The main morphological features observed were: no perceptible lips; an open lumen to the esophagus; an intestine with no perceptible lumen and a long tapering tail with an open cavity. The free-living first stage larvae range in length from 438.7 to 522.0 microns. They are capable of attaching by their tails but no glands could be ascertained in the tail utilizing oil immersion, phase microscopy. These larvae would be a good subject for study by electron microscopy.

First stage larvae fall slowly through the water column and wriggle actively on the substrate. They attach by their tails to debris present and at times to one another. Their anterior ends always extend outward. After one hour from 2 to several hundred

may be attached to a given, minute piece of detritus (Figs. 7 and 8.) This phenomenon, "Clumping," occurred readily when larvae were confined to petri dishes, 9 cm in diameter, containing water to a 1 cm depth. Preliminary experiments indicate the clumps of 7 or more larvae tend to attract cyclopoid copepods more rapidly than single larvae. Clumping may be an adaptation which increases the probability of these larvae being contacted and ingested by intermediate hosts.

Active free-living first stage larvae were taken immediately upon release from the parent female worms and studied for longevity at different temperatures. They were maintained in distilled water in constant temperature cabinets at 20 to 25°C which approximately brackets the summer temperature of Lake Erie. They remained active for 7 days at 20°C and all of them had ceased movement in 10 days, they remained active for 5 days at 25°C and had ceased active movement in 7 days at this temperature. When larvae cease active movement they are not dead but lie coiled in the bottom of containers, they could be stimulated to move only by touch or by creating a current in their immediate vicinity. All larvae were beyond stimulation for movement in 14 days at 20°C and 18 days at 25°C and they were presumed to be dead. First stage larvae can be maintained at 15°C for as long as 22 days with only a 10% loss of the population. After 22 days the number of living larvae drops quickly and 27 days only 5 to 7% show any life signs when disturbed. These observations are from three replicates and the percentages are averages. Larvae were utilized for infection experiments only during their active periods. Free-living, first stage larvae placed in a refrigerator at 5°C ceased to move but regained their active movements when warmed to 20°C. First stage larvae can be stored at 5°C for 16 days and still remain infective for the first intermediate host.

Because other nematodes of the Superfamily Dracunculoidea and the Family Philometridae had been demonstrated to utilize cyclopoid copepods as intermediate hosts these curstaceans were chosen for experimental infections. Cyclopoid copepods were collected by plankton tow and cultured by feeding them Paramecium. The copepods were placed in small petri dishes with approximately 300 active, free-living, first stage larvae. Cyclopoid copepods readily ingest active, first stage larvae. The ingested larvae penetrated the wall of the alimentary canal of the copepod within 10 to 45 minutes and entered the haemocoel. More than 3,000 copepods were infected in this manner during the summer of 1972

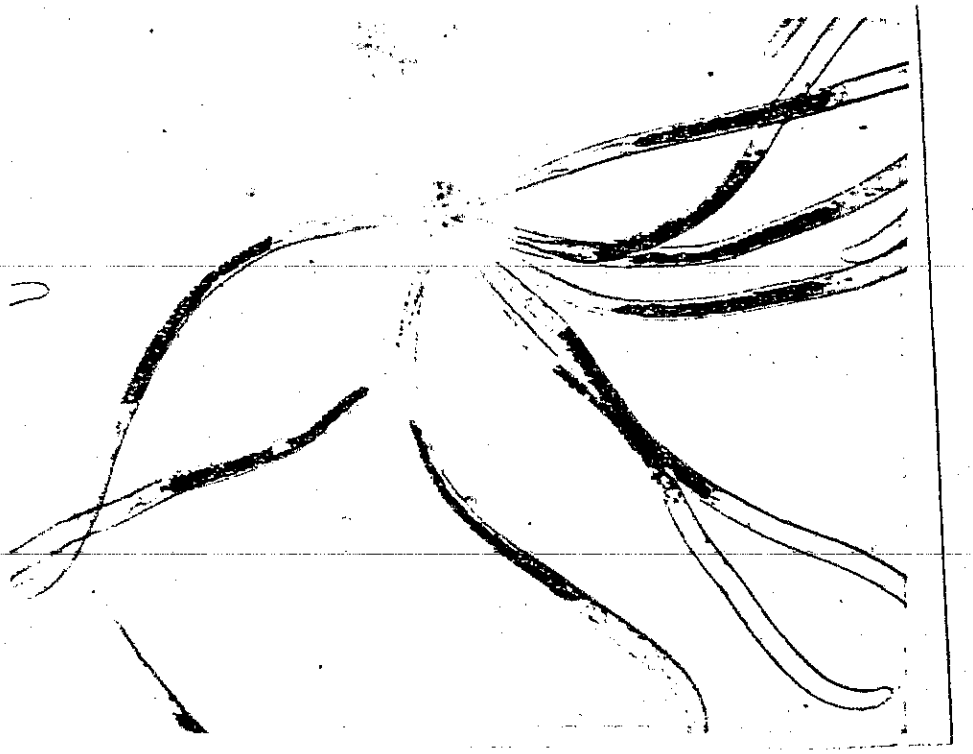


Figure 7. First stage larvae of Philometra sp. attached to detritus by their tails.

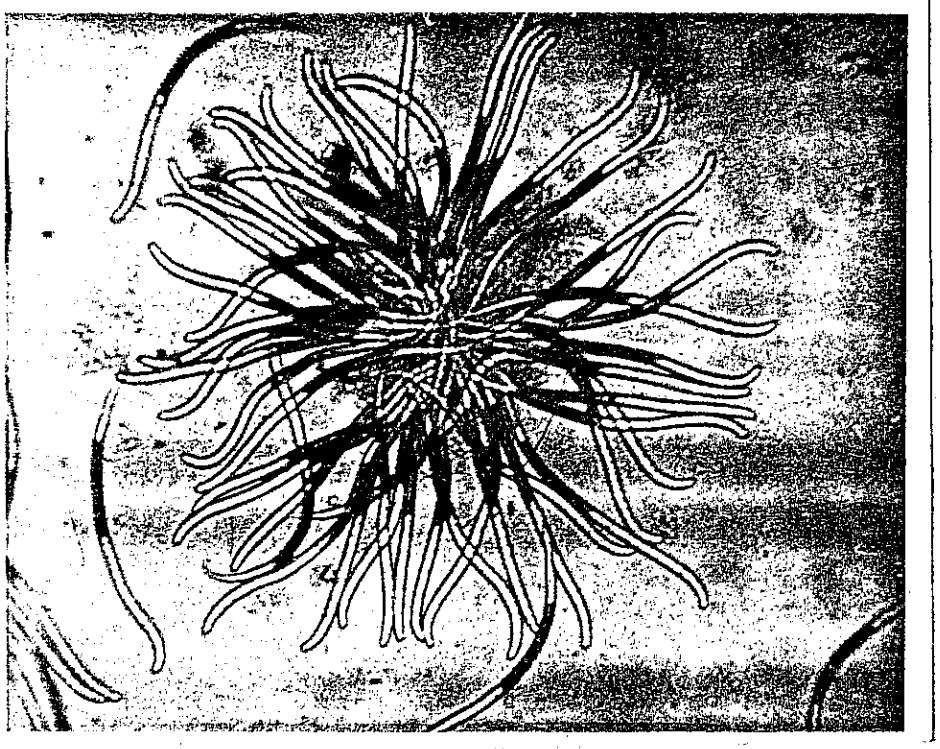
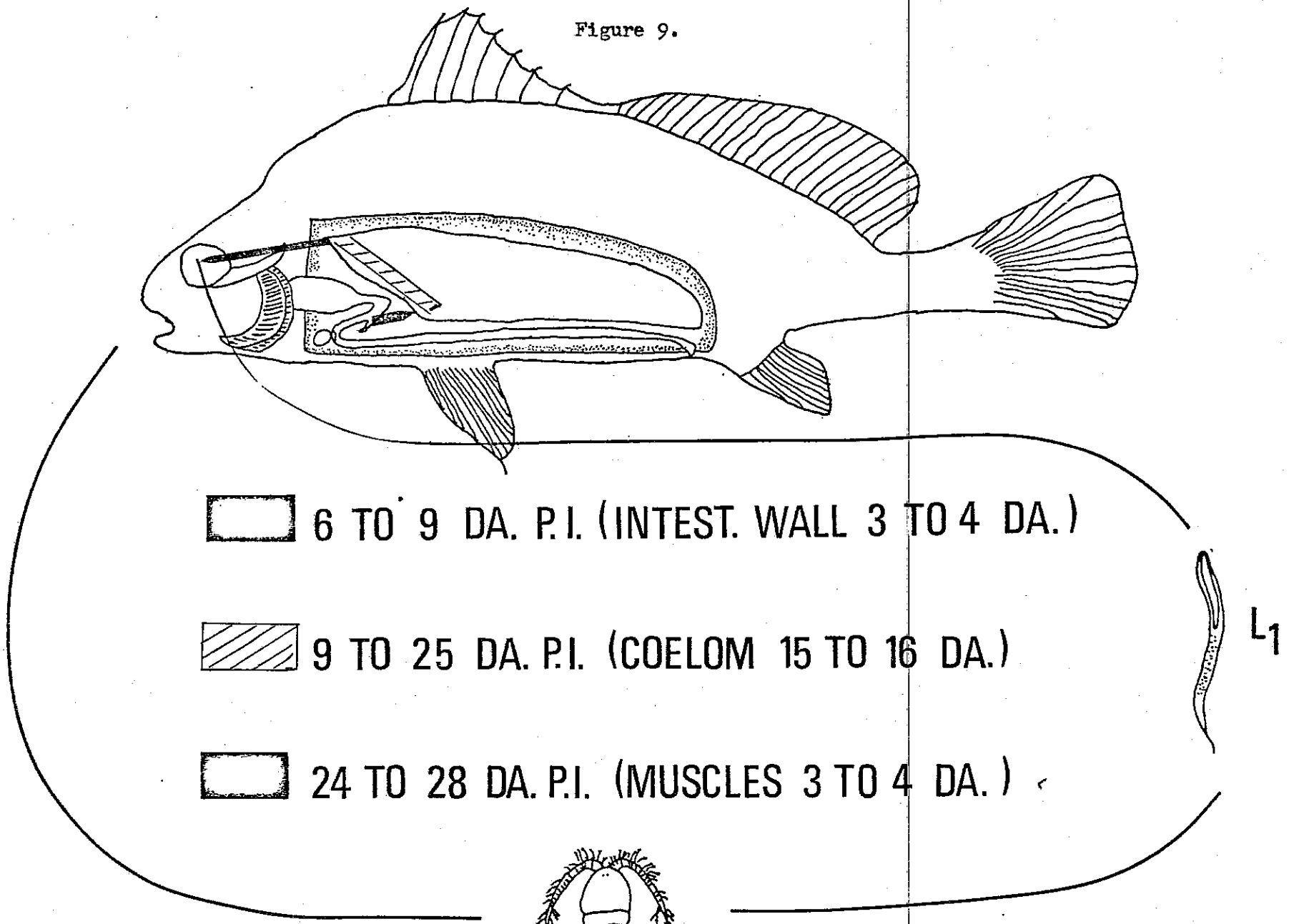


Figure 8. Clumped first stage larvae of Philometra sp.

Figure 9.


6 TO 9 DA. P.I. (INTEST. WALL 3 TO 4 DA.)
9 TO 25 DA. P.I. (COELOM 15 TO 16 DA.)
24 TO 28 DA. P.I. (MUSCLES 3 TO 4 DA.)
L1
L1, L2, L3

and 2,000 more during the summers of 1973 and 1974 and used for further studies.

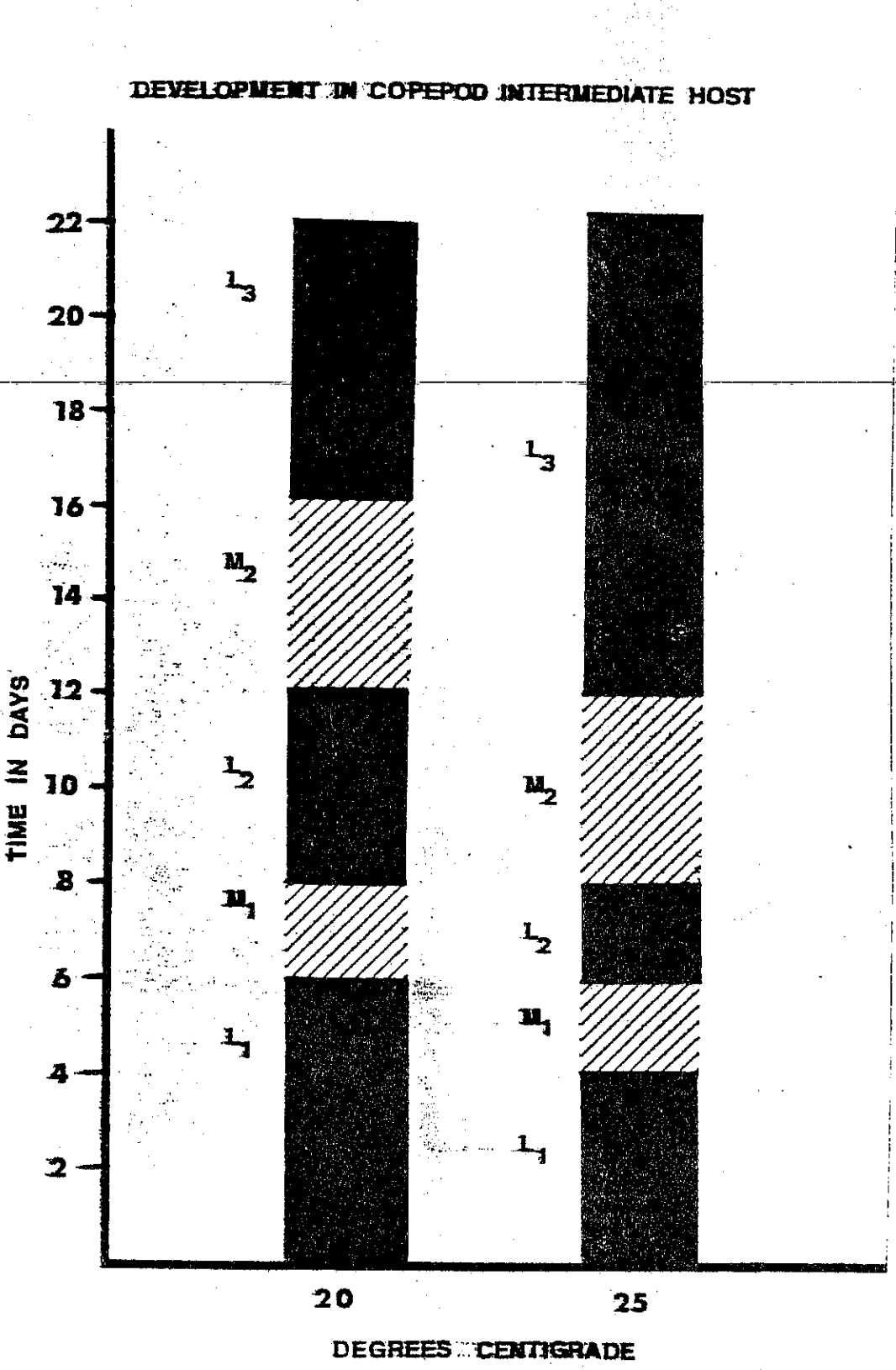
The series of infected copepods were maintained, one at 20°C and one at 25°C. Infected copepods from each series were crushed on consecutive days and the parasitic larval stages examined microscopically for molting and development. Each of these was studied for morphological changes and photomicrograhed. Parasitic first stage larvae undergo a development of the esophageal structures and a closure of the esophageal lumen and the open area in the tail fills with tissue. The first cuticular molt occurs 4 to 6 days after infection at 25°C and 6 to 8 days post-infection at 20°C (Fig. 10.)

The larvae are now referred to as second stage larvae. The second stage larvae undergo a marked lengthing of the esophagus and development of intestinal cells, the esophagus is twice the length of the intestine. The second molt occurs 8 to 12 days after infection at 25°C and 12 to 16 days at 20°C. The second molt is always marked by anterior stomal structures in the molting cuticle. The larvae are referred to as third stage after completing the second molt. In third stage larvae the esophagus is always more than twice the length of the intestine. The third stage larvae remain alive for 20 or more days in the haemocoel and are infective for fish definitive hosts. A single copepod may harbour from 1 to 24 larvae in its haemocoel and survive but only 10 larvae were used because of the possibility of stress to the copepod host.

Both living and preserved specimens of cyclopoid copepods infected during the summer were carefully studied and identified. Most prevalent among the infected copepods was Cyclops vernalis but Cyclops bicuspidatus and Orthocyclops modestus were also infected. Plankton samples taken 3 times per week during each summer indicated that C. vernalis and C. bicuspidatus were the most common cyclopoid copepods in the Bass Island region of Lake Erie. These quantitative plankton samples also enabled us to determine that free-living, first stage larvae are released from streaming female Philometra sp. at the time when the copepod population was reaching its peak each summer.

Figures 11, 12 and 13 illustrate graphically the cyclopoid copepod blooms in the Bass Island Region of Lake Erie and the synchronized release of the first stage larvae of Philometra sp. In 1972 (Fig. 11) active first stage larvae were found for the first time in the uteri of gravid females on 28 June and were present in many of the females recovered through 18 August. The earliest

Figure 10


DEVELOPMENT IN COPEPOD INTERMEDIATE HOST
TIME IN DAYS
22
20
18
16
14
12
10
8
6
4
2
L3
M2
L2
M1
L1
L3
M2
L2
M1
L1
20
25
DEGREES CENTIGRADE

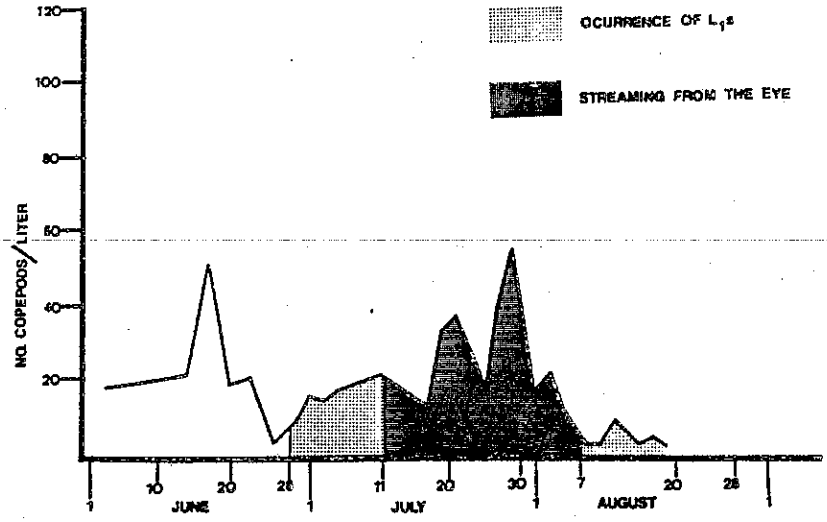


Figure 11. Summer 1972. Biosynchronization of copepod bloom and development of active first stage larvae of Philometra sp. and their release by streaming adult females.

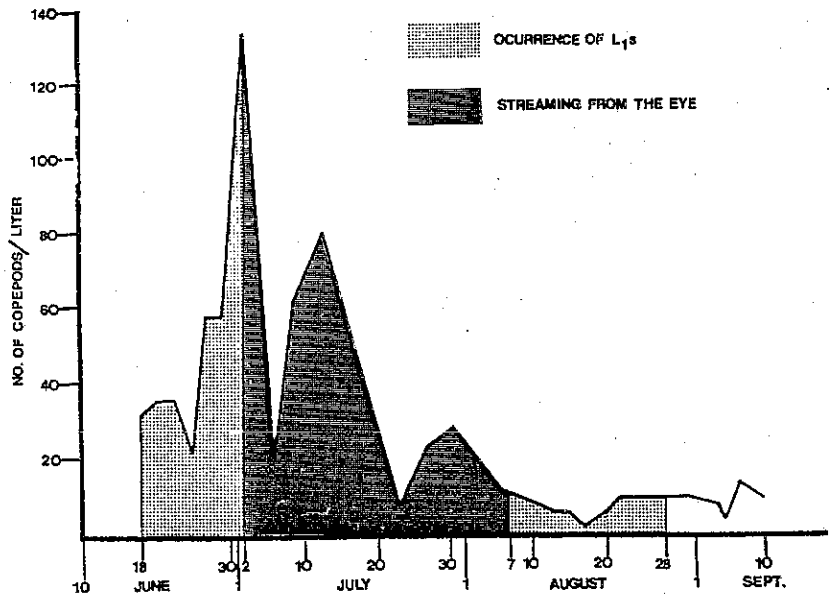


Figure 12. Summer 1973. Biosynchronization of copepod bloom and development of active first stage larvae of Philometra sp. and their release by streaming adult females.

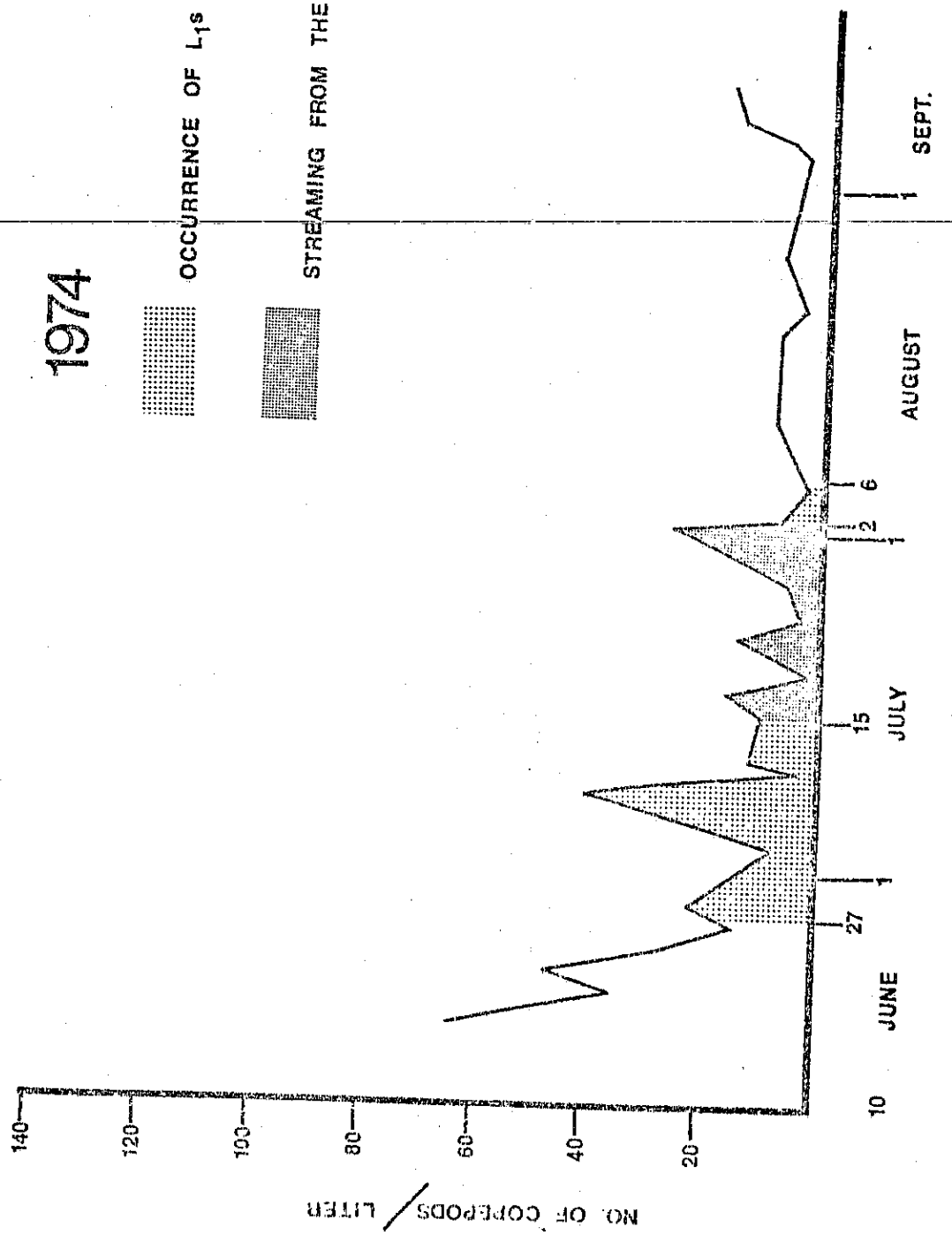

1974
OCCURRENCE OF L_1s
STREAMING FROM THE EYE
NO. OF COPEPODS / LITER
140
120
100
80
60
40
20
10
JUNE
27
1
15
JULY
1
2
6
AUGUST
1
SEPT.

Figure 13.

date for collection of freshwater-drum with females streaming from the eyes, thus releasing larvae into the water, was 11 July 1972. Fish with gravid female Philometra sp. streaming from the eyes were collected through 7 August 1972. The period of development of larvae and the streaming and release of larvae was correlated and synchronized with the summer peak of cyclopoid copepods. In 1973 active first stage larvae were found in the uteri of gravid female worms at the earliest date of 18 June and they were in the uteri of females collected through 28 August. Freshwater-drum with females streaming from the eyes, releasing larvae, were collected during the summer of 1973 from 2 July through 7 August. In 1973 the streaming and larval release occurred on the highest peak of copepod bloom and continued through the next major peak (Fig. 12) again correlated and synchronized with July period of high copepod abundance. In 1974 streaming and release of larvae occurred from 15 July through 2 August, a shorter and later period than in 1973 but similar to 1972 (Fig. 13.) In general the patterns for the 3 years were very similar and the principle of biosynchronization was clear but there were some differences in the 3 years which indicate that some external ecological factor determines the timing.

The following is a comparison of the three years: 1) In 1972, the copepod density never reached more than 80 per liter, in 1973, copepods were more abundant over a longer period of time reaching peak densities of 140 per liter. 2) In 1973, the first stage larvae of Philometra sp. were present 10 days earlier than in 1972, and the period of streaming lasted at least 10 days longer in 1973 (see Figs. 11 and 12.) These differences fit with other differences noted for the two years in earlier reports and are attributed to differences in water temperature. During the summer of 1972 water temperatures were lower than normal during June and July. In 1972, there was no time period overlap of gravid females with new juveniles entering the freshwater-drum population (see Fig. 11) but in 1973 a very slight overlap of these two stages occurred (see Fig. 12.) In 1974, the periods of larval release and streaming of adult females was very similar to 1972 (Fig. 13.) The spring and early summer water temperatures were also very similar.

In all 3 years the newly developing "young of the year" freshwater-drum reached a natatory and feeding stage and entered the Lake Erie population during the last of July and early August, just in time to feed on infected copepods.

When possible infection experiments paralleled, as nearly as possible, the natural situation as it occurred in western Lake Erie. Copepods which were to be used for experimental infections of Aplodinotus grunniens were infected during the last two weeks of July. These copepods contained fully developed, infective third stage larvae by mid-August. This same process was assumed to be taking place in the Lake at the same time. After 7 August, in all years, no female Philometra sp. were found streaming from the conjunctivas of the eyes of naturally infected Aplodinotus grunniens.

The primary purposes of experimental infections of fishes was to determine the routes of migration of different stages of Philometra sp. in the fish host, the different tissues involved, and the timing of development of the nematode stages. Fish utilized in infection experiments were collected, transported, stabilized, screened, anesthetized and infected as outlined in the procedures section of this report. Experimental infections of freshwater-drum were begun on 1 August 1972 and 1974, and on 14 August 1973. The fish utilized ranged in size from 13.2 to 19.3 cm in length.

The following outline represents a combination of the results of experiments in 1972 and 1973. A total of 74 fish were utilized, 36 in 1972, and 38 in 1973. Eight fish each year were used as placebo control fish, they were anesthetized and administered distilled water via stomach tubes.

Fish Number	Day Post-infection	
(1)	1	Visual autopsy, no larvae recovered
(2)	2	Visual atuopsy, 2 larvae in lumen of duodenum
(3)	3	Pepsin digest, no larvae recovered
(4)	3*	Section of duodenum, larvae present in lumen
(5)	4	Section of duodenum, larvae present in lumen
(6)	4*	Sectioning reveals no larvae
(7)	5*	Visual autopsy, no larvae in lumen
(8)	6	Visual autopsy, larvae in lumen
(9)	6*	Digest of alimentary canal, 2 larvae present
(10)	6*	Sectioning of gut, larvae present in muscles
(11)	7	Pepsin digest, 5 larvae present in digest of wal
(12)	7*	Sectioning of gut, no larvae observed
(13)	9	Pepsin digest, 1 larvae present
(14)	10	Pepsin digest, 1 larvae present, 2 larvae free in coelom
(15)	11*	5 larvae free in coelom
(16)	12*	4 larvae in coelom, no larvae in pepsin digest
(17)	16	1 larvae in coelom, no larvae in pepsin digest
(18)	16*	3 larvae in coelom
(19)	18	2 larvae in coelom, on gas bladder
(20)	18*	4 larvae in coelom, on gas bladder
(21)	20	1 larvae in coelom, on gas bladder
(22)	20*	1 larvae in coelom, on gas bladder
(23)	21*	3 larvae in coelom, on gas bladder
(24)	22	2 larvae in coelom, on gas bladder
(25)	24*	2 larvae in coelom, on gas bladder
(26)	26*	2 larvae, along the parasphenoid bone anterior to the gas bladder, no larvae in the coelom or eyes
(27)	27*	One juvenile in the right eye, 1 juvenile in the left eye
(28)	33*	2 young adults in the left eye
(29)	33*	1 young adult in the right eye
(30)	35*	1 young adult in the left eye
(31)	36*	2 young adults in the right eye, and 2 juveniles in the left eye
(32)	38*	2 young adults in the left eye
(33)	40*	2 young adults in the right eye
(34)	44*	1 young adult in the right eye, 2 juveniles in the left eye

* Indicates fish experimentally infected during the summer of 1973.
16 fish were utilized as placebo control fish.
24 other fish, 16 in 1972 and 8 in 1973, were experimentally infected and were found negative.

Twenty fish were experimentally infected in 1974 and the results further confirm those in the preceding outline.

The data outlined above enables one to state more definitively the times of migration within the fish definitive host. Third stage larvae are released in the lumen of the duodenum and are present there from 1 to 6 days post-infection (present in lumen of gut 6 days.) The larvae migrate through the intestinal wall from 6 to 10 days post-infection (present in wall of the gut 3 to 4 days,) and they remain in the body cavity or coelom from 9 to 25 days post-infection (present in the coelom 15 to 16 days.) After 15 to 16 days in the coelom the larvae migrate anteriorly along the muscles of the parasphenoid bone. In freshwater-drum the parasphenoid bone extends from the coelom, adjacent to the anterior end of the gas bladder, anteriorly and forms the medial base of both eye sockets. The larvae can be found along these muscles from 24 to 28 days post-infection (3 to 4 days migration through these tissues.) After 28 days post-infection the larvae enter the tissues surrounding the orbit of the eye and many enter the connective tissues of the lumen. As we shall demonstrate later in this report the female nematodes remain in the orbit, its surrounding tissues and in the eye muscles and tissue 10 to 11 months before streaming from the eye.

Six Perca flavescens fed copepods containing third stage infective larvae of Philometra sp. from the freshwater-drum were autopsied and all were negative for larvae of any type in the body cavities or eyes. These larvae are apparently uninfective for Perca flavescens which harbours a different species of philometrid nematode in its body cavity.

During 1972, 1973, and 1974 autopsies of drum from natural populations yielded parallel results which confirmed the experimental findings. In 1972, pepsin digests of the gut walls of naturally infected fish yielded larvae in August and larvae were also found free in the coelom; in September, larvae were found in the coelom and in mid and late September in the eyes; in October juveniles and young adults were found in the eyes. In 1973 and 1974, the results were very similar to 1972 except that the timing of stages was more spread. Larvae were recovered from the coelom and discovered along the muscles of the parasphenoid bone in August. Juveniles were already present in the eyes by late August. Juveniles and young adults were present in the eyes through the September and October collections.

In all three years 1+ and 2+ class freshwater-drum were recovered which had both young philometrid adults and encapsulations of previous infections in the same eye. This indicates that reinfection of the same fish and the same eye does occur.

Living nematode specimens were studied as soon as possible after removal from the fishes and photographed. The majority of nematodes from each sample were fixed, processed and arranged as sets according to tissue site and date recovered. Each set was arranged in series according to length, from shortest to longest, and studied in that order. Careful study of the sets, in series, of living and preserved specimens provided useful information concerning the development of philometrid worms in the body of Aplodinotus grunniens. Sections were correlated with the times, whole mounted nematode stages and tissues involved. A study of these sections enhanced our understanding of the parasitic nematode stages, tissues involved and the histopathology. Sections were prepared of uninfected fish for comparative purposes.

Third stage larvae are released by digestion into the lumen of the alimentary canal. Larvae were found in sections of the walls of the pyloric ceacae and the anterior intestine 2 to 4 days post-infection.

Philometrid larvae removed from the coelom and from films of liquid on the gas bladder 12 to 17 days post-infection were molting to become 4th stage larvae. Fourth stage larvae were also recovered from films of liquid on the gas bladder of naturally infected freshwater-drum during late August and early September. The lips were beginning to form but there were no discernable cephalic papillae. The esophagus was not yet fully developed and no giant esophageal gland could be seen. The esophageal intestinal valve was only weakly developed and no distinct cells could be seen. The gonoducts were developing and they were well beyond the genital rudiment stage but no sexual differentiation was apparent. The posterior end was rounded, not squared off in ventral view as in later stages. There were no caudal papillae. The intestine ends in a strand, perhaps representing the atrophied rectum, and this strand joined the hypodermis very near the tip of the tail. There was no functional anus even at this stage of development. These worms ranged in length from 0.7 to 1.5 mm in length.

The migration route along the muscles of the parasphenoid bone was discovered by studying cross-sections of the head of a

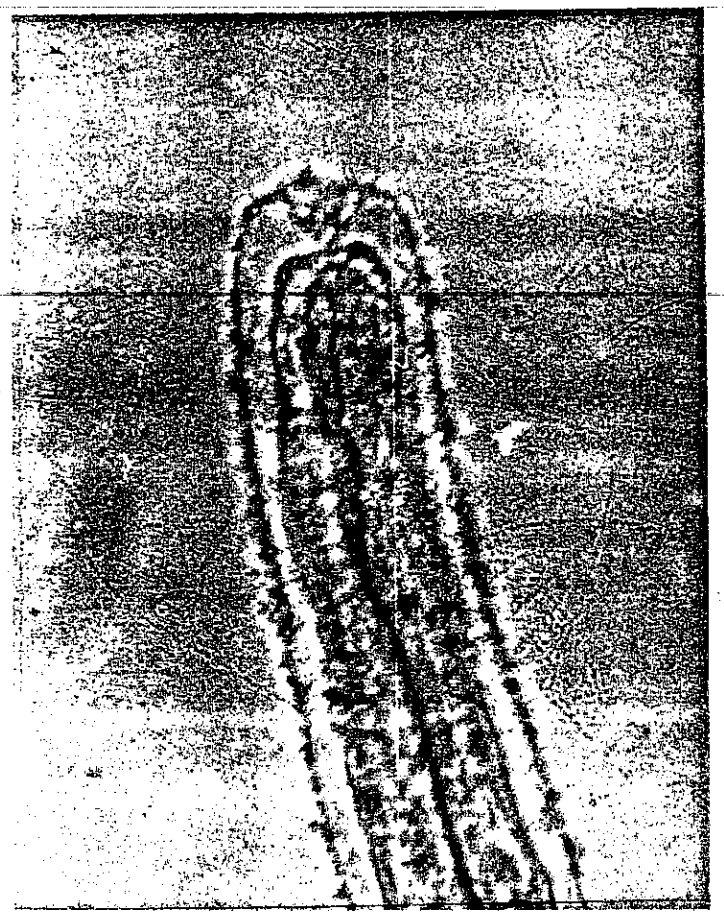


Figure 14. Fourth stage larva of Philometra sp. undergoing the fourth molt.

naturally infected fish taken 4 October 1972. During 1973, juvenile nematodes were found at this site in both experimentally and naturally infected fishes (Fig. 9.) Study of the few whole specimens recovered from this site shows that they were more advanced in their development but not yet mature adults, some had reached a length of 3 millimeters.

The nematodes of this species of Philometra molt for the fourth time and sexual differentiation occurs very quickly after they reach the orbit of the eyes and the tissues surrounding the orbit. Most nematodes taken from the orbit and its tissues in September and October were definitely mature adults. When nematodes of both living and preserved sets and series were examined both adult males and females were present. This is the first time that males of this species have been discovered. Living and fixed male specimens were studied in detail and photographed. The males were definitely mature, they had a fully developed single testis which was packed with spermatocytes. The vas deferens was packed with spermatozoa and there were well developed copulatory structures. There are two spicules and a very thin gubernaculum, the spicules are used in copulation. The sex ratio was one male per approximately 18 females. Males range from 2769 to 2918 microns in length, average length 2818 microns.

Fifty-two species of Philometra have been described from fishes from different parts of the world, only three have known males. Five species have been described from freshwater fishes in North America and none of these have known males. One closely related genus, Philometroides, occurs in the skin and fins of suckers in the United States and this male was described in an unpublished doctoral dissertation at Colorado State University (Daley, 1966.) We have hesitated from the beginning of this project to give a species name to the Philometra which we have recovered from the eyes of freshwater-drum in Lake Erie. Since male specimens are now available it can be correctly identified and described in detail.

The living females were studied and photographed as soon as possible after removal from the eyes. The preserved females of each set were studied according to length as an ascending series. The smallest females, late September and early October, were only slightly longer than the males, 3241.2 microns to 3617 microns (mean, 3377microns) in length. By 1 November the females range from 8,527.2 microns, 0.85 cm, to 51,493.4 microns, 5.2 cm, in length; the

mean length is 31,089.4 microns, 3.1 cm. The magnitude of increase in length was 9.2 times in 1974. This demonstrates that there was growth of the females during the autumn months. The smaller females have a well developed vulva (genital opening) and a duct-like vagina which leads from the vulva to the uteri, there are oviducts and reflexed ovaries at the anterior and posterior ends of the uteri. (Fig. 15) Females with a functional vulva ranged from 3240 to 5029 microns in length. (mean 3.63 microns) Copulation occurs very soon after the juveniles reach the eyes. Copulation with the male must take place while there is a functional vulva. After copulation there is a copulatory plug in the vulva and the uteri become a single longitudinal sac-like structure, extending the length of the body between the two oviducts, containing many spermatozoa. Following copulation the vulva and the vagina atrophy and the vulva is replaced by cuticular tissue (Figs. 16 and 17.) This explains the absence of a vulva in mature, gravid females recovered later in the spring and summer months. These females when they stream from the eyes during the summer have no other mechanism for release of larvae through the body wall except for the prolapsis of the uterus, there is no vulva.

Males die soon after copulating with females and become encapsulated with connective tissue. Very small fresh encapsulations were found in the eyes in late October of 1973 and these contained males of this species. Other small encapsulations were removed from the eyes of 0+ class fish in May 1973, microdissection of these capsules revealed the characteristic spicules and gubernacula of the males of this species. Other small encapsulations removed at this time appear to be small philometrid females which did not survive over the winter.

After copulation while the females continue to grow, oocytes leave the ovaries, enter the oviducts, and begin to enter the uterus where they enlarge and are fertilized and form zygotes. As the females become progressively larger the uterus completely fills with zygotes. In all 3 years the females taken in late October were all fertilized and their uteri filled with zygotes. With the onset of winter temperatures the post-fertilization females ceased growing and there was no further development of the zygotes. Post-fertilization females recovered in April of 1973 ranged from 3.4 cm to 6.4 cm (mean 4.0 cm) in length. These early spring post-fertilization females had their uteri packed with zygotes, none had yet started cleavage.

Study of these ascending series of autumn females also made other morphogenic developments and comparisons evident. Cephalic

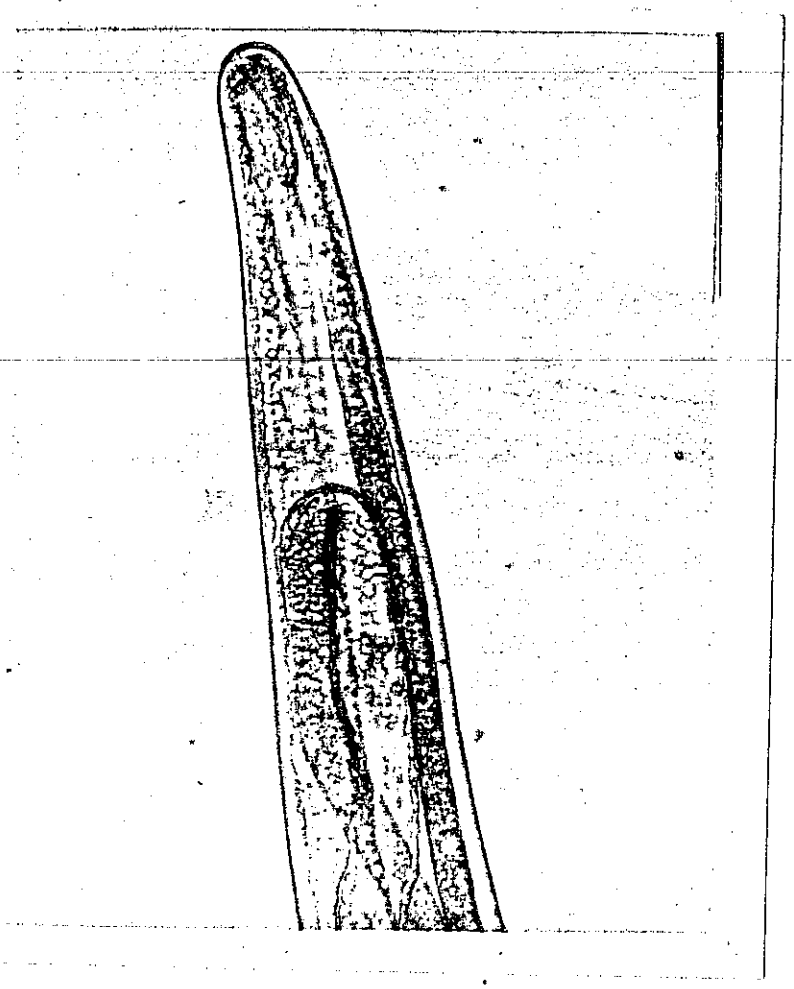


Figure 15. Posterior end of young adult female, posterior loop of the uterus, lateral view.

Figure 16. Vulva copulatory plug, young adult female, lateral view.

Figure 17. Vulva is replaced by cuticle, lateral view.

papillae begin to show development on the smaller specimens of the series. An examination of en face preparations shows a distribution of papillae typical of the genus Philometra, 8 large papillae in the outer circle and 4 papillae in the inner circle with 2 lateral amphids.

Two caudal papillae so prominent in the fully grown, mature, gravid females begin their development at this time. These caudal papillae are visible both in lateral and dorsal-ventral view of the larger specimens examined. In ventral view the tail is blunted and unrounded. None of these post-fertilization females had a functional anus.

The cells forming the cardia or esophageal-intestinal valve, become quite evident in smaller specimens of the series. Study of larger specimens shows that 4 very large uninucleate cells comprise this organ at this stage of development. These cells are evident in both whole mounts and cross and longitudinal sections.

In almost all of the young adults and autumn post-fertilization females a large gland cell is quite prominent in the middle one-third of the esophageal length. This gland cell was demonstrated in sections, and in preparations of the esophagus dissected and mounted alone. We have also observed other smaller gland cells and many prominent nuclei in the esophageal tissue utilizing sections and phase microscopy. These structures have been photographed. From a functional point of view it could be hypothesized that the giant cell secretes enzymes which enable these nematodes to feed and migrate through tissues. In future work this gland may merit a histochemical study. If it does secrete enzymes they may be related to the pathology which occurs in the eye tissues of the fish host.

Observations from gross examinations for gross pathological affects in the eyes of 0+ and 1+ freshwater-drum taken in October indicated that young adult worms were present in the connective tissues surrounding the eye socket, attached to muscles and to the sclera of the eye globe. Small hemorrhages were often present where the nematodes attached or were burrowed into tissue.

Heads of fish infected with young adult worms were taken in early October and sections were prepared and compared with sections of uninfected fish for histopathological affects. The procedures were as given earlier in the report. In sections, worms

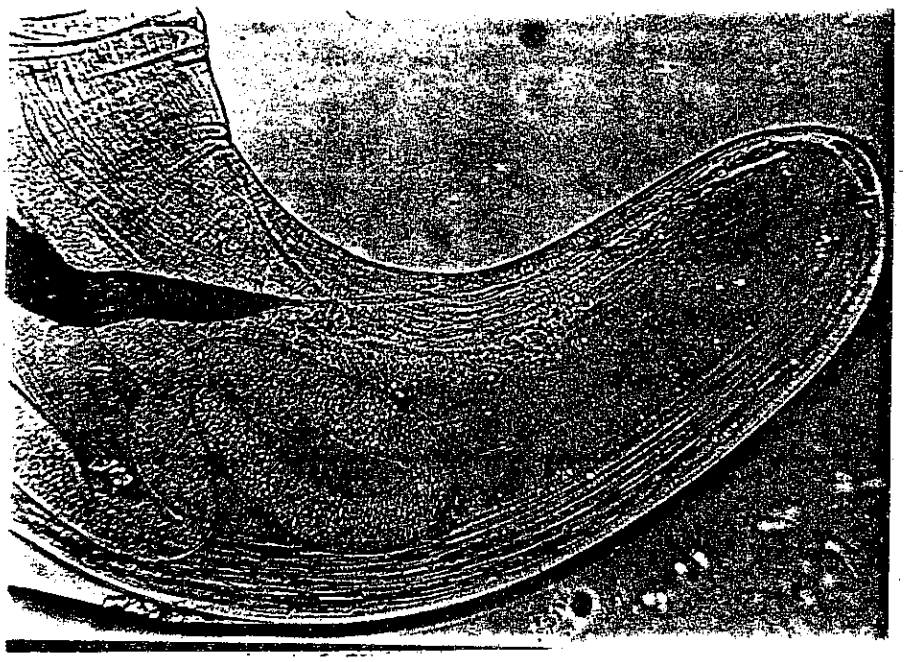


Figure 18. Posterior end, female, Philometra sp.

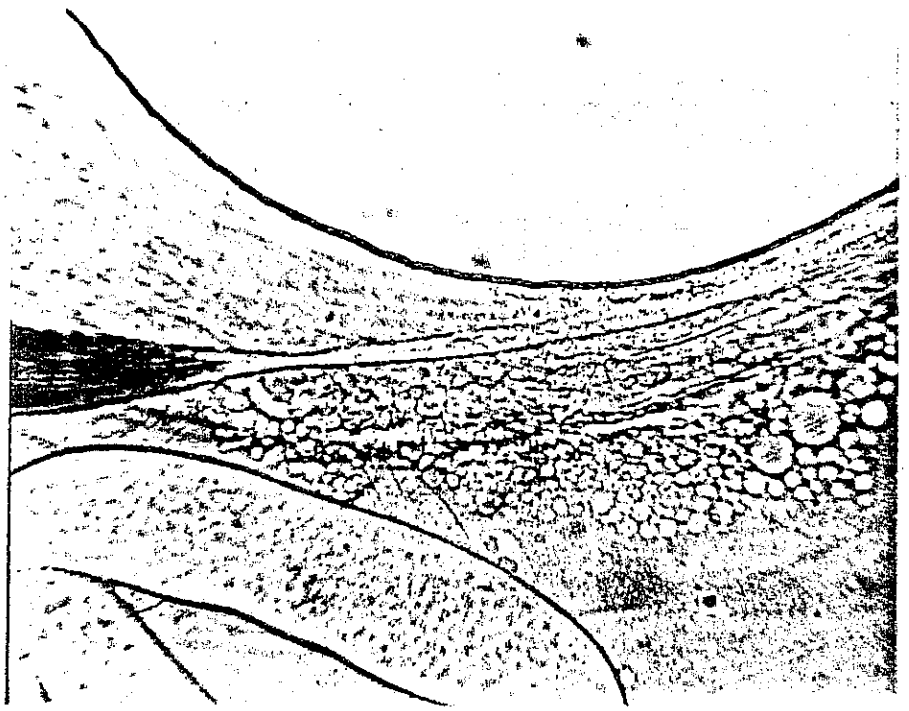


Figure 19. Region of the atrophied rectum attaching to body wall.

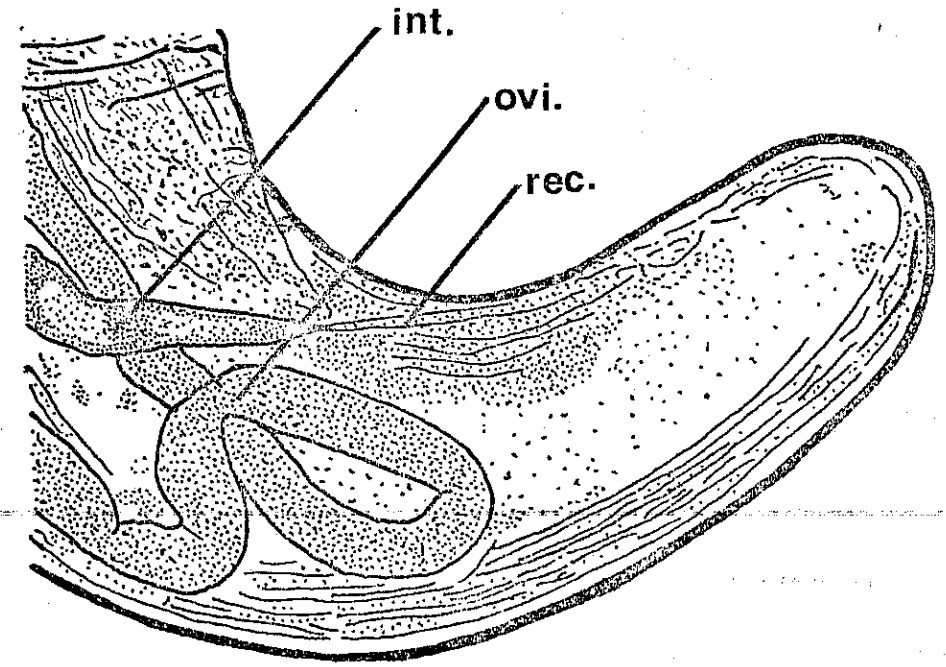


Figure 20. Legend: int. intestine; ovi. oviduct; rec, atrophied rectum.

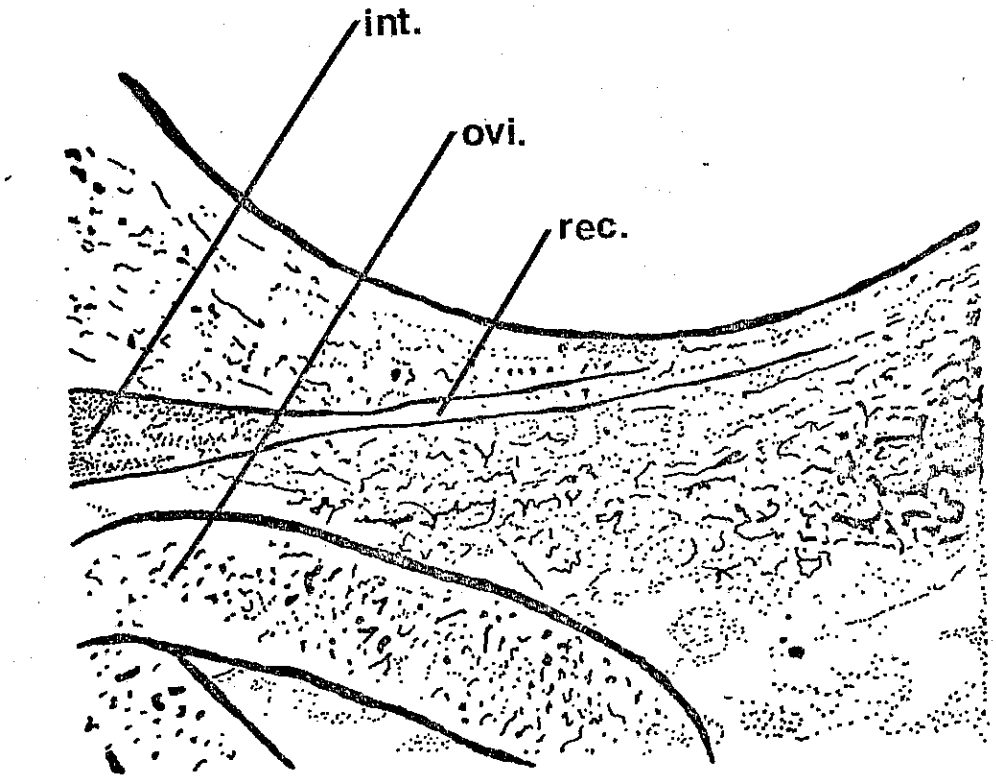


Figure 21. Legend: int. intestine; ovi. oviduct; rec, atrophied rectum.

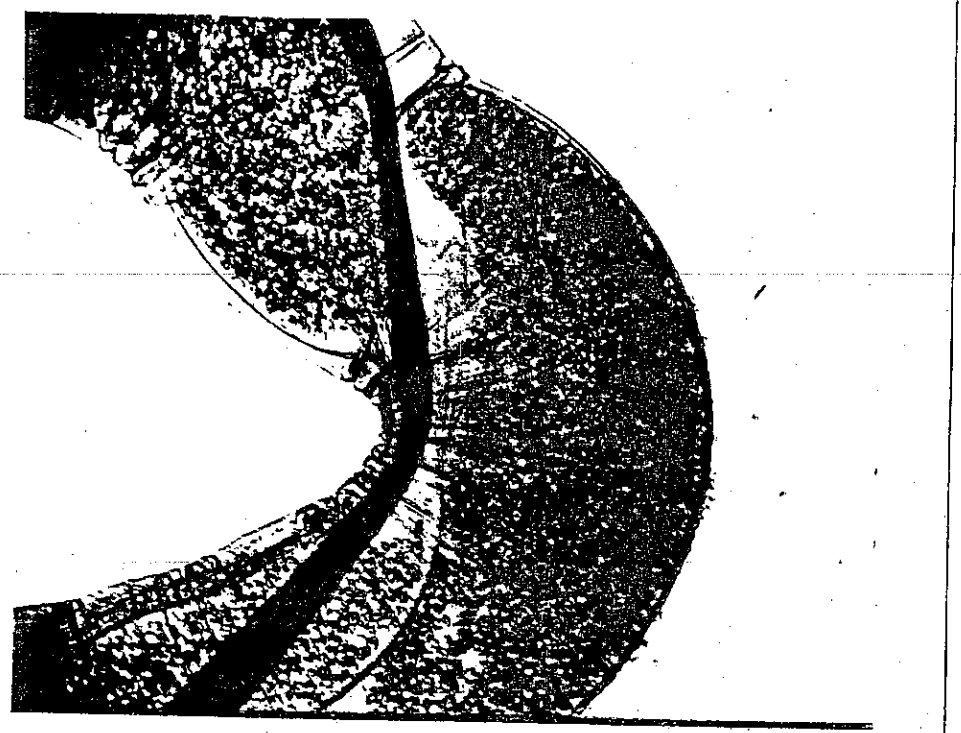


Figure 22. Early spring female packed with zygotes.

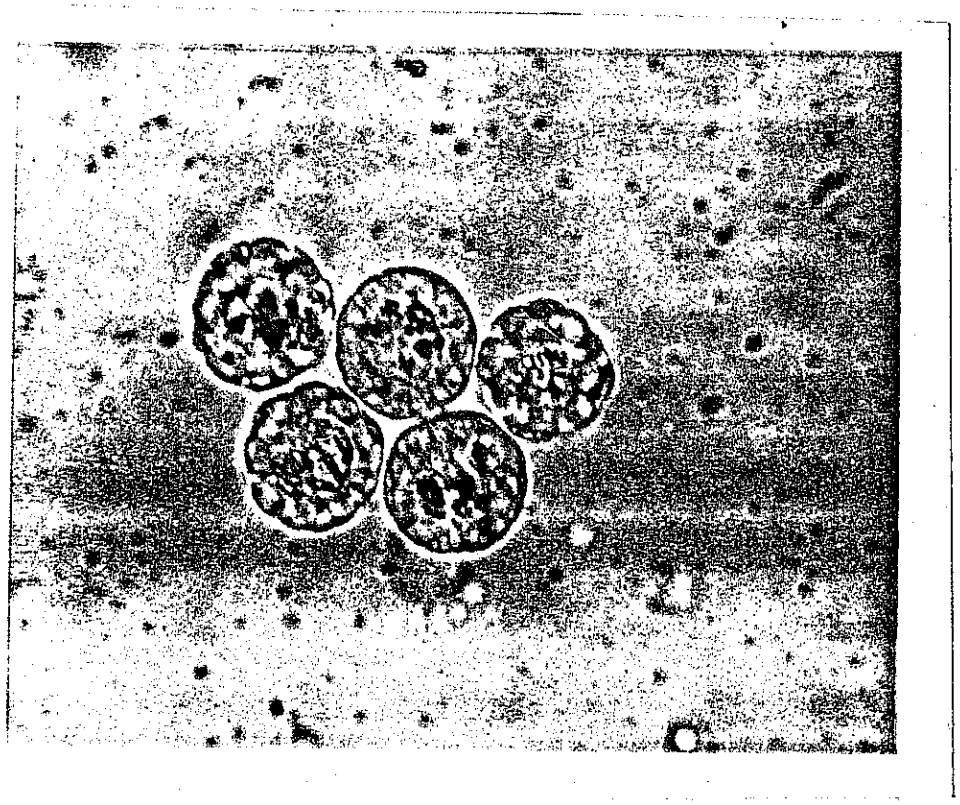


Figure 23. Blastula stages.

Figure 24. Anterior end of a June female.

were found entering the orbit of the eye from the muscles of the parasphenoid bone, usually on a posterio-ventral angle. They invade the cavity of the orbit of the eye and invade and damage the following tissues. 1) The epithelial membranes surrounding the orbit and the mucus membranes within the orbit. 2) They invade and erode the rectus and other muscle tissues. 3) They invade and erode the nerves. 4) They invade the tissues of the tunica external and the sclera. 5) They puncture capillaries and there are usually hemorrhages surrounding them, often leucocytic reactions occur. These young adult worms feed primarily on blood of the fish host. Sections of the worms in situ and singly show the intestine packed with fish erythrocytes. This is easily established by a comparison of the cells in the lumen of the intestine of a worm with the red blood cells in capillaries of undamaged fish tissue. They are identical.

This establishes that the histopathology caused by these worms during their early stages in the autumn months may be very severe particularly when there were infections of 10 or more worms in one eye. These parthological conditions were photographed using 35 mm transparency film.

As mentioned earlier with the onset of winter temperatures the post-fertilization females in the eyes cease growth and development and there is no further development of the zygotes in their uteri. The majority of these females reach a length of 3.1 cm to 5.2 cm by early November in fish in Lake Erie. Fish taken from the same population the next April contained females in their eyes which ranged from 3.4 cm to 6.4 cm (mean 4.0cm) in length and all embryos were still in the zygote stage but the uteri were packed with thousands of these forms (Fig. 22.) As we shall show later in this report the "young of the year" fish hosts grow very little during this same period as they overwinter. After a short period of time in May as the waters of the lake warm the female nematodes begin to grow in length. The growth to the full length and the concomitant intrauterine development of larvae occurs in spring and early summer months as described in the first part of this report (Fig. 8.) These females reach a length of 13 to 23 cm (mean 16.2 cm) by the time they stream from the eyes in July. They are referred to as grand females, when they contain developing embryos and larvigerous females when they contain active first stage larvae. Gravid females 9 to 13.5 cm (mean 11.5 cm) taken in late June and early July still show some cephalic papillae but they are degenerating as large ridges form at the anterior end and the lips fuse and become indistinct (Fig. 24.) Two large caudal

papillae can still be seen clearly in most specimens in whole mounts and the tail is truncate. Internally the intestine is still filled with fish erythrocytes and the intestine attaches to the body wall by a solid ligament (Figs. 18, 19, 20 and 21.) Sections of the posterior part of the body show that the muscles are beginning to atrophy.

Larvigerous females were taken from the eyes in July just before streaming reached its height for comparative morphological studies, 13 to 23 cm (mean 16.2 cm) in length. The specimens were studied in cleared whole mounts, in section and by stereo-electron scan. Both photomicrographs and stereo-micrographs were prepared. These oldest and terminal larvigerous females have a functional esophagus and they may have ceased feeding as the intestine was filled with only digesting material and no fish erythrocytes with intact membranes could be found. The lips which were present in the young adults were now degenerate and fused and no cephalic papillae were evident even in stereo-scan micrographs (Figs. 25 and 26.) The muscles of the body wall were still functional from the intestinal-esophageal valve anterior but other body wall muscles and hypodermal tissue appeared degenerate. The hypodermal areas between the muscle quadrants were thinner in these older females than it was in the young adults of the previous spring even though these females were much longer and have a greater diameter. The tail was now rounded and the caudal papillae were difficult to discern. The uterus when filled with active first stage larvae has a very thin wall, in most places only one cell layer thick. The females at this time literally become long thin-walled bags of tissue filled with thousands of first stage larvae. Since only the anterior end was functional we have assumed that it was involved in the processes of making the opening through the conjunctiva of the eye. The anterior end however usually stays anchored in the eye tissues and it is this portion of the worm which becomes encapsulated by connective tissue.

Sections of heads of freshwater-drum with "popped-eyes" collected in mid-July showed damage and histopathology different from that caused by the small young adults during the autumn months. Much of the same tissue damage was still present but it was more extensive. Muscle tissue, nerve tissue, the tissues of the sclera and the tunica were more eroded. The capillaries showed more extensive damage and the areas of hemorrhage were greater. Often hemorrhages appear as large clots in gross examinations. In some sections there was invasion of the optic artery. Damage and erosion of the choroid glands was present. There was a leucocytic invasion of the connective tissues and the

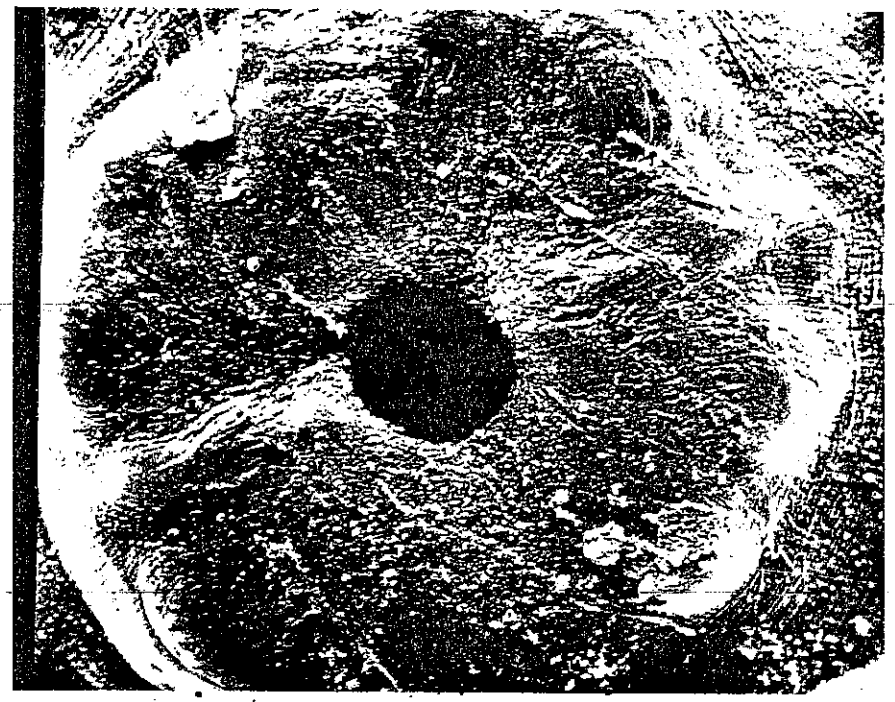


Figure 25. Stereo-scan photograph, 1275X, En face view.

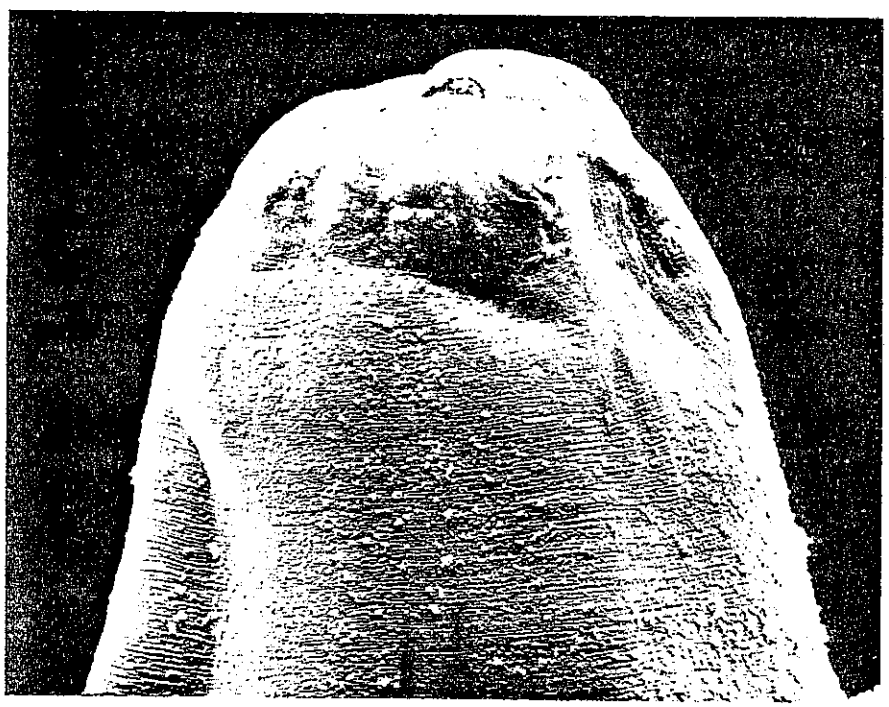


Figure 26. Stereo-scan photograph, 765X, Ant. End Lateral view.

areas surrounding the worms showed massive concentrations of leucocytes. This probably accounts for much of the inflamation syndrome seen in gross external examinations. The adults were seen with most of their bodies in the orbit lumen and they lay coiled completely around the eye ball. This was true in both sections and in gross examinations. In multiple infections the "pop-eye" syndrome was always evident. At this time one of the major problems is the great pressure built up in the eye due to the great size of the nematodes and inflamatory reactions. Sections through eyes with multiple infections showed great compression damage and break down of tissue caused by these pressures.

As many as 9 larvigerous females have been removed from one eye and double eye infections are more prevalent than single eye infections (Figs. 61, 62 and 63.) In cases where the exopthalmic syndrome was present the pressure was always great enough to make the eye swell and extend from the socket stretching the muscles, external tissues and exerting pressure on the optic nerve and retina. These conditions considered together with the demonstrated histopathology make it apparent that normal functions are greatly impaired or completely lost in freshwater-drum which exhibit the syndrome. Experiments with flashing strobe lights in a completely darkened room indicate the fish suffering the syndrome are at least temporarily blinded during the terminal portion of the infection. Infected fish also showed significantly different reactions in a thermal gradient.

The recovery of encapsulations from 1+ age class and older fish does indicate that many freshwater-drum survive the infection if they are not lost from the population because of complications of the infection or preyed upon by larger predacious fishes to which they may be easier prey. However, statistical tests (Mann-Whitney U) fitted to a Crofton Model (Crofton, 1971) indicate that as many as 16% of the 0+ class infected fish may be lost from the population.

In late December 1973 I was given two Aplodinotus grunniens taken in Sandusky Bay and maintained in a large aquarium. The aquarium was held at the temperature of Lake Erie water until 3 November 1973 and maintained at a temperature of 22°C until 20 December 1973. The first fish, 11 cm in length, 0+ age class had the globe, muscles and conjunctiva of both eyes completely missing, only a stub of the optic nerve of each eye was present. The fish was still alive but swimming in an abnormal manner. The orbits of both eyes were carefully examined and a small fragment of a nonencapsulated philometrid worm was found in the left orbit.

The orbits of both eyes were photographed (Figs. 27 and 28.) We would admit that there is a possibility that a heavy case of philometrosis (multiple infection) could have been responsible for the loss of the eyes, particularly as development procedes more rapidly at higher temperatures as is demonstrated in the case of the second fish, but there may be still another explanation. This fish was kept in a large aquarium with other fish of different genera. We have noticed that when freshwater-drum with streaming adult females are kept in aquaria the worms will float off the sides of the infected fish and other fish will tend to feed on them. This could lead to an almost complete loss of the eyes. This is only speculation at this time but it could have some management implications if freshwater-drum were crowded together with other fishes in small warm water ponds. It seems unlikely that philometrosis would account for complete loss of the eyes in any other manner.

The second fish, 13.4 cm, 0+ age class, was in an early stage of the double "pop-eye" syndrome. Both right and left eyes were slightly swollen and reddened from inflamation; philometrid nematodes could be clearly seen through the conjunctiva in the ventro-anterior corner of both eyes. Necropsy yielded one philometrid from each eye, both nematodes were females, 7.9 cm and 8.2 cm in length and both had already attained a bright red color.

This growth both in length and morphogensis of Philometra sp. in the infected fish maintained at warmer temperatures was equivalent to that attained by philometrid nematodes over wintering in the eyes of naturally infected fish and recovered in May of 1972 and 1973. These two female worms contained embryos undergoing development in their uteri; early cleavage stages in the first and last blastula stages in the second. These stages of embryonic development also parallel stages seen in female worms during May or June in natural infections. It has been demonstrated experimentally that temperature affects the growth and development of the first three larval stages in copepods and I have stated before that I believe that temperature has an effect on the growth, development, population regulation and environmental synchrony of the adults of Philometra sp. Measurements of Philometra sp. taken in the late fall and early spring have shown little or no growth occurs in the eyes of naturally infected fish during the winter months. Most growth in length and embryonic development was arrested during the period November through late April. This period of arrested development provides the timing in Lake Erie which leads to an annual life cycle and a

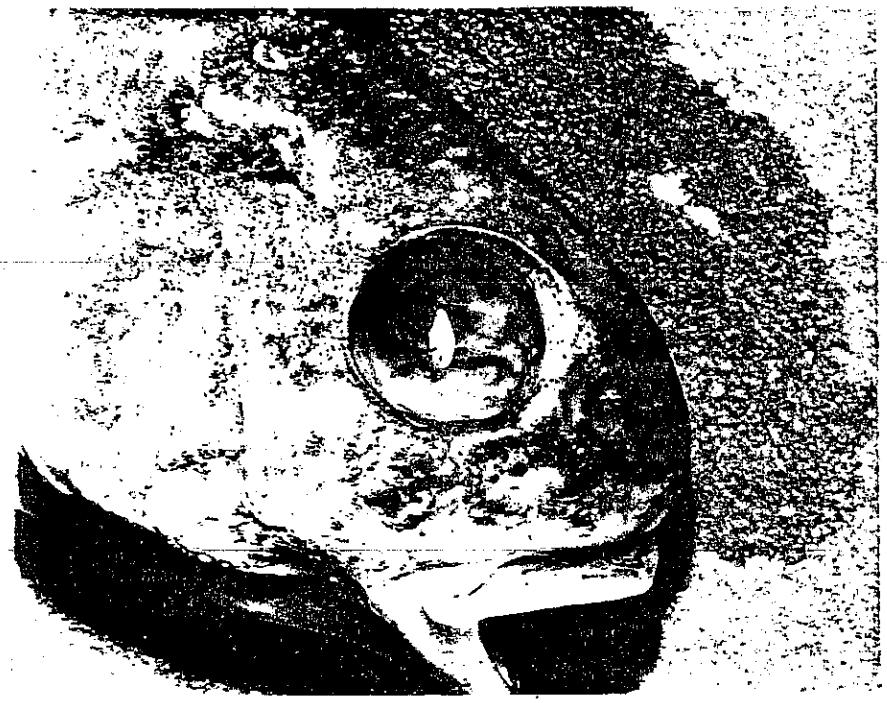


Figure 27. Right orbit of the eye, freshwater-drum.

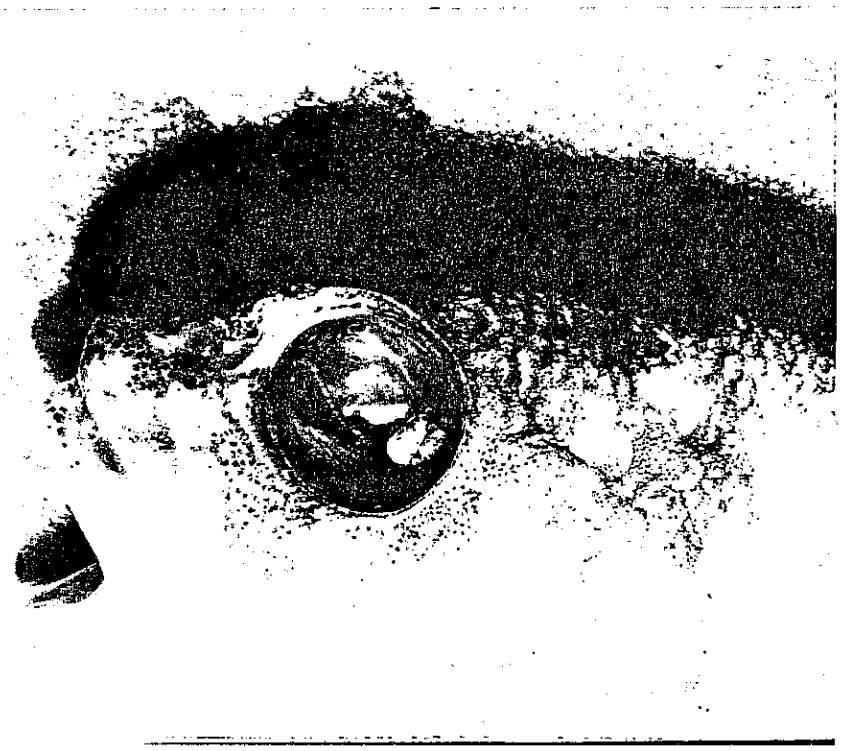


Figure 28. Left orbit of the eye, freshwater-drum.

synchrony of release of larvae at a time when there is more than adequate supply of copepod intermediate hosts present. Extrapolating from our data concerning worms removed from fish eyes under natural temperature conditions we can estimate worms from these fish maintained at warmer temperatures would have completed their development and released larvae 6 months earlier than those in Lake Erie.

These findings could have some significance in management of freshwater-drum and portend rather serious consequences if this nematode is spread to freshwater-drum in warmer climates.

Some investigators such as Rasheed (Rasheed, S. 1963,) have maintained that it is enough to describe and identify species of the nematode family Philometridae using only the characteristics of the mature, gravid females. We do not agree with this because as we have demonstrated there is a gradual development and in some species, such as that in the eyes of freshwater-drum there is an annual transmission cycle where fully developed females occur only in one period of the year as in Lake Erie. Description and identification of Philometridae should also be based on male characteristics, and in the vulvular stage of the young adult females whenever possible.

In the course of our work for this project we have also recovered a philometrid nematode from the body cavity of Perca flavescens. We have identified this nematode as Philometra cylindracea Ward and Magath, 1916. This nematode was inadequately described and no males are yet know. Bangham first reported this nematode from yellow perch in Lake Erie in 1939 (Bangham and Hunter, 1939.) We are convinced that even by Rasheeds criteria that the species in the eye of the sheepshead is not P. cylindracea. There are consistent differences in the adult females: the shape of the body; the shape of the head; the number of cephalic papillae; the shape of the tail; the shape, position and size of the caudal papillae; the shape and structure of the esophagus and cardium. Infection experiments using copepods infected with larvae from sheepshead were negative when these copepods were fed to 6 Perca flavescens. A study and comparison of larval and juvenile stages from the body cavity of Perca flavescens and the eyes of Aplodinotus grunniens made during the year further strengthens our belief and contention that these are two different species. The larval specimens from perch have characteristics which correspond with those of adult specimens from perch. Those from the eyes of freshwater-drum have characteristics which correspond with the

adults from the eyes of freshwater-drum. We have prepared a redescription of the females and a description of the male of P. cylindracea and the publication is now in press (Ashmead and Crites, 1975.)

Population Investigations of Philometra sp. In the project proposal statements were made to the effect that the incidence and intensity of infection of parasites in different size and age classes of natural populations of fish hosts should be checked throughout the year when possible. We pointed out that such information is usually missing in studies dealing with parasites of fishes and that this type of data could be useful to fisheries biologists and in fisheries management. This section of the report presents quantitative data for infections of Philometra sp. in the eyes of freshwater-drum from Lake Erie. It presents the frequency distributions of freshwater-drum examined from June through October 1972, and May through October 1973, and the frequency distribution of populations of Philometra sp. infecting them. It also presents the data concerning specific months of the year when infections occurred and specific size and age classes of freshwater-drum infected.

The scatter-diagram graphs which are presented here were obtained in the following manner. A single computer card was punched in FORTRAN computer language for each freshwater-drum collected from natural populations in Lake Erie and autopsied for evidence of philometrid infection. Experimentally infected fish and fish examined by digestion techniques were not included in this data. More than 1,000 freshwater-drum were examined in the sampling season of 1972 and 838 fitted this category and are represented on the graphs. In 1973 2,190 freshwater-drum fitted this category, 2,176 were plotted by the computer, 14 were too large to fit the computerized composition of the scatter-diagram graphs. In 1974, 857 Aplodinotus grunniens were autopsied and 796 fell within the bounds for computerized proportioning, a total of 3662 freshwater-drum examined and the data analyzed over the three year period. This data and the OMNITAB programs have been imprinted on tape discs and are recallable. The data was taken directly from research notes, Volumes 1 through 8 and punched on cards in the exact sequential order of examination after determining the program code to be used. The OMNITAB program was used to plot all scatter-diagrams on an IBM 370/165 computer.

All graphs, figures and tables presented include fishes from open lake trawls taken between Green and Rattlesnake Islands,

Lake Erie, trawls off Locust Point, Lake Erie and the Cold Creek samples collected in Sandusky Bay.

Length and Age Class of Freshwater-Drum Examined: Conversion of lengths to age classes of freshwater-drum from Lake Erie presents some problems and we have accepted the measurements given by more expert investigators in recent works.

Russell Scholl and Carl Baker (personal communication) provided us with average length measurements of age classes of freshwater-drum taken at West Reef Lake Erie. Their data shows:

0	YOY	-to October-	16.12 cm ave.
1+	2 annuli	7.2 inches ave.	=18.27 cm ave.
2+	3 annuli	8.6 inches ave.	=21.82 cm ave.
3+	4 annuli	9.6 inches ave.	=24.36 cm ave.
4+	5 annuli	10.4 inches ave.	=26.40 cm ave.
5+	6 annuli	11.1 inches ave.	=28.17 cm ave.
6+	7 annuli	12.0 inches ave.	=30.46 cm ave.
7+	8 annuli	13.4 inches ave.	=34.00 cm ave.

Tubb, 1972, and Herdendorf and Hair, 1972, gives the following data for freshwater-drum collected at Locust Point, Ohio, Lake Erie. (R.A. Tubb, 1972.)

Their data for freshwater-drum shows:

Age Class	Range in Cm	Mean Total Length in Cm
0+	3.8-14.2	$\bar{x}$- 9.9
1+	13.0-19.1	$\bar{x}$-16.3
2+	16.3-23.9	$\bar{x}$-21.6
3+	22.6-35.6	$\bar{x}$-29.7
4+	25.9-39.1	$\bar{x}$-31.2
5+	31.0-41.9	$\bar{x}$-37.1
6+	31.2-42.9	$\bar{x}$-38.4

Frequency Distributions of Freshwater-Drum Examined in 1972 and 1973: The following graphs, Figures 29, 30, 31, and 32 are computer generated scatter-diagrams which display the length distribution of fish autopsied versus the time of year sampled. In these diagrams fish are represented by either a single dot or a numeral at a given point. When a numeral is displayed this represents the number of fish (more than one) at a given length autopsied at a given time. In all 4 diagrams the length of the freshwater-drum in centimeters is given on the ordinate and the month of the year is represented on the abscissa. In the 1973 readout (Graph B) the first M on the abscissa represents March 1973 and the second M represents May 1973.

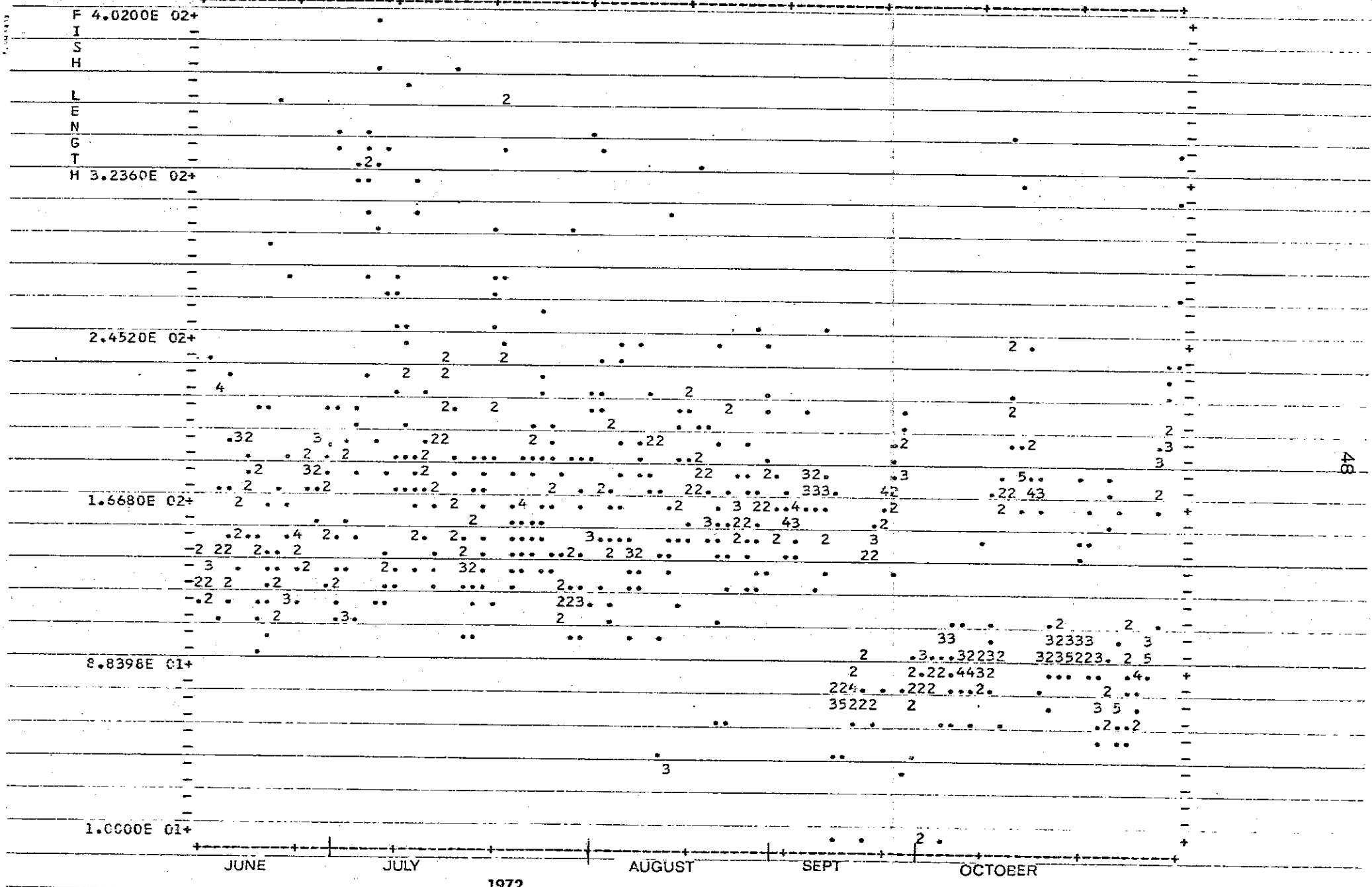
In 1972 (Figures 29 and 31) we felt that our samples were biased toward 0+ through 3+ to 4+ age class fish. The 1973 scatter-diagram (Fig. 30) illustrates that we had a more than adequate sample of fish from these age classes and that we also had adequate samples of freshwater-drum of the age classes 4+ through 7+. As will be demonstrated the incidence and intensity of infection with Philometra sp. in the eyes is most prevalent in age classes 0+ through 2+ regardless of the source of age class data. As all the scatter-diagrams indicate our sample size was very strong for these age classes in all three years. Data and statistical analyses of 1973 autopsies should be very valid for age classes 0+ through 7+ and comparisons and comparative analyses of 1972, 1973, and 1974 data should be valid for age classes 0+ through 5+.

In each year there is a definite break or gap between "young of the year" freshwater-drum, represented in the lower right-hand corner of each diagram, and the older age classes of freshwater-drum. A comparison of these groups of "young of the year" on the four diagrams demonstrates two interesting differences. In 1972, and 1974, the small "young of the year" entered our samples in mid August and grew to a maximum length of 11.62 cm by 30 October 1972. In 1973, the small "young of the year" entered our samples in mid July, a full month earlier, reaching from 14.91 to 16.6 cm in length by 18 October 1973. In 1973, the "young of the year" were present sooner and grew to longer lengths. We believe that this may represent the effect of warmer water temperatures in Lake Erie during the summer of 1973. Another inference which can be made is that with warmer water temperatures the pelagic embryos of freshwater-drum develop more rapidly.

OMNITAB PHILOMETRA STUDY
1972 FISH LENGTH/TIME

Figure 29

ABS- COLUMN 1; ORD- COLUMN 2 (.),
TOTAL NO. OF PTS. PLOTTED IS 796 AND NO. NOT PLOTTED BECAUSE THEY FALL OUTSIDE OF BOUNDS IS 3

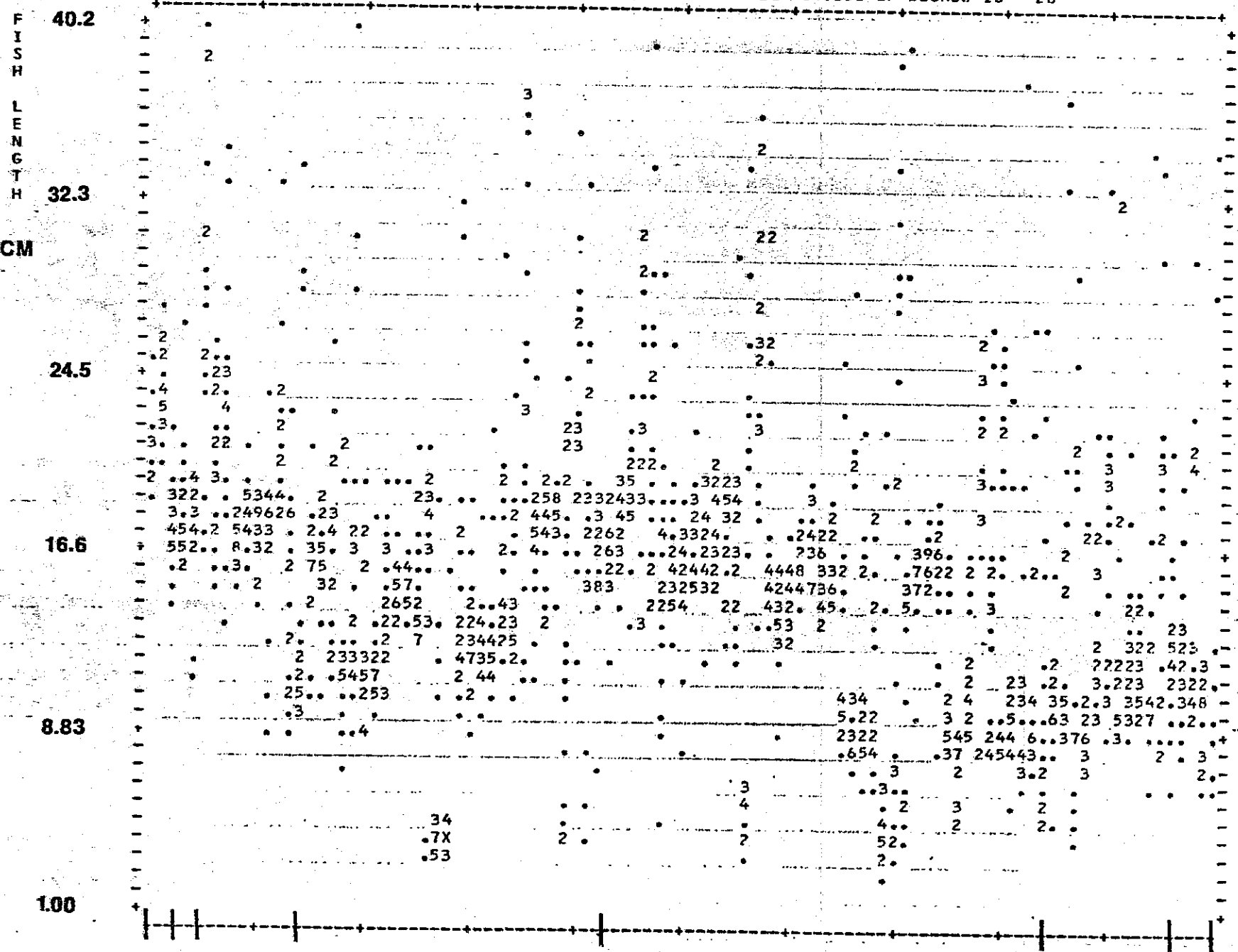


1973 OMNITAB PHILOMETRA STUDY PAGE 1

FISH LENGTH/TIME

Figure 30

ABS- COLUMN 1; ORD- COLUMN 2 (.),
TOTAL NO. OF PTS. PLOTTED IS 1893 AND NO. NOT PLOTTED BECAUSE THEY FALL OUTSIDE OF BOUNDS IS 28

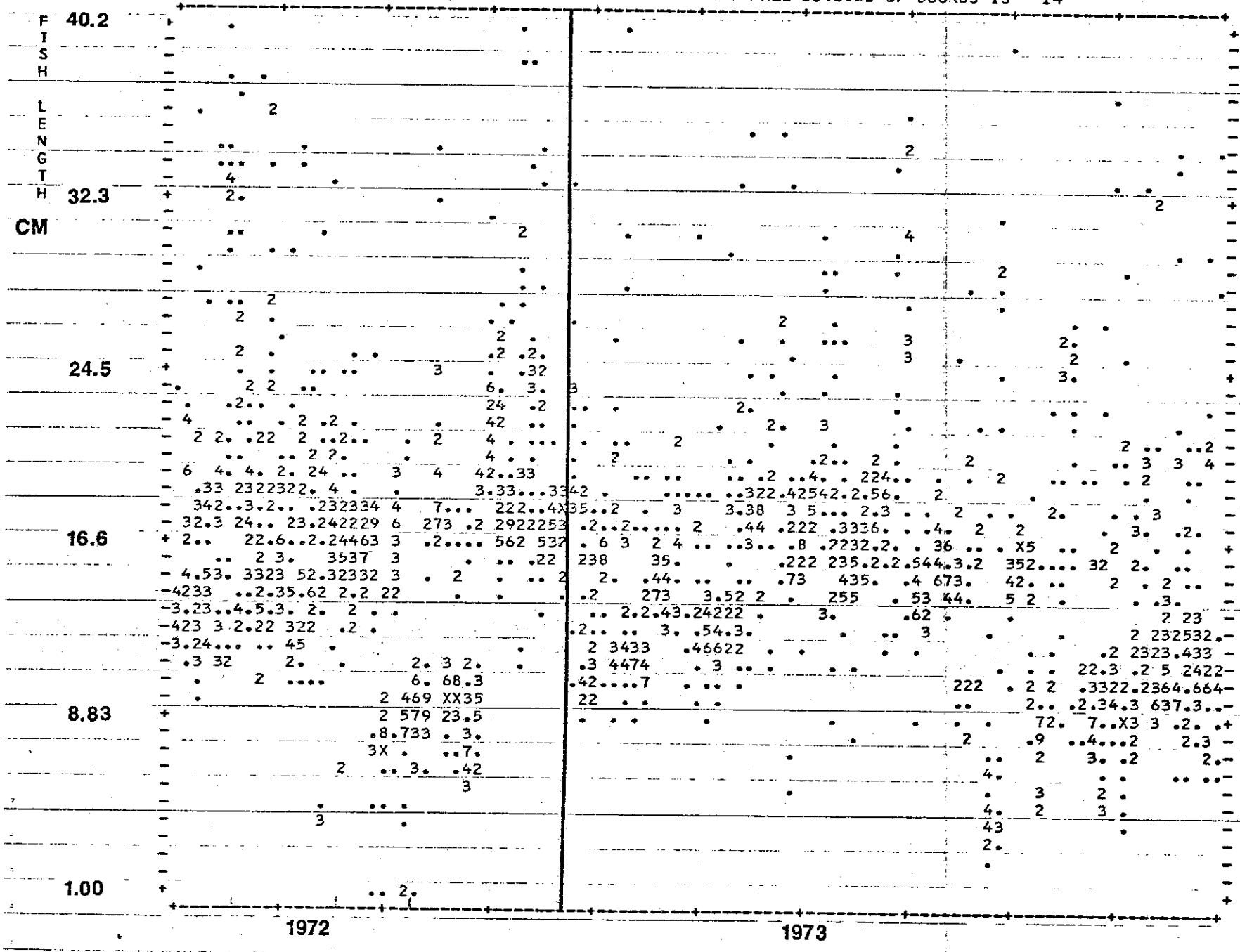


1973 OMNITAB PHILOMETRA STUDY PAGE 1

FISH LENGTH / TIME

Figure 31

ABS- COLUMN 1; ORD- COLUMN 2 (.),
TOTAL NO. OF PTS. PLOTTED IS 2176 AND NO. NOT PLOTTED BECAUSE THEY FALL OUTSIDE OF BOUNDS IS 14



1974 OMNITAB PHILOMETRA STUDY **fish length vs time** Figure 32 PAGE 1

ABS- COLUMN 1; ORD- COLUMN 2 (-).
TOTAL NO. OF PTS. PLOTTED IS 835 AND NO. NOT PLOTTED BECAUSE THEY FALL OUTSIDE OF BOUNDS IS 2

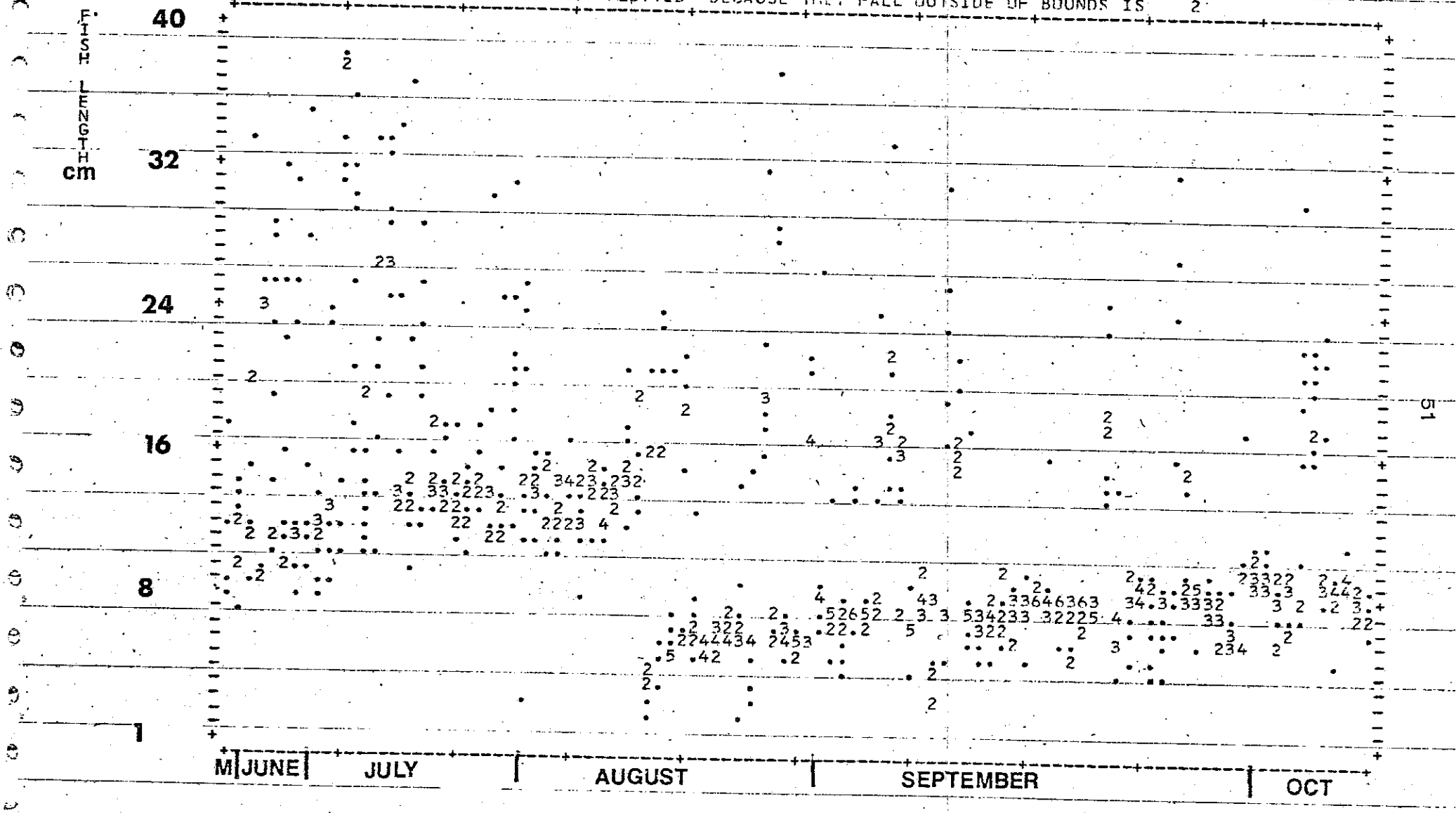


Figure 31 illustrates that "young of the year" sheepshead grow at a much reduced rate during the winter months. Most of the growth in length is suspended during the period October through April. Growth takes place more rapidly again after the waters warm in May.

Since 0+ and 1+ age classes of freshwater-drum have been demonstrated to carry the greatest burden of philometrid nematode infection; combination scatter-diagrams, such as Figure 32, become useful for predictive purposes. They make it possible to predict what length fish will harbor the major burden of adults of Philometra during the next spring and summer.

Frequency Distribution of Adult, Gravid Females

Figure 33: A computer generated scatter-diagram showing frequency distribution of gravid and larvigerous females in different lengths of freshwater-drum hosts in 1972.

Careful inspection of Figure 33 shows that the majority of fish infected with adult females were collected in 1972 from fish measuring 9 to 14 cm in length, 0+ age class. There is a second, less frequently and less intensely infected group of fish, measuring from 16 to 20 cm, 1+ and 2+ age class freshwater-drum and a few scattered infections occur in larger and older fish.

Figure 34: A computer generated scatter-diagram showing the frequency distribution of gravid and larvigerous females in different lengths of freshwater-drum hosts in 1973.

Inspection of Figure 34 shows that the same phenomena occurred in 1973. The size and age class distribution of freshwater-drum hosts infected is almost identical with those in 1972. The only difference indicated is that in 1973 a few 0+ class fish had a total of as many as 20 adult worms in both eyes.

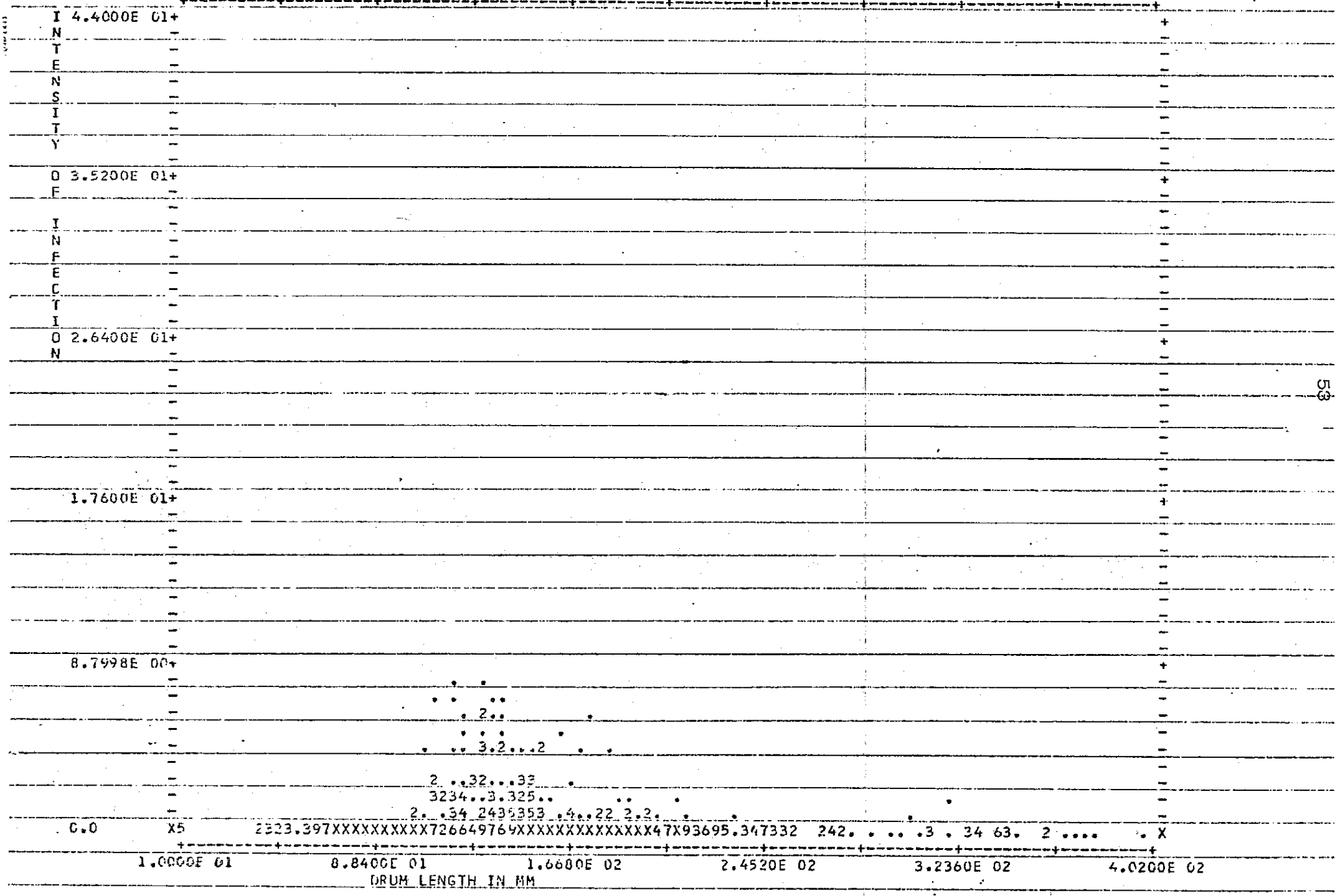
Figure 35: A computer generated scatter-diagram showing the frequency distributions of gravid and larvigerous females for the years 1972 and 1973 combined plotted against length of freshwater-drum hosts.

Figure 36: A computer generated scatter-diagram showing the frequency distributions of gravid and larvigerous females for the years 1972 and 1973 combined plotted against length of freshwater-drum hosts. Figure 36 is identical to Figure 35 except

OMNITAB PHILOMETRA FREQUENCY DISTRIBUTION
SUMMER 1972 FOR ADULT FEMALES

Figure 33

ABS- COLUMN 1; ORD- COLUMN 2 (.).
TOTAL NO. OF PTS. PLOTTED IS 796 AND NO. NOT PLOTTED BECAUSE THEY FALL OUTSIDE OF BOUNDS IS 3

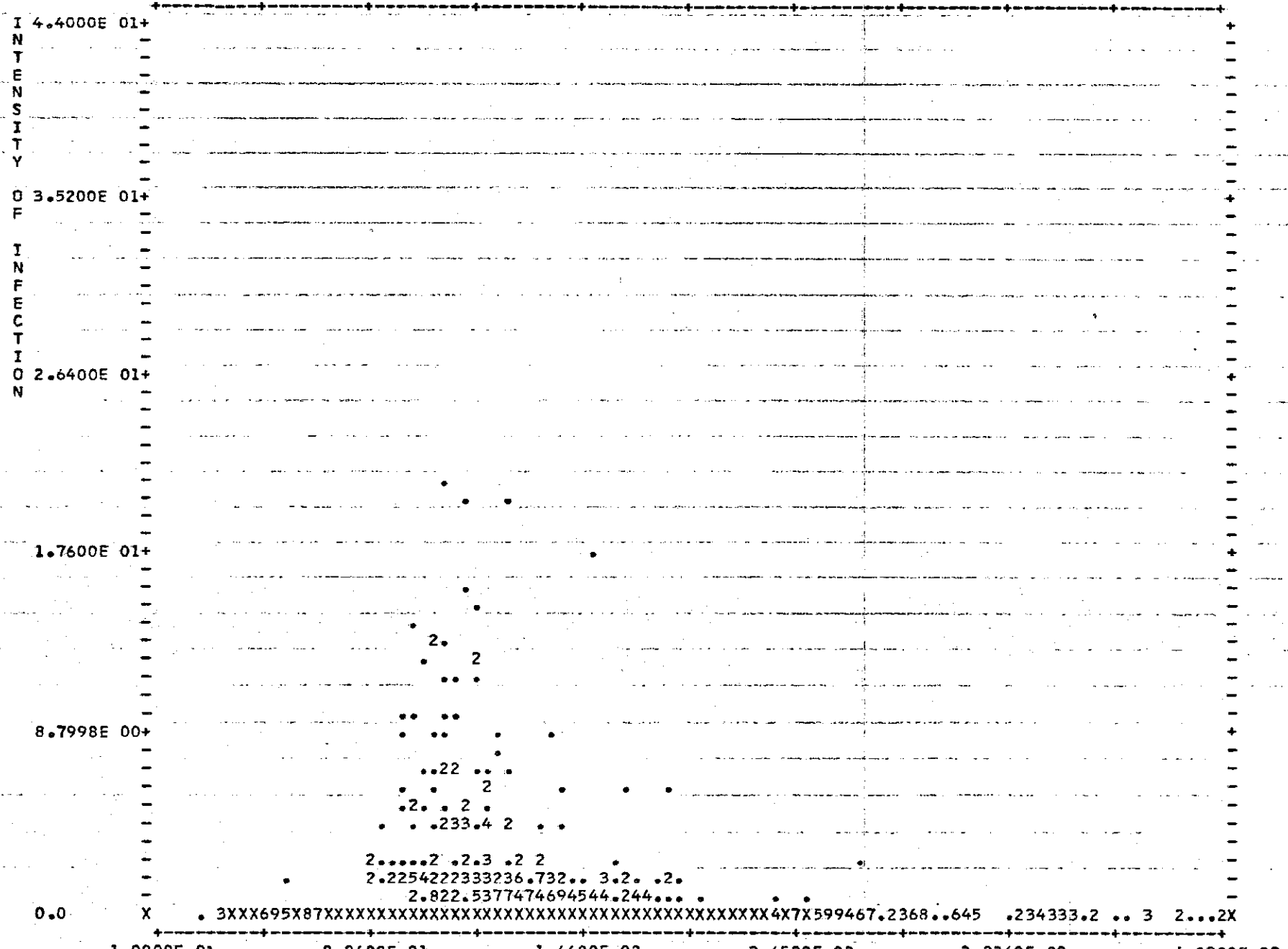


OMNITAB PHILOMETRA STUDY

ADULTS - Gravid and Larvigerous

Figure 34

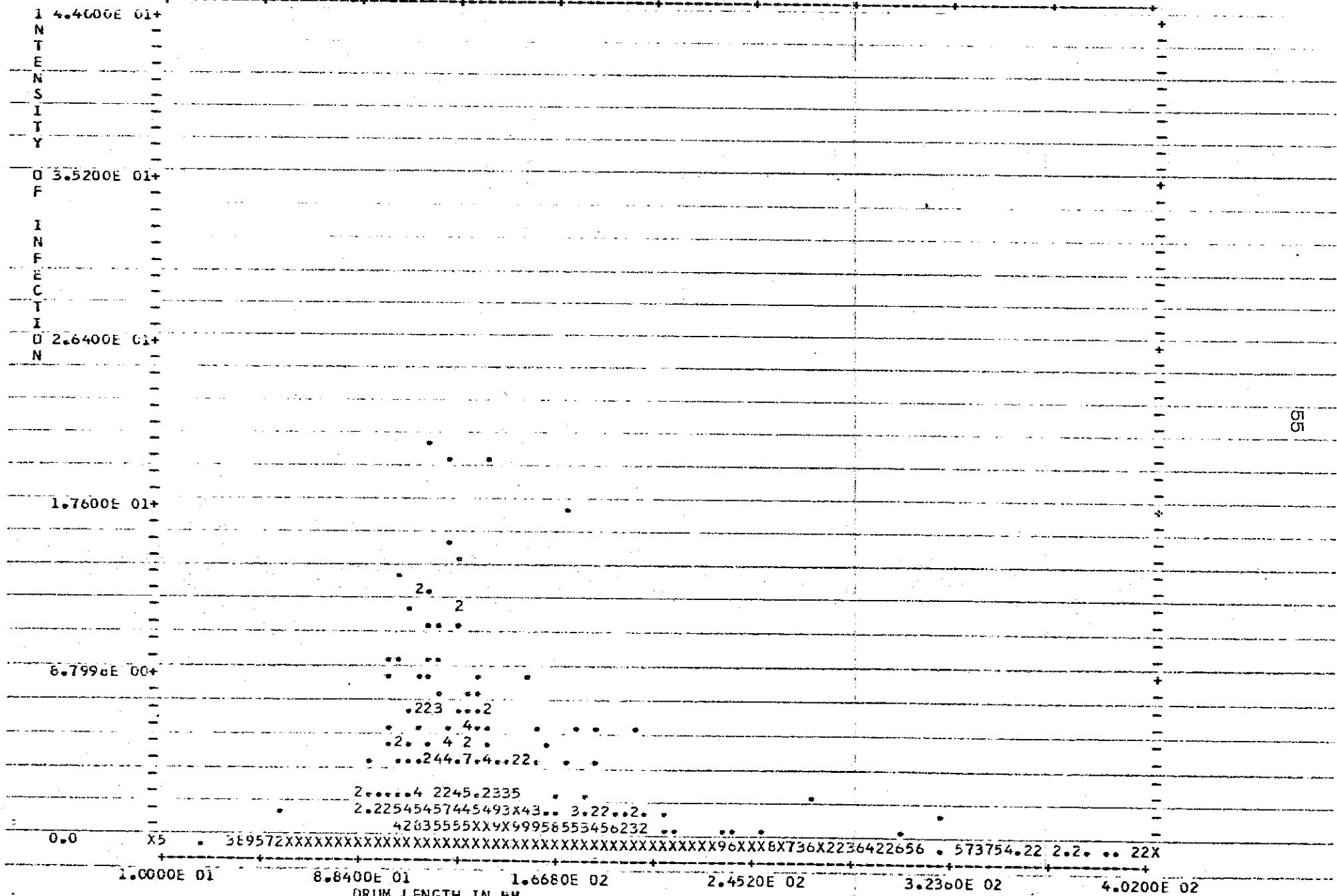
ABS- COLUMN 1; ORD- COLUMN 2 (.),
TOTAL NO. OF PTS. PLOTTED IS 1889 AND NO. NOT PLOTTED BECAUSE THEY FALL OUTSIDE OF BOUNDS IS 19

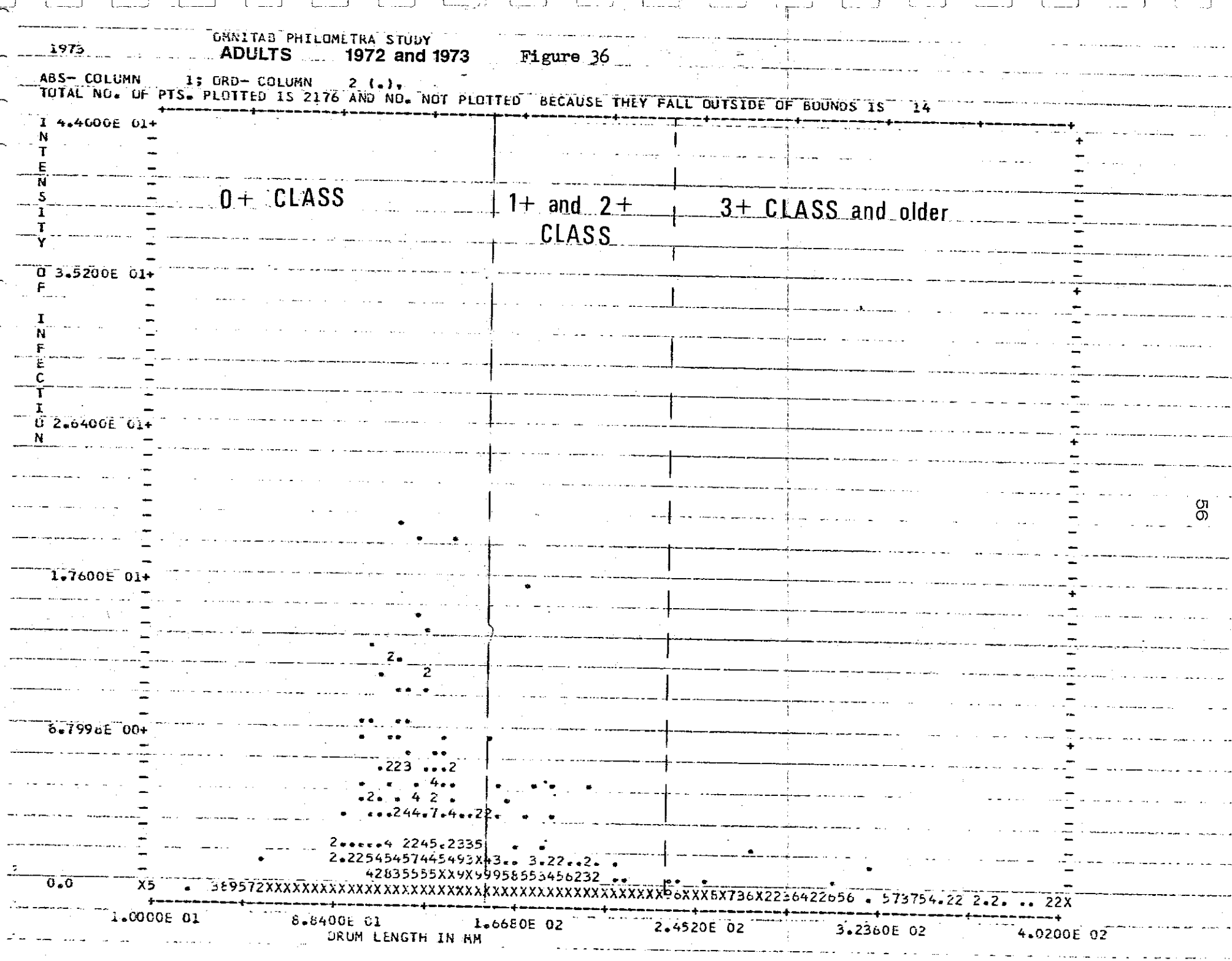


OMNITAB PHILOMETRA STUDY
ADULTS 1972 and 1973

Figure 35

ABS- COLUMN 1; ORD- COLUMN 2 (.),
TOTAL NO. OF PTS. PLOTTED IS 2176 AND NO. NOT PLOTTED BECAUSE THEY FALL OUTSIDE OF BOUNDS IS 14




1975
ORNITAB PHILOMETRA STUDY
ADULTS 1972 and 1973
Figure 36
ABS- COLUMN 1; ORD- COLUMN 2 (.),
TOTAL NO. OF PTS. PLOTTED IS 2176 AND NO. NOT PLOTTED BECAUSE THEY FALL OUTSIDE OF BOUNDS IS 14
INTENSITY OF INFECTION
4.4000E 01+
3.5200E 01+
2.6400E 01+
1.7600E 01+
8.7996E 00+
0.0
0+ CLASS
1+ and 2+ CLASS
3+ CLASS and older
1.0000E 01
8.8400E 01
1.6680E 02
2.4520E 02
3.2360E 02
4.0200E 02
DRUM LENGTH IN MM

that the age-class distribution of freshwater-drum hosts is indicated as well as the lengths.

The last two Figures (35 and 36) show the total of all females collected in both years, 1972 and 1973, in relation to the length of freshwater-drum from which they were removed. It is obvious that the majority of adults were removed from fish up to 20 cm in length. In Fig. 36 the vertical lines separate the age classes of freshwater-drum based on the age-length data of Tubb, Hair and Herdendorf. It demonstrates clearly that the heaviest infections occur in freshwater-drum ranging in length from 9 to 16 cm, 0+ class fish.

Figure 37: A computer generated scatter-diagram showing the frequency distribution of gravid and larvigerous females in 1974. Again, the majority of infection occurred in fishes 9 to 16 cm in length.

Graphs 33, 34, 35, 36 and 37 again confirm our earlier reports that "young of the year" sheepshead are most heavily infected with adult worms having fed on infected copepods the previous fall. This data also fits with a study of the size ranges of freshwater-drum feeding on copepods conducted by Price (J.W. Price 1963.) The slight incidence of worms in larger and older fish may be due to accidental ingestion of copepods with other foods.

Figure 38: A computer generated scatter-diagram showing the frequency distribution of gravid and larvigerous females plotted against time, June through October 1972.

Inspection of this scatter-diagram shows that the majority of freshwater-drum were collected during June and July of 1972, only four adult females were collected during August 1972 and our research notes reveal that these were spent females. This data correlates with our evidence showing that the peak of females streaming from the eyes of infected drum occurred on 26 July 1972.

Figure 39: A computer generated scatter-diagram showing the frequency distribution of gravid and larvigerous females plotted against time, May through October 1973.

Inspection of this figure reveals some differences in 1973 but in general it correlates very closely with Figure 38. In 1973 most of the adult worms were again present in the eyes of drum in June and July. Careful inspection shows that the intensity of

Figure 37

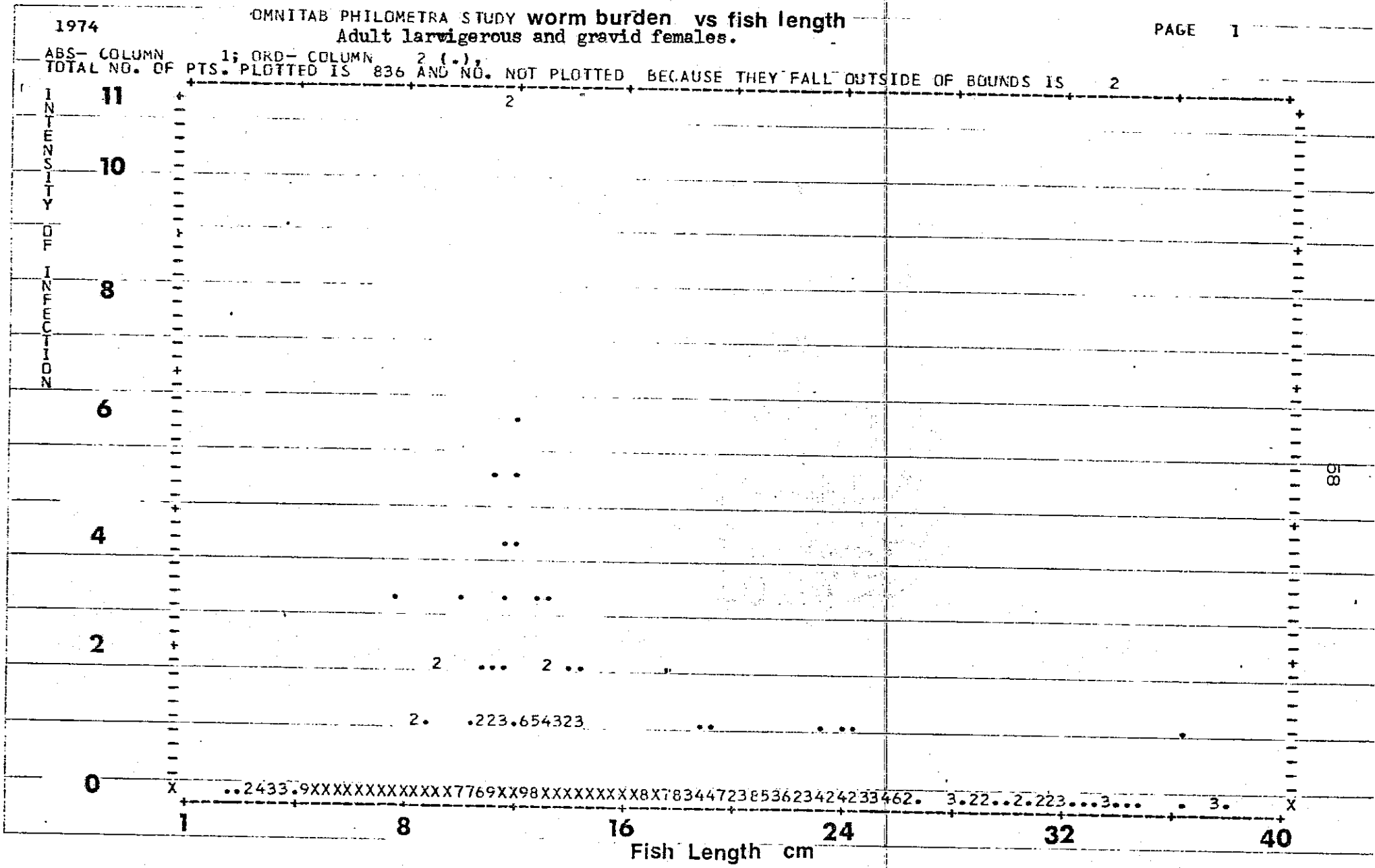

1974
OMNITAB PHILOMETRA STUDY worm burden vs fish length
Adult larwigerous and gravid females.
PAGE 1
ABS- COLUMN 1; ORD- COLUMN 2 (.),
TOTAL NO. OF PTS. PLOTTED IS 836 AND NO. NOT PLOTTED BECAUSE THEY FALL OUTSIDE OF BOUNDS IS 2
INTENSITY OF INFECTION
11
10
8
6
4
2
0
1
8
16
24
32
40
Fish Length cm

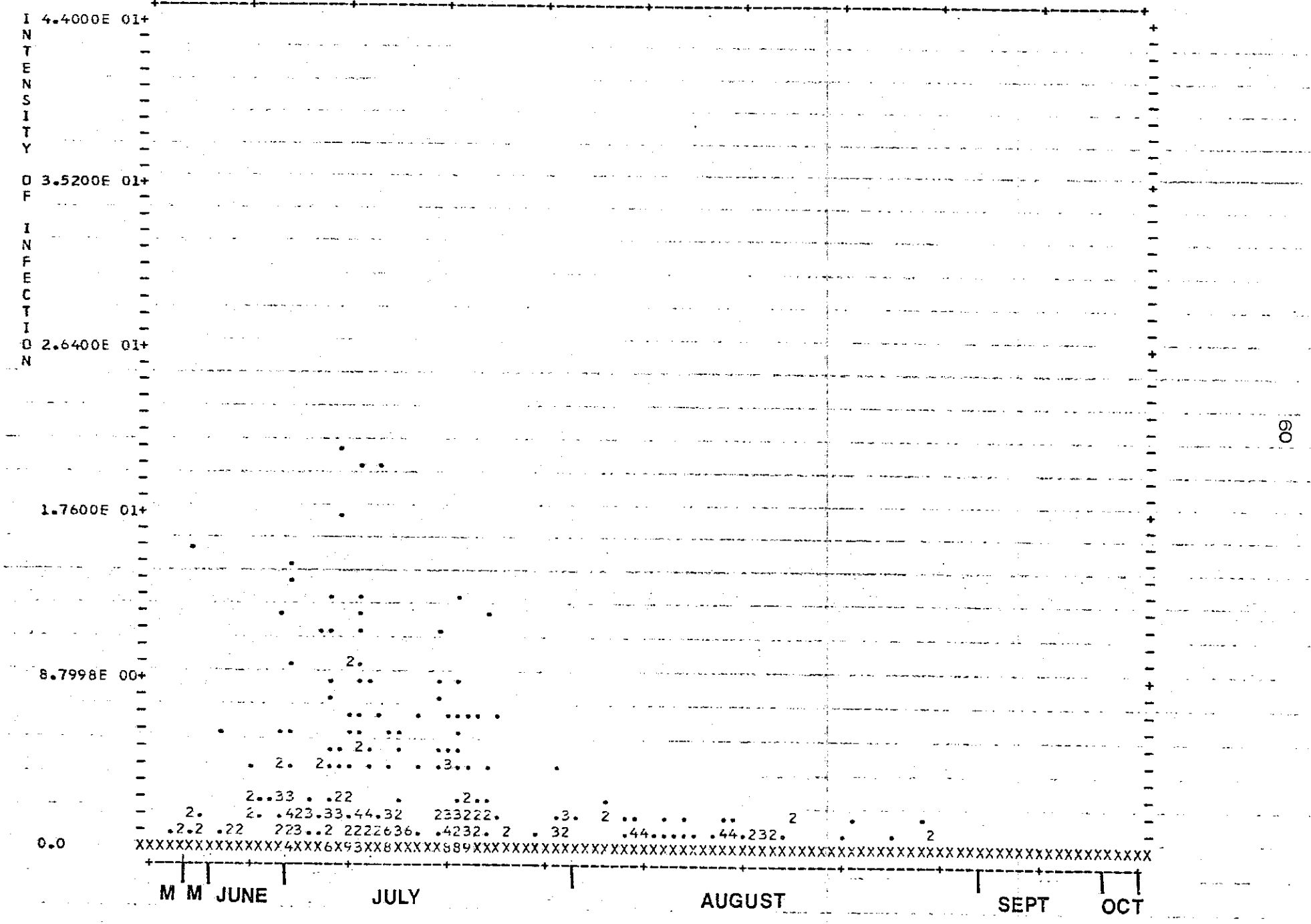
Figure 39

OMNITAB PHILOMETRA STUDY

1973 **ADULTS** (Gravid and Larvigerous)

PAGE 1

ABS- COLUMN 1; ORD- COLUMN 2 (.),
TOTAL NO. OF PTS. PLOTTED IS 1921 AND NO. NOT PLOTTED BECAUSE THEY FALL OUTSIDE OF BOUNDS IS 0



infection (worms per fish) may have been higher in 1973 but the sample size was also greater. Timewise fewer worms were recovered during June 1973 but more were taken in July 1973 and adult philometrids were more prevalent in August 1973. Streaming of adult female worms reached its peak from 24 July to 26 July 1973. The slight variations noted here and the extension of the females into August 1973 were noted earlier in the report and it is believed that they were caused by the warmer temperatures of 1973 versus 1972.

Figure 40: A computer generated scatter-diagram illustrating the distribution of gravid and larvigerous females, May through October 1974. This diagram indicates that the distribution was very similar to that of 1972. No larvigerous females were collected after 15 August.

In summary, these data and analyses help to answer concisely two questions. One, what lengths and age classes of freshwater-drum are most heavily infected with adult, post-fertilization and gravid females philometrids? Two, when are these adult female worms most prevalent in the eyes of freshwater-drum in Lake Erie? These Graphs illustrate and confirm our consistent reports that the greatest number adult, gravid and larvigerous female worms of Philometra sp. occur in the eyes of 0+ class Aplodinotus grunniens and that they are present in 1+ and 2+ class freshwater-drum. The adult females, having overwintered in the eyes, are present in them at the beginning of the collecting season, March, and they are present through July and decline rapidly in numbers during August. This fits well with the increase of "popeye" syndrome in 0+, 1+ and 2+ class fish during June and July. After the peak of streaming from the eyes in late July only spent females are found in the eyes during much of August and this marks the end of one annual generation of Philometra sp. in sheepshead in Lake Erie. The portion of the adult female which remains in the eye becomes encapsulated with connective tissue.

Frequency Distributions of Encapsulations

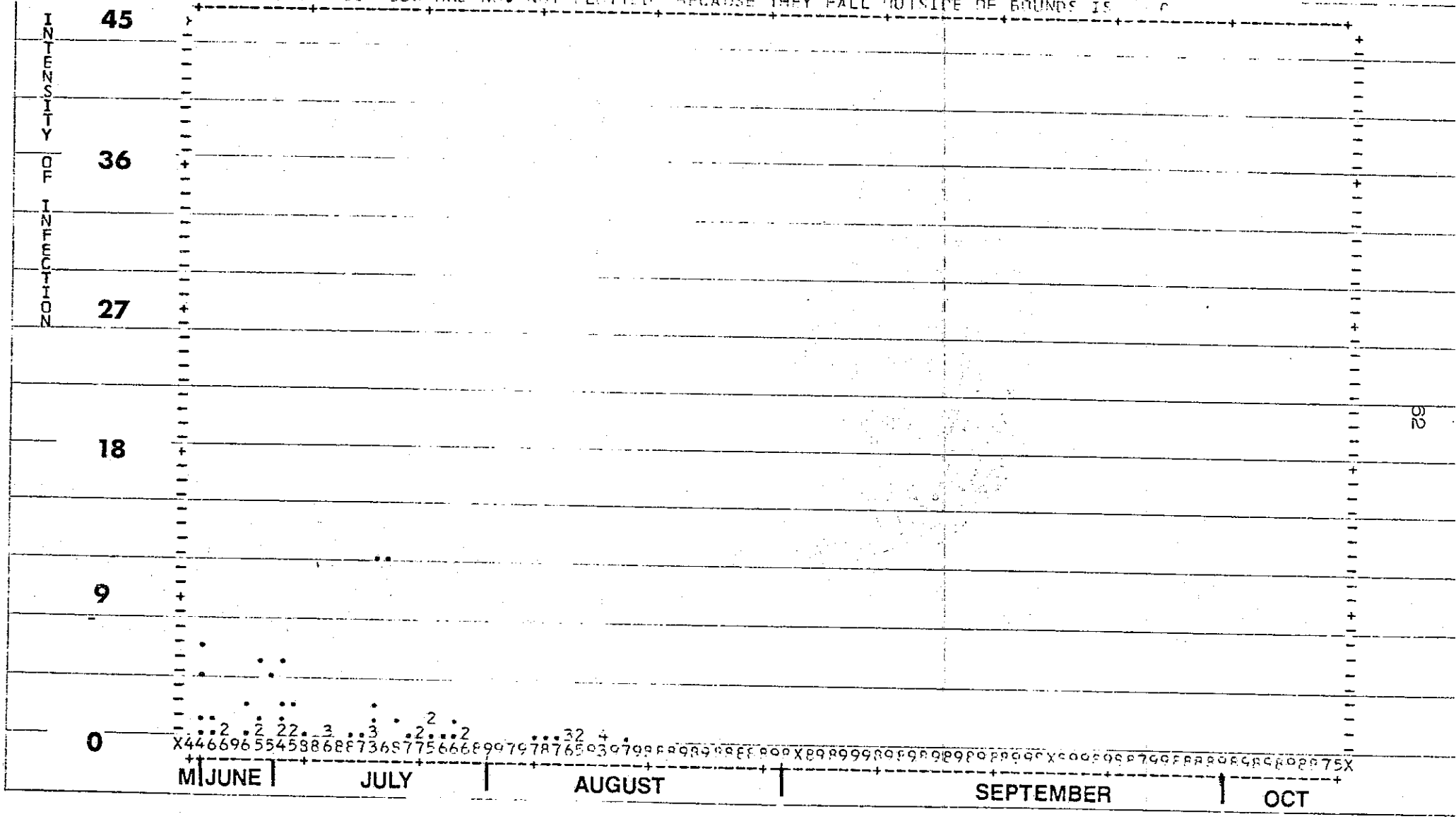
Figure 41: A computer generated scatter-diagram showing the frequency distribution of encapsulations in the eyes of different lengths of freshwater-drum from Lake Erie collected June through October 1972.

Figure 42: A computer generated scatter-diagram showing the frequency distribution of encapsulations in the eyes of different lengths of freshwater-drum from Lake Erie collected March through October 1973.

Figure 40

1974 OMNITAB PHILOMETRA STUDY **worm burden vs time**
Adult Worms, gravid and larvigerous.

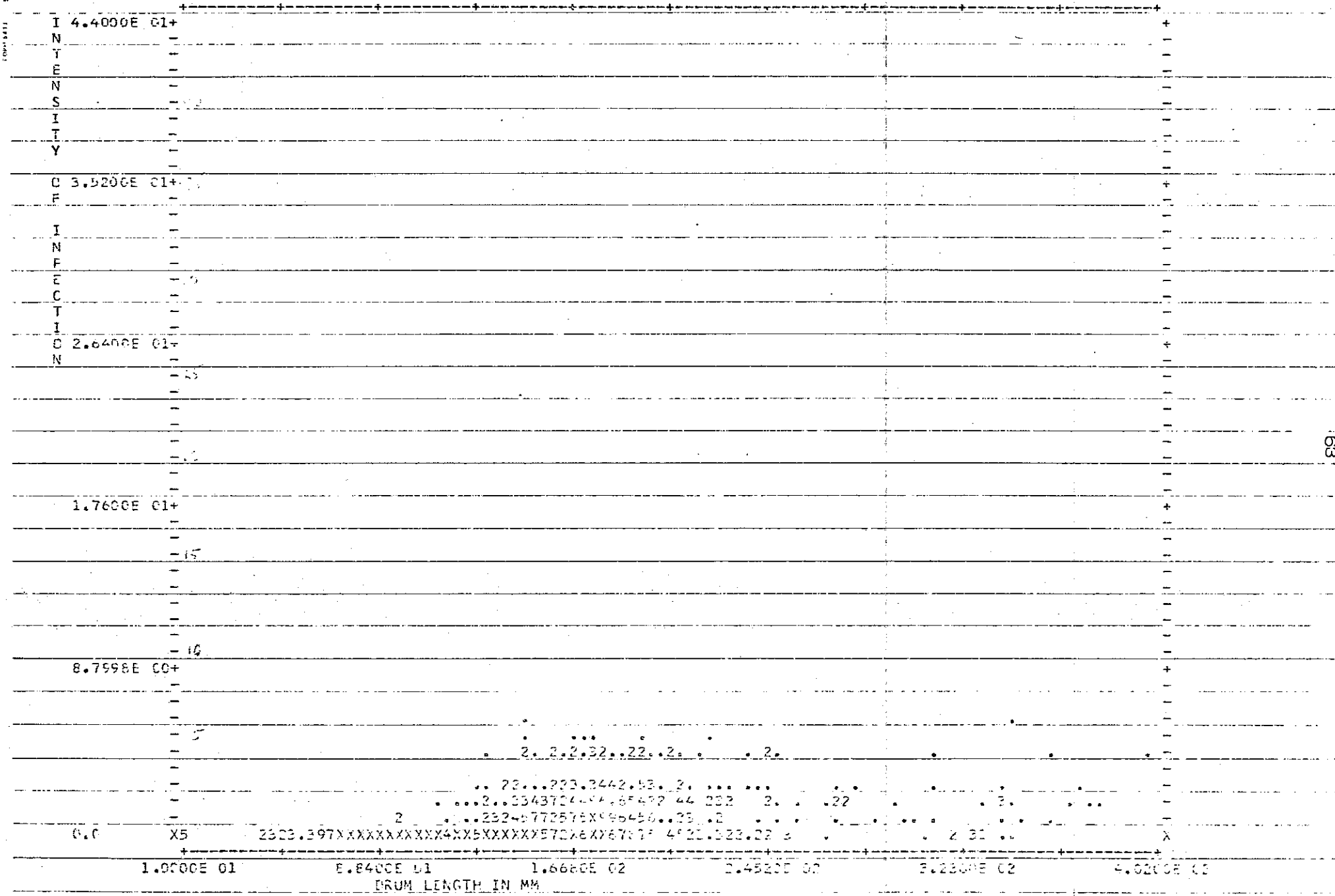
ABS- COLUMN 1; ORD- COLUMN 2 (.).
TOTAL NO. OF PTS. PLOTTED IS 838 AND NO. NOT PLOTTED BECAUSE THEY FALL OUTSIDE OF BOUNDS IS 0



SUMMER 1972 — SPHITAB PHILOMETRA FREQUENCY DISTRIBUTION FOR ENCAPSULATIONS

Figure 41

ABS- COLUMN 1; ORD- COLUMN 4 (.).
TOTAL NO. OF PTS. PLOTTED IS 796 AND NO. NOT PLOTTED BECAUSE THEY FALL OUTSIDE OF BOUNDS IS 3



ENCAPSULATIONS Figure 42

ABS- COLUMN 1; ORD- COLUMN 2 (.),
TOTAL NO. OF PTS. PLOTTED IS 1887 AND NO. NOT PLOTTED BECAUSE THEY FALL OUTSIDE OF BOUNDS IS 14

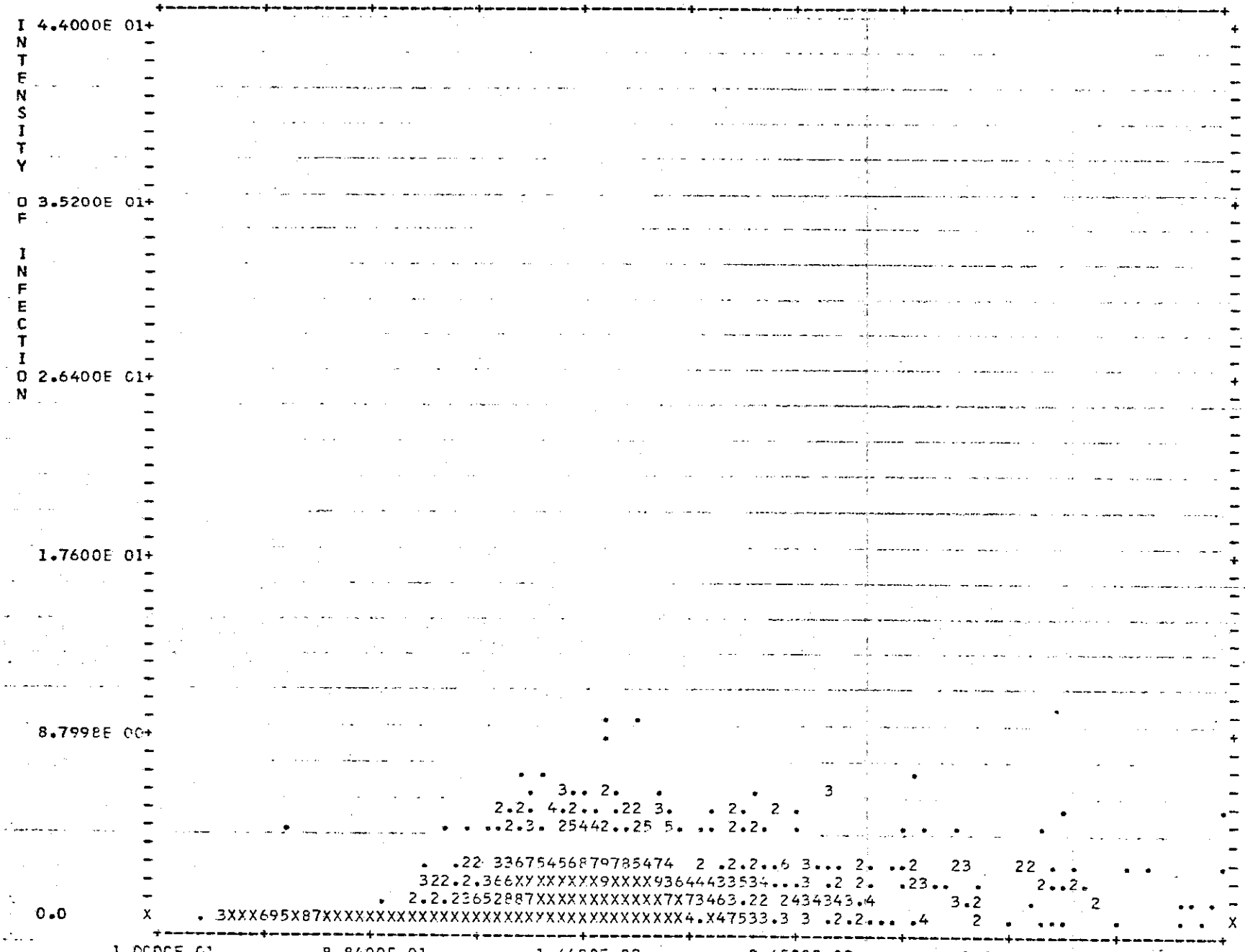


Figure 43: A computer generated scatter-diagram illustrating the combined frequency distributions for encapsulations collected from the eyes of freshwater-drum from Lake Erie during 1972 and 1973.

Figure 44: A computer generated scatter-diagram illustrating the frequency distribution of encapsulations in the eyes of different lengths of freshwater-drum collected from Lake Erie in 1974.

Inspection and comparisons of Figures 41, 42, 43, and 44 show no significant differences in the years 1972, 1973, and 1974. Encapsulations of the portion of the adult female worms occur in all sizes and age classes of freshwater-drum except "young of the year." Small drum 3 to 10 cm in length had no encapsulations as can be seen on all three of these graphs. Encapsulations begin after mid July in freshwater-drum 10 to 14 cm in length, are maintained at high levels in fish through 28 or 29 cm in length, but are found in all length and age classes of fish. Small drum 10 to 14 cm are the 0+ age class fish infected the previous autumn and they contain living adult females as shown on previous graphs. They do not contain large encapsulations (portions of large adult females which have streamed) until after July. Fish of lengths above 10 cm and age classes 1+ or older exhibit encapsulations indicating previous infections.

Figure 45: A computer generated scatter-diagram showing the frequency distribution of encapsulations in the eyes of freshwater-drum from Lake Erie plotted against time, June through October 1972.

Figure 46: A computer generated scatter-diagram showing the frequency distribution of encapsulations in the eyes of freshwater-drum from Lake Erie plotted against time, March through October 1973.

Figure 47: A computer generated scatter-diagram showing the frequency distribution of encapsulations in the eyes of freshwater-drum plotted against time, March through October 1974.

A comparison of Figures 45, 46, and 47 shows that they exhibit an identical pattern. The larger number of points plotted for 1973, Figure 46, is due to the larger sample size in 1973. These two graphs illustrate that encapsulations occur in the eyes of freshwater-drum in Lake Erie at all times of the year. This can be explained as reported earlier, the encapsulations occur only in previously infected fish and are composed of tough connective tissue which may persist in the eyes for years.

OMNITAB PHILOMETRA STUDY

ENCAPSULATIONS 1972 AND 1973

Figure 43

ABS- COLUMN 1; ORD- COLUMN 2 (.),
TOTAL NO. OF PTS. PLOTTED IS 2176 AND NO. NOT PLOTTED BECAUSE THEY FALL OUTSIDE OF BOUNDS IS 14

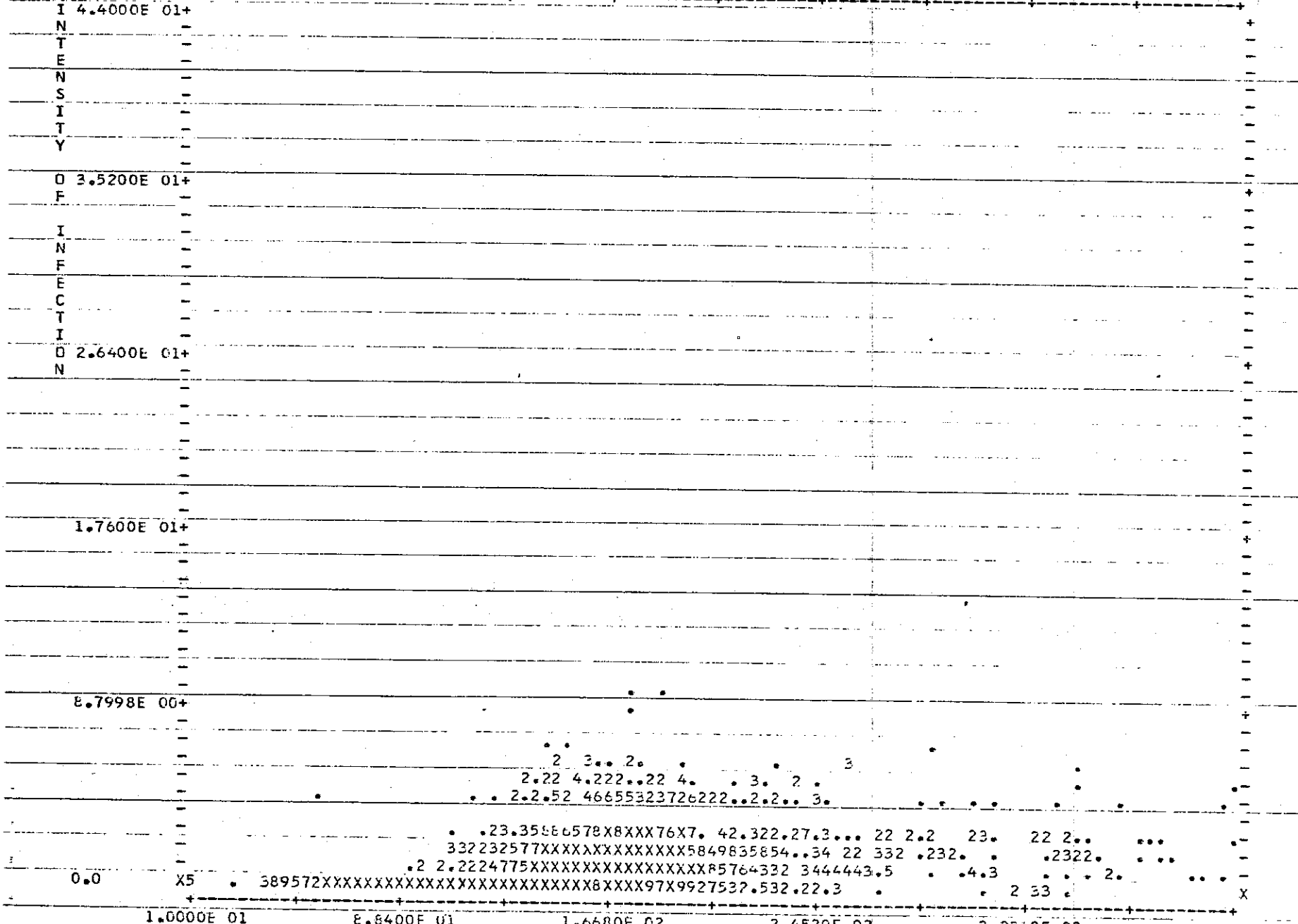
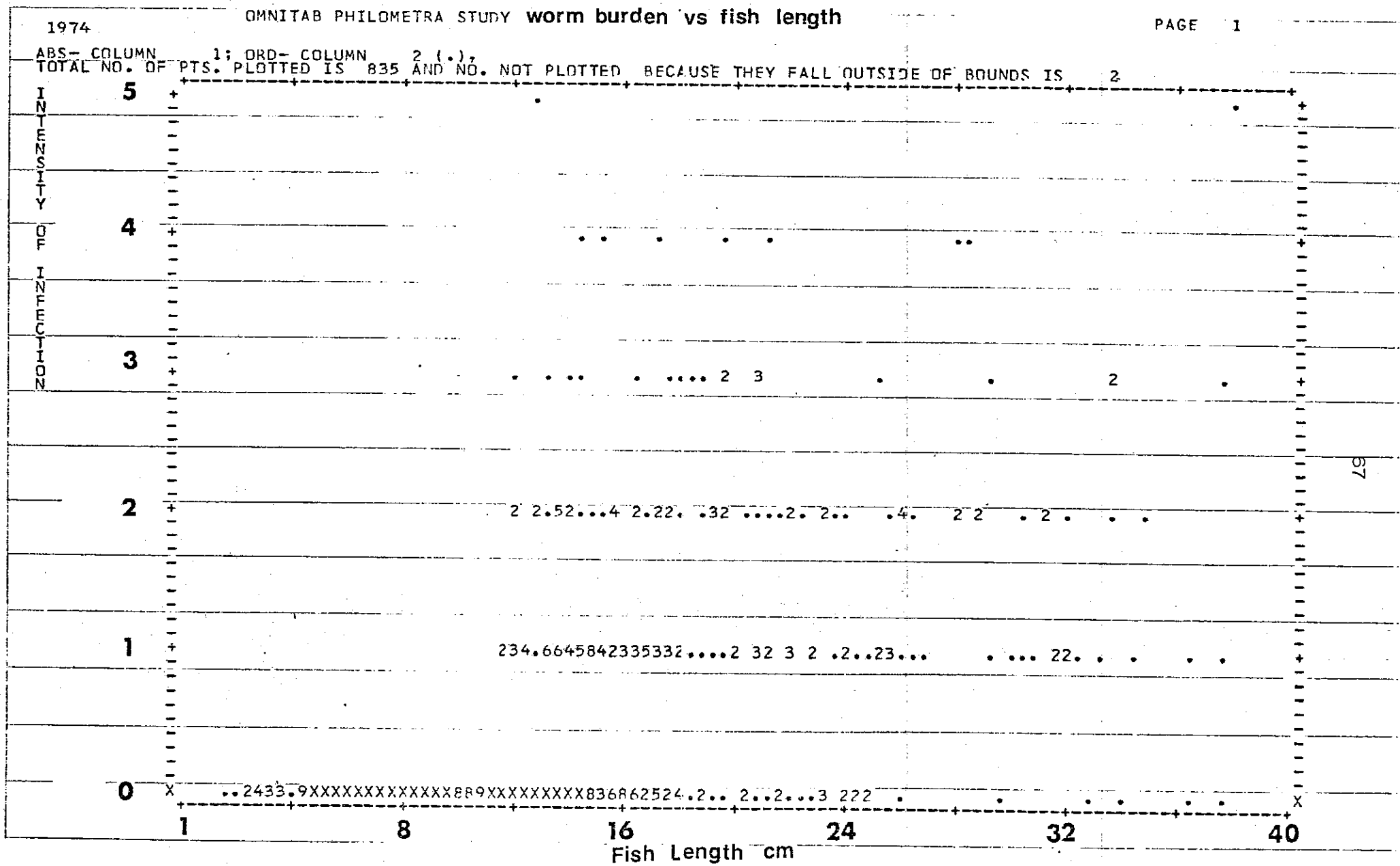
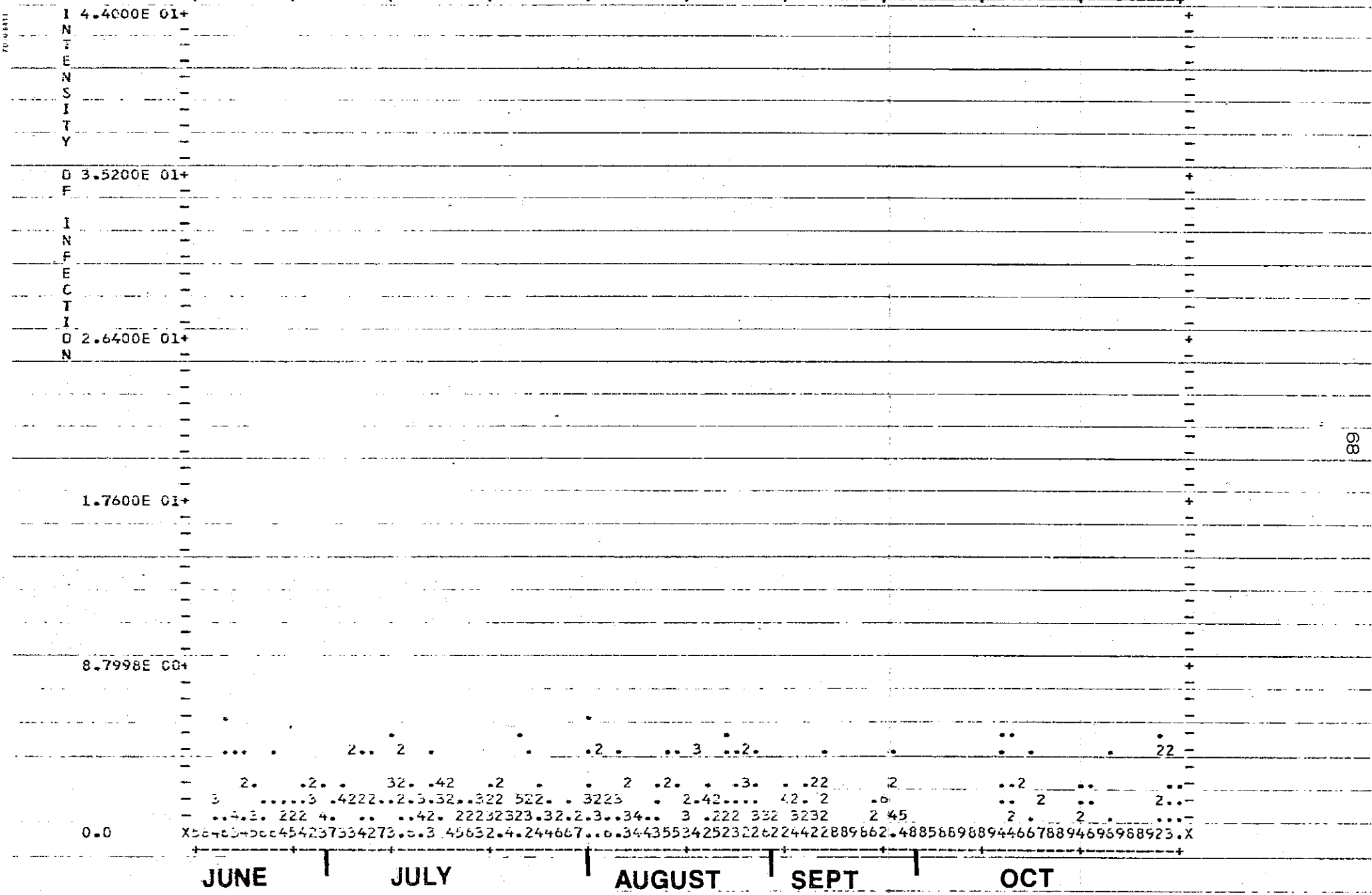


Figure 44


1974
OMNITAB PHILOMETRA STUDY worm burden vs fish length
PAGE 1
ABS- COLUMN 1; ORD- COLUMN 2 (.),
TOTAL NO. OF PTS. PLOTTED IS 835 AND NO. NOT PLOTTED BECAUSE THEY FALL OUTSIDE OF BOUNDS IS 2
INTENSITY OF INFECTION
5
4
3
2
1
0
1
8
16
24
32
40
Fish Length cm

1972 ORNITAB PHILOMETRA FREQUENCY DISTRIBUTION FOR ENCAPSULATIONS Figure 45

ABS- COLUMN 1; ORD- COLUMN 4 (.),
TOTAL NO. OF PTS. PLOTTED IS 799 AND NO. NOT PLOTTED BECAUSE THEY FALL OUTSIDE OF BOUNDS IS 0



1973 OMNITAB PHILOMETRA STUDY
ENCAPSULATIONS

Figure 46

PAGE 1

ABS- COLUMN 1; ORD- COLUMN 2 (.),
TOTAL NO. OF PTS. PLOTTED IS 1921 AND NO. NOT PLOTTED BECAUSE THEY FALL OUTSIDE OF BOUNDS IS 0

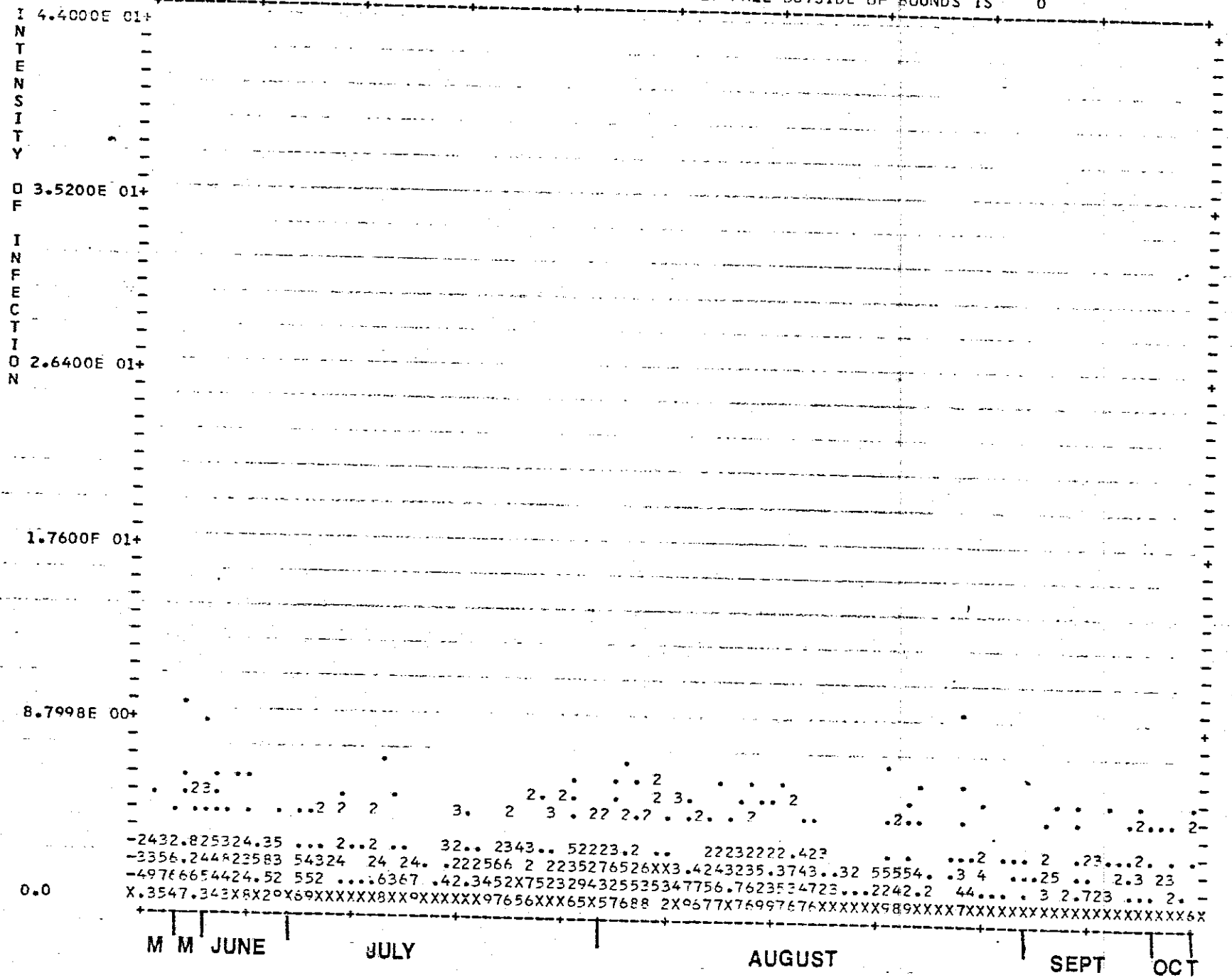
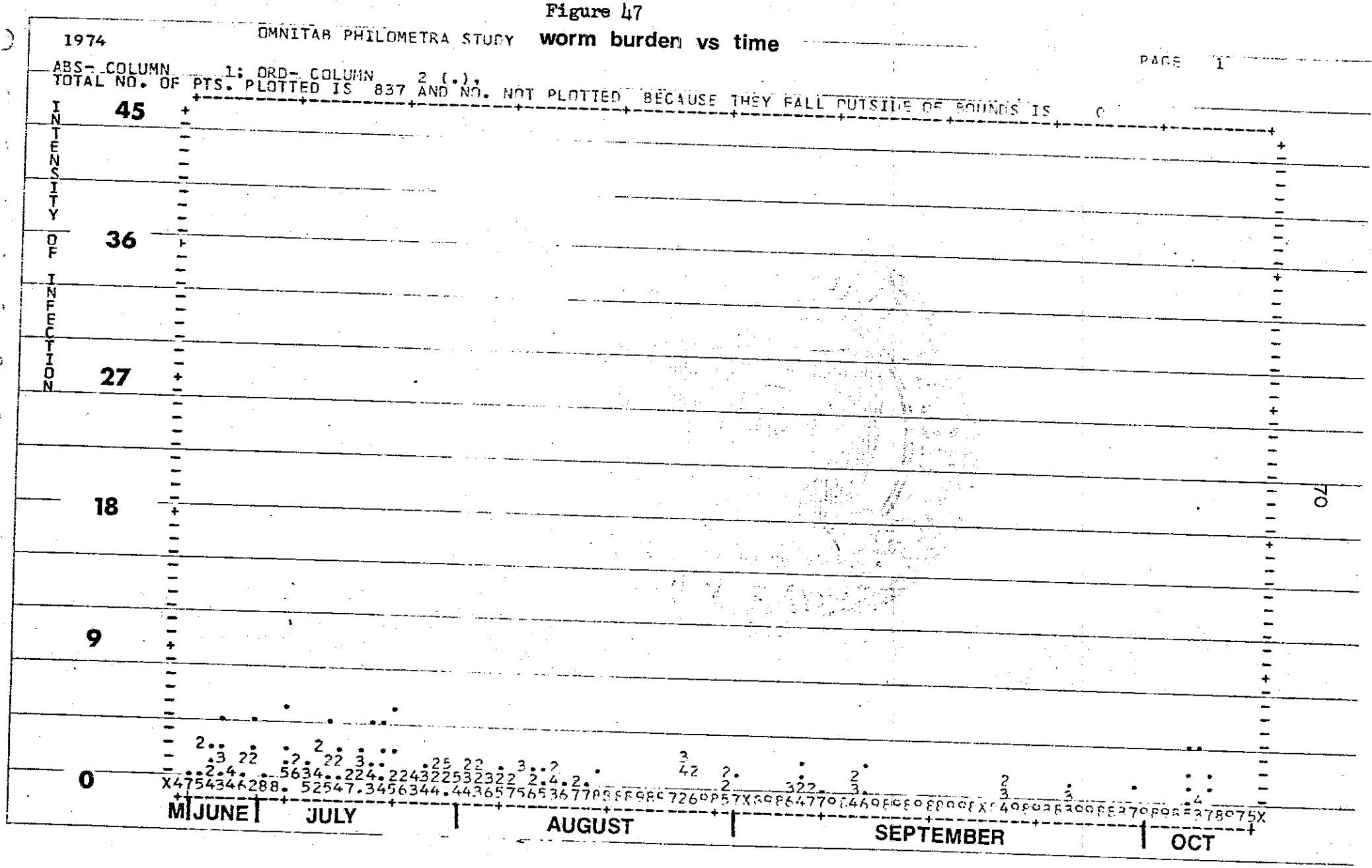


Figure 47

worm burden vs time



Frequency Distribution of Juvenile and Young Adult Worms

Figure 48: A computer generated scatter-diagram showing the frequency distribution of juvenile and young adult worms removed from the eyes of different lengths of freshwater-drum hosts from Lake Erie in 1972. Distribution of 0+ age class and older fish indicated.

Figure 49: A computer generated scatter-diagram showing the frequency distribution of juvenile and young adult worms removed from the eyes of different lengths of freshwater-drum hosts from Lake Erie in 1973. Distribution in 0+ class and older fish indicated.

Figure 50: A computer generated scatter-diagram showing the frequency distribution of juvenile and young adult worms removed from the eyes of different lengths of freshwater-drum hosts in Lake Erie. Data from 1972 and 1973 combined. Distribution in 0+ class and older fish indicated.

Figure 51: A computer generated scatter-diagram showing the frequency distribution of juvenile and young adult worms removed from the eyes of different lengths of freshwater-drum hosts in Lake Erie in 1974.

A comparison of Figures 49, 50, and 51 demonstrates a very similar pattern for the frequency distributions of juvenile philometrids in 1972 and 1973 but there are two differences which should be noted.
1. The intensity of infection was greater in 1972 and 1974 than 1973. We believe that this may be related to the period of time when the adult females streamed from the eyes releasing their larvae. These periods were slightly different in the 3 years. Figs. 11 and 12 show that in 1972 there was a more tight fit synchronization of streaming and copepod blooms.
2. In 1972, the vast majority of juveniles occurred in freshwater-drum which ranged in length from 3 to 10 cm, 0+ age class fish, these were "young of the year" which developed over the summer. A small number of juveniles occurred in fish 14 to 18 cm in length and were probably 1+ and possibly 2+ age class fish. In 1973, the majority of juveniles occurred in drum 8 to 14 cm in length and a smaller number occurred in 14 to 18 cm length fish. In 1973 "young of the year" sheepshead entered the Lake Erie population a full month to month and a half earlier than in 1972 and grew to a larger size. This may be correlated with the warmer temperature of 1973. Streaming in 1974 was more similar to 1972.

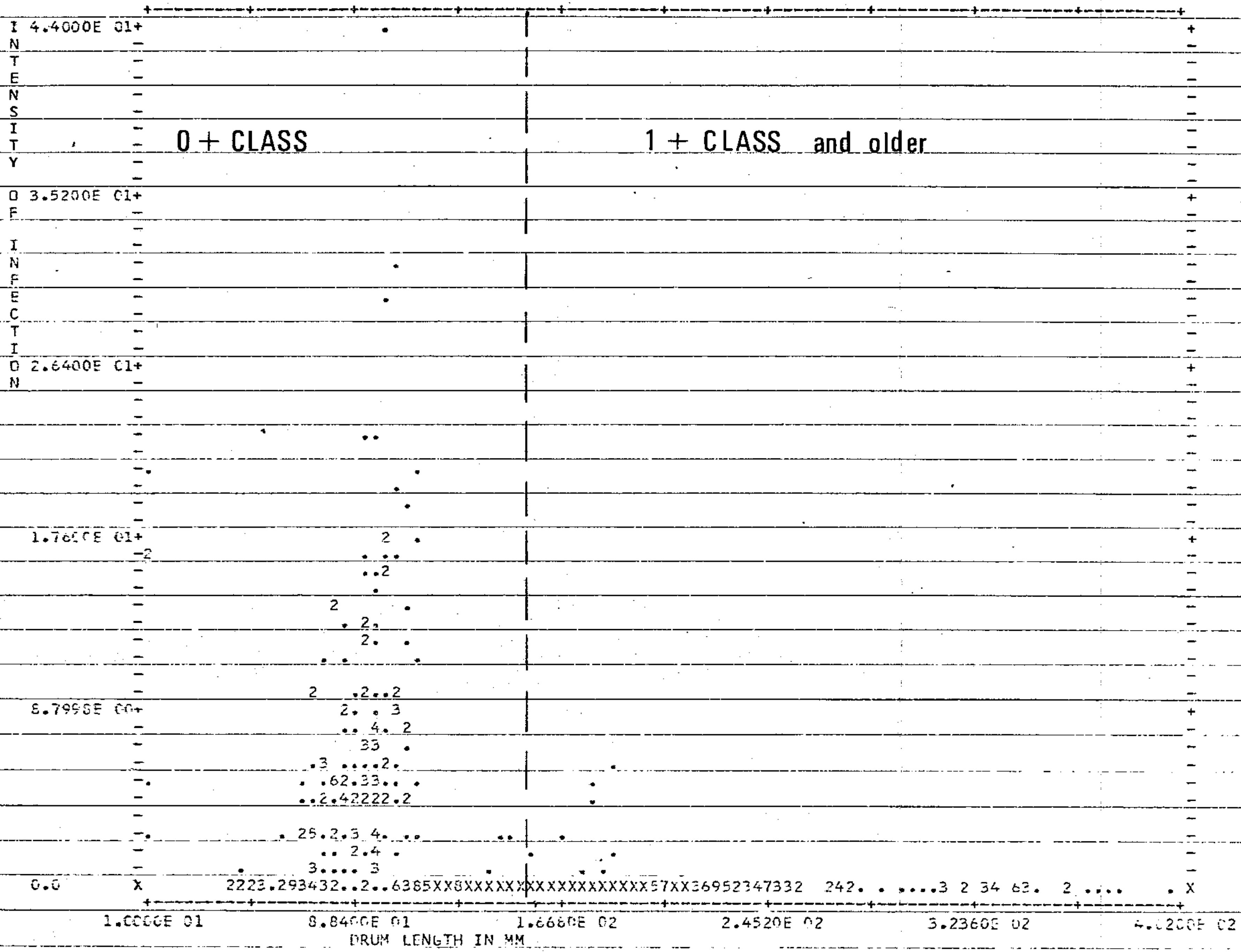
Figure 50 which combines the data concerning juvenile distribution for 1972 and 1973 indicates that a vast majority of

SUMMER 1972 ... TAB ... ONE ... FREQUENCY DISTRIBUTION FOR JUVENILES

Figure 48

ABS- COLUMN 1; ORD- COLUMN 3 (.),

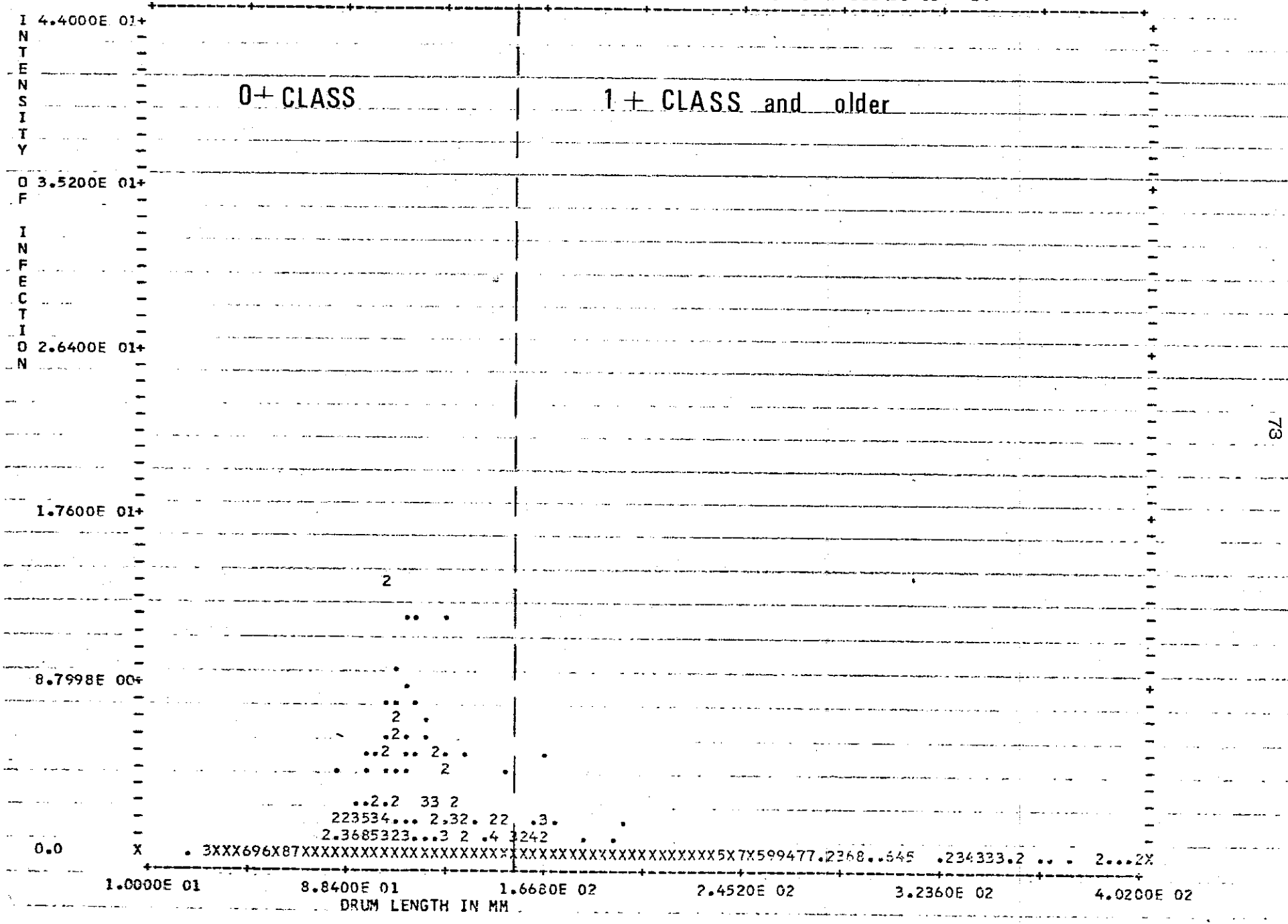
TOTAL NO. OF PTS. PLOTTED IS 796 AND NO. NOT PLOTTED BECAUSE THEY FALL OUTSIDE OF BOUNDS IS 3



1973 OMNITAB PHILOMETRA STUDY
JUVENILES

Figure 49

ABS- COLUMN 1; ORD- COLUMN 2 (.),
TOTAL NO. OF PTS. PLOTTED IS 1887 AND NO. NOT PLOTTED BECAUSE THEY FALL OUTSIDE OF BOUNDS IS 14



OMNITAB PHILOMETRA STUDY

1973 **FOR JUVENILES** 1972 and 1973 Figure 50

ABS- COLUMN 1; ORD- COLUMN 2 (.),
TOTAL NO. OF PTS. PLOTTED IS 2176 AND NO. NOT PLOTTED BECAUSE THEY FALL OUTSIDE OF BOUNDS IS 14

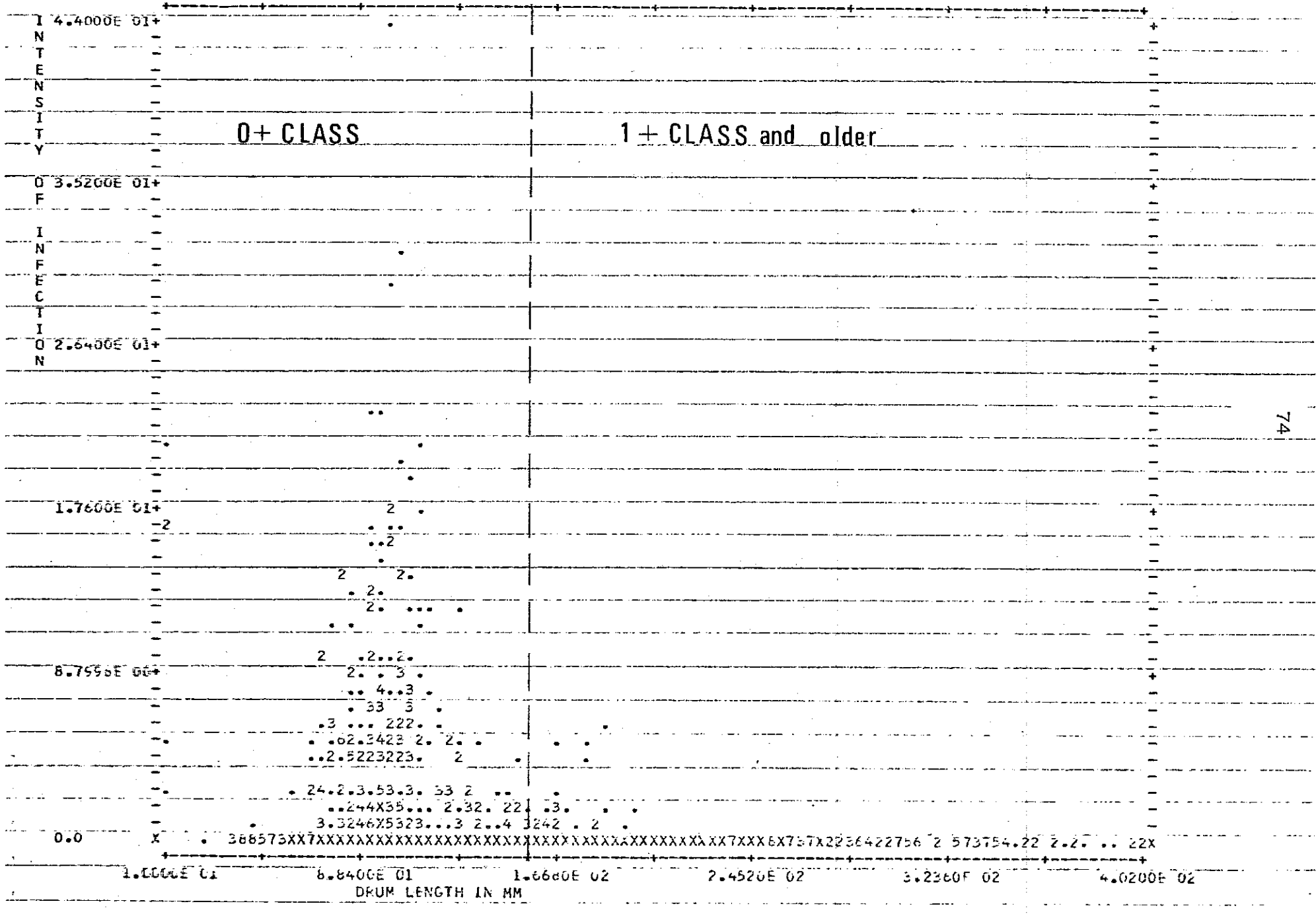
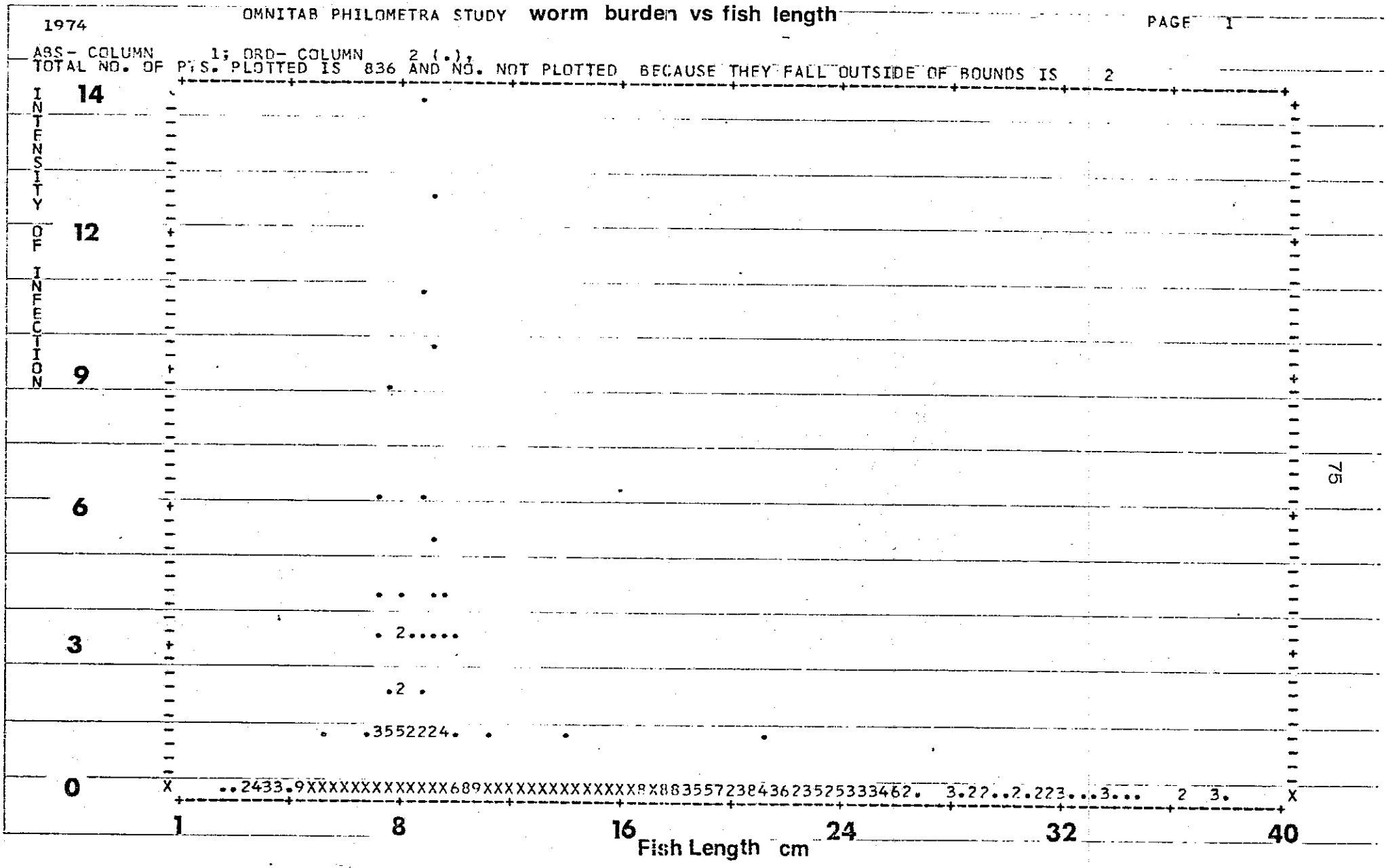


Figure 51


1974 OMNITAB PHILOMETRA STUDY worm burden vs fish length PAGE 1
ABS- COLUMN 1; ORD- COLUMN 2 (.),
TOTAL NO. OF PTS. PLOTTED IS 836 AND NO. NOT PLOTTED BECAUSE THEY FALL OUTSIDE OF BOUNDS IS 2
INTENSITY OF INFECTION
14
12
9
6
3
0
1
8
16
24
32
40
Fish Length cm
75

the freshwater-drum infected, even with temperature differences, range from 3 to 16 cm in length and are 0+ class drum. Price (J.W. Price, 1963) demonstrates that this size freshwater-drum feed heavily upon copepods.

Figure 52: A computer generated scatter-diagram of the frequency distribution of philometrid juveniles and young adults recovered from the eyes of freshwater-drum in Lake Erie in 1972. Intensity of infection (worm burden) is plotted against time at which they were recovered.

Figure 53: A computer generated scatter-diagram of the frequency distribution of philometrid juveniles and young adults recovered from the eyes of freshwater-drum in Lake Erie in 1973. Intensity of infection (worm burden) is plotted against time at which they were recovered.

Figure 54: A computer generated scatter-diagram of the frequency distribution of philometrid juveniles and young adults recovered from the eyes of freshwater-drum in 1974. Intensity of infection is plotted against time at which they were recovered.

A comparison of Figures 52, 53, and 54 shows only two differences worth noting. In 1972, the juveniles and young adults were not present in the eyes until September and they were present in great numbers. In 1973 and 1974, the juveniles and young adults were present in the eyes by 10-15 August, 25 to 20 days earlier than in 1972. In general, the patterns are similar with the occurrence of juveniles and young adults in the late summer and fall months.

Composite Summary Graphs of Frequency Distributions of Various Stages

Figures 55, 56, and 57: These are composite diagrams for the years 1972, 1973, and 1974 respectively. They illustrate the frequencies of adult females, encapsulations, and juveniles and young adult philometrid nematodes removed from the eyes of infected freshwater-drum hosts in each year. Uninfected fishes are not shown. Intensity of infection is plotted against length of fish hosts.

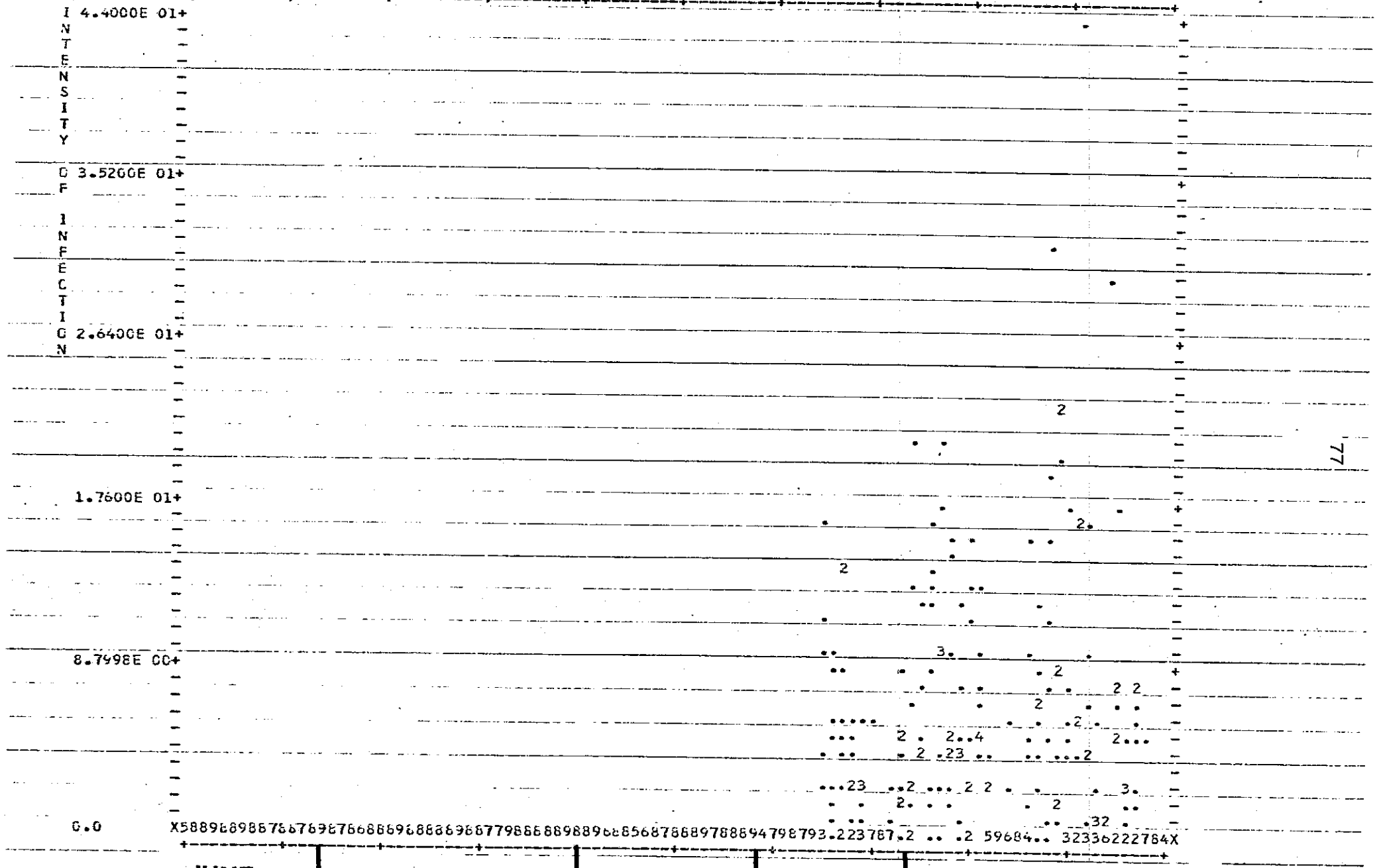
Study of Figure 55 emphasizes the distinctiveness of the three stages of philometrid infection in freshwater-drum from Lake Erie in 1972. Juveniles and young adults occur in fish 3 to 8 cm in length, 0+ age class drum. Adults occur primarily in fish 9 to 16 cm in length, 0+, 1+ and 2+ age classes of drum. Large

1972

OMNITAB PHILOMETRA FREQUENCY DISTRIBUTION
FOR JUVENILES

Figure 52

ABS- COLUMN 1; ORD- COLUMN 3 (.),
TOTAL NO. OF PTS. PLOTTED IS 799 AND NO. NOT PLOTTED BECAUSE THEY FALL OUTSIDE OF BOUNDS IS 0



JUVENILES

Figure 53

ABS- COLUMN 1; ORD- COLUMN 2 (.).
TOTAL NO. OF PTS. PLOTTED IS 1921 AND NO. NOT PLOTTED BECAUSE THEY FALL OUTSIDE OF BOUNDS IS 0

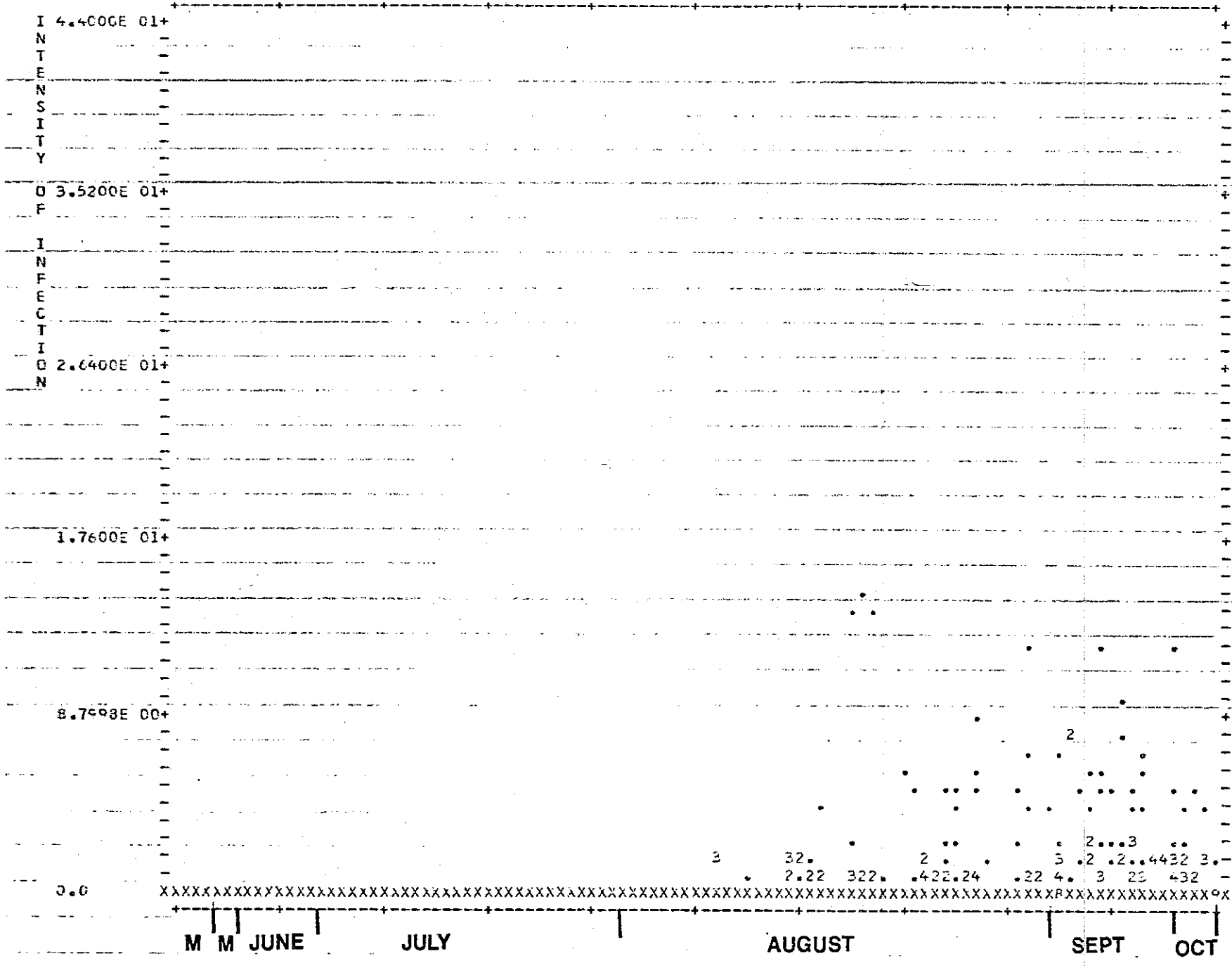


Figure 54

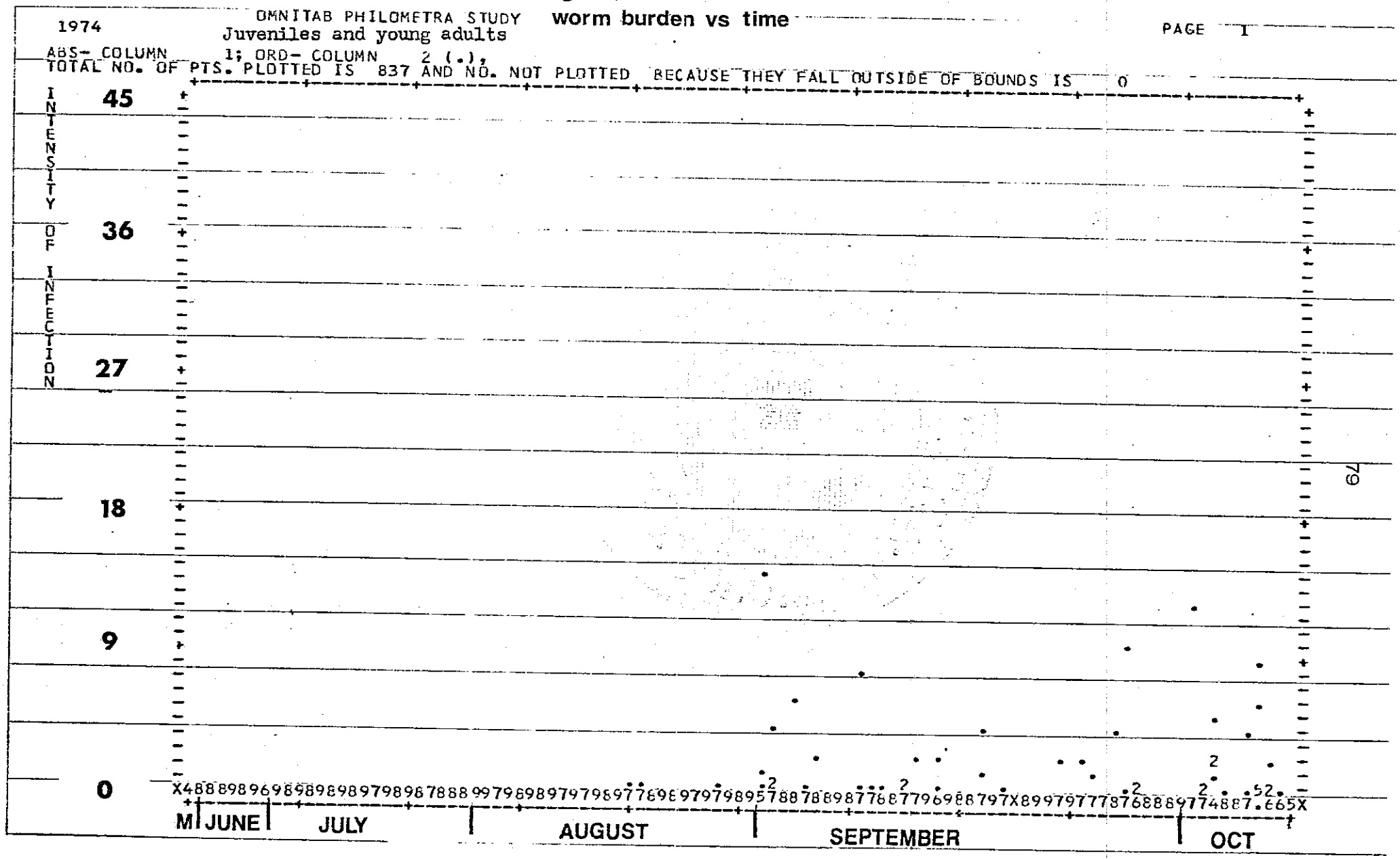
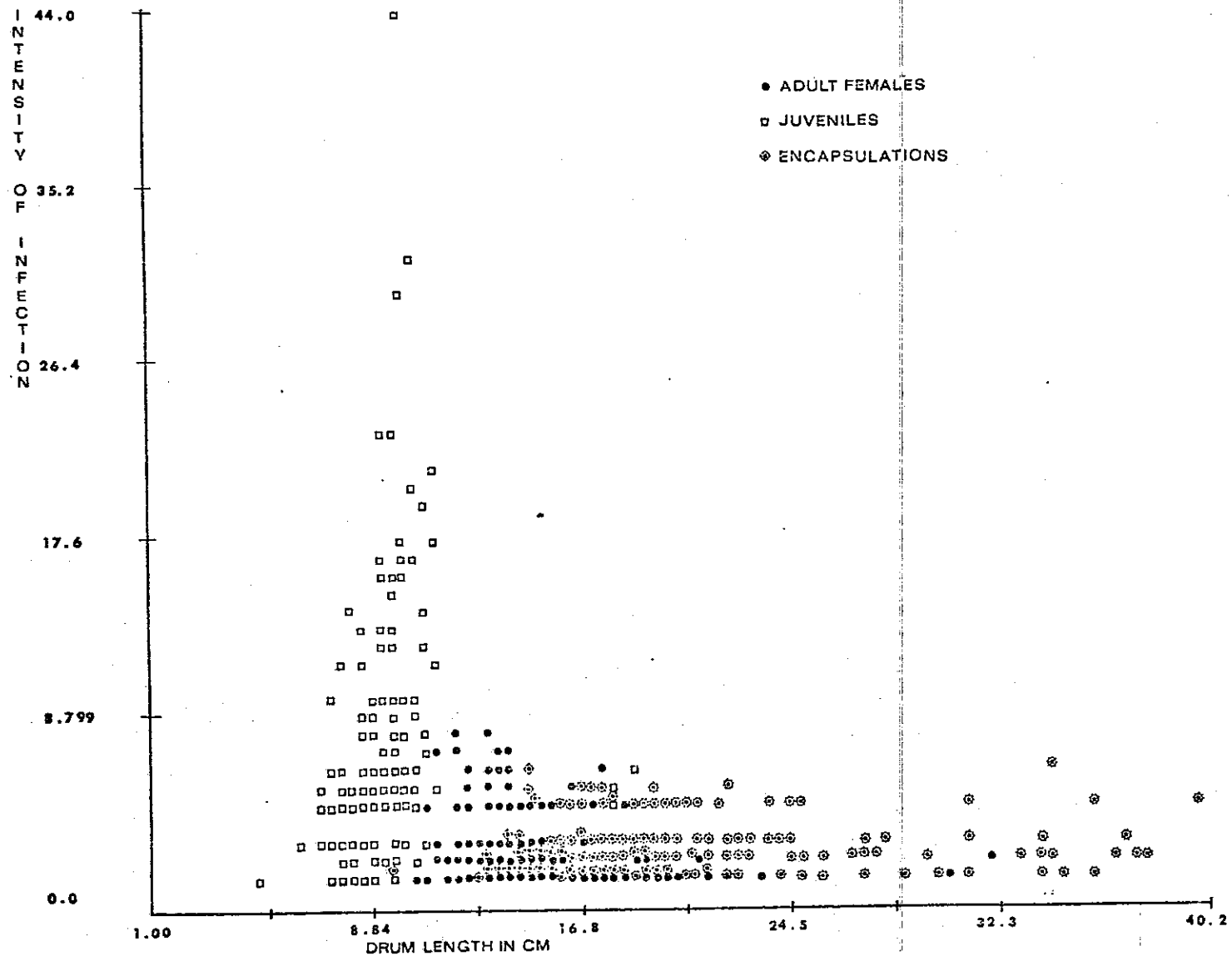
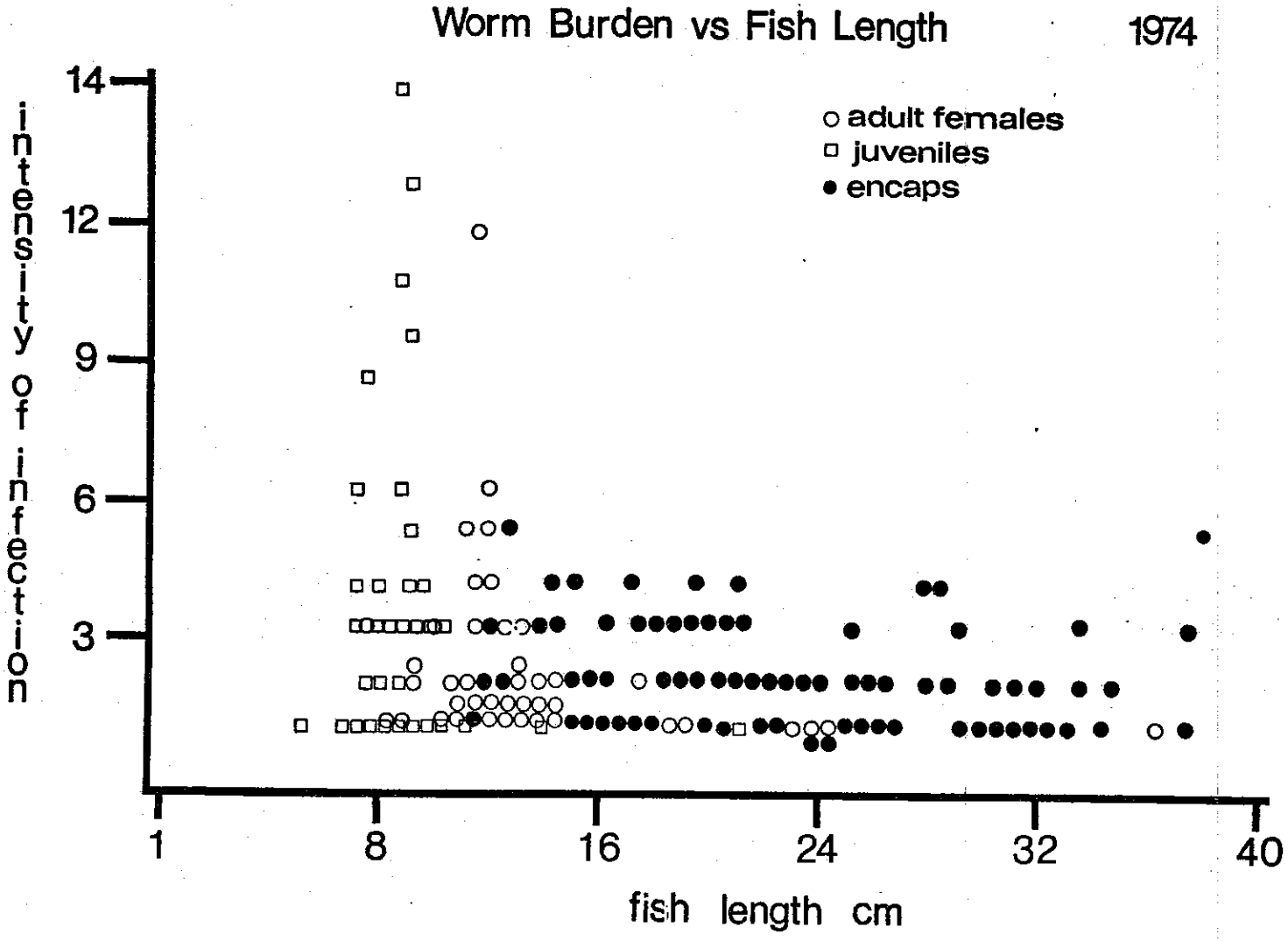

1974
OMNITAB PHILOMETRA STUDY
Juveniles and young adults
worm burden vs time
PAGE 1
ABS- COLUMN 1; ORD- COLUMN 2 (.),
TOTAL NO. OF PTS. PLOTTED IS 837 AND NO. NOT PLOTTED BECAUSE THEY FALL OUTSIDE OF BOUNDS IS 0
INTENSITY OF INFECTION
45
36
27
18
9
0
M
JUNE
JULY
AUGUST
SEPTEMBER
OCT

Figure 55



1972

Figure 57


Worm Burden vs Fish Length
1974
adult females
juveniles
encaps
intensity of infection
14
12
9
6
3
1
8
16
24
32
40
fish length cm

encapsulations are not found in small drum, "young of the year," and occur in 0+, 1+ and 2+ freshwater-drum in Lake Erie only the females have been present and they persist in the older age classes of fish hosts.

Study of Figures 56 and 57 shows the same phenomena as those demonstrated in Figure 55 but there is a distinct overlap of adult females and juvenile philometrids in fish 7 to 13 cm in length, 0+ age class fish. There is also an overlap of adult females with fresh encapsulations in fish 11 to 20 cm in length. This telescoping or overlapping effect was due to the fact that fish hosts grew faster in the summer of 1973 and the mature juvenile worms spread over a greater period of time in 1973 (Figure 56.) These differences, as discussed earlier, may be caused by differences in temperature during the two collecting seasons. The data for 1974 (Figure 57) falls between the years 1972 and 1973.

Figures 58, 59 and 60: Composite scatter-diagrams of data for the years 1972, 1973, and 1974 respectively. The frequencies of infections with gravid and larvigerous females, encapsulations, and juvenile and young adults removed from the eyes of freshwater-drum in Lake Erie in each year are plotted against the time of the year when they were collected. Uninfected fish are not shown.

Inspection of Figure 58 illustrates the seasonal sequence of events in the life history of Philometra sp. in the eyes of freshwater-drum from Lake Erie in 1972. Larvigerous females occurred in June, July and tapered off in early August. The larvigerous females had reached their peak of streaming on July 26, 1972. There is a period in late August and early September of 1972 when there were no living worms found in the eyes, this marks the end of the 1971-1972 generation. At this time larval stages were in the copepod intermediate hosts and in the body cavities of infected fish hosts. In early September, 1972, the juveniles which had been in copepods, and later ingested, or in the body cavities of infected sheepshead migrated to the eyes and were found there as young adults throughout the remainder of the 1972 collecting season. These were a part of the young adult generation which overwintered in the eyes and grew to adult, gravid and larvigerous females in the spring and early summer of 1973 (Figure 59.) The philometrid stages had a rather clumped and distinct distribution during the collecting season of 1972.

Inspection of Figure 59 reveals the same general pattern and distribution of life history stages during 1973. However, the adult females persisted longer in the summer of 1973 and there is some

Figure 58

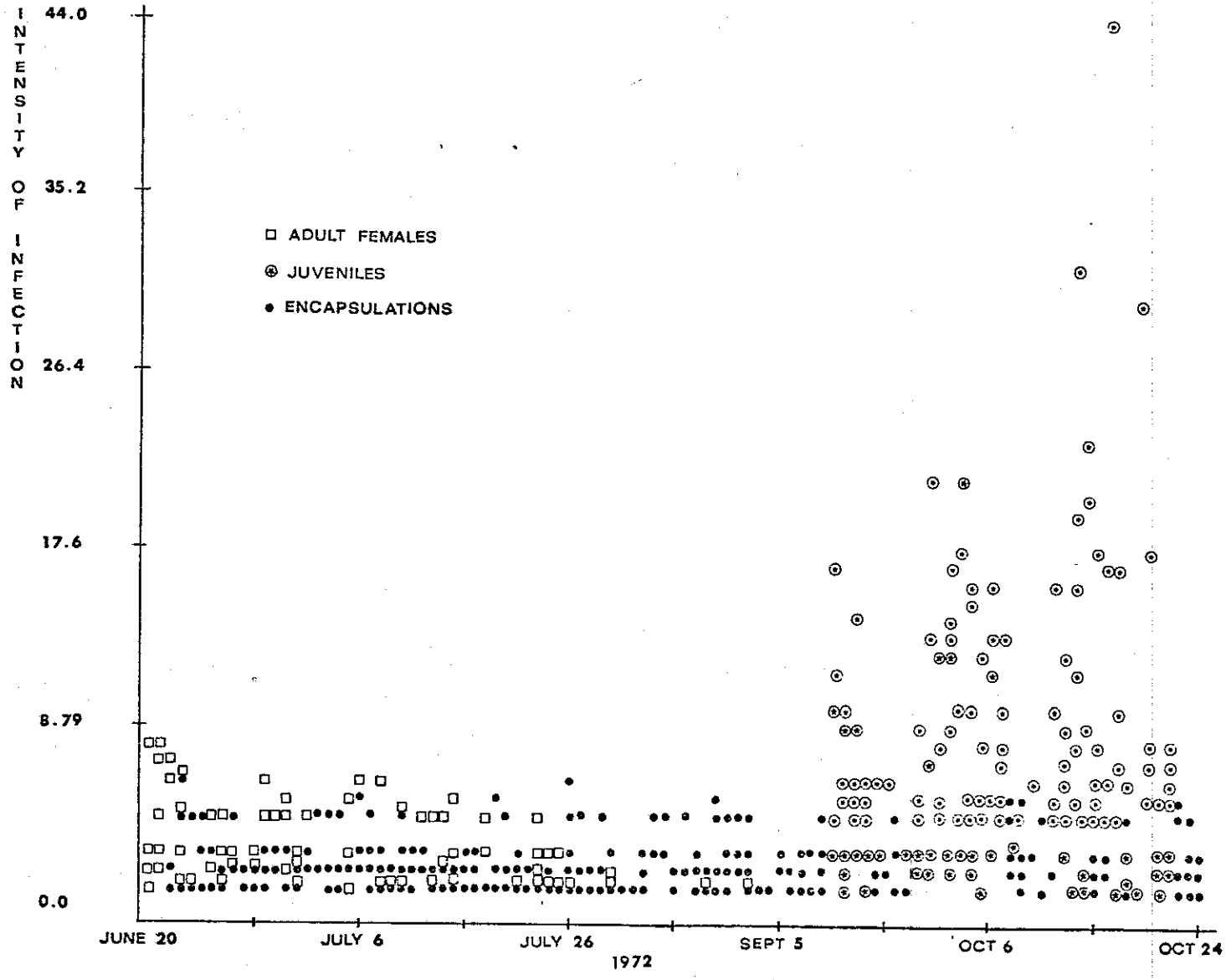

INTENSITY OF INFECTION
44.0
35.2
26.4
17.6
8.79
0.0
ADULT FEMALES
JUVENILES
ENCAPSULATIONS
JUNE 20
JULY 6
JULY 26
SEPT 5
OCT 6
OCT 24
1972

Figure 59

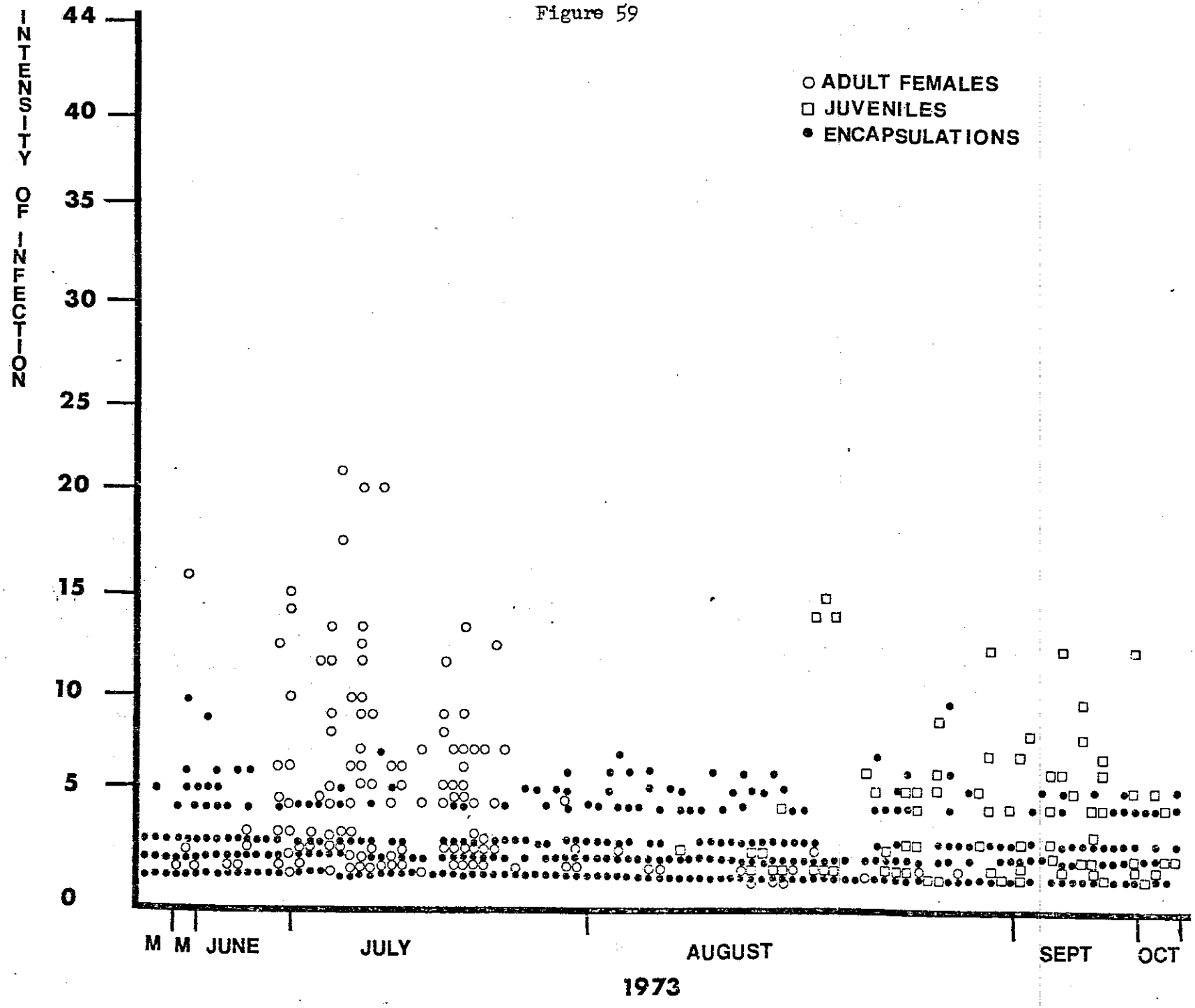
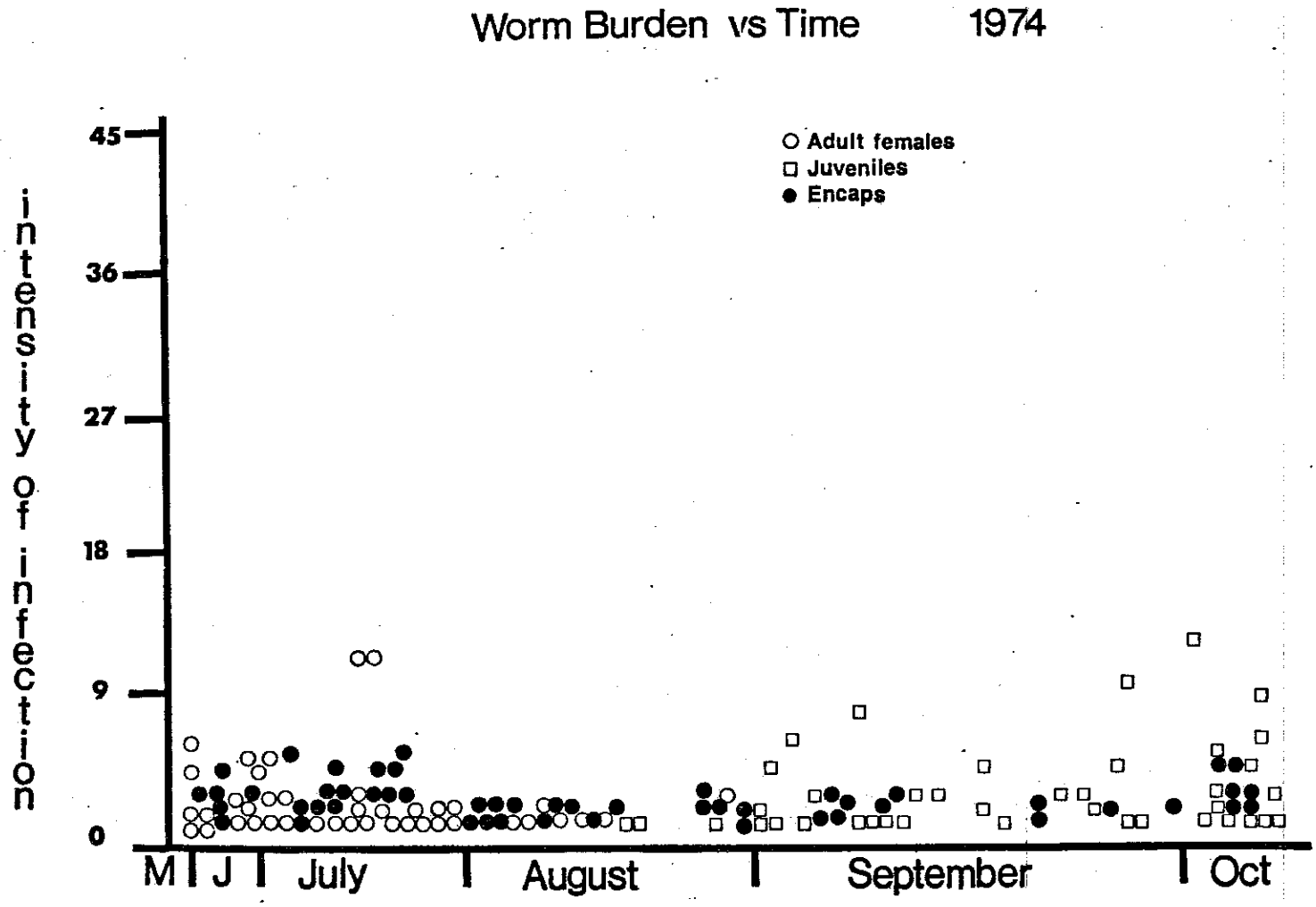

INTENSITY OF INFECTION
44
40
35
30
25
20
15
10
5
0
○ ADULT FEMALES
□ JUVENILES
• ENCAPSULATIONS
M M JUNE
JULY
AUGUST
SEPT
OCT
1973

Figure 60


Worm Burden vs Time 1974
Adult females
Juveniles
Encaps
intensity of infection
45
36
27
18
9
0
M
J
July
August
September
Oct

overlap with the new generation of juveniles entering the eyes in drum collected in August 1973, probably a temperature effect. It is interesting to note that the great number of juveniles and young adults in the fall of 1972 gave rise to a large number of adults in the spring and summer of 1973. The pattern is repeated in 1974 (Figure 60.)

It should be emphasized that the general patterns of distribution in length of fish hosts and of seasonal distributions is very similar for all three years. This species of Philometra primarily infects 0+ class fish and it has one generation per year.

The next section of this report will be devoted to statistics derived from our use of computerized OMNITAB programs and from more simple calculations . Presented here are the total numbers and percentages of freshwater-drum naturally infected with three categories of Philometra sp.: juvenile and young adult worms from the autumn samples; gravid and larvigerous females from the spring and summer samples and encapsulations of previous infections from entire sampling seasons. The data utilized were compiled from our visual autopsies of 3,561 freshwater-drum during the summers of 1972, 1973, and 1974. The data correlates with Figures 29 through 60. Where further explanation or interpretation is deemed necessary a short discussion follows the appropriate tables.

The following tables present the actual numbers of freshwater-drum from Lake Erie autopsied, the numbers of different stages of Philometra sp. recovered, the actual and mean worm burdens, and the incidence (percentage) of freshwater-drum infected during the years 1972, 1973 and 1974. In each table the numbers for the years 1972 through 1974 are stated in series: the number for 1972 is stated first, the number for 1973 follows in parentheses; the number for 1974 is last in brackets.

TABLE 3

TOTAL NUMBER OF ALL Aplodinotus grunniens EXAMINED FROM
JUNE THROUGH OCTOBER 1972
MARCH THROUGH OCTOBER 1973
MARCH THROUGH OCTOBER 1974

1.	Total number of freshwater-drum visually autopsied	799	(1925)	[837]
2.	Total number of freshwater-drum experimentally infected	39	(39)	[20]
3.	Total number of freshwater-drum examined by digestion technique	30	(15)	[10]
	TOTAL	868	(1979)	[867]

TABLE 4

TOTAL NUMBER OF INFECTED Aplodinotus grunniens FROM
JUNE THROUGH OCTOBER 1972
MARCH THROUGH OCTOBER 1973
MARCH THROUGH OCTOBER 1974

1.	Total number of fish infected with juveniles	175	(158)	[65]
2.	Total number of fish infected with mature females	141	(275)	[60]
3.	Total number of fish infected with encapsulations	334	(886)	[198]
4.	Total number of fish infected with juveniles and adults (mature females)	317	(433)	[125]
5.	Total number of fish infected with Philometra sp., all three stages (This includes some fish with both living mature females and encapsulations in the same eye.)	600	(1319)	[323]

Discussion: Items 1, 2 and 3 in Table 4 will correlate with the frequency distributions, Figures 30 through 60 given earlier in the report. Item 5 above, as noted, includes some fish which were infected with both mature females worms and encapsulations at the same time. Later in our overall percentage data these two categories are separated out.

TABLE 5

TOTAL NUMBER OF INFECTED Aplodinotus grunniens
CATEGORIZED BY EYE INFECTED

1.	Total number of fish infected with mature females (adults) in the right eye only	46	(111) [25]
2.	Total number of fish infected with mature females (adults) in the left eye only	66	(102) [22]
3.	Total number of fish infected with juveniles in the right eye only	14	(47) [20]
4.	Total number of fish infected with juveniles in the left eye only	14	(42) [16]
5.	Total number of fish infected with encapsulations in the right eye only	69	(210) [51]
6.	Total number of fish infected with encapsulations in the left eye only	98	(200) [68]
7.	Total number of fish infected with mature females (adults) in the right eye (double and single)	95	(202) [38]
8.	Total number of fish infected with mature females (adults) in the left eye (double and single)	106	(197) [35]
9.	Total number of fish infected with juveniles in the right eye (double and single)	162	(116) [49]
10.	Total number of fish infected with juveniles in the left eye (double and single)	162	(111) [45]
11.	Total number of fish infected with encapsulations in the right eye (double and single)	235	(686) [130]
12.	Total number of fish infected with encapsulations in the left eye (double and single)	264	(676) [147]
13.	Total number of fish infected with mature females (adults) in both eyes (double)	60	(124) [13]
14.	Total number of fish infected with juveniles in both eyes (double)	148	(69) [29]
15.	Total number of fish infected with encapsulations in both eyes (double)	166	(476) [79]

TABLE 6

TOTAL NUMBER OF *Philometra* STAGES PRESENT
IN ALL *Aplodinotus grunniens* AUTOPSIED FROM
JUNE THROUGH OCTOBER 1972
MARCH THROUGH OCTOBER 1973
MARCH THROUGH OCTOBER 1974

1.	Total number of adults in the right eye only	46	(111)	[37]
2.	Total number of adults in the left eye only	66	(102)	[25]
3.	Total number of juveniles in the right eye only	37	(66)	[22]
4.	Total number of juveniles in the left eye only	31	(67)	[22]
5.	Total number of encapsulations in the right eye only	86	(248)	[54]
6.	Total number of encapsulations in the left eye only	129	(250)	[78]
7.	Total number of adults in double eye infections	235	(671)	[54]
8.	Total number of juveniles in double eye infections	1218	(313)	[187]
9.	Total number of encapsulations in double eye infections	479	(1413)	[196]
10.	Total number of adults in the right eye (double and single eye infections	167	(782)	[91]
11.	Total number of adults in the left eye (double and single eye infections)	180	(773)	[79]
12.	Total number of juveniles in the right eye (double and single eye infections)	591	(379)	[209]
13.	Total number of juveniles in the left eye (double and single infections)	695	(380)	[209]
14.	Total number of encapsulations in the right eye (double and single eye infections)	330	(1661)	[250]
15.	Total number of encapsulations in the left eye (double and single eye infections)	364	(1663)	[274]

TABLE 7

SUMMARY OF SOME IMPORTANT TOTALS

	DOUBLE EYE INFECTIONS		INFECTIONS RIGHT EYE ONLY		INFECTIONS LEFT EYE ONLY	
	Worms	Fish	Worms	Fish	Worms	Fish
Adults	235 (671) [54]	60 (124) [13]	46 (111) [37]	35 (78) [25]	66 (102) [25]	46 (73) [22]
Juveniles	1218 (313) [187]	148 (69) [29]	37 (66) [22]	14 (47) [20]	31 (67) [22]	14 (42) [16]
Encapsulations	479 (1413) [196]	166 (476) [79]	86 (248) [54]	69 (210) [51]	129 (250) [78]	98 (200) [68]

TABLE 8

INTENSITY OF INFECTIONS WITH MATURE FEMALES (ADULTS)
DURING THE PERIOD
JUNE THROUGH JULY 1972
MARCH THROUGH OCTOBER 1973
MARCH THROUGH OCTOBER 1974

Total number of worms in the right eye only	46	(111)	[37]
Total number of fish with worms in the right eye only	35	(78)	[25]
Average intensity of infection in the right eye only	1.31*	(1.42)*	[1.48]
Total number of worms in the left eye only	66	(102)	[25]
Total number of fish with worms in the left eye only	46	(73)	[22]
Average intensity of infection in the left eye only	1.43*	(1.39)*	[1.14]
Total number of worms in double eye infections only	235	(671)	[54]
Total number of fish with double eye infections only	60	(124)	[13]
Average intensity of infection with adult worms in double eye infections only	3.91	(5.41)	[4.15]
Total number of right eye infections (single and double)	167	(432)	[91]
Total number of fish with right eye infections (single and double)	95	(202)	[38]
Average intensity of infection in the right eye (single and double)	1.75*	(2.13)*	[2.39]
Total number of left eye infections (single and double)	180	(452)	[79]
Total number of fish with left eye infections (single and double)	106	(197)	[35]
Average intensity of infection in the left eye (single and double)	1.69*	(2.29)*	[2.26]

Discussion: The data presented here (Table 8) demonstrates that there is no preference for either right or left eyes. It also indicates that the average intensity of infection is higher when both eyes are infected.

* Tested using Chi-Square there is no significant difference at the 5% level between categories, not tested for yearly differences.

TABLE 9

INTENSITY OF INFECTIONS WITH "JUVENILE" WORMS
DURING THE PERIOD
AUGUST THROUGH SEPTEMBER 1972
MARCH THROUGH OCTOBER 1973
MARCH THROUGH OCTOBER 1974

Total number of worms in the right eye only	37	(66)	[22]
Total number of fish with worms in the right eye only	14	(47)	[20]
Average intensity of infection with juveniles in the right eye only	2.64*	(1.40)*	[1.10]
Total number of worms in the left eye infections only	31	(67)	[22]
Total number of fish with worms in the left eye only	14	(42)	[16]
Average intensity of infection with juveniles in the left eye only	2.21*	(1.59)*	[1.38]
Total number of worms in double eye infections only	1218	(313)	[187]
Total number of fish with double eye infections only	148	(69)	[29]
Average intensity of infection with juvenile worms in double infections only	8.20	(4.53)	[6.45]
Total number of right eye infections (single and double)	591	(207)	[209]
Total number of fish with right eye infections (single and double)	162	(116)	[49]
Average intensity of infection in the right eye (single and double)	3.03	(1.78)	[4.27]
Total number of left eye infections (single and double)	695	(239)	[209]
Total number of fish with left eye infections (single and double)	162	(111)	[45]
Average intensity of infection in the left eye (single and double)	4.28	(2.15)	[4.64]

Discussion: The data presented in Table 9 illustrates that there is no strong preference by juvenile philometrids for either right or left eyes. It also indicates that the average intensity was higher for double eye infections although this phenomenon was not as marked in 1973 as it was in 1972 and 1974. When the average intensity for juveniles in the fall of 1972, particularly the double eye infections (Table 9,) is compared with the crop of adult female worms to which they gave rise in the spring and summer of 1973 (Table 8, in parentheses) the intensity was higher for juvenile worms. This indicates that there was some loss of post-fertilization female worms over the winter period. This may indicate a density dependent factor was operating in the eye orbit of the freshwater-drum host. The same phenomenon occurred in 1974.

There was a decidedly lower mean intensity of juveniles in 1973 than in 1972. This was probably due to the larger number of "young of the year" sheepshead which reached a feeding stage after the period of biosynchronization of the streaming female worms and infection of copepods in 1973. It is interesting to note that this lower intensity was balanced by the density dependent factor, noted above, when considering the mean burden of gravid adult female Philometra sp. in the spring and summer of 1974.

TABLE 10

INTENSITY OF INFECTIONS WITH ENCAPSULATIONS
OF SPENT FEMALE WORMS
JUNE THROUGH OCTOBER 1972
MARCH THROUGH OCTOBER 1973
MARCH THROUGH OCTOBER 1974

Total number of encapsulations in the right eye only	86	(248)	[54]
Total number of fish with encapsulations in the right eye only	69	(210)	[51]
Average intensity of infection with encapsulations in the right eye only	1.25*	(1.18)*	[1.06]
Total number of encapsulations in the left eye only	129	(250)	[78]
Total number of fish with encapsulations in the left eye only	98	(200)	[68]
Average intensity of infection with encapsulations in the left eye only	1.32*	(1.25)*	[1.15]
Total number of encapsulations in double eye infections only	479	(1413)	[196]
Total number of fish with encapsulations in double eye infections only.	166	(476)	[79]
Average intensity of infection with encapsulations in double eye infections only	2.88	(2.96)	[2.48]
Total number of encapsulations in right eye infections (single and double)	330	(942)	[250]
Total number of fish with right eye infections with encapsulations (single and double)	235	(686)	[130]
Average intensity of infection encapsulations in the right eye (single and double)	1.40*	(1.37)*	[1.92]
Total number of encapsulations in left eye infections (single and double)	364	(969)	[274]
Total number of fish with encapsulations in left eye (single and double)	264	(676)	[78]
Average intensity of infection with encapsulations in the left eye (single and double)	1.38*	(1.43)*	[1.56]

Discussion: The data presented here correlates with that given in Table 8, there is no significant preference for either right or left eyes for encapsulations. This is to be expected as the mature gravid females which partially stream from the eyes' show no preference. As in the case of the mature females there is a slightly higher intensity and incidence of infection for double eye infections. The pattern exhibited is exactly the same for the years 1972, 1973, and 1974.

* Tested using Chi-Square there is no significant difference at the 5% level between the categories or years.

TABLE 11

INCIDENCE OF INFECTION (PERCENTAGE) OF ALL STAGES OF Philometra sp. IN Aplodinotus grunniens
JUNE THROUGH OCTOBER 1972
MARCH THROUGH OCTOBER 1973
MARCH THROUGH OCTOBER 1974

1.	Percent of all fish infected with juveniles	22%	(8.20%)	[8.0%]
2.	Percent of all fish infected with adults	17.5%	(14.28%)	[7.0%]
3.	Percent of all fish infected with encapsulations	42%	(46%)	[24%]
4.	Percent of all fish infected with juveniles and adults	39.7%	(22.4%)	[15%]
5.	Percent of all fish infected with Philometra (all three stages). Adults and encapsulations figured separately.	82%	(68.48%)	[39%]
6.	Percent of all fish infected with Philometra (all three stages)	76.4%	(68.5%)	[39%]
7.	Percent of all fish with adults in the right eye only	4.37%	(4.05%)	[4.18%]
8.	Percent of all fish infected with adults in the left eye only	5.7%	(3.79%)	[2.62%]
9.	Percent of all fish infected with adults in both eyes only	7.5%	(6.40%)	[1.55%]
10.	Percent of all fish with juveniles in the right eye only	1.7%	(2.44%)	[2.38%]
11.	Percent of all fish with juveniles in the left eye only	1.7%	(2.18%)	[1.91%]
12.	Percent of all fish with juveniles in both eyes	18.5%	(3.58%)	[3.46%]

Continues

TABLE 11 (Continued)

13.	Percent of all fish with encapsulations in the right eye only	8.6%	(10.9%) [6.09%]
14.	Percent of all fish with encapsulations in the left eye only	12.2%	(10.3%) [8.12%]
15.	Percent of all fish with encapsulations in both eyes	20.8%	(24.7%) [9.43%]
16.	Percent of fish infected with adult females that have right eye infections only	24.8%	(28.36%) [17.7%
17.	Percent of fish infected with adult females that have left eye infections only	32.5%	(26.5%) [15.6%]
18.	Percent of fish infected with adult females that have double eye infections	42.5%	(45.0%) [9.2%]
19.	Percent of fish infected with juveniles that have infections in the right eye only	8.0%	(29.7%) [11.2%]
20.	Percent of all fish infected with juveniles that have infections in the left eye only	8.0%	(26.5%) [9.1%]
21.	Percent of fish infected with juveniles that have double eye infections	84.5%	(43.6%) [16.5%]
22.	Percent of fish infected with encapsulations that have have right eye infections only	20.6%	(23.7%) [15.2]
23.	Percent of fish infected with encapsulations that have left eye infections only	29.3%	(22.5%) [23.3]
24.	Percent of fish infected with encapsulations that have double eye infections	50.2%	(53.7%) [58.6]

Discussion: These percentages of incidence figures correlate quite well with the frequency distribution, Figures 30-60. The apparent disagreement between items 2 and 3, Table 11, can easily be explained if one considers the adult females were present in only June and July of 1972 and June, July and August of 1973 and 1974, while encapsulations were present in most age classes of fish throughout the entire sampling period each year.

In general, the data presented in Tables 8, 9, and 10 indicates no preference for right or left eyes when intensity of infection is considered. In the last table (Table 11) items 10, 11, 12 and 19, 20, 21 indicate that there was no preference for right and left eyes in incidence (prevalence) of the occurrence of juveniles but double eye infections were more prevalent. While the pattern of infection was similar there was a higher percentage of double eye infections in 1972 and higher percentage of single eye infection in 1973. However, all other items, in the series 7 through 24 indicate that there was a slight preference for incidence in left eye over right eye infections for adult females and encapsulations in 1972. In 1973, the preference, if any, was reversed and there is some indication that there is no significant preference when the two years are averaged. This was confirmed by the data from 1974. The incidence remains higher for double eye infections in 1973 and 1974 and was thus significantly higher for all years. Both incidence and intensity of infection of double eye infections was greater in all 3 years.

The data for right, left and double eye infections in 1972, 1973, and 1974 for the three life history phases (juveniles, adult, gravid females and encapsulations) is illustrated in the following summary graphs.

Figure 61 shows the mean intensity of infection for right eye only, left eye only and double eye only infections for Philometra sp. The data is from infected fish of all sizes autopsied during the period June through October 1972. Figure 62 shows the same data for fish autopsied for the period March through October 1973 and Figure 63 the data for 1974. In 1972 (Figure 61) there was no significant difference (tested with chi-square) between right and left eye infections for juveniles, adults and encapsulations. The same is true for 1973 and 1974 (Figures 62 and 63.) In all years the mean intensity for double eye infections by juveniles and adults was much greater which indicates a true preference for double eye infections. In general encapsulation intensities exhibit the same pattern and are greater for double eye infections.

Figures 64, 65 and 66 illustrate the incidence, percentage, of fish infected by the three life history phases of Philometra sp. Double eye infections by juveniles was higher than single eye infections this was particularly marked in 1972, Figure 64. In all years except 1974 encapsulations parallel the adults which gave rise to them.

Actually the juveniles from the autumn of 1972, Figures 61 and 64, gave rise to the adult females in the summer of 1973, Figures 62 and 64. This indicates that there was not only a loss

Figure 61

INTENSITY OF INFECTION FOR FISH INFECTED WITH PHILOMETRA 1972

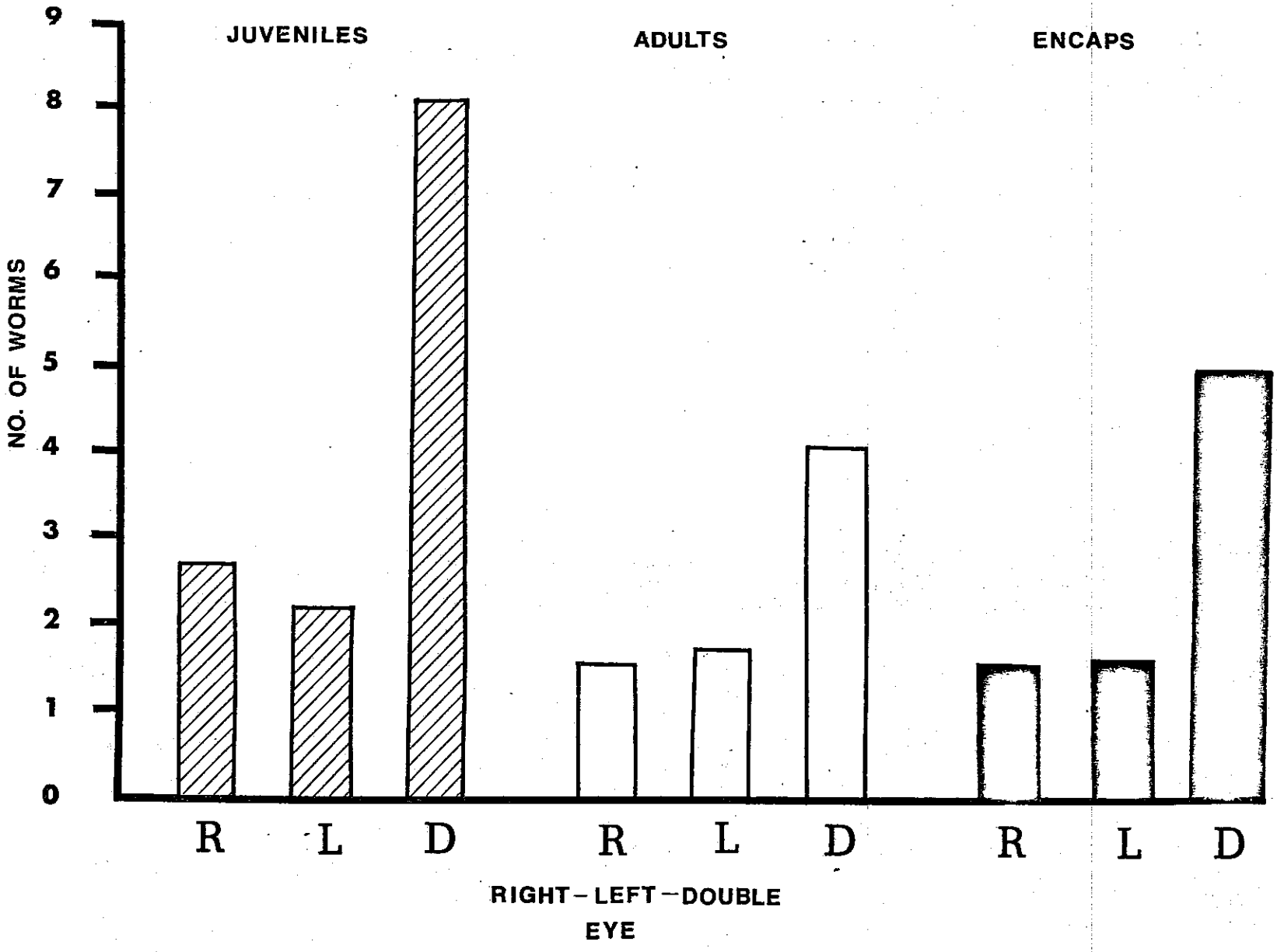


Figure 62

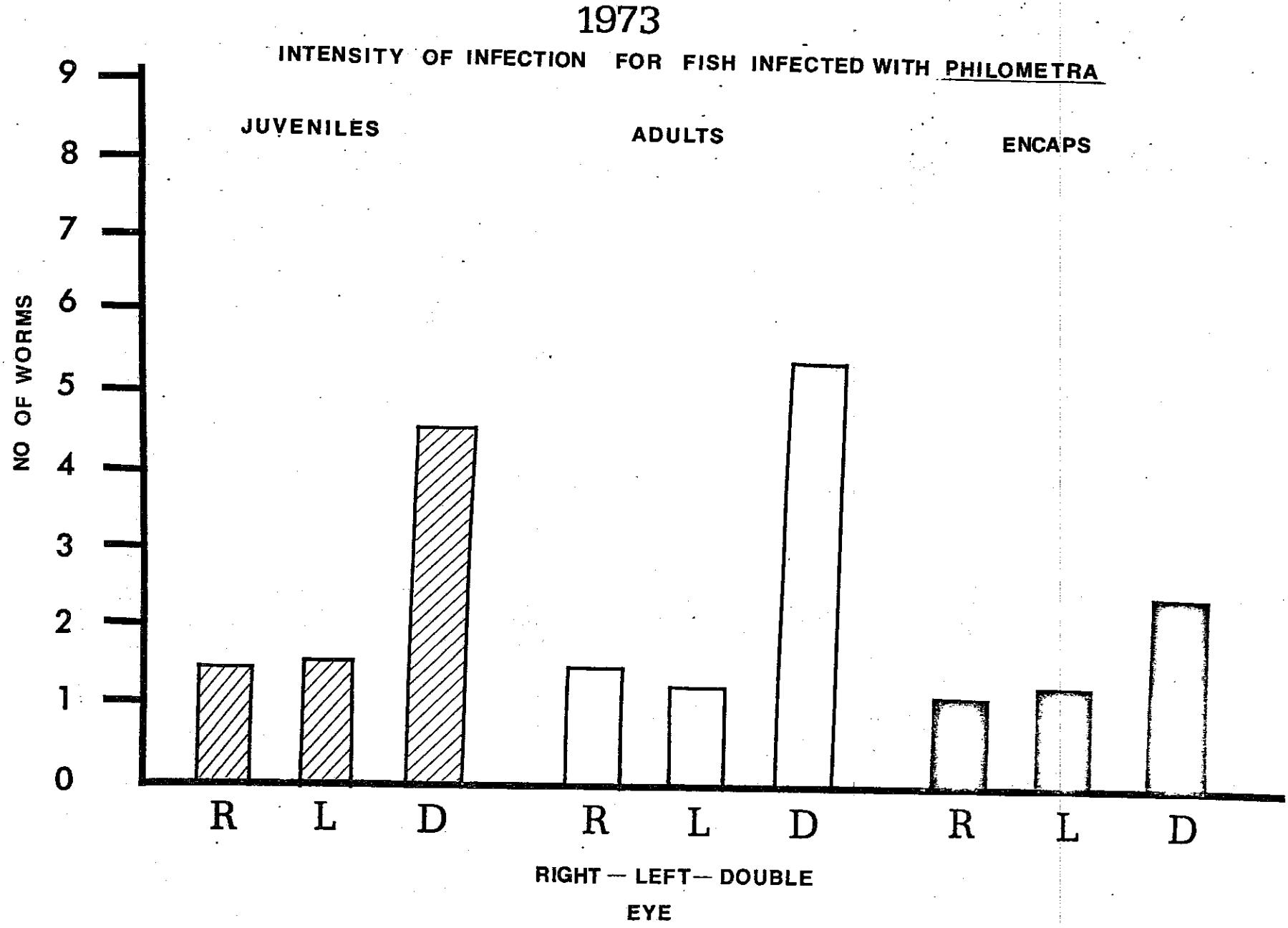

1973
INTENSITY OF INFECTION FOR FISH INFECTED WITH PHILOMETRA
JUVENILES
ADULTS
ENCAPS
NO OF WORMS
0
1
2
3
4
5
6
7
8
9
R
L
D
R
L
D
R
L
D
RIGHT — LEFT — DOUBLE
EYE

Figure 63

Intentensity Of Infection For Fish With Philometra 1974

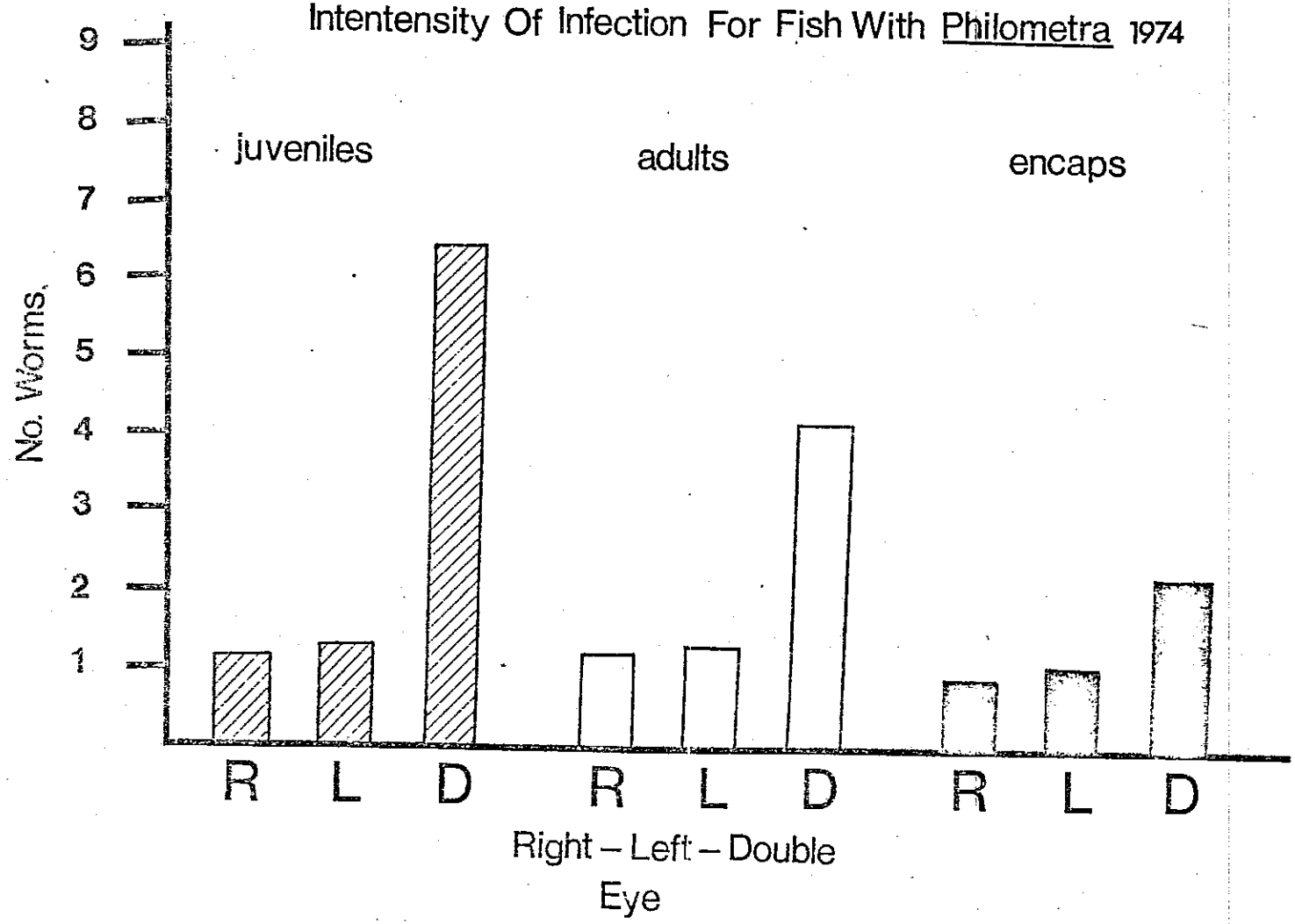


Figure 64

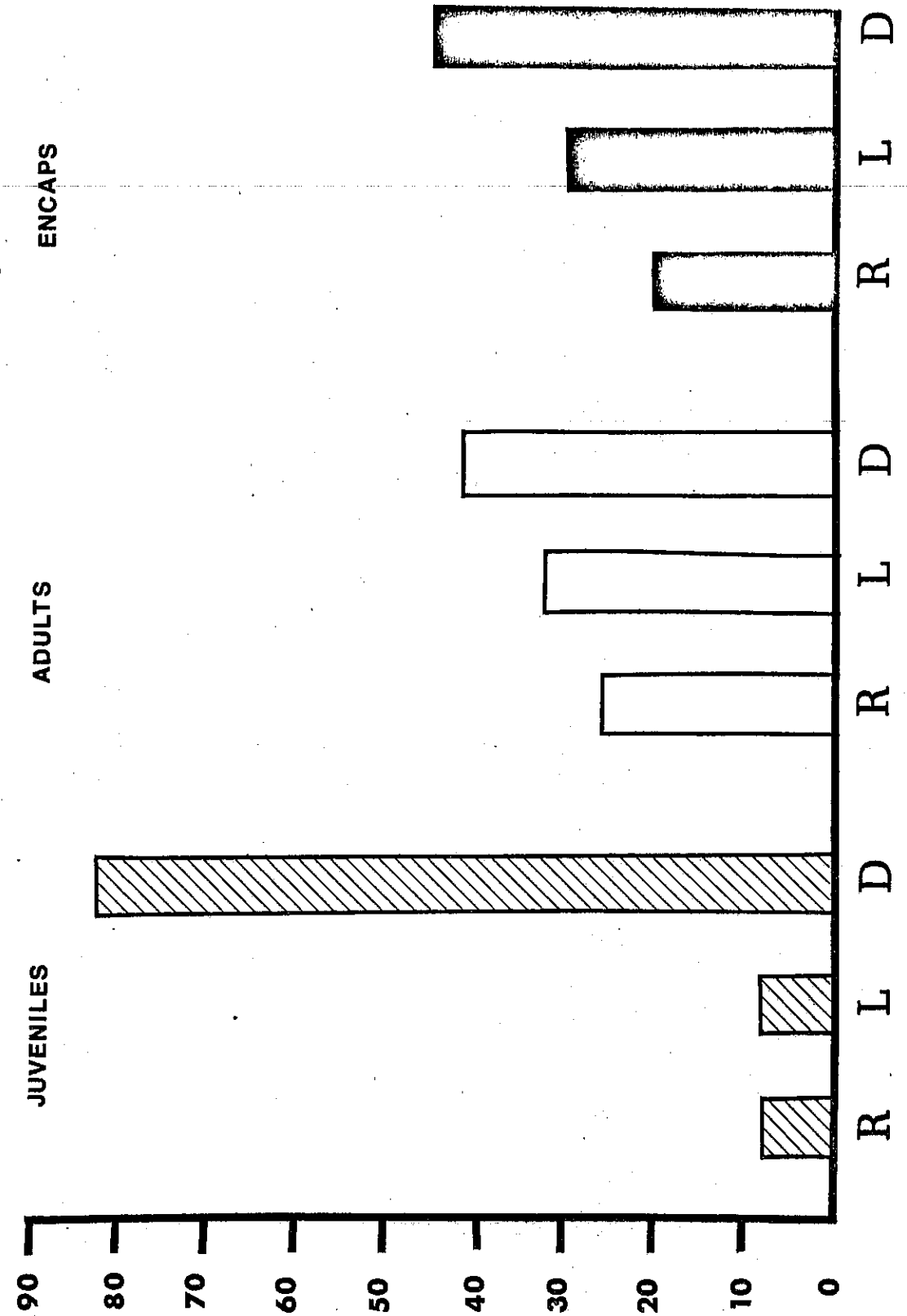

% INCIDENCE OF INFECTION IN EYES OF INFECTED FISH 1972
JUVENILES
ADULTS
ENCAPS
%
90
80
70
60
50
40
30
20
10
0
R L D R L D R L D
RIGHT—LEFT—DOUBLE EYE

Figure 65

% INCIDENCE OF INFECTION IN EYES OF INFECTED FISH 1973

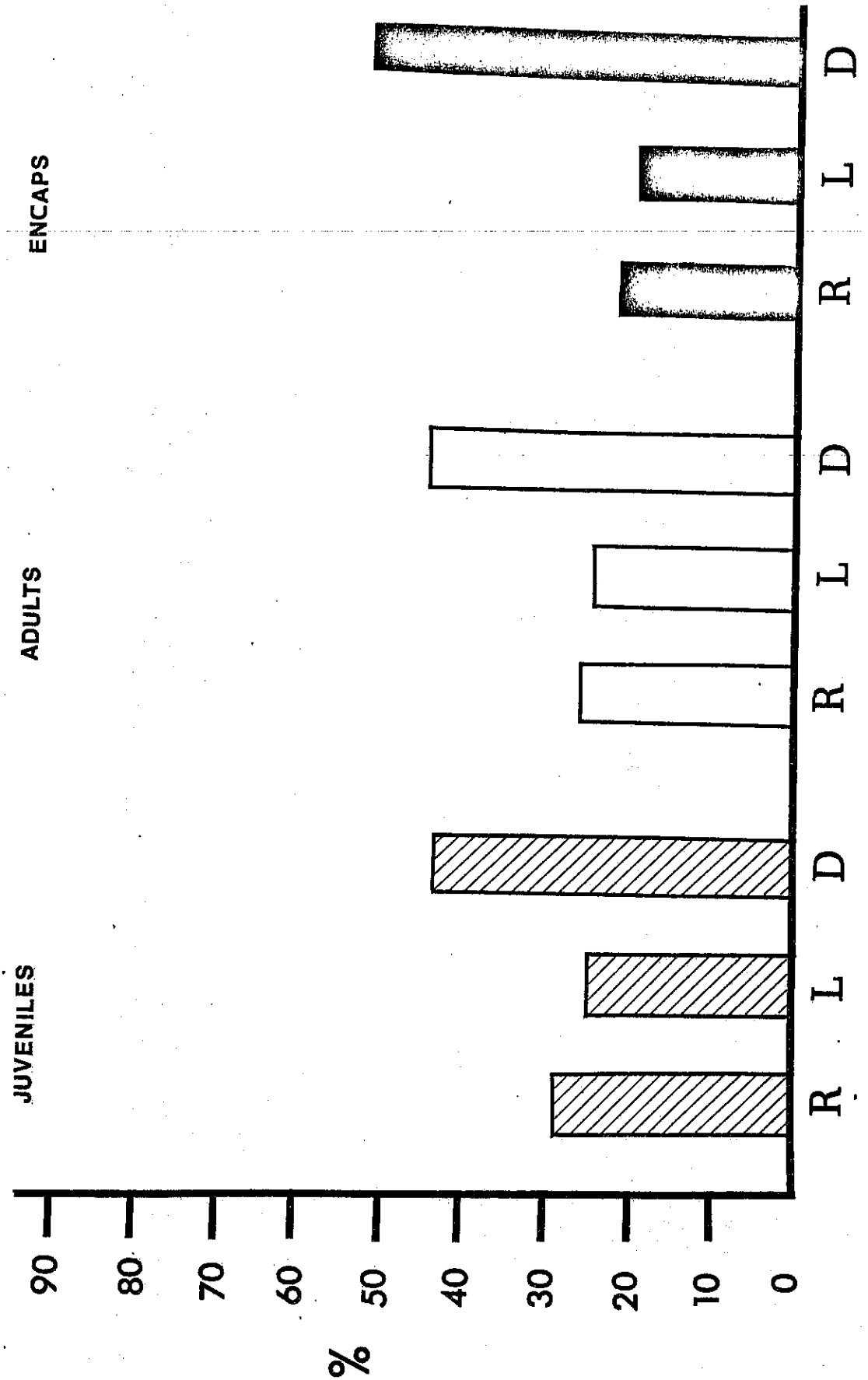


Figure 66

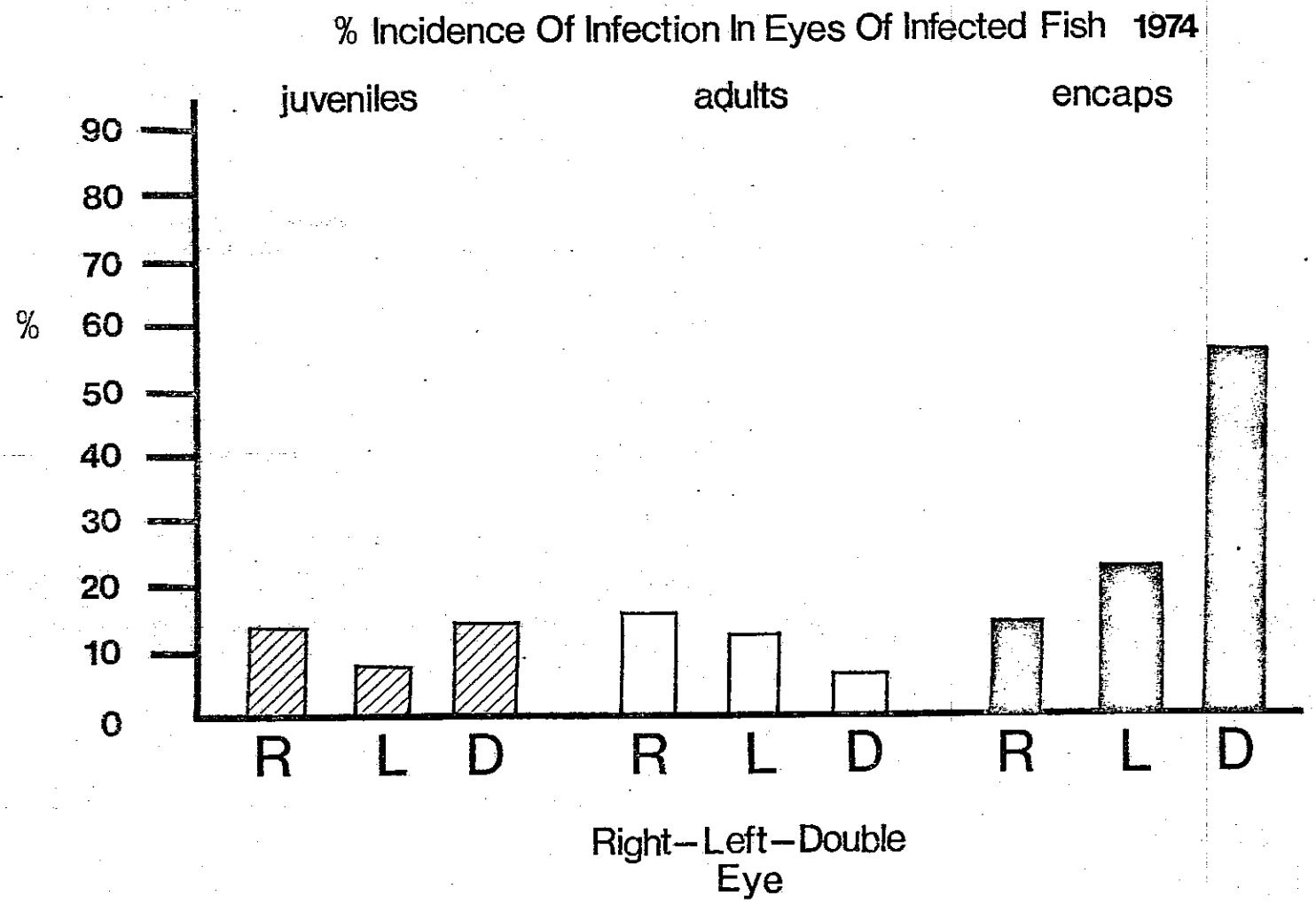

% Incidence Of Infection In Eyes Of Infected Fish 1974
juveniles
adults
encaps
90
80
70
%
60
50
40
30
20
10
0
R
L
D
R
L
D
R
L
D
Right--Left--Double Eye

TABLE 12

FISH INFECTED WITH JUVENILE WORMS

SIZE CLASS	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER
0—5 CM	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.251 MEAN WORMS= -0.0 VARIANCE=****** STD.DEV.=******	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.251 MEAN WORMS= -0.0 VARIANCE= 0.000 STD.DEV.=******	NO. OF FISH= 4.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 6.0 NO. INFECTED= 5.0 FRACTION=0.833 MEAN WORMS= 7.8 VARIANCE=66.139 STD.DEV.= 8.133	NO. OF FISH= 1.0 NO. INFECTED= 1.0 FRACTION=1.000 MEAN WORMS= 17.0 VARIANCE= 0.000 STD.DEV.= 0.016
5.1—10	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.886 MEAN WORMS= -0.0 VARIANCE=****** STD.DEV.=-0.000	NO. OF FISH= 2.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 50.0 NO. INFECTED= 42.0 FRACTION=0.840 MEAN WORMS= 4.5 VARIANCE=13.690 STD.DEV.= 3.700	NO. OF FISH=105.0 NO. INFECTED= 83.0 FRACTION=0.790 MEAN WORMS= 5.4 VARIANCE=28.697 STD.DEV.= 5.357
10.1—15	NO. OF FISH= 51.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 77.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 38.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 13.0 NO. INFECTED= 3.0 FRACTION=0.231 MEAN WORMS= 0.5 VARIANCE= 1.172 STD.DEV.= 1.082	NO. OF FISH= 35.0 NO. INFECTED= 34.0 FRACTION=0.971 MEAN WORMS= 11.3 VARIANCE=85.540 STD.DEV.= 9.249
15.1—20	NO. OF FISH= 42.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 74.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 69.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 55.0 NO. INFECTED= 1.0 FRACTION=0.018 MEAN WORMS= 0.0 VARIANCE= 0.071 STD.DEV.= 0.267	NO. OF FISH= 47.0 NO. INFECTED= 7.0 FRACTION=0.149 MEAN WORMS= 0.5 VARIANCE= 1.738 STD.DEV.= 1.318
20.1—25	NO. OF FISH= 9.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 30.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 24.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 3.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 12.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
25.1—30	NO. OF FISH= 2.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 12.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
30.1—35	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=***** MEAN WORMS= 2.0 VARIANCE= 0.000 STD.DEV.=******	NO. OF FISH= 17.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 3.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=***** MEAN WORMS=***** VARIANCE=****** STD.DEV.=******	NO. OF FISH= 4.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
35.1—40	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 6.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=***** MEAN WORMS=***** VARIANCE=****** STD.DEV.=******	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=***** MEAN WORMS=***** VARIANCE=****** STD.DEV.=-0.000	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.000 MEAN WORMS= 0.0 VARIANCE= 0.000 STD.DEV.=-0.000
40.1—45	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=***** MEAN WORMS= 0.1 VARIANCE=****** STD.DEV.=******	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=***** MEAN WORMS= 1.1 VARIANCE=****** STD.DEV.=******	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=-.000 MEAN WORMS= -0.0 VARIANCE=****** STD.DEV.=******	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=***** MEAN WORMS=***** VARIANCE= 0.000 STD.DEV.=******
45.1+	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.251 MEAN WORMS= -0.0 VARIANCE=****** STD.DEV.=******	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=***** MEAN WORMS= 64.2 VARIANCE=****** STD.DEV.= 0.000	NO. OF FISH= 2.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=-.000 MEAN WORMS= -0.0 VARIANCE=-0.000 STD.DEV.=-7.907	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.000 MEAN WORMS= -0.0 VARIANCE= 0.000 STD.DEV.=******
	TOTAL FISH=107.0 INFECTED FISH= 0.0 FRACTION=0.0	TOTAL FISH=216.0 INFECTED FISH= 0.0 FRACTION=0.0	TOTAL FISH=143.0 INFECTED FISH= 0.0 FRACTION=0.0	TOTAL FISH=128.0 INFECTED FISH= 51.0 FRACTION=0.398	TOTAL FISH=205.0 INFECTED FISH=125.0 FRACTION=0.610

TABLE 13

1973

O.C.

FISH INFECTED WITH JUVENILE WORMS PAGE 2

SIZE CLASS cm	MARCH	MAY	JUNE	JULY	AUGUST
0 — 5	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 37.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 21.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
5.1 — 10	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 25.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH=164.0 NO. INFECTED= 19.0 FRACTION=0.116 MEAN WORMS= 0.2 VARIANCE= 0.377 STD.DEV.= 0.614
10.1 — 15	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 11.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH=221.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH=166.0 NO. INFECTED= 37.0 FRACTION=0.223 MEAN WORMS= 0.8 VARIANCE= 5.083 STD.DEV.= 2.255
15.1 — 20	NO. OF FISH= 8.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 35.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH=109.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH=200.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH=349.0 NO. INFECTED= 22.0 FRACTION=0.063 MEAN WORMS= 0.1 VARIANCE= 0.224 STD.DEV.= 0.474
20.1 — 25	NO. OF FISH= 23.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 2.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 34.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 36.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 53.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
25.1 — 30	NO. OF FISH= 6.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 9.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 15.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 33.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
30.1 — 35	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 6.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 8.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 13.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
35.1 — 40	NO. OF FISH= 0.0 NO. INFECTED= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0	NO. OF FISH= 2.0 NO. INFECTED= 0.0	NO. OF FISH= 3.0 NO. INFECTED= 0.0	NO. OF FISH= 4.0 NO. INFECTED= 0.0

TABLE 13 (continued)

cm					
O.C.	FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
40.1 — 45	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 5.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 4.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
45.1+	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 3.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
	TOTAL FISH= 37.0 INFECTED FISH= 0.0 FRACTION=0.0	TOTAL FISH= 39.0 INFECTED FISH= 0.0 FRACTION=0.0	TOTAL FISH=180.0 INFECTED FISH= 0.0 FRACTION=0.0	TOTAL FISH=550.0 INFECTED FISH= 0.0 FRACTION=0.0	TOTAL FISH=803.0 INFECTED FISH= 78.0 FRACTION=0.097

TABLE 13 (continued)

SIZE CLASS cm	FISH INFECTED WITH JUVENILE WORMS SEPTEMBER	OCTOBER
0—5	NO. OF FISH= 3.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
5.1—10	NO. OF FISH= 68.0 NO. INFECTED= 17.0 FRACTION=0.250 MEAN WORMS= 0.5 VARIANCE= 0.867 STD.DEV.= 0.931	NO. OF FISH= 16.0 NO. INFECTED= 3.0 FRACTION=0.188 MEAN WORMS= 0.3 VARIANCE= 0.313 STD.DEV.= 0.559
10.1—15	NO. OF FISH= 71.0 NO. INFECTED= 31.0 FRACTION=0.437 MEAN WORMS= 1.9 VARIANCE= 7.931 STD.DEV.= 2.816	NO. OF FISH= 51.0 NO. INFECTED= 22.0 FRACTION=0.431 MEAN WORMS= 1.2 VARIANCE= 4.211 STD.DEV.= 2.052
15.1—20	NO. OF FISH= 31.0 NO. INFECTED= 1.0 FRACTION=0.032 MEAN WORMS= 0.1 VARIANCE= 0.125 STD.DEV.= 0.353	NO. OF FISH= 5.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
20.1—25	NO. OF FISH= 19.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 8.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
25.1—30	NO. OF FISH= 3.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
30.1—35	NO. OF FISH= 7.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
35.1—40	NO. OF FISH= 1.0 NO. INFECTED= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0

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TABLE 13 (continue)

cm

	FRACTION=0.0	FRACTION=0.0
	MEAN WORMS= 0.0	MEAN WORMS= 0.0
	VARIANCE= 0.0	VARIANCE= 0.0
	STD.DEV.= 0.0	STD.DEV.= 0.0
40.1—45	NO. OF FISH= 0.0	NO. OF FISH= 0.0
	NO. INFECTED= 0.0	NO. INFECTED= 0.0
	FRACTION=0.0	FRACTION=0.0
	MEAN WORMS= 0.0	MEAN WORMS= 0.0
	VARIANCE= 0.0	VARIANCE= 0.0
	STD.DEV.= 0.0	STD.DEV.= 0.0
45+	NO. OF FISH= 1.0	NO. OF FISH= 0.0
	NO. INFECTED= 0.0	NO. INFECTED= 0.0
	FRACTION=0.0	FRACTION=0.0
	MEAN WORMS= 0.0	MEAN WORMS= 0.0
	VARIANCE= 0.0	VARIANCE= 0.0
	STD.DEV.= 0.0	STD.DEV.= 0.0
	TOTAL FISH=204.0	TOTAL FISH= 81.0
	INFECTED FISH= 49.0	INFECTED FISH= 25.0
	FRACTION=0.240	FRACTION=0.309

TABLE 14

FISH INFECTED WITH JUVENILE WORMS — PAGE 2

SIZE CLASS	MAY	JUNE	JULY	AUGUST
0—5	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 9.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
5.1—10	NO. OF FISH= 2.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 5.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 3.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 84.0 NO. INFECTED= 2.0 FRACTION=0.024 MEAN WORMS= 0.0 VARIANCE= 0.023 STD.DEV.= 0.152
10.1—15	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 24.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 79.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 55.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
15.1—20	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 6.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 25.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 48.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
20.1—25	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 8.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 16.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 12.0 NO. INFECTED= 1.0 FRACTION=0.083 MEAN WORMS= 0.1 VARIANCE= 0.076 STD.DEV.= 0.276
25.1—30	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 7.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 14.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 3.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
30.1—35	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 3.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 12.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
35.1—40	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 5.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
40.1—45	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
45.1—50	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
	TOTAL FISH= 4.0 INFECTED FISH= 0.0 FRACTION=0.0	TOTAL FISH= 55.0 INFECTED FISH= 0.0 FRACTION=0.0	TOTAL FISH=155.0 INFECTED FISH= 0.0 FRACTION=0.0	TOTAL FISH=213.0 INFECTED FISH= 3.0 FRACTION=0.014

Length of Freshwater-drum in Cm.

TABLE 14 (continued)

Continued. Length of Freshwater-drum in Cm.

SIZE CLASS	FISH INFECTED WITH JUVENILE WORMS SEPTEMBER	OCTOBER
0—5	NO. OF FISH= 5.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
5.1—10	NO. OF FISH=245.0 NO. INFECTED= 24.0 FRACTION=0.098 MEAN WORMS= 0.3 VARIANCE= 1.894 STD.DEV.= 1.376	NO. OF FISH= 46.0 NO. INFECTED= 10.0 FRACTION=0.217 MEAN WORMS= 0.5 VARIANCE= 2.120 STD.DEV.= 1.456
10.1—15	NO. OF FISH= 18.0 NO. INFECTED= 5.0 FRACTION=0.278 MEAN WORMS= 0.6 VARIANCE= 1.247 STD.DEV.= 1.117	NO. OF FISH= 27.0 NO. INFECTED= 9.0 FRACTION=0.333 MEAN WORMS= 1.1 VARIANCE= 6.217 STD.DEV.= 2.493
15.1—20	NO. OF FISH= 34.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 6.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
20.1—25	NO. OF FISH= 9.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 9.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
25.1—30	NO. OF FISH= 3.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
30.1—35	NO. OF FISH= 3.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
35.1—40	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
40.1—45	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
45.1—50	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
	TOTAL FISH=318.0 INFECTED FISH= 29.0 FRACTION=0.091	TOTAL FISH= 89.0 INFECTED FISH= 19.0 FRACTION=0.213

TABLE 15

FISH INFECTED WITH ADULT WORMS

SIZE CLASS	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER
0–5 CM	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.251 MEAN WORMS= -0.0 VARIANCE=****** STD.DEV.=******	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.251 MEAN WORMS= -0.0 VARIANCE= 0.000 STD.DEV.=******	NO. OF FISH= 4.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 6.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
5.1–10	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.886 MEAN WORMS= -0.0 VARIANCE=****** STD.DEV.=-0.000	NO. OF FISH= 2.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 50.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH=105.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
10.1–15	NO. OF FISH= 51.0 NO. INFECTED= 39.0 FRACTION=0.765 MEAN WORMS= 2.6 VARIANCE= 5.567 STD.DEV.= 2.359	NO. OF FISH= 77.0 NO. INFECTED= 49.0 FRACTION=0.636 MEAN WORMS= 1.5 VARIANCE= 2.715 STD.DEV.= 1.648	NO. OF FISH= 38.0 NO. INFECTED= 10.0 FRACTION=0.263 MEAN WORMS= 0.4 VARIANCE= 0.496 STD.DEV.= 0.704	NO. OF FISH= 13.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 35.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
15.1–20	NO. OF FISH= 42.0 NO. INFECTED= 15.0 FRACTION=0.357 MEAN WORMS= 0.8 VARIANCE= 2.091 STD.DEV.= 1.446	NO. OF FISH= 74.0 NO. INFECTED= 21.0 FRACTION=0.284 MEAN WORMS= 0.5 VARIANCE= 1.007 STD.DEV.= 1.003	NO. OF FISH= 69.0 NO. INFECTED= 1.0 FRACTION=0.014 MEAN WORMS= 0.0 VARIANCE= 0.014 STD.DEV.= 0.120	NO. OF FISH= 55.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 47.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
20.1–25	NO. OF FISH= 9.0 NO. INFECTED= 1.0 FRACTION=0.111 MEAN WORMS= 0.2 VARIANCE= 0.395 STD.DEV.= 0.629	NO. OF FISH= 30.0 NO. INFECTED= 2.0 FRACTION=0.067 MEAN WORMS= 0.1 VARIANCE= 0.062 STD.DEV.= 0.249	NO. OF FISH= 24.0 NO. INFECTED= 1.0 FRACTION=0.042 MEAN WORMS= 0.0 VARIANCE= 0.040 STD.DEV.= 0.200	NO. OF FISH= 3.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 12.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
25.1–30	NO. OF FISH= 2.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 12.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
30.1–35	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=***** MEAN WORMS= 2.0 VARIANCE= 0.000 STD.DEV.=******	NO. OF FISH= 17.0 NO. INFECTED= 2.0 FRACTION=0.118 MEAN WORMS= 0.2 VARIANCE= 0.263 STD.DEV.= 0.513	NO. OF FISH= 3.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=***** MEAN WORMS=***** VARIANCE=****** STD.DEV.=******	NO. OF FISH= 4.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
35.1–40	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 6.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=***** MEAN WORMS=***** VARIANCE=****** STD.DEV.=******	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=***** MEAN WORMS=***** VARIANCE=****** STD.DEV.=-0.000	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.000 MEAN WORMS= 0.0 VARIANCE= 0.000 STD.DEV.=-0.000
40.1–45	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=***** MEAN WORMS= 0.1 VARIANCE=****** STD.DEV.=******	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=***** MEAN WORMS= 1.1 VARIANCE=****** STD.DEV.=******	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=-.000 MEAN WORMS= -0.0 VARIANCE=****** STD.DEV.=******	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=***** MEAN WORMS=***** VARIANCE= 0.000 STD.DEV.=******
45.1 +	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.251 MEAN WORMS= -0.0 VARIANCE=****** STD.DEV.=******	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=***** MEAN WORMS= 64.2 VARIANCE=****** STD.DEV.= 0.000	NO. OF FISH= 2.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=-.000 MEAN WORMS= -0.0 VARIANCE=-0.000 STD.DEV.=-7.907	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.000 MEAN WORMS= -0.0 VARIANCE= 0.000 STD.DEV.=******
	TOTAL FISH=107.0 INFECTED FISH= 55.0 FRACTION=0.514	TOTAL FISH=216.0 INFECTED FISH= 74.0 FRACTION=0.343	TOTAL FISH=143.0 INFECTED FISH= 12.0 FRACTION=0.084	TOTAL FISH=128.0 INFECTED FISH= 0.0 FRACTION=0.0	TOTAL FISH=205.0 INFECTED FISH= 0.0 FRACTION=0.0

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O.C.

FISH INFECTED WITH ADULT WORMS PAGE 1

SIZE CLASS cm	MARCH	MAY	JUNE	JULY	AUGUST
0—5	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 37.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 21.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
5.1—10	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 25.0 NO. INFECTED= 13.0 FRACTION=0.520 MEAN WORMS= 1.6 VARIANCE= 4.640 STD.DEV.= 2.154	NO. OF FISH=164.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
10.1—15	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 11.0 NO. INFECTED= 10.0 FRACTION=0.909 MEAN WORMS= 4.9 VARIANCE=20.992 STD.DEV.= 4.582	NO. OF FISH=221.0 NO. INFECTED=130.0 FRACTION=0.588 MEAN WORMS= 2.6 VARIANCE=14.913 STD.DEV.= 3.862	NO. OF FISH=166.0 NO. INFECTED= 28.0 FRACTION=0.169 MEAN WORMS= 0.2 VARIANCE= 0.340 STD.DEV.= 0.583
15.1—20	NO. OF FISH= 8.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 35.0 NO. INFECTED= 2.0 FRACTION=0.057 MEAN WORMS= 0.1 VARIANCE= 0.054 STD.DEV.= 0.232	NO. OF FISH=109.0 NO. INFECTED= 14.0 FRACTION=0.128 MEAN WORMS= 0.2 VARIANCE= 0.515 STD.DEV.= 0.718	NO. OF FISH=200.0 NO. INFECTED= 32.0 FRACTION=0.160 MEAN WORMS= 0.4 VARIANCE= 2.633 STD.DEV.= 1.623	NO. OF FISH=349.0 NO. INFECTED= 29.0 FRACTION=0.083 MEAN WORMS= 0.1 VARIANCE= 0.166 STD.DEV.= 0.407
20.1—25	NO. OF FISH= 23.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 2.0 NO. INFECTED= 1.0 FRACTION=0.500 MEAN WORMS= 0.5 VARIANCE= 0.250 STD.DEV.= 0.500	NO. OF FISH= 34.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 36.0 NO. INFECTED= 2.0 FRACTION=0.056 MEAN WORMS= 0.1 VARIANCE= 0.132 STD.DEV.= 0.363	NO. OF FISH= 53.0 NO. INFECTED= 1.0 FRACTION=0.019 MEAN WORMS= 0.0 VARIANCE= 0.019 STD.DEV.= 0.136
25.1—30	NO. OF FISH= 6.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 9.0 NO. INFECTED= 2.0 FRACTION=0.222 MEAN WORMS= 0.4 VARIANCE= 0.914 STD.DEV.= 0.956	NO. OF FISH= 15.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 33.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
30.1—35	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 6.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 8.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 13.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
35.1—40	NO. OF FISH= 0.0 NO. INFECTED= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0	NO. OF FISH= 2.0 NO. INFECTED= 0.0	NO. OF FISH= 3.0 NO. INFECTED= 0.0	NO. OF FISH= 4.0 NO. INFECTED= 0.0

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TABLE 16 (continued)

cm

O.C.	FRACTION=0.0	FRACTION=0.0	FRACTION=0.0	FRACTION=0.0	FRACTION=0.0
	MEAN WORMS= 0.0	MEAN WORMS= 0.0	MEAN WORMS= 0.0	MEAN WORMS= 0.0	MEAN WORMS= 0.0
	VARIANCE= 0.0	VARIANCE= 0.0	VARIANCE= 0.0	VARIANCE= 0.0	VARIANCE= 0.0
	STD.DEV.= 0.0	STD.DEV.= 0.0	STD.DEV.= 0.0	STD.DEV.= 0.0	STD.DEV.= 0.0
40.1—45	NO. OF FISH= 0.0	NO. OF FISH= 0.0	NO. OF FISH= 5.0	NO. OF FISH= 4.0	NO. OF FISH= 0.0
	NO. INFECTED= 0.0	NO. INFECTED= 0.0	NO. INFECTED= 0.0	NO. INFECTED= 0.0	NO. INFECTED= 0.0
	FRACTION=0.0	FRACTION=0.0	FRACTION=0.0	FRACTION=0.0	FRACTION=0.0
	MEAN WORMS= 0.0	MEAN WORMS= 0.0	MEAN WORMS= 0.0	MEAN WORMS= 0.0	MEAN WORMS= 0.0
	VARIANCE= 0.0	VARIANCE= 0.0	VARIANCE= 0.0	VARIANCE= 0.0	VARIANCE= 0.0
	STD.DEV.= 0.0	STD.DEV.= 0.0	STD.DEV.= 0.0	STD.DEV.= 0.0	STD.DEV.= 0.0
45.1 +	NO. OF FISH= 0.0	NO. OF FISH= 0.0	NO. OF FISH= 3.0	NO. OF FISH= 1.0	NO. OF FISH= 0.0
	NO. INFECTED= 0.0	NO. INFECTED= 0.0	NO. INFECTED= 0.0	NO. INFECTED= 1.0	NO. INFECTED= 0.0
	FRACTION=0.0	FRACTION=0.0	FRACTION=0.0	FRACTION=1.000	FRACTION=0.0
	MEAN WORMS= 0.0	MEAN WORMS= 0.0	MEAN WORMS= 0.0	MEAN WORMS= 1.0	MEAN WORMS= 0.0
	VARIANCE= 0.0	VARIANCE= 0.0	VARIANCE= 0.0	VARIANCE= 0.0	VARIANCE= 0.0
	STD.DEV.= 0.0	STD.DEV.= 0.0	STD.DEV.= 0.0	STD.DEV.= 0.0	STD.DEV.= 0.0
	TOTAL FISH= 37.0	TOTAL FISH= 39.0	TOTAL FISH=180.0	TOTAL FISH=550.0	TOTAL FISH=803.0
	INFECTED FISH= 0.0	INFECTED FISH= 3.0	INFECTED FISH= 26.0	INFECTED FISH=178.0	INFECTED FISH= 58.0
	FRACTION=0.0	FRACTION=0.077	FRACTION=0.144	FRACTION=0.324	FRACTION=0.072

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TABLE 16 (continued)

FISH INFECTED WITH ADULT WORMS

SIZE CLASS cm	SEPTEMBER	OCTOBER
0—5	NO. OF FISH= 3.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
5.1—10	NO. OF FISH= 68.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 16.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
10.1—15	NO. OF FISH= 71.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 51.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
15.1—20	NO. OF FISH= 31.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 5.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
20.1—25	NO. OF FISH= 19.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 8.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
25.1—30	NO. OF FISH= 3.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
30.1—35	NO. OF FISH= 7.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
35.1—40	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0

1973

cm

o.c.

TABLE 16 (continued)

	FRACTION=0.0	FRACTION=0.0
	MEAN WORMS= 0.0	MEAN WORMS= 0.0
	VARIANCE= 0.0	VARIANCE= 0.0
	STD.DEV.= 0.0	STD.DEV.= 0.0
40.1—45	NO. OF FISH= 0.0	NO. OF FISH= 0.0
	NO. INFECTED= 0.0	NO. INFECTED= 0.0
	FRACTION=0.0	FRACTION=0.0
	MEAN WORMS= 0.0	MEAN WORMS= 0.0
	VARIANCE= 0.0	VARIANCE= 0.0
	STD.DEV.= 0.0	STD.DEV.= 0.0
45+	NO. OF FISH= 1.0	NO. OF FISH= 0.0
	NO. INFECTED= 0.0	NO. INFECTED= 0.0
	FRACTION=0.0	FRACTION=0.0
	MEAN WORMS= 0.0	MEAN WORMS= 0.0
	VARIANCE= 0.0	VARIANCE= 0.0
	STD.DEV.= 0.0	STD.DEV.= 0.0
	TOTAL FISH=204.0	TOTAL FISH= 81.0
	INFECTED FISH= 0.0	INFECTED FISH= 0.0
	FRACTION=0.0	FRACTION=0.0

TABLE 17

Continued. Length of Freshwater-drum in Cm.

SIZE CLASS	FISH INFECTED WITH ADULT WORMS SEPTEMBER	OCTOBER
0—5	NO. OF FISH= 5.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
5.1—10	NO. OF FISH=245.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 46.0 NO. INFECTED= 1.0 FRACTION=0.022 MEAN WORMS= 0.0 VARIANCE= 0.065 STD.DEV.= 0.292
10.1—15	NO. OF FISH= 18.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 27.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
15.1—20	NO. OF FISH= 34.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 6.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
20.1—25	NO. OF FISH= 9.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 9.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
25.1—30	NO. OF FISH= 3.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
30.1—35	NO. OF FISH= 3.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
35.1—40	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
40.1—45	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
45.1—50	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
	TOTAL FISH=318.0 INFECTED FISH= 0.0 FRACTION=0.0	TOTAL FISH= 89.0 INFECTED FISH= 1.0 FRACTION=0.011

TABLE 18

SIZE CLASS	FISH INFECTED WITH ENCAPSULATED WORMS JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER
0—5 CM	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.251 MEAN WORMS= -0.0 VARIANCE=****** STD.DEV.=******	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.251 MEAN WORMS= -0.0 VARIANCE= 0.000 STD.DEV.=******	NO. OF FISH= 4.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 6.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
5.1—10	NO. OF FISH= 1.0 NO. INFECTED= 1.0 FRACTION=1.000 MEAN WORMS= 1.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.886 MEAN WORMS= -0.0 VARIANCE=****** STD.DEV.=-0.000	NO. OF FISH= 2.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 50.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH=105.0 NO. INFECTED= 1.0 FRACTION=0.010 MEAN WORMS= 0.0 VARIANCE= 0.009 STD.DEV.= 0.097
10.1—15	NO. OF FISH= 51.0 NO. INFECTED= 6.0 FRACTION=0.118 MEAN WORMS= 0.3 VARIANCE= 1.070 STD.DEV.= 1.035	NO. OF FISH= 77.0 NO. INFECTED= 17.0 FRACTION=0.221 MEAN WORMS= 0.3 VARIANCE= 0.457 STD.DEV.= 0.676	NO. OF FISH= 38.0 NO. INFECTED= 21.0 FRACTION=0.553 MEAN WORMS= 1.3 VARIANCE= 2.036 STD.DEV.= 1.427	NO. OF FISH= 13.0 NO. INFECTED= 7.0 FRACTION=0.538 MEAN WORMS= 1.1 VARIANCE= 1.609 STD.DEV.= 1.269	NO. OF FISH= 35.0 NO. INFECTED= 1.0 FRACTION=0.029 MEAN WORMS= 0.1 VARIANCE= 0.250 STD.DEV.= 0.500
15.1—20	NO. OF FISH= 42.0 NO. INFECTED= 27.0 FRACTION=0.643 MEAN WORMS= 1.1 VARIANCE= 1.248 STD.DEV.= 1.117	NO. OF FISH= 74.0 NO. INFECTED= 51.0 FRACTION=0.689 MEAN WORMS= 1.4 VARIANCE= 1.732 STD.DEV.= 1.316	NO. OF FISH= 69.0 NO. INFECTED= 46.0 FRACTION=0.667 MEAN WORMS= 1.2 VARIANCE= 1.490 STD.DEV.= 1.221	NO. OF FISH= 55.0 NO. INFECTED= 40.0 FRACTION=0.727 MEAN WORMS= 1.3 VARIANCE= 1.159 STD.DEV.= 1.077	NO. OF FISH= 47.0 NO. INFECTED= 27.0 FRACTION=0.574 MEAN WORMS= 1.5 VARIANCE= 2.675 STD.DEV.= 1.636
20.1—25	NO. OF FISH= 9.0 NO. INFECTED= 7.0 FRACTION=0.778 MEAN WORMS= 1.4 VARIANCE= 0.914 STD.DEV.= 0.956	NO. OF FISH= 30.0 NO. INFECTED= 26.0 FRACTION=0.867 MEAN WORMS= 1.9 VARIANCE= 1.490 STD.DEV.= 1.221	NO. OF FISH= 24.0 NO. INFECTED= 13.0 FRACTION=0.542 MEAN WORMS= 1.5 VARIANCE= 2.582 STD.DEV.= 1.607	NO. OF FISH= 3.0 NO. INFECTED= 1.0 FRACTION=0.333 MEAN WORMS= 0.3 VARIANCE= 0.222 STD.DEV.= 0.471	NO. OF FISH= 12.0 NO. INFECTED= 7.0 FRACTION=0.583 MEAN WORMS= 1.7 VARIANCE= 2.389 STD.DEV.= 1.546
25.1—30	NO. OF FISH= 2.0 NO. INFECTED= 2.0 FRACTION=1.000 MEAN WORMS= 1.5 VARIANCE= 0.250 STD.DEV.= 0.500	NO. OF FISH= 12.0 NO. INFECTED= 9.0 FRACTION=0.750 MEAN WORMS= 1.4 VARIANCE= 0.910 STD.DEV.= 0.954	NO. OF FISH= 1.0 NO. INFECTED= 1.0 FRACTION=1.000 MEAN WORMS= 1.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 1.0 FRACTION=1.000 MEAN WORMS= 3.0 VARIANCE= 0.000 STD.DEV.= 0.003
30.1—35	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=***** MEAN WORMS= 2.0 VARIANCE= 0.000 STD.DEV.=******	NO. OF FISH= 17.0 NO. INFECTED= 9.0 FRACTION=0.529 MEAN WORMS= 1.3 VARIANCE= 2.796 STD.DEV.= 1.672	NO. OF FISH= 3.0 NO. INFECTED= 2.0 FRACTION=0.667 MEAN WORMS= 1.7 VARIANCE= 1.556 STD.DEV.= 1.247	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=***** MEAN WORMS=***** VARIANCE=****** STD.DEV.=******	NO. OF FISH= 4.0 NO. INFECTED= 3.0 FRACTION=0.750 MEAN WORMS= 1.5 VARIANCE= 1.250 STD.DEV.= 1.118
35.1—40	NO. OF FISH= 1.0 NO. INFECTED= 1.0 FRACTION=1.000 MEAN WORMS= 4.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 6.0 NO. INFECTED= 6.0 FRACTION=1.000 MEAN WORMS= 2.3 VARIANCE= 0.889 STD.DEV.= 0.943	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=***** MEAN WORMS=***** VARIANCE=****** STD.DEV.=******	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=***** MEAN WORMS=***** VARIANCE=****** STD.DEV.=-0.000	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.000 MEAN WORMS= 0.0 VARIANCE= 0.000 STD.DEV.=-0.000
40.1—45	NO. OF FISH= 1.0 NO. INFECTED= 1.0 FRACTION=1.000 MEAN WORMS= 4.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=***** MEAN WORMS= 0.1 VARIANCE=****** STD.DEV.=******	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=***** MEAN WORMS= 1.1 VARIANCE=****** STD.DEV.=******	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=-.000 MEAN WORMS= -0.0 VARIANCE=****** STD.DEV.=******	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=***** MEAN WORMS=***** VARIANCE= 0.000 STD.DEV.=******
45.+	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.251 MEAN WORMS= -0.0 VARIANCE=****** STD.DEV.=******	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=***** MEAN WORMS= 64.2 VARIANCE=****** STD.DEV.= 0.000	NO. OF FISH= 2.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=-.000 MEAN WORMS= -0.0 VARIANCE=-0.000 STD.DEV.=-7.907	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.000 MEAN WORMS= -0.0 VARIANCE= 0.000 STD.DEV.=******
	TOTAL FISH=107.0 INFECTED FISH= 45.0 FRACTION=0.421	TOTAL FISH=216.0 INFECTED FISH=118.0 FRACTION=0.546	TOTAL FISH=143.0 INFECTED FISH= 83.0 FRACTION=0.580	TOTAL FISH=128.0 INFECTED FISH= 48.0 FRACTION=0.375	TOTAL FISH=205.0 INFECTED FISH= 40.0 FRACTION=0.195

TABLE 19

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PAGE 3

SIZE CLASS cm	FISH INFECTED WITH ENCAPSULATED FORMS MARCH	MAY	JUNE	JULY	AUGUST
0—5	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 37.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 21.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
5.1—10	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 25.0 NO. INFECTED= 2.0 FRACTION=0.080 MEAN WORMS= 0.2 VARIANCE= 0.640 STD.DEV.= 0.810	NO. OF FISH=164.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
10.1—15	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 1.0 FRACTION=1.000 MEAN WORMS= 1.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 11.0 NO. INFECTED= 2.0 FRACTION=0.182 MEAN WORMS= 0.4 VARIANCE= 0.777 STD.DEV.= 0.881	NO. OF FISH=221.0 NO. INFECTED= 65.0 FRACTION=0.294 MEAN WORMS= 0.6 VARIANCE= 1.270 STD.DEV.= 1.127	NO. OF FISH=166.0 NO. INFECTED= 72.0 FRACTION=0.434 MEAN WORMS= 1.0 VARIANCE= 2.131 STD.DEV.= 1.460
15.1—20	NO. OF FISH= 8.0 NO. INFECTED= 7.0 FRACTION=0.875 MEAN WORMS= 1.6 VARIANCE= 0.984 STD.DEV.= 0.992	NO. OF FISH= 35.0 NO. INFECTED= 27.0 FRACTION=0.771 MEAN WORMS= 1.9 VARIANCE= 3.930 STD.DEV.= 1.982	NO. OF FISH=109.0 NO. INFECTED= 73.0 FRACTION=0.670 MEAN WORMS= 1.5 VARIANCE= 2.451 STD.DEV.= 1.566	NO. OF FISH=200.0 NO. INFECTED=118.0 FRACTION=0.590 MEAN WORMS= 1.2 VARIANCE= 1.744 STD.DEV.= 1.321	NO. OF FISH=349.0 NO. INFECTED=232.0 FRACTION=0.665 MEAN WORMS= 1.4 VARIANCE= 2.195 STD.DEV.= 1.482
20.1—25	NO. OF FISH= 23.0 NO. INFECTED= 19.0 FRACTION=0.826 MEAN WORMS= 1.6 VARIANCE= 1.456 STD.DEV.= 1.206	NO. OF FISH= 2.0 NO. INFECTED= 1.0 FRACTION=0.500 MEAN WORMS= 0.5 VARIANCE= 0.250 STD.DEV.= 0.500	NO. OF FISH= 34.0 NO. INFECTED= 28.0 FRACTION=0.824 MEAN WORMS= 2.0 VARIANCE= 2.499 STD.DEV.= 1.581	NO. OF FISH= 36.0 NO. INFECTED= 23.0 FRACTION=0.639 MEAN WORMS= 1.6 VARIANCE= 2.453 STD.DEV.= 1.566	NO. OF FISH= 53.0 NO. INFECTED= 31.0 FRACTION=0.585 MEAN WORMS= 1.0 VARIANCE= 1.745 STD.DEV.= 1.116
25.1—30	NO. OF FISH= 6.0 NO. INFECTED= 6.0 FRACTION=1.000 MEAN WORMS= 1.3 VARIANCE= 0.556 STD.DEV.= 0.745	NO. OF FISH= 1.0 NO. INFECTED= 1.0 FRACTION=1.000 MEAN WORMS= 1.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 9.0 NO. INFECTED= 9.0 FRACTION=1.000 MEAN WORMS= 2.3 VARIANCE= 0.667 STD.DEV.= 0.816	NO. OF FISH= 15.0 NO. INFECTED= 8.0 FRACTION=0.533 MEAN WORMS= 1.3 VARIANCE= 2.729 STD.DEV.= 1.652	NO. OF FISH= 33.0 NO. INFECTED= 26.0 FRACTION=0.788 MEAN WORMS= 1.9 VARIANCE= 3.113 STD.DEV.= 1.764
30.1—35	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 6.0 NO. INFECTED= 6.0 FRACTION=1.000 MEAN WORMS= 3.0 VARIANCE= 1.000 STD.DEV.= 1.000	NO. OF FISH= 8.0 NO. INFECTED= 5.0 FRACTION=0.625 MEAN WORMS= 1.1 VARIANCE= 1.109 STD.DEV.= 1.053	NO. OF FISH= 13.0 NO. INFECTED= 10.0 FRACTION=0.769 MEAN WORMS= 1.3 VARIANCE= 0.482 STD.DEV.= 0.991
35.1—40	NO. OF FISH= 0.0 NO. INFECTED= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0	NO. OF FISH= 2.0 NO. INFECTED= 2.0	NO. OF FISH= 3.0 NO. INFECTED= 2.0	NO. OF FISH= 4.0 NO. INFECTED= 2.0

1973

TABLE 19 (continued)

cm

o.c.	FRACTION=0.0	FRACTION=0.0	FRACTION=1.000	FRACTION=0.667	FRACTION=0.500
	MEAN WORMS= 0.0	MEAN WORMS= 0.0	MEAN WORMS= 1.0	MEAN WORMS= 0.7	MEAN WORMS= 1.0
	VARIANCE= 0.0	VARIANCE= 0.0	VARIANCE= 0.0	VARIANCE= 0.222	VARIANCE= 1.500
	STD.DEV.= 0.0	STD.DEV.= 0.0	STD.DEV.= 0.0	STD.DEV.= 0.471	STD.DEV.= 1.225
40.1—45	NO. OF FISH= 0.0	NO. OF FISH= 0.0	NO. OF FISH= 5.0	NO. OF FISH= 4.0	NO. OF FISH= 0.0
	NO. INFECTED= 0.0	NO. INFECTED= 0.0	NO. INFECTED= 4.0	NO. INFECTED= 3.0	NO. INFECTED= 0.0
	FRACTION=0.0	FRACTION=0.0	FRACTION=0.800	FRACTION=0.750	FRACTION=0.0
	MEAN WORMS= 0.0	MEAN WORMS= 0.0	MEAN WORMS= 2.0	MEAN WORMS= 2.3	MEAN WORMS= 0.0
	VARIANCE= 0.0	VARIANCE= 0.0	VARIANCE= 2.000	VARIANCE= 3.188	VARIANCE= 0.0
	STD.DEV.= 0.0	STD.DEV.= 0.0	STD.DEV.= 1.414	STD.DEV.= 1.785	STD.DEV.= 0.0
45+	NO. OF FISH= 0.0	NO. OF FISH= 0.0	NO. OF FISH= 3.0	NO. OF FISH= 1.0	NO. OF FISH= 0.0
	NO. INFECTED= 0.0	NO. INFECTED= 0.0	NO. INFECTED= 2.0	NO. INFECTED= 0.0	NO. INFECTED= 0.0
	FRACTION=0.0	FRACTION=0.0	FRACTION=0.667	FRACTION=0.0	FRACTION=0.0
	MEAN WORMS= 0.0	MEAN WORMS= 0.0	MEAN WORMS= 2.3	MEAN WORMS= 0.0	MEAN WORMS= 0.0
	VARIANCE= 0.0	VARIANCE= 0.0	VARIANCE= 4.222	VARIANCE= 0.0	VARIANCE= 0.0
	STD.DEV.= 0.0	STD.DEV.= 0.0	STD.DEV.= 2.055	STD.DEV.= 0.0	STD.DEV.= 0.0
	TOTAL FISH= 37.0	TOTAL FISH= 39.0	TOTAL FISH=180.0	TOTAL FISH=550.0	TOTAL FISH=803.0
	INFECTED FISH= 32.0	INFECTED FISH= 30.0	INFECTED FISH=126.0	INFECTED FISH=226.0	INFECTED FISH=373.0
	FRACTION=0.865	FRACTION=0.769	FRACTION=0.700	FRACTION=0.411	FRACTION=0.465

TABLE 19 (continued)

1973

SIZE CLASS cm	FISH INFECTED WITH ENCAPSULATED WORMS SEPTEMBER	OCTOBER
0—5	NO. OF FISH= 3.0	NO. OF FISH= 0.0
	NO. INFECTED= 0.0	NO. INFECTED= 0.0
	FRACTION=0.0	FRACTION=0.0
	MEAN WORMS= 0.0	MEAN WORMS= 0.0
	VARIANCE= 0.0	VARIANCE= 0.0
	STD.DEV.= 0.0	STD.DEV.= 0.0
5.1—10	NO. OF FISH= 68.0	NO. OF FISH= 16.0
	NO. INFECTED= 0.0	NO. INFECTED= 0.0
	FRACTION=0.0	FRACTION=0.0
	MEAN WORMS= 0.0	MEAN WORMS= 0.0
	VARIANCE= 0.0	VARIANCE= 0.0
	STD.DEV.= 0.0	STD.DEV.= 0.0
10.1—15	NO. OF FISH= 71.0	NO. OF FISH= 51.0
	NO. INFECTED= 7.0	NO. INFECTED= 2.0
	FRACTION=0.099	FRACTION=0.039
	MEAN WORMS= 0.2	MEAN WORMS= 0.1
	VARIANCE= 0.589	VARIANCE= 0.190
	STD.DEV.= 0.768	STD.DEV.= 0.436
15.1—20	NO. OF FISH= 31.0	NO. OF FISH= 5.0
	NO. INFECTED= 24.0	NO. INFECTED= 5.0
	FRACTION=0.774	FRACTION=1.000
	MEAN WORMS= 1.7	MEAN WORMS= 2.6
	VARIANCE= 1.767	VARIANCE= 1.440
	STD.DEV.= 1.329	STD.DEV.= 1.200
20.1—25	NO. OF FISH= 19.0	NO. OF FISH= 8.0
	NO. INFECTED= 13.0	NO. INFECTED= 6.0
	FRACTION=0.684	FRACTION=0.750
	MEAN WORMS= 1.4	MEAN WORMS= 1.5
	VARIANCE= 1.917	VARIANCE= 1.500
	STD.DEV.= 1.385	STD.DEV.= 1.225
25.1—30	NO. OF FISH= 3.0	NO. OF FISH= 0.0
	NO. INFECTED= 2.0	NO. INFECTED= 0.0
	FRACTION=0.667	FRACTION=0.0
	MEAN WORMS= 1.3	MEAN WORMS= 0.0
	VARIANCE= 0.889	VARIANCE= 0.0
	STD.DEV.= 0.943	STD.DEV.= 0.0
30.1—35	NO. OF FISH= 7.0	NO. OF FISH= 1.0
	NO. INFECTED= 7.0	NO. INFECTED= 1.0
	FRACTION=1.000	FRACTION=1.000
	MEAN WORMS= 3.0	MEAN WORMS= 3.0
	VARIANCE= 0.571	VARIANCE= 0.000
	STD.DEV.= 0.756	STD.DEV.= 0.003
35.1—40	NO. OF FISH= 1.0	NO. OF FISH= 0.0
	NO. INFECTED= 1.0	NO. INFECTED= 0.0

1973

cm

TABLE 19 (continued)

o.c.	FRACTION=1.000 MEAN WORMS= 3.0 VARIANCE= 0.000 STD.DEV.= 0.003	FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0			
40.1–45	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0			
45+	NO. OF FISH= 1.0 NO. INFECTED= 1.0 FRACTION=1.000 MEAN WORMS= 4.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0			
	TOTAL FISH=204.0 INFECTED FISH= 55.0 FRACTION=0.270	TOTAL FISH= 81.0 INFECTED FISH= 14.0 FRACTION=0.173	).0 = 0.0	TOTAL FISH= 0.0 INFECTED FISH= 0.0 FRACTION=0.003	TOTAL FISH= 0.0 INFECTED FISH= 0.0 FRACTION=0.000

TABLE 20

Continued. Length of Freshwater-drum in Cm.

SIZE CLASS	FISH INFECTED WITH ENCAPSULATED WORMS SEPTEMBER	OCTOBER
0—5	NO. OF FISH= 5.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
5.0—10	NO. OF FISH=245.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 46.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
10.1—15	NO. OF FISH= 18.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 27.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
15.1—20	NO. OF FISH= 34.0 NO. INFECTED= 15.0 FRACTION=0.441 MEAN WORMS= 0.6 VARIANCE= 0.699 STD.DEV.= 0.836	NO. OF FISH= 6.0 NO. INFECTED= 2.0 FRACTION=0.333 MEAN WORMS= 0.7 VARIANCE= 1.222 STD.DEV.= 1.106
20.1—25	NO. OF FISH= 9.0 NO. INFECTED= 7.0 FRACTION=0.778 MEAN WORMS= 1.1 VARIANCE= 0.765 STD.DEV.= 0.875	NO. OF FISH= 9.0 NO. INFECTED= 8.0 FRACTION=0.889 MEAN WORMS= 2.0 VARIANCE= 1.778 STD.DEV.= 1.333
25.1—30	NO. OF FISH= 3.0 NO. INFECTED= 2.0 FRACTION=0.667 MEAN WORMS= 1.0 VARIANCE= 0.667 STD.DEV.= 0.816	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
30.1—35	NO. OF FISH= 3.0 NO. INFECTED= 2.0 FRACTION=0.667 MEAN WORMS= 0.7 VARIANCE= 0.222 STD.DEV.= 0.471	NO. OF FISH= 1.0 NO. INFECTED= 1.0 FRACTION=1.000 MEAN WORMS= 1.0 VARIANCE= 0.0 STD.DEV.= 0.0
35.1—40	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
40.1—45	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
45.1—50	NO. OF FISH= 1.0 NO. INFECTED= 1.0 FRACTION=1.000 MEAN WORMS= 1.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
	TOTAL FISH=318.0 INFECTED FISH= 27.0 FRACTION=0.085	TOTAL FISH= 89.0 INFECTED FISH= 11.0 FRACTION=0.124

TABLE 20

FISH INFECTED WITH ENCAPSULATED WORMS PAGE 3

SIZE CLASS	MAY	JUNE	JULY	AUGUST
0—5	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 9.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
5.1—10	NO. OF FISH= 2.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 5.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 3.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 84.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
10.1—15	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 24.0 NO. INFECTED= 1.0 FRACTION=0.042 MEAN WORMS= 0.0 VARIANCE= 0.040 STD.DEV.= 0.200	NO. OF FISH= 79.0 NO. INFECTED= 28.0 FRACTION=0.354 MEAN WORMS= 0.6 VARIANCE= 1.049 STD.DEV.= 1.024	NO. OF FISH= 55.0 NO. INFECTED= 16.0 FRACTION=0.291 MEAN WORMS= 0.4 VARIANCE= 0.418 STD.DEV.= 0.646
15.1—20	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 6.0 NO. INFECTED= 5.0 FRACTION=0.833 MEAN WORMS= 1.7 VARIANCE= 1.889 STD.DEV.= 1.374	NO. OF FISH= 25.0 NO. INFECTED= 16.0 FRACTION=0.640 MEAN WORMS= 1.0 VARIANCE= 0.998 STD.DEV.= 0.999	NO. OF FISH= 48.0 NO. INFECTED= 28.0 FRACTION=0.583 MEAN WORMS= 0.9 VARIANCE= 0.802 STD.DEV.= 0.895
20.1—25	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 8.0 NO. INFECTED= 6.0 FRACTION=0.750 MEAN WORMS= 1.4 VARIANCE= 0.984 STD.DEV.= 0.992	NO. OF FISH= 16.0 NO. INFECTED= 12.0 FRACTION=0.750 MEAN WORMS= 1.2 VARIANCE= 0.777 STD.DEV.= 0.882	NO. OF FISH= 12.0 NO. INFECTED= 4.0 FRACTION=0.333 MEAN WORMS= 0.7 VARIANCE= 1.056 STD.DEV.= 1.027
25.1—30	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 7.0 NO. INFECTED= 7.0 FRACTION=1.000 MEAN WORMS= 2.1 VARIANCE= 0.980 STD.DEV.= 0.990	NO. OF FISH= 14.0 NO. INFECTED= 12.0 FRACTION=0.857 MEAN WORMS= 1.4 VARIANCE= 1.102 STD.DEV.= 1.050	NO. OF FISH= 3.0 NO. INFECTED= 3.0 FRACTION=1.000 MEAN WORMS= 2.0 VARIANCE= 0.0 STD.DEV.= 0.0
30.1—35	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 3.0 NO. INFECTED= 3.0 FRACTION=1.000 MEAN WORMS= 2.0 VARIANCE= 0.667 STD.DEV.= 0.816	NO. OF FISH= 12.0 NO. INFECTED= 10.0 FRACTION=0.833 MEAN WORMS= 1.3 VARIANCE= 0.689 STD.DEV.= 0.829	NO. OF FISH= 1.0 NO. INFECTED= 1.0 FRACTION=1.000 MEAN WORMS= 2.0 VARIANCE= 0.0 STD.DEV.= 0.0
35.1—40	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 1.0 FRACTION=1.000 MEAN WORMS= 2.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 5.0 NO. INFECTED= 4.0 FRACTION=0.800 MEAN WORMS= 2.0 VARIANCE= 3.200 STD.DEV.= 1.789	NO. OF FISH= 1.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
40.1—45	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 1.0 NO. INFECTED= 1.0 FRACTION=1.000 MEAN WORMS= 1.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
45.1—50	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0	NO. OF FISH= 0.0 NO. INFECTED= 0.0 FRACTION=0.0 MEAN WORMS= 0.0 VARIANCE= 0.0 STD.DEV.= 0.0
	TOTAL FISH= 4.0 INFECTED FISH= 0.0 FRACTION=0.0	TOTAL FISH= 55.0 INFECTED FISH= 24.0 FRACTION=0.436	TOTAL FISH=155.0 INFECTED FISH= 82.0 FRACTION=0.529	TOTAL FISH=213.0 INFECTED FISH= 52.0 FRACTION=0.244

Length of Freshwater-drum in Cm.

of male philometrid nematodes but also a loss of post-fertilization females over the winter period. As mentioned earlier, there was apparently a density dependent factor operating in the eyes which comes into force as the post-fertilization adult female worms begin their period of greatest growth in the early spring. In our early spring samples both years we did find medium sized and fairly fresh encapsulations in the eyes. Sections of these encapsulations indicated that these were female worms.

Analysis of the Frequency of Monthly Occurrence of Philometra sp.

Much of the remainder of the findings section of this report will be devoted to an analysis of data for infections of freshwater-drum on a monthly basis. The occurrence of Philometra sp. parasitic in different size-classes are given for each month during the collecting periods of 1972, 1973, and 1974. A computer program was developed using FORTRAN language to compare this data in usable form. Tables 12, 13, and 14 for juvenile and young adult stages, 15, 16, and 17 for gravid, and larvigerous females and 18, 19, and 20 for encapsulations are the result. On each of these tables there is a box or group of figures for each size-class for each month of the year. Each box except the last one for each month is composed of five lines, each line is self-explanatory with perhaps the exception of the line reading "fraction," the fraction is the percentage of infection for that class in that month. Smaller and more concise tables were developed from each of the larger ones giving the essential data for purposes of discussion.

Tables 21, 22, 23, 24, 25, 26, 27, 28 and 29 were derived from Tables 12, 13 and 14.

TABLE 21
INTENSITY OF INFECTION OF FISH WITH
JUVENILE AND YOUNG ADULT WORMS (Mean Worm Burden)-1972

Size Class cm	June	July	August	Sept.	October
0.0-5	0	0	0	7.8	17.0*
5.1-10	0	0	0	4.8	5.4
10.1-15	0	0	0	0.5	11.2
15.1-20	0	0	0	0.0	0.5

*Indicates a single infection

Analysis of Table 21 indicates that freshwater-drum from 0 through 20 centimeters long harboured juvenile stages and that these stages were present in the fish only in September and October of 1972. This correlates exactly with frequency distributions presented earlier. This Table also illustrates that, in general, as the fish increase in size the mean worm burden decreases. These freshwater-drum would be of the 0+, 1+ and 2+ age classes and the 0+ and 1+ age classes carried the greatest mean burden of Philometra juveniles.

TABLE 22
INTENSITY OF INFECTION OF FISH WITH JUVENILE AND YOUNG ADULT WORMS (Mean Worm Burden)-1973

Size Class cm	Mar	May	June	July	Aug	Sept	Oct
0.0-5	0	0	0	0	0	0	0
5.1-10	0	0	0	0	0.7	0.5	0.3
10.1-15	0	0	0	0	0.8	1.9	1.2
15.1-20	0	0	0	0	0.1	0.1*	0

*Indicates single infection

TABLE 23
INTENSITY OF INFECTION OF FISH WITH JUVENILE AND YOUNG ADULT (Mean Worm Burden)-1974

Size Class cm	June	July	August	Sept.	October
0.0-5	0	0	0	0	0
5.1-10	0	0	9.0	0.3	0.5
10.1-15	0	0	0	0.6	1.1
15.1-20	0	0	0	0	0
20.1-25	0	0	1.1	0	0

Tables 22 and 23 show that the infections occurred earlier in 1973 and 1974 as compared with 1972 (Table 21) and fish infected were larger in size. This reflects greater dispersion of fish hosts during 1973 and 1974. However, the freshwater-drum infected were still 0+, 1+ and 2+ age class fishes and the greatest burden still occurred in fish of the smaller sizes.

TABLE 24
INCIDENCE (PERCENTAGE) OF FISH INFECTED WITH JUVENILE WORMS-1972

Size Class cm	June	July	August	September	October
0.0-5	0	0	0	83.3%	100.0%*
5.1-10	0	0	0	84.0%	83.0%
10.1-15	0	0	0	23.1%	97.1%
15.1-20	0	0	0	1.8%	14.9%

*Indicates single infection

TABLE 25
INCIDENCE (PERCENTAGE) OF FISH INFECTED WITH JUVENILE WORMS-1973

Size Class cm	Mar	May	June	July	Aug	Sept	Oct
0.0-5	0	0	0	0	0	0	0
5.1-10	0	0	0	0	11.6%	25.0%	18.8%
10.1-15	0	0	0	0	22.3%	43.7%	43.1%
15.1-20	0	0	0	0	6.3%	3.2%	0

TABLE 26
INCIDENCE (PERCENTAGE) OF FISH INFECTED
WITH JUVENILE AND YOUNG ADULT WORMS - 1974

Size Class cm	June	July	August	September	October
0.0-5	0	0	0	0	0
5.1-1-	0	0	7.4%	9.8%	21.7%
10.1-15	0	0	0	27.8%	33.8%
15.1-20	0	0	0	0	0
20.1-25	0	0	1.3	0	0

Tables 24, 25, and 26 correlate well with Figures 48 through 60. Inspections show that fish 5.1-15 cm in length had the greatest incidence of infection with juvenile and young adults in all years 1972, 1973, and 1974. The infections were greater in October than in September. The smaller fish also carried the greatest worm burdens (Tables 21, 22 and 23.) It is these size classes of fish which feed most readily on plankton. In fish larger than 15 cm the incidence and intensity of juveniles and young adults declines very rapidly. Most size classes had a lower incidence of juveniles and young adults in 1974. These three tables, and the three tables which follow (Tables 27, 28 and 29) show that the population was more dispersed in 1973 and 1974, this correlates with the summary Figures 67 through 73. In general worm burden and incidence (percentage of infection) was lower in 1973 and 1974.

TABLE 27
TOTAL MONTHLY INCIDENCE OF INFECTION OF ALL FISH OF ALL SIZE CLASSES FOR 1972, JUVENILE AND YOUNG ADULT WORMS

June	July	August	September	October
0	0	0	39.8%	61.0%

TABLE 28
TOTAL MONTHLY INCIDENCE OF INFECTION OF ALL FISH OF ALL SIZE CLASSES FOR 1973, JUVENILE AND YOUNG ADULT WORMS

Mar.	May	June	July	August	September	October
0	0	0	0	9.7%	24.0%	30.9%

TABLE 29
TOTAL MONTHLY INCIDENCE OF INFECTION OF ALL FISH OF ALL SIZE CLASSES FOR 1974, JUVENILE AND YOUNG ADULT WORMS

June	July	August	September	October
0	0	0.01%	19.0%	29.0%

Tables 27, 28, and 29 show the total monthly incidence of infection of freshwater-drum with juvenile and young adult philometrid nematodes. It is clear that as in the earlier scatter-diagrams, graphs and tables these stages do not occur until mid-August and that they are present through October with increasing frequency. This is probably because of a cumulative affect as these stages feed on infected copepods and the time required for migration of larval worms to the eyes. It is also demonstrated in these three tables that with the greater dispersion of the philometrid population that there was a lower total incidence of infection in 1973 and 1974 than 1972. Since these are the worms which overwinter in the eyes and grow to gravid and larvigerous females the next spring and summer some predictions can be made from data collected concerning them each year. In the 1973 Annual Report, it was predicted that there would be a lower population of gravid and larvigerous females in the spring and summer of 1974. This prediction proved to be true. Caution, however, should be exercised in making such predictions, for water temperatures and overcrowding may also influence the number of females which eventually stream from the eyes.

Tables 30, 31, 32, 33, 34 and 35 were derived from data presented in Tables 15, 16 and 17.

TABLE 30
MONTHLY INTENSITY OF INFECTION OF FRESHWATER-DRUM SAMPLES IN 1972. MEAN BURDEN OF GRAVID AND LARVIGEROUS FEMALE WORMS

Size Class* cm	June	July	August	Sept	October
10.1-15	2.6	1.5	0.4	0	0
15.1-20	0.8	0.5	0.0	0	0
20.1-25	0.1	0.1	0.0	0	0

*All other size classes were negative or had only single infections.

TABLE 31
MONTHLY INTENSITY OF INFECTION OF FRESHWATER-DRUM SAMPLES IN 1973.
MEAN BURDEN OF GRAVID AND LARVIGEROUS FEMALE WORMS

Size Class* cm	Mar	May	June	July	Aug	Sept	Oct
0.0–5	0	0	0	0	0	0	0
5.1–10	0	0	0	1.6	0	0	0
10.1–15	0	0	4.9	2.6	0.2	0	0
15.1–20	0	0.1	0.2	0.4	0.1	0	0
20.1–25	0	0.5	0	0.1	0	0	0

*All other size classes were negative or had only single infections.

TABLE 32
MONTHLY INTENSITY OF INFECTION OF FRESHWATER-DRUM SAMPLES IN 1974.
MEAN BURDEN OF GRAVID AND LARVIGEROUS FEMALE WORMS

Size Class cm	June	July	August	September	October
0–5	0	0	0	0	0
5.1–10	0.4	0.3	0	0	0
10.1–15	1.2	0.8	0.2	0	0
15.1–20	0	0.1	0.1	0	0
20.1–25	0.1	0.1	0.1	0	0
35.1–40	0	0.2	0	0	0

These three tables also correlate exactly with all graphs of philometrid stages in this report.

Study of Tables 30, 31 and 32 demonstrates that in all years freshwater-drum 10 through 25 cm harboured the vast majority of adult, gravid female worms in the months of May, June, July and the first two weeks in August. Fish 10 to 20 cm long carried the greatest mean worm burdens. Very few fish over 20 cm in length were even infected. After the first two weeks in August the gravid, adult females had streamed from the eyes, thus in most cases the intensities were less in July than in June. Again the dispersion of adult, gravid females was greater in 1973 and 1974 than 1972 and there were heavier worm burdens in larger fish in 1973. Indeed the fish hosts may have grown more rapidly in 1973.

TABLE 35
MONTHLY INCIDENCE (PERCENTAGE) OF FRESHWATER-DRUM SAMPLED INFECTED WITH GRAVID AND LARVIGEROUS FEMALE WORMS, 1974

Size Class cm	June	July	August	September	October
0.0-5	0	0	0	0	0
5.1-10	40.0%	33.0%	1.2%	0	0
10.1-15	45.8%	36.7%	1.8%	0	0
15.1-20	0	08.0%	4.2%	0	0
20.1-25	12.5%	06.3%	08.3%	0	0
35.1-40	0	20.0%	0	0	0

These three tables also correlate with the graphs presented earlier and they correlate closely with Tables 30, 31, and 32. These tables display incidence of infections. The same spring and summer months were involved as far as gravid and larvigerous female philometrid nematodes were concerned. The highest percentages of infection by the adult females each month were in fish 10 to 20 cm in length and the percentages, in general, decrease in longer fish. In 1973 and 1974, more longer fish autopsied were infected than in 1972. Again as shown by these tables the number of adult gravid females generally decreases June to July to August.

TABLE 36
TOTAL MONTHLY INCIDENCE OF INFECTION OF ALL FISH OF ALL SIZE CLASSES FOR 1972, GRAVID AND LARVIGEROUS FEMALE WORMS

June	July	August	September	October
51.4%	34.4%	8.4%	0	0

TABLE 37
TOTAL MONTHLY INCIDENCE OF INFECTION OF ALL FISH OF ALL SIZE CLASSES FOR 1973, GRAVID AND LARVIGEROUS FEMALE WORMS

March	May	June	July	August	September	October
0	7.7%	14.4%	32.4%	7.2%	0	0

TABLE 38
TOTAL MONTHLY INCIDENCE OF INFECTION OF ALL FISH OF ALL SIZE CLASSES FOR 1974, GRAVID AND LARVIGEROUS FEMALE WORMS

May	June	July	August	September	October
0	25.5%	21.9%	06.6%	0	0

These tables show that incidence decreases June, July to August when the total percentages of infection are considered. This was expected as females begin their streaming in July. The exception to this general picture was the statistic for June 1973 (Table 37.) Our sample size here was large enough but it was biased toward larger fishes, 15.1-25 cm in length. The open lake trawls and Cold Creek samples did not yield many smaller fish during June 1973. We were aware of this and during July we moved to more shallow water off Locust Point and began to recover smaller fish (Table 37.) This phenomenon did not occur in 1972 and 1974. Comparison of the three tables does show a lower incidence of infection with adult worms in 1973, and this was followed, as shown earlier, with a lower incidence of juveniles in 1973.

Encapsulation for the months of the years 1972, 1973, and 1974 are displayed in Tables 18, 19 and 20 respectively. Smaller freshwater-drum, 5 to 15 cm, usually do not have encapsulations until after August since they have been infected for the first time and encapsulations are present only after the adult, gravid, female worms have streamed from the eyes. The incidence and intensity of encapsulations is greatest in fish 15 cm or longer, fish which are 1+ age class or older. The encapsulations persist and are present in high percentages of fish up to 35 cm in length, 2+, 3+, 4+ and 5+ age classes throughout the entire year. Encapsulations exist in even older and longer fish but they are much more scattered and the incidence and intensities are much lower. Reinfections can and do occur, living worms were recovered from eyes which also had encapsulations, but these are most prevalent in 1+ and 2+ age class fish. We have inferred from this that encapsulations persist for at least four years and perhaps longer within the eyes of fish which recover from infections.

Figures 67, 68, and 69 are summary graphs, they are based on the total average percentages of all size classes of fish infected with the three life history phases.

Figure 67

PHILOMETRA SP. IN APLODINOTUS GRUNNIENS

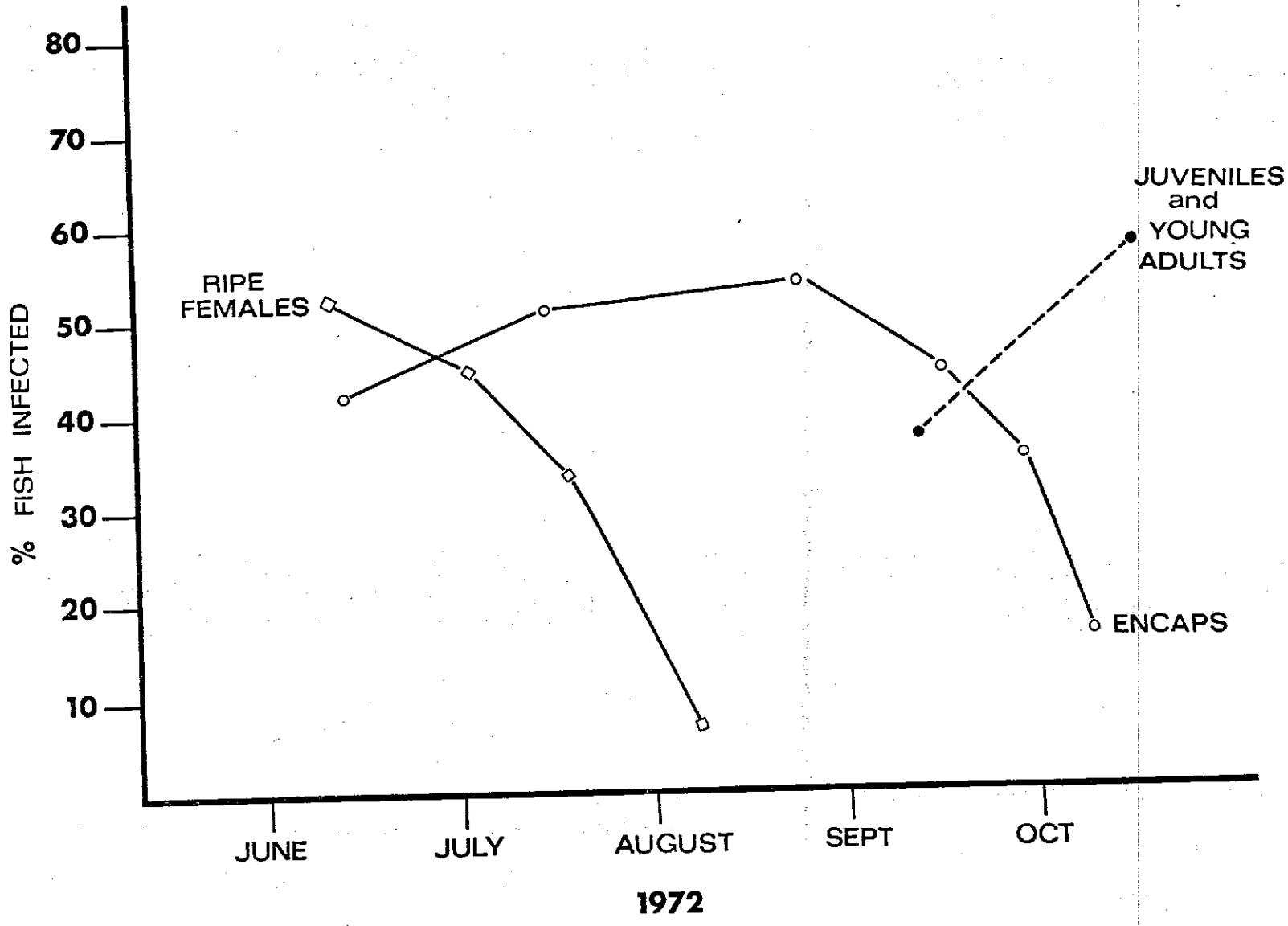


Figure 68

PHILOMETRA SP. IN APLODINOTUS GRUNNIENS

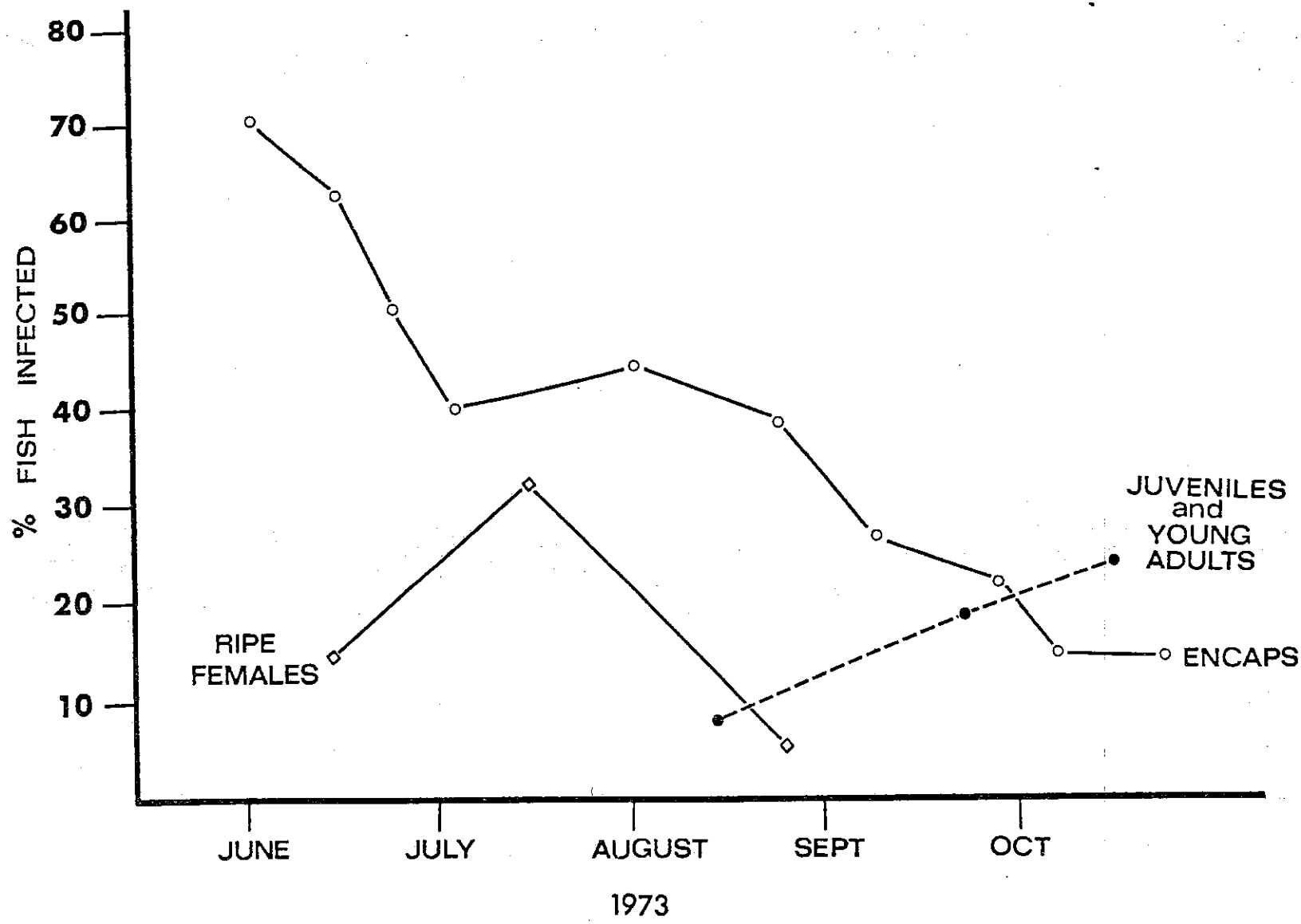


Figure 70

% PHILOMETRA INFECTION VS. FISH SIZE

1972

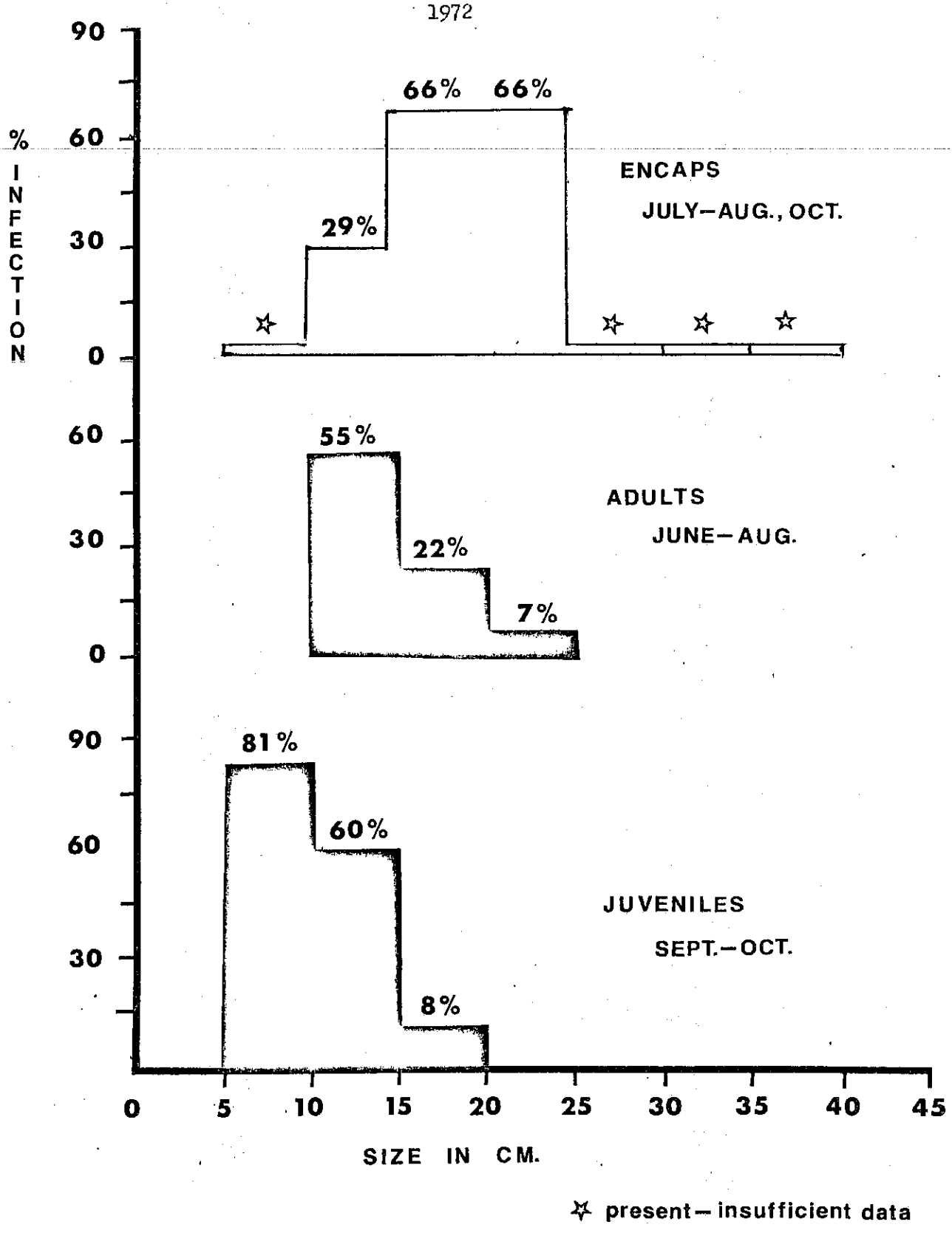


Figure 71

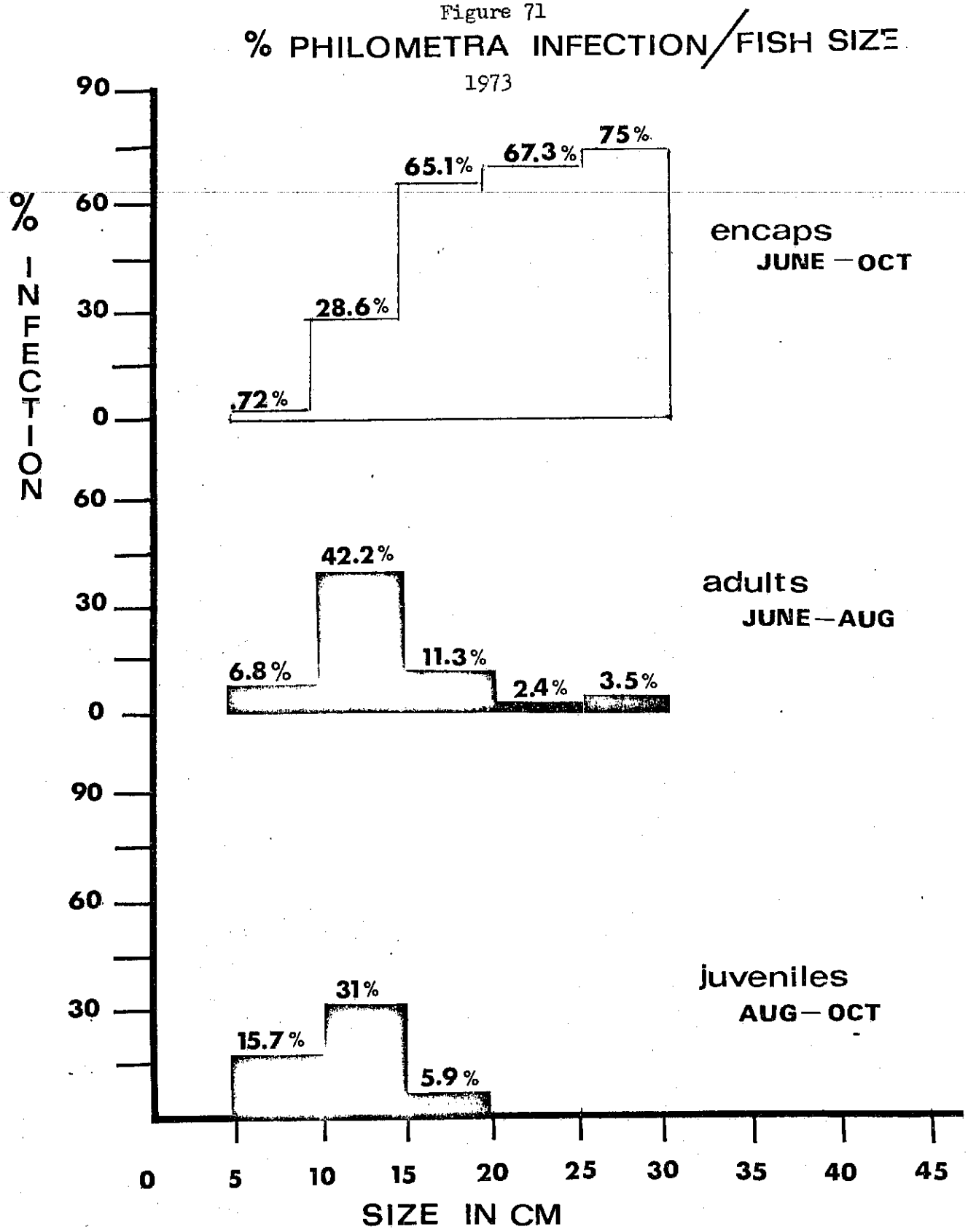
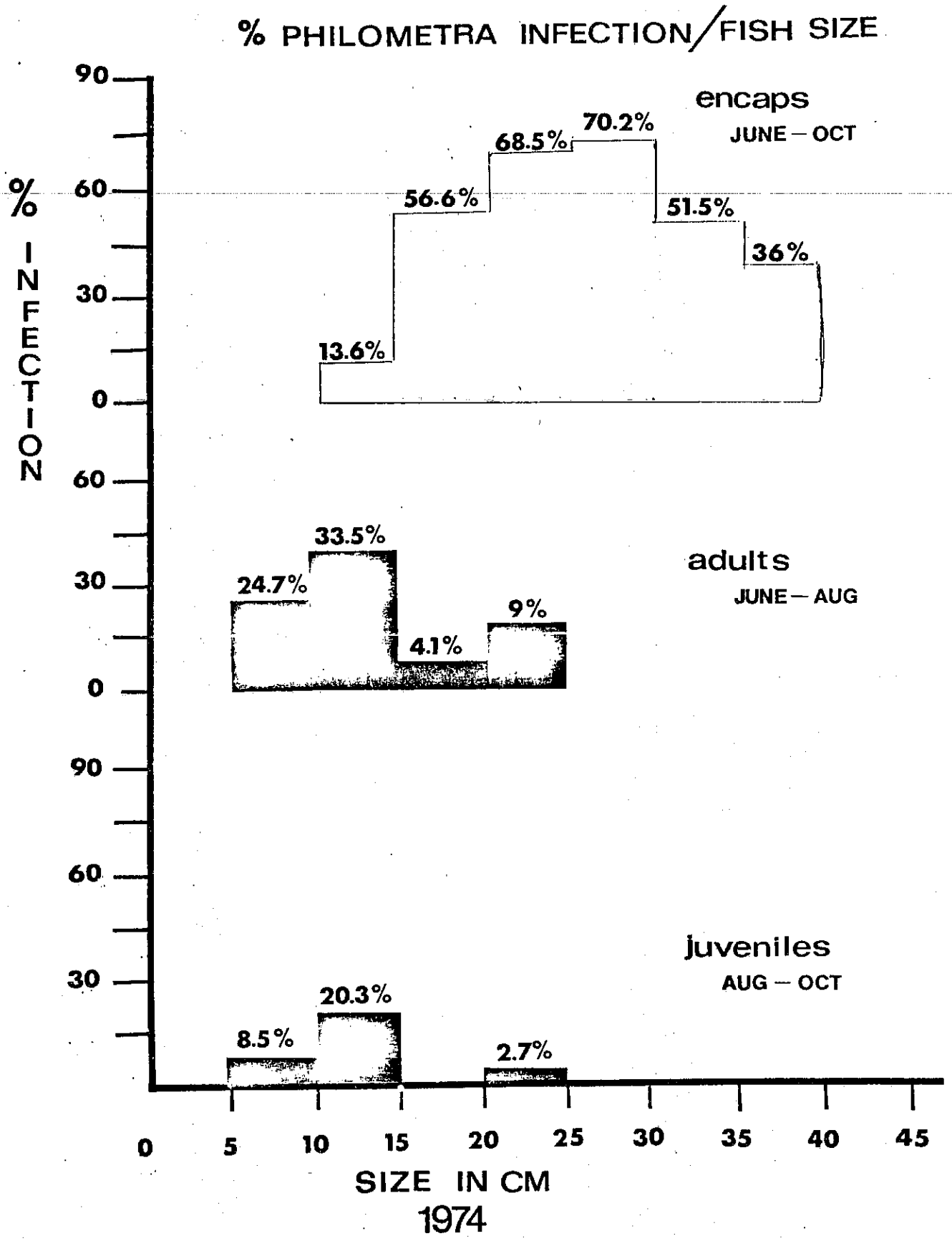

% PHILOMETRA INFECTION/FISH SIZE
1973
% INFECTION
90
60
30
0
60
30
0
90
60
30
.72%
28.6%
65.1%
67.3%
75%
encaps
JUNE — OCT
6.8%
42.2%
11.3%
2.4%
3.5%
adults
JUNE — AUG
15.7%
31%
5.9%
juveniles
AUG — OCT
0
5
10
15
20
25
30
35
40
45
SIZE IN CM

Figure 72


% PHILOMETRA INFECTION/FISH SIZE
% INFECTION
90
60
30
0
60
30
0
90
60
30
encaps
JUNE – OCT
13.6%
56.6%
68.5%
70.2%
51.5%
36%
adults
JUNE – AUG
24.7%
33.5%
4.1%
9%
juveniles
AUG – OCT
8.5%
20.3%
2.7%
0
5
10
15
20
25
30
35
40
45
SIZE IN CM
1974

dependence which operates in each infected eye. One must also recall that each gravid, adult female worm which survives will stream from the eye and release 70,000+ first stage larvae into the lake water. All stages in the eye of the fish host are pathogenic. Over-dispersion with the resultant lower mean worm burden may lead to a continued success of philometrid worms with less harm to the fish host, ecologically and evolutionarily a successful situation.

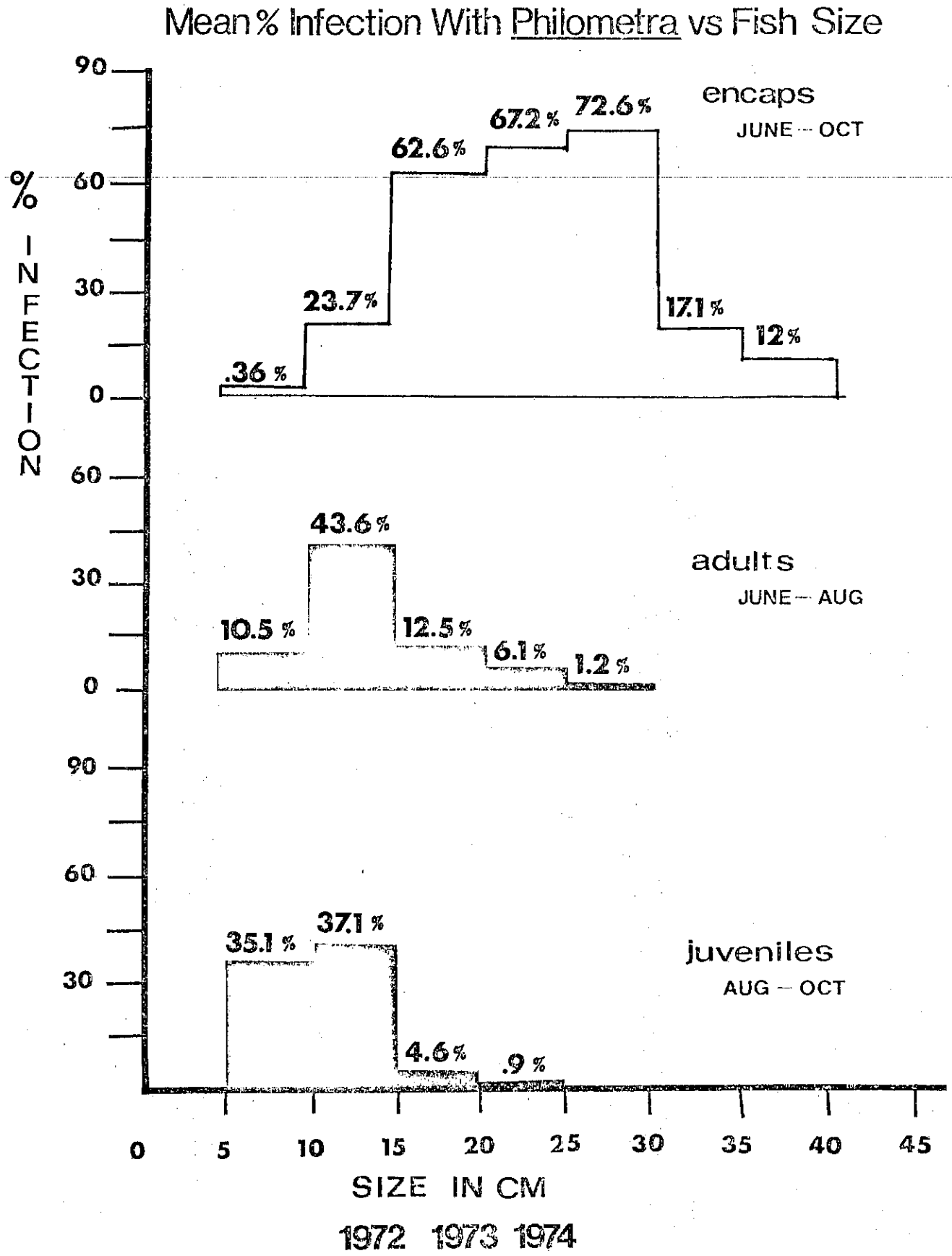
Consideration of these factors could be important in fisheries management of freshwater-drum. What would be the results if small freshwater-drum were stocked in small ponds, which warm more quickly, stay warm longer and have high populations of cyclopoid copepods throughout the year and are often present with predaceous fishes? This parasite would probably be spread to other waters. In more confined situations under the conditions stated above it could maintain itself readily and some years it might have a serious affect on stocked freshwater-drum populations.

Figures 70, 71, 72 and 73 illustrate material already discussed in the tables and scatter-diagrams. Figures 70, 71 and 72 are summary graphs of the years 1972, 1973 and 1974 respectively. Figure 73 is a summary graph showing the average incidence of infection in different sizes of freshwater-drum with the different life history stages of Philometra sp. for the three years combined.

These graphs illustrate that the majority of gravid and larvigerous females occurred in freshwater-drum 5 to 20 cm in length during June and July. They also show that the majority of juveniles and young adults occur in freshwater-drum 5 to 15 cm in length during September and October. The highest percentages of infection occurred during 1972 but in general the pattern of infection is clear and it is similar throughout the years of study. It would seem from the material presented in the entire report and summarized in Figures 70, 71, 72 and 73 that the most propitious time for shipping uninfected freshwater-drum from Lake Erie would be in the autumn after the last week in August. The fish could be left in the waters of destination over winter. If the fish taken were a minimum of 20 cm in length the probability of shipping uninfected fish would be greatly lessened. If freshwater-drum are to be shipped from Lake Erie in the spring the fish should be at least 20 cm long and there are still distinct probabilities that some infected freshwater-drum would be transported.

The statistics, descriptions and analyses presented here confirm our opinions given in reports throughout the course of this study, and they indicate some problems for further investigations.

Figure 73


Mean % Infection With Philometra vs Fish Size
% INFECTION
90
60
30
0
encaps
JUNE -- OCT
.36 %
23.7%
62.6%
67.2 %
72.6 %
17.1 %
12 %
adults
JUNE -- AUG
10.5 %
43.6 %
12.5 %
6.1 %
1.2 %
juveniles
AUG -- OCT
35.1 %
37.1 %
4.6 %
.9 %
0 5 10 15 20 25 30 35 40 45
SIZE IN CM
1972 1973 1974

RECOMMENDATIONS

The analyses of data from the three years of this study provide much of the knowledge needed to understand the basic biology of the nematode parasite, Philometra sp., infecting the eyes of Aplodinotus grunniens in Lake Erie. The information concerning the pathology, transmission cycle, seasonal distribution of developing and mature stages, and the population biology furnish a foundation for recommendations and future studies.

When freshwater-drum are shipped alive from Lake Erie the infection is likely to spread, at present it is known only from Lake Erie. If freshwater-drum from Lake Erie are used to stock small eutrophic ponds containing other predaceous fishes they may suffer sizeable losses unless care is taken concerning the size of fish utilized and perhaps the time of the year when they are stocked. Freshwater-drum should be at least 20 cm long if they are to be taken alive from Lake Erie and used for stocking, even at this length there is a slight probability of shipping infected fishes. If possible, and when feasible in keeping with other management practices, these fish should be taken after the first three weeks in August. Fish at least 20 cm long taken at this season would have the lowest probability of being infected.

Within Lake Erie the situation is different from that described above. While this nematode is pathogenic and may account for as much as a 16 percent loss to "young of the year" freshwater-drum in western Lake Erie it is well adapted and some of the fish survive the infection. Despite losses, there is always a margin of uninfected 0+ class fish in the freshwater-drum population which develop either before or after the nematode larvae are found in the copepod intermediate hosts. These fish do not become infected. The population of Philometra sp. and the populations of freshwater-drum are fairly balanced in Lake Erie under normal conditions. This does not mean that infections with this nematode do not cause harm or that they could not reach epidemic portions if there are temporary environmental changes in certain years or if conditions change in the future in a manner which favors the transmission of the nematode. Some monitoring might be worthwhile particularly if Lake Erie becomes subject to more thermal pollution.

Small 0+ and 1+ class freshwater-drum should be selected with care to make sure that they have no signs of the infection syndrome if they are to be used for experiments, bio-assays or thermal studies. In this study tests demonstrated that freshwater-drum

exhibiting the exopthalmic syndrome are at least temporarily blinded and some are permanently blinded. These fish react abnormally to light. Freshwater-drum taken in the spring and early summer from Lake Erie containing developing worms in the eyes react differently in a thermal gradient.

The information resulting from this study has been organized into several manuscripts which are now in preparation for publication or in print. These publications will be concerned with the following subjects: (1) a formal description of this species which we plan to name Philometra banghami; (2) the transmission cycle and life history; (3) the morphogenesis of Philometra sp. in freshwater-drum; (4) the histopathology and pathological effects in the eyes and other tissues of freshwater-drum; (5) the behavior of the free-living first stage larvae and (6) the population biology. For comparative purposes a manuscript redescribing the female and describing the male of Philometra cylindracea from the yellow perch in Lake Erie has been submitted for publication and accepted (Ashmead and Crites, 1975).

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Area Research

Approved By: _____
Federal Aid Coordinator

Date: 31 July 1975

Date: _____

FEDERAL AID IN SPORT FISH RESTORATION

FINAL REPORT

STATE OF:	Ohio
PROJECT NO.:	F-48-R-1, 2, 3
PERIOD COVERED:	June 1, 1972 - May 31, 1975
PROJECT TITLE:	Impact of Parasitic Worms on Lake Erie Fishes
STUDY NO:	II
STUDY TITLE:	Impact of Camallanus oxycephalus on Lake Erie Fishes
PREPARED BY:	Paul C. Stromberg and John L. Crites
DATE:	31 July 1975

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ABSTRACT

The study of the impact of Camallanus oxycephalus involved four areas of intensive investigation; 1) identity of the parasite, 2) life history, 3) pathology and 4) ecology and interactions of the population in the western Lake Erie system. The identity of the parasite was determined by study and redescription of numerous adult specimens. The life history of the parasite was studied experimentally and in the natural environment. The intermediate host is Cyclops bicuspidatus and Cyclops vernalis. Although many species of aquatic arthropods eat the larvae, only copepods may transmit the infection. The efficiency of infection of copepods is very high. Fish may become infected by eating copepods with the infective larvae. Large predatory species may also acquire the infection by eating smaller infected fish.

The first stage larvae may live for 24 - 39 days at normal extreme summer water temperatures. The ability to infect the copepod decreases rapidly. No penetration occurs after 18 days at 25°C. The larvae are also relatively intolerant to NaCl solutions. Survival was significantly altered at 1% NaCl and only lasted 3 days at 3% NaCl. Free-living larvae are very active and experiments indicated that this activity "attracts" copepods. Penetration of the copepod gut was primarily a mechanical phenomenon.

No pathogenesis was observed which could be directly attributed to the worms. The intestinal mucosa is penetrated and the nematodes feed on blood and tissue. Mechanical injury, especially in heavy infections, could lead to secondary invasion of pathogens and fish death. Extremely heavy infections might also kill fish.

Analysis of past records strongly indicates that this parasite has become more abundant in western Lake Erie. This may be due, in part, to changes in the plankton and benthic communities. The great increase in prevalence coincides with the rise in abundance of gizzard shad and alewife. Both fish are infected with C. oxycephalus, particularly shad, and transmit the disease to fish that eat them.

The population biology and ecology of C. oxycephalus in western Lake Erie was intensively investigated in white bass. The parasite is distributed as a negative binomial through all segments of the fish population in all seasons. No difference exists between infections of male and female fish. Larger fish generally are more heavily infected. Approximately 80 - 90% of all adult bass are infected. The worms live for only one year and die in July when larvae are dispersed. The heaviest infections are found in July and August when most worms are larvae. The mean worm burden decrease from August to the following

July. This reduction in the worm population is believed to be caused by worm mortality which may be related to the population density of the parasite. Growth of females is arrested over winter, then proceeds at a very rapid rate during April, May and June. Male and female nematodes copulate in autumn but eggs are not fertilized until April. Larvae are released simultaneously. Dispersal in the population occurs during the maximum density period of the copepod intermediate host. Relative success of each parasite generation may be effected by the abundance of copepods, gizzard shad and alewife, water temperature and other factors affecting the timing of parasite transmission.

BACKGROUND

Camallanus oxycephalus is a common and widespread nematode in fishes in eastern North America. It occurs in 52 species of fishes, most of which are cypriniform and perciform. This parasite has been reported from as many as 26 species in Lake Erie. Since its first report in Lake Erie in 1927, it has apparently attracted much attention from sportsmen and commercial fishermen. During late spring and summer, these worms, which are bright red in color, protrude from the rectum of fish and are readily observable. Fish infected with this parasite are unsightly and discourage fishermen.

In a preliminary study of C. oxycephalus in western Lake Erie, we were impressed with the great abundance of this worm. Some populations of fishes, such as white bass and crappies were 100% during some seasons and often very heavily infected. Comparison of our findings with previous studies suggested that the prevalence of C. oxycephalus has increased sharply in recent years. In addition, our data also suggested that the intensity of infection increased as well.

The great abundance of this parasite and the accurate information available to us concerning the history of this species in Lake Erie presented a unique opportunity to study this parasite population and its interaction with Lake Erie fish. Because white bass were common, heavily infected and easily collected, much of our work centers around this fish. Other fish, however, were studied, to accurately assess the host-parasite interactions in the Lake Erie system. The gathering of information on pathology, transmission, life history, morphology abundance and population ecology would allow us to construct certain hypotheses about how this population of parasites maintains abundance in western Lake Erie. Such insights might develop into generalizations and useful information concerning parasite communities in aquatic systems. This information could be applied to management practices

to control the spread of harmful parasitic species in general and of C. oxycephalus in particular.

OBJECTIVES

This very broad and inclusive study of a host-parasite interaction was divided into three jobs, each with specific objectives. These jobs were entitled: 1) descriptive investigations, 2) experimental investigations and 3) population investigations. Under job one, our first objective was to describe accurately the species Camallanus oxycephalus (Ward and Magath, 1916). Although this worm has been commonly collected for about 60 years, its specific description has been poor, making identification nearly impossible. Also under job one, we planned to describe any evidence of disease associated with the attachment of this worm to host gut tissue. The experimental aspects of this study revolved about the transmission pathways of the parasite, its life history and development and an analysis of the biology of the dispersal agent and its interaction with the lake environment. The third job is broadly defined. It includes the gathering of information on the incidence, intensity of infection and seasonality of the parasite and correlating this data with information gathered from Jobs I and II in order to construct hypotheses about population regulation, transmission and factors contributing to parasite success in western Lake Erie.

PROCEDURES

This project was in residence at the Franz Theodore Stone Laboratory at Put-in-Bay, Ohio from June through mid-September. During the last part of September, the laboratory was moved to the parasitological laboratory in the zoology department of The Ohio State University at Columbus. The project worked out of Columbus for the remainder of the year. Fish for the study were obtained from 14 locations in western Lake Erie and Sandusky Bay. They were collected by otter trawl, commercial trap nets, commercial shore seines, gill nets, fyke nets, minnow seines and angling. Young-of-the-year fish for experimental procedures were taken with dip nets, minnow seines and shallow water otter trawl, maintained in laboratory holding tanks and treated with Nox-Ich and Maracyn to prevent disease. These fish were fed frozen minnows and chopped earthworms. Invertebrate animals for experimental work were obtained with plankton nets and dip nets. Copepods were maintained in 3 gal. canisters and fed Paramecium and brine shrimp.

First stage larvae of C. oxycephalus were maintained in filtered lake water and stored in constant temperature cabinets at 15°, 20°, and 25°C. Fresh larvae were offered to various invertebrates in finger bowls. Quantified infection experiments with copepods were conducted by exposing 10 larvae to a single copepod in 2 ml of lake water for 24 hrs. Development of the larval stages of C. oxycephalus was studied every 24 hrs. by sacrificing infected copepods and studying living worms.

Fish were infected with Camallanus in several ways. Some young-of-the-year white bass and yellow perch were anesthetized with Quinaldine (.025 ml/3 gal) and infected with a stomach tube. Infected copepods were also exposed to minnow fry and these fry exposed to young fish, passing the infection along. Development of Camallanus within white bass and yellow perch was studied by sacrificing fish every 48 hours and studying living and preserved worms. Drawings were made with the aid of a drawing tube mounted upon a wild microscope.

Population data was gathered by sampling white bass, yellow perch and freshwater drum bi-weekly. Data on fish size, sex, season, incidence, intensity of infection, site selection and structure of worm population was kept. Approximately 6,600 worms were measured and body volume calculated. In addition, 13 other species of fishes were surveyed for Camallanus. Data from all fishes were keypunched on computer cards and analyzed using standard statistical programs on the IBM 370/165 at Ohio State University. Plankton samples were taken 3 times per week between May and September with a 2 l Kemmerer water bottle.

White bass intestines containing Camallanus adults and larvae were fixed for histological examination by placing them in alcoholic Bouin's for at least 24 hours. Bouin's was washed from the tissue with 70% ethanol. Tissue was dehydrated and embedded in paraplast and sectioned at 6 - 10 microns. Sections were stained with Mallory's triple stain and Hematoxylin and Eosin.

FINDINGS

Intermediate Host Determination and Larval Development

Gravid female nematodes were placed in lake water, allowed to rupture and active, first-stage larvae were collected and exposed to various aquatic invertebrates (Table 1). Although many of these arthropods ate the larvae, penetration through the gut wall and migration

TABLE 1

EXPERIMENTAL DETERMINATION OF THE INTERMEDIATE HOST

Potential Host	Larvae Eaten	Larvae Penetrated
Gammarus	+	–
Hyalella	+	–
Asellus	+	–
Cyclops	+	+
Diaptomus	+	+
Daphnia	–	–
Bosmina	–	–
Chironomus	+	–
Cricotopus	+	–
Stenonema	–	–
Ostracoda	+	–

to the hemocoel occurred only in copepods. Active larvae were frequently observed moving about in amphipod and midge larvae guts, but no penetration occurred. Larvae were allowed to remain with these arthropods for several days and specimens dissected periodically but no nematodes were found in any animals except copepods.

When penetration occurred, it was usually within 2 hours. Larvae thrashed vigorously in the copepod gut, punched a hole in the gut wall and slipped into the hemocoel quickly. First-stage larvae remain active within the hemocoel for several days, but become quiescent. Shortly after penetration, the anterior portion of the thin-walled esophagus thickens, becoming more distinct. The posterior portion becomes lined with cells indistinguishable from the intestinal cells. The intestinal wall becomes thicker with more cells and the lumen becomes wider and straightens out. The genital primordium remains unchanged.

The first molt occurs on the third day post infection at 25°C and on the fifth day post infection at 20°C. The molt is readily observed at the posterior end of the larva. The stoma of the second-stage larva is round and the minute dorsal spine is absent.

A great change in the anterior end of the worm occurs during development of the second-stage larva. The buccal cavity inflates, laterally, forming the buccal valves, and the esophagus moves posteriorly. The stoma changes from a circular opening to a dorso-ventral oval. The anterior part of the esophagus becomes muscular and the

posterior portion differentiates from the intestine.

The second molt occurs on the sixth day after infection at 25°C and on the tenth day after infection at 20°C. This molt can be easily detected because the third-stage larva has 3 mucranes on the tail. The stoma is dorso-ventrally elongated but no circumoval papillae are present. The buccal capsule is composed of two weakly sclerotized lateral valves, pale yellow in color, and divided into two chambers. The esophagus is distinctly divided into an anterior muscular portion and a posterior glandular portion. The entire worm has a distinct orange color.

The third-stage larvae lie coiled and inactive in the copepod hemocoel. If the copepod contains only one or two worms, they are usually found dorsal to the gut. The larvae are immediately infective after the third molt. Infected copepods were placed in 1 ml of 1) Ringer's Solution, 2) Pepsin-HCl + Ringer's and 3) Fish Bile + Ringer's in separate depression slides to determine what released escape activity of the larvae. Copepods died quickly in each solution. Larvae remained coiled and motionless in Ringer's Solution for several hours. Immersion in the pepsin-HCl solution caused some larval activity after 35 minutes and continued for 3 hours, but larvae remained coiled. The bile solution caused larval activity within 5 - 10 minutes. Larvae uncoiled and moved about so violently that it caused flexing of the entire dead copepod body. These larvae moved throughout the body pushing their heads into appendages and lashing vigorously about the hemocoel. No larvae escaped, although activity continued for 3 hours. Some infected copeopds were placed in pepsin-HCl for 1 hour, then in the bile solution. A high percentage of larvae escaped from these copepods through the tail.

Spottail shiner fry (Notropis hudsonius) were collected with a dip net and exposed to infected copepods in large finger bowls for three days. Dissection of these fish revealed developing third-stage Camallanus larvae. Shiner fry exposed to infected copepods were placed into a 15 gal aquarium with 5 yearling yellow perch (Perca Flavescens). The perch were observed feeding upon the shiner fry. All the perch were sacrificed after 5 days and 4 of these fish contained developing third-stage larvae. All control fish were free of infection.

Third-stage larvae from fish are morphologically similar to larvae from copepods except they are larger (maximum length - 1.241 mm as opposed to .671 mm). The third molt occurs 9 - 10 days after infection. The most striking change following this molt is in the buccal capsule. Its color is a deep bronze, the ridges are longitudinal and the two chambers are fused into a single one. Five pairs

of circumoval papillae occur around the stoma. The tail has three small mucrones.

The final molt occurs at different times for the male and female. The male fourth-stage larvae reach a maximum length of 1.90 mm and molt on the seventeenth or eighteenth day after infection at 26°C. The female larvae attain a maximum length of 2.60 mm and molt 24 days after infection. Adult worms have dorsal and ventral tridents associated with the buccal capsule. The tail is rounded and smooth, without mucrones.

The experimental determination of the life history elucidates several important points on the transmission of Camallanus oxycephalus. One, infective first stage larvae must be eaten by the intermediate host. Transmission of this parasite depends, in part, upon the trophic dynamics of this host. Two, the intermediate host may only be a copepod. Although many aquatic arthropods eat these larvae, development to the stage infective to fish occurs only in copepods. This is a relatively important finding because transmission of the parasite can be narrowed to the interaction between the plankton community and fish. The parasite contacts the fish populations through a single route. Three, the infection may be passed from one fish to another. This, too, is a significant finding of this study. The ability to be transferred in such a manner provides a zone of overlap between plankton feeding fish and fish eating fish, thus widening the potential range of hosts. Many fish, far removed from plankton feeding, are open to infection if they feed upon forage fish. It is likely that the large sport fish, such as white bass, black bass, yellow perch, walleye, northern pike, drum, crappies and rockbass become infected by eating infected forage species.

Summary of the Experimental Life History (Fig. 1)

1. Ovoviviparous females exposed to lake water when fully gravid rupture and release 7,000 - 10,000 infective first-stage larvae.

2. Active first-stage larvae are eaten by a variety of aquatic arthropods, but penetration through the gut wall into the hemocoel occurs only in copepods.

3. There is little observable development until the first molt, which occurs 3 - 5 days after infection.

4. Many morphological changes begin to take place in the second-stage larva. Most notably, the buccal capsule begins to form.

5. The second molt occurs 6 - 10 days after infection.

6. Third-stage larvae are distinct. The buccal capsule is divided into two chambers, the tail terminates in three mucrones and living worms are bright orange in color.

7. Escape activity of the third-stage larvae is stimulated by bile. Actual escape may depend in part on the softening of the copepod exoskeleton by pepsin and HCl.

8. Infection may be acquired by large fish either by directly ingesting infected copepods or eating smaller fishes carrying the worms. The parasite may be passed from plankton to plantivorous fish to piscivorous fish in this manner.

9. The third molt occurs on the eighteenth day after infection. Fourth-stage larvae have a one chambered buccal capsule, circumoral papillae and three mucrones on the tail.

10. The final molt occurs on the eighteenth day after infection for males and 24 days after infection for females. Adult nematodes have dorsal and ventral tridents associated with the buccal capsule and a smooth, blunt tail.

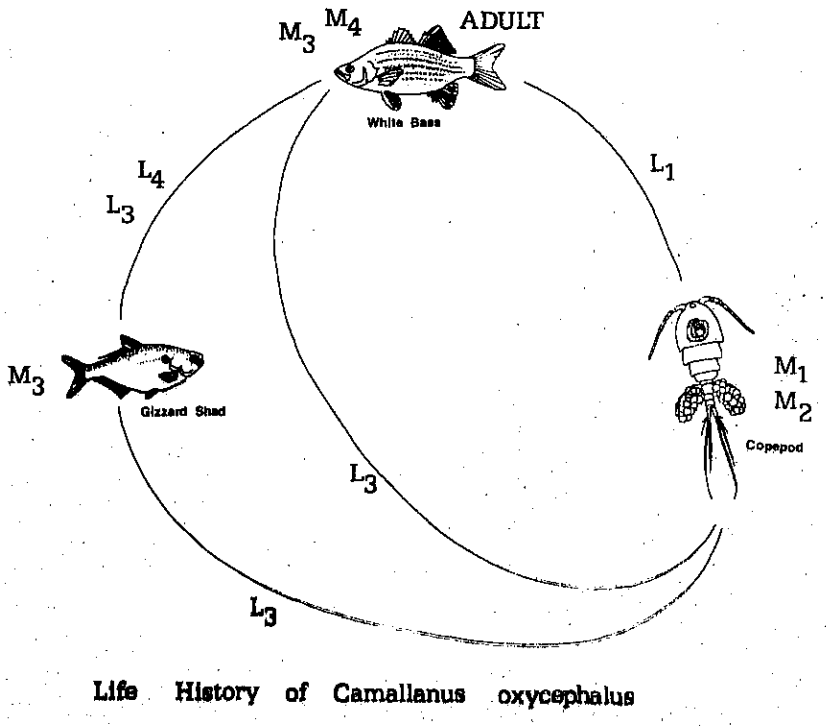

Figure 1. Life History of Camallanus oxycephalus.

Biology of the Dispersal Agent

Because the ability of the first-stage larva to infect the intermediate host may be affected by its interaction with the environment, a series of experiments were conducted with the larvae to determine the nature of the interaction and its effect on infectivity. Among the most important are the effect of temperature on survival, the relationship between infectivity and age, salinity tolerance and the relationship between larval activity and consumption rate by the intermediate host.

To determine a basal efficiency of infection, copepods (Cyclops bicuspidatus, C. vernalis, Diaptomus sp.) were exposed to freshly released infective larvae of Camallanus oxycephalus. Table 2 summarizes these experiments.

TABLE 2

INFECTION OF COPEPODS BY FIRST-STAGE LARVAE

Copepod	No. L, exposed	No. L, eaten	No. L, penetrated
Cyclops	800	508 (.635)	360 Pe = .709
Diaptomus	108	1 (.009)	0

It is clear that cyclopoid copepods ingest Camallanus larvae much more readily than do calanoid copepods. This is probably related to the feeding habits of the copepods, since cyclopoids are predacious and scavengers, while calanoids are filter feeders. Although we were able to produce infections in calanoid copepods, these experiments allow us to confine epidemiological considerations of the intermediate host to the dynamics of cyclopoid copepod populations. The experiments also revealed a very high efficiency of infection (Pe) by the dispersal agent. This high efficiency is an important factor in the local abundance of C. oxycephalus in western Lake Erie.

Survival of the infective dispersal agent was definitely affected by temperature. Figure 2 shows the survivorship curve determined at 20°C and 25°C. These temperatures were selected because they approximate the extreme water temperatures in western Lake Erie during June and July when larvae are in the water. Larvae survived 39 days at 20°C but only lived 24 days at 25°C. More than half of

the larvae lived 28 days at the lower temperature, while 50% survival was reached between 17 and 18 days at the higher temperature. Not only did the larvae survive for a longer period of time at 20°C but the decline in survival was more gradual than at 25°C. Figure 3 shows the effect of NaCl concentration on larval survivorship. A 1% solution of NaCl was clearly detrimental to larvae, significantly decreasing their survival. Higher concentrations of NaCl reduced survival more rapidly. Most larvae exposed to 3% NaCl were crenated and exhibited abnormal twitching after 24 hours.

Although the larvae survived 39 days and 24 days in filtered lake water at 20° and 25° respectively, the ability to penetrate the copepod gut wall was lost before the larvae died (Figs. 2 and 4). The penetration efficiency decreased logarithmically with the age of the larvae. The decrease was rapid at both temperatures. No larvae penetrated the gut wall after 18 days at 25°C. Penetration efficiency had not reached 0 when the 20° experiment was terminated at 28 days.

The initial rate of larval activity was high. Fresh, undisturbed larvae exhibited a mean rate of 130 undulations per minute. The decline in spontaneous activity was linear and the slope was -8.55 (Fig. 5). Spontaneous activity ceased after 16 days at 25°C. The mean excitatory rate was consistantly higher than the mean spontaneous rate, but was maintained for less than 1 minute after the larvae were disturbed. The maximum excitatory rate was 142 undulations per minute, slightly less than a 10% increase over the spontaneous rate. The slope of the excitatory line (-7.61) was nearly equal to that of the spontaneous line. No excitatory activity could be elicited after 18 days.

The relationship between the mean spontaneous activity rate and the number of larvae eaten by Cyclops (Fig. 6) was a positive linear function. Statistical analysis of the regression coefficient indicated that this was a highly significant relationship ($F = 8.06$, $P < .01$). This suggests that active larvae are more likely to be eaten by copepods than sluggish or inactive larvae. The expenditure of energy in maintaining a constant movement by the larvae, then appears "ecologically justified" since this movement apparently attracts copepods and significantly increases the probability of intermediate host infection. The conservation of energy resources by Camallanus larvae, for prolonged survival at a low activity rate does not seem an effective tactic because larvae liberated into the plankton fall rapidly through the water column and are probably lost in the sediments. In addition, cyclopoid copepods are abundant for only several weeks during the year. Thus, copepods and infective dispersal agents overlap temporally for only a short time, and there would be little selective advantage in prolonged survival of larvae.

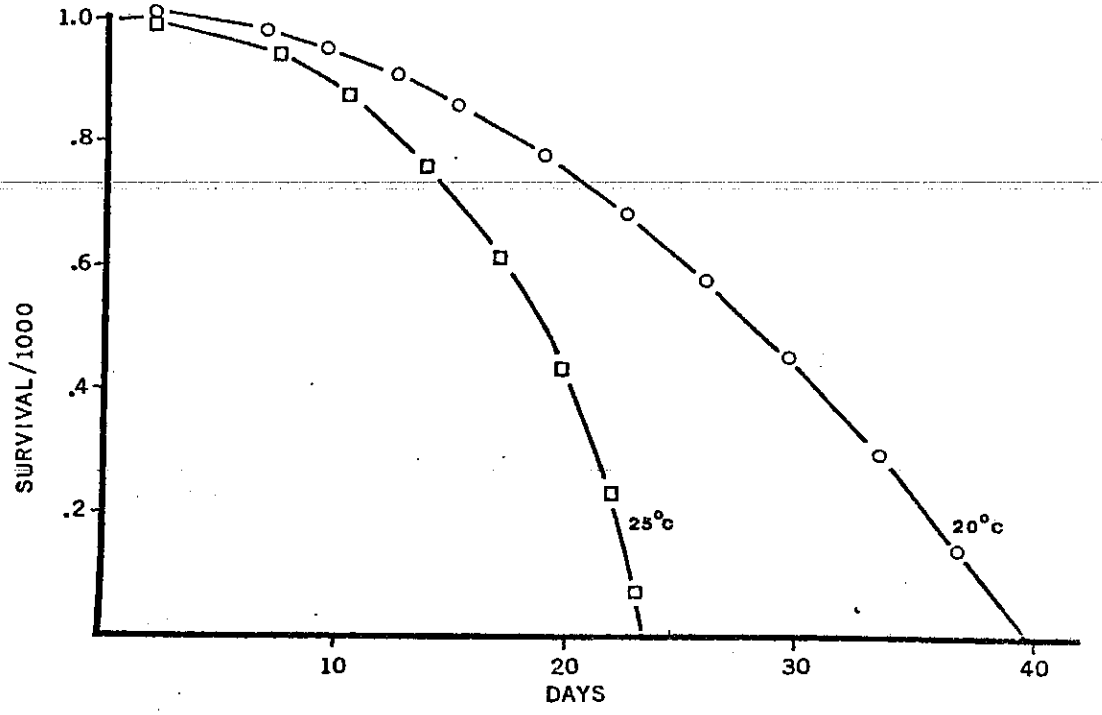

Figure 2. Survival of first-stage larvae of C. oxycephalus at 20°C and 25°C

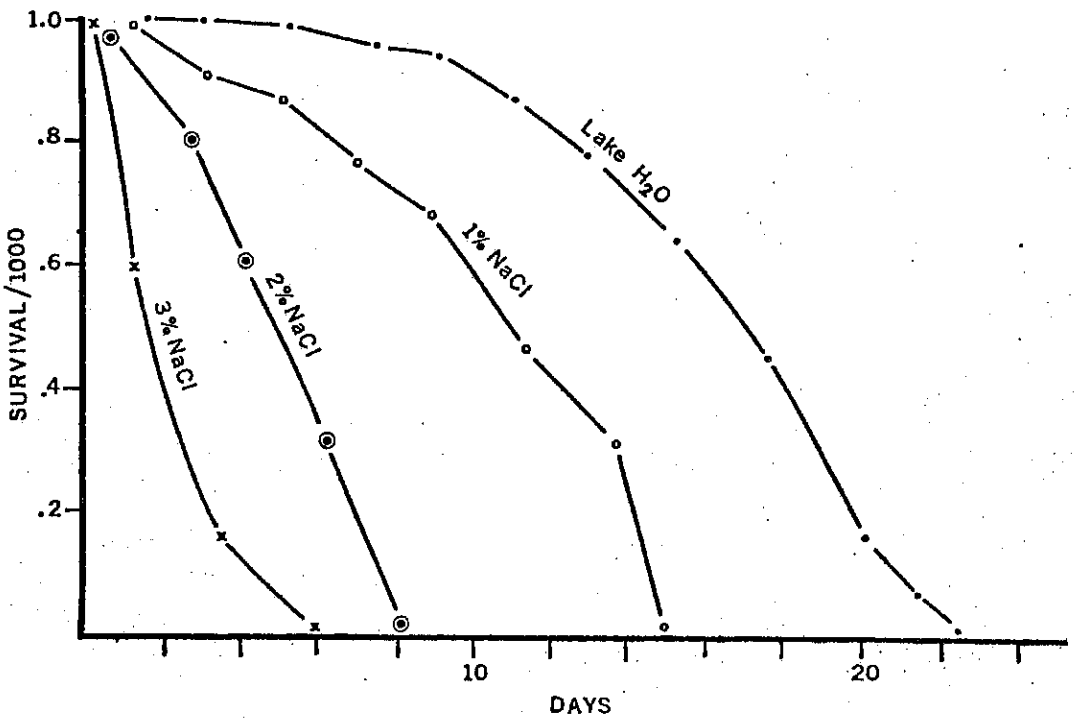

Figure 3. Survival of first-stage larvae of C. oxycephalus in saline solutions at 25°C

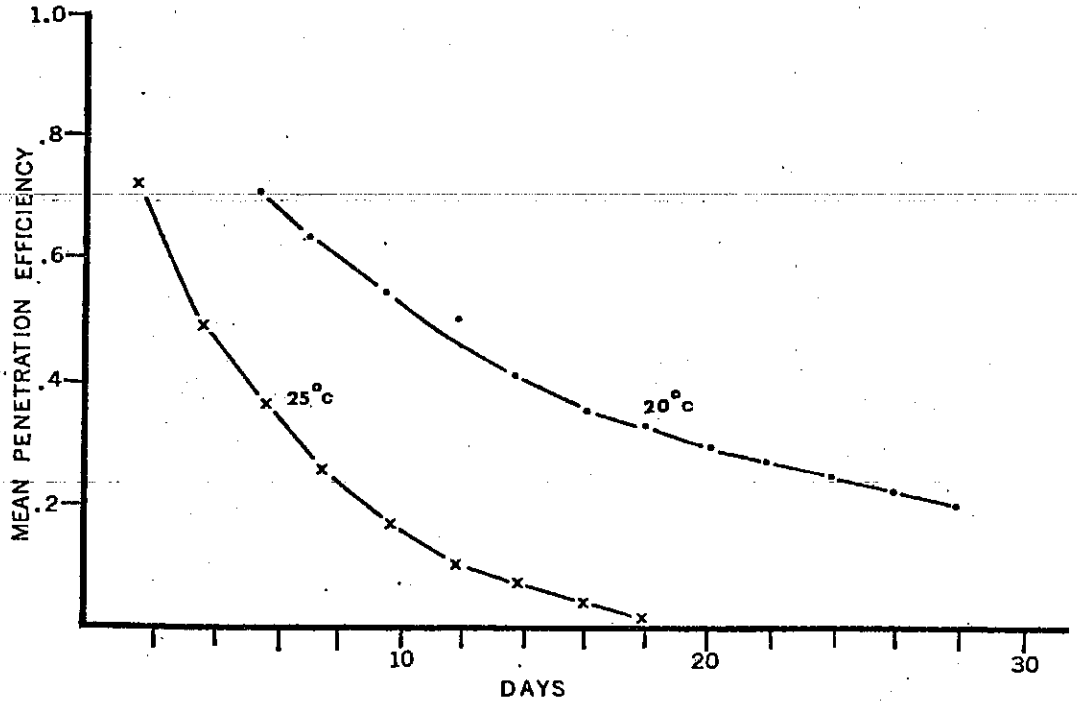

Figure 4. Relationship between penetration efficiency and age of C. oxycephalus larvae at 20°C and 25°C

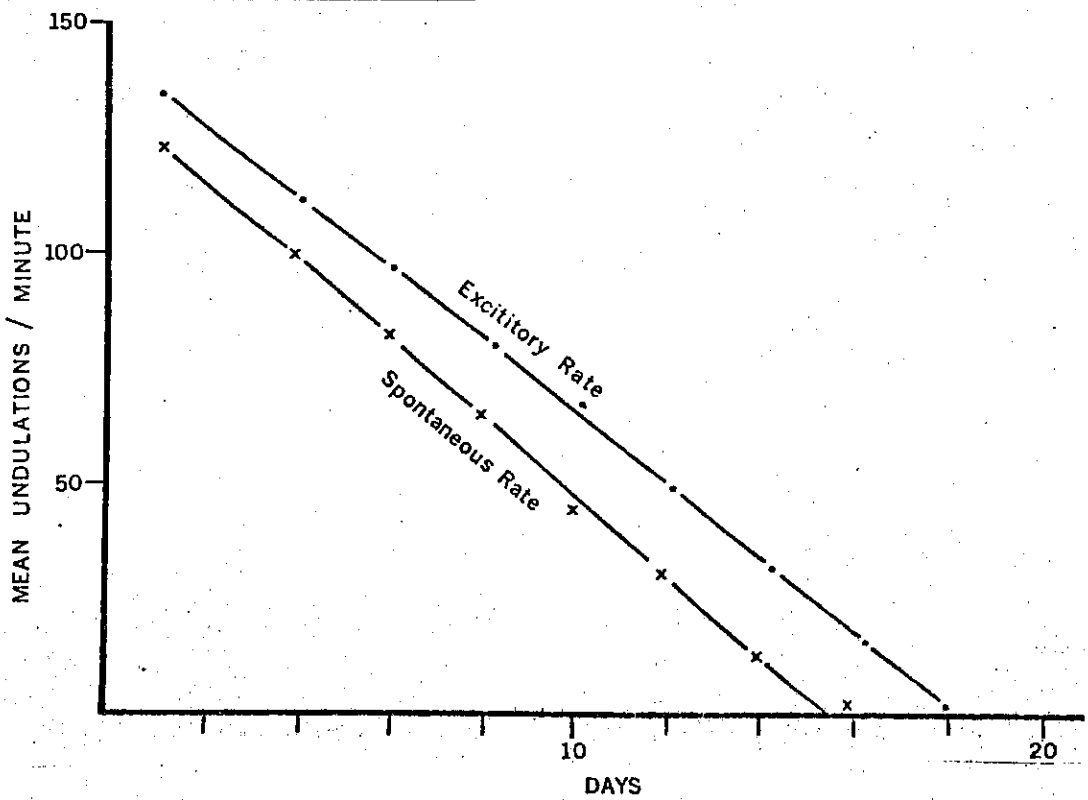

Figure 5. Relationship between rate of activity and age of first-stage larvae of C. oxycephalus at 25°C

Because the rate of activity declines linearly while the penetration efficiency declines logarithmically (Figs. 4 and 5), it was initially thought that larval activity alone did not account for penetration of the copepod gut wall. Analysis of the log transformation values for penetration efficiency and activity and their relationship (Fig. 7), however, yielded a highly significant correlation coefficient ($t = 4.22$; $P < .01$). This indicates that larval activity is the most important factor in penetration of the copepod gut wall.

Pathogenicity in Fish

Repeated analyses of the histopathology associated with adult Camallanus oxycephalus has not revealed any definite, significant disease in white bass. The nematodes clearly penetrate the rectal mucosa, grasp the submucosa and feed upon blood and tissue fluid. The reaction to this invasion is minimal and there is limited damage associated with it. Although no heavy infections were studied histologically, it is unlikely that there is any significant structural damage caused by Camallanus. The minimal structural damage to the rectal mucosa, however, might be sufficient to allow secondary invasion of normal bacterial flora and subsequent disease. There are other cases on record of parasite-disease complexes caused in this manner. This was not investigated, but might be the subject in future investigations of any white bass mortality.

Changes in the Prevalence of C. oxycephalus in Lake Erie

Seventeen species of fishes were examined for C. oxycephalus in western Lake Erie. Sixteen of these fishes were found to be parasitized. These data were compared to the previous findings of Bangham and Hunter (1939) and Bangham (1957) to ascertain if any trend could be seen in the abundance of this parasite. Figures 8 - 11 illustrate the nature and magnitude of the frequency of infection in important Lake Erie fishes. Tables 3 and 4 show the precise frequency values of Camallanus in Lake Erie fish among the three studies and the statistical analysis of the changes in prevalence.

It is clear immediately that the frequency of occurrence of C. oxycephalus was quite high in many of the 1972 fish. Highest frequencies occurred in predatory species such as Morone chrysops, Pomoxis spp., Stizostedion vitreum. Low frequencies occurred in plankton feeders with the exception of Dorosoma cepedianum and young-of-the-year drum (Aplodinotus grunniens).

A comparison of the prevalence of C. oxycephalus between 1927 and 1957 (Table 4) reveals a significant decrease in three species; drum, troutperch (P. omiscomaycus) and spotfin shiner (N. spilopterus).

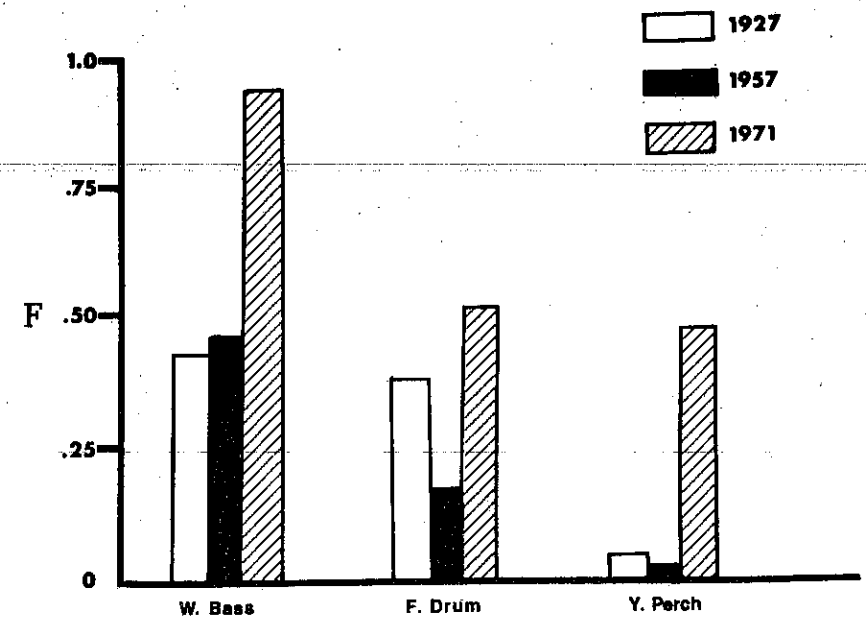

Figure 8. Frequency of C. oxycephalus in Lake Erie fish

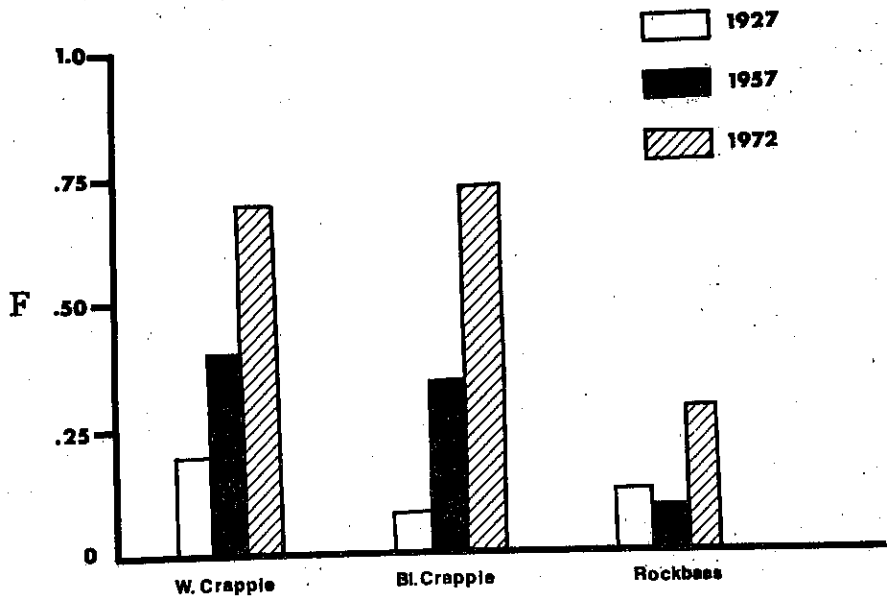

Figure 9. Frequency of C. oxycephalus in Lake Erie fish

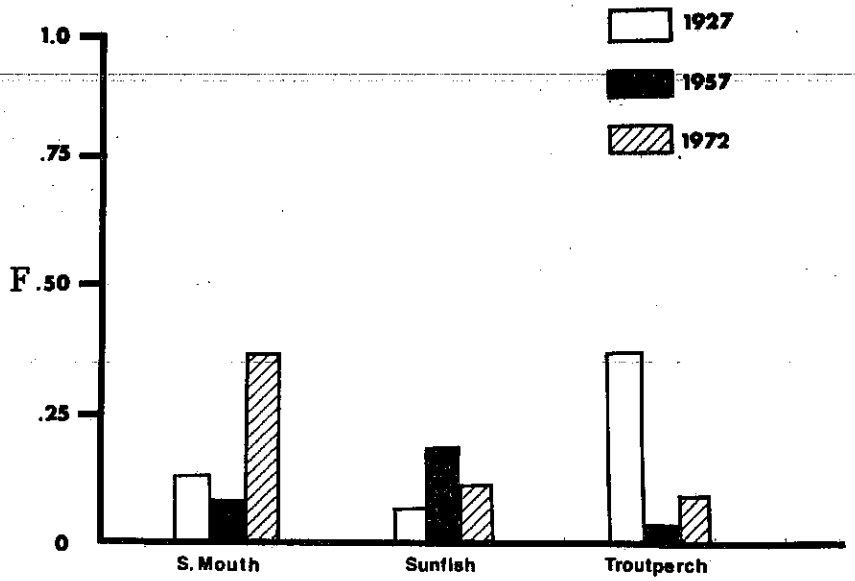

Figure 10. Frequency of C. oxycephalus in Lake Erie fish

Figure 11. Frequency of C. oxycephalus in Lake Erie fish

TABLE 3

CHANGES IN THE FREQUENCY OF *Camallanus oxycephalus* IN LAKE ERIE FISHES 1957 - 1972

Fish	1957 N	1957 F	1972 N	1972 F	z
Morone chrysops (Adult)	53	.472	83	.952	+6.233**
M. chrysops (YOY)	--	----	170	.641	
Aplodinotus grunniens (Adult)	88	.143	67	.507	+4.727**
A. grunniens (YOY)	--	----	300	.500	
Perca flavescens (Adult)	93	.054	114	.475	+8.816**
P. flavescens (YOY)	--	----	64	.016	
Pomoxis annularis	53	.396	48	.708	+3.270**
P. nigromaculatus	29	.310	37	.730	+3.428**
Ambloplites rupestris	75	.107	22	.273	+1.983**
Lepomis gibbosus	58	.069	28	.107	+0.606
Micropterus dolomieui	55	.078	40	.375	+3.574**
Stizostedion vitreum	33	.212	13	.691	+3.092**
Percopsis omiscomaycus	63	.032	13	.077	+0.761
Ictalurus punctatus	39	.026	57	.157	+2.047**
Notropis hudsonius	77	.013	60	0	-0.619
N. atherinoides	39	0	240	.011	+0.523
N. spilopterus	68	.015	24	.167	-2.923**
Osmerus mordax (YOY)	61	0	50	.040	+1.633
Dorosoma cepedianum (YOY)	27	0	360	.533	+5.333**
Alosa pseudoharengus (YOY)	14	0	190	.121	+1.388

N - sample size; F - frequency of infection; ** denotes significant z-value

TABLE 4

CHANGES IN THE FREQUENCY OF *Camallanus oxycephalus* IN LAKE ERIE FISHES 1927 - 1957

Fish	1927 N	1927 F	1957 N	1957 F	z
Morone chrysops (Adult)	32	.469	53	.472	-0.006
M. chrysops (YOY)	9	.220	--	----	
Aplodinotus grunniens (Adult)	45	.400	88	.143	-2.888**
Perca flavescens (Adult)	45	.022	93	.054	+1.000
P. flavescens (YOY)	15	0	--	----	
Pomoxis annularis	17	.231	53	.396	+1.231
P. nigromaculatus	9	.111	29	.310	+1.192
Ambloplites rupestris	12	.116	75	.107	-0.090
Lepomis macrochirus	10	.100	74	.311	+1.384
L. gibbosus	23	.043	58	.069	+0.441
Micropterus dolomieui	80	.125	55	.078	-0.855
M. salmoides	24	.041	40	.175	+1.576
Stizostedion vitreum	48	.104	33	.212	+1.403
Etheostoma/Percina	93	.161	127	.102	-0.418
Percopsis omiscomaycus	46	.369	63	.032	-4.746**
Ictalurus punctatus	29	.034	39	.026	-0.178
Notropis hudsonius	83	.036	77	.013	-0.958
N. atherinoides	81	.012	39	0	-0.706
N. spilopterus	49	.101	68	.015	-2.300**

N - sample size; F - frequency of infection; ** denotes significant z-value

The prevalence of the worm remained essentially unchanged in 14 species of fish between 1927 and 1957. A study of the 1957 and 1972 data, however, shows a significant increase of Camallanus in 11 species of Lake Erie fish (Table 3). The prevalence of Camallanus remained unchanged in 6 species and declined in none. It is apparent that C. oxycephalus has become more common in Lake Erie since 1957. Troutperch were the only fish to exhibit a decline in prevalence of the parasite since 1927. Although no data for young-of-the-year white bass are available for 1957, direct comparison of the 1972 data with the 1927 values revealed a significant increase in Camallanus ($z = +2.586$). A similar comparison of yellow perch showed that although adult fish are more frequently parasitized, the frequency in young-of-the-year has not changed significantly ($z = +.498$).

In addition to the increase in frequency of infection of white bass, the data suggest that the intensity of infection has also risen. Only 18% of the 1927 white bass carried more than 10 worms each, while 45% of the 1972 adult white bass had at least 10 worms. Thus, the mean worm burden in the white bass population appears to have increased.

The magnitude of the changes in prevalence of C. oxycephalus in the fish community as a whole is illustrated in Fig. 12. This figure compares the sequence of infection frequencies ordered from highest to lowest for each year studied. The numbers do not correspond to specific fish, so that the changes in specific fish is not illustrated. Rather, the graph represents the shift in abundance of the parasite population. It is clear from this figure that no significant change in prevalence of C. oxycephalus occurred in the fish community between 1927 and 1957. The curves for these two years are characterized by relatively low frequencies of infection, sloping gradually from a high of about .47 to .01. The 1972 curve represents a significant increase of great magnitude in the prevalence of the parasite. Several groups of fish can be defined based upon infection frequency. One species (white bass) occurs at the .90 level, three species (white crappie, black crappie, walleye) occur around .70 and three species (freshwater drum, gizzard shad and yellow perch) occur around .50. The 1972 curve declines sharply after these seven high frequency values through two intermediate frequencies (.40 - .20). The remaining seven values are low, occurring around or below .15. The fish exhibiting the low frequencies of infection are all plankton feeders except the catfish and pumpkinseed. In contrast, the 1927 - 1957 curves have only a single species in the high frequency group (ca. .50), three or four species in the intermediate group and the majority (12) in the low frequency group.

Based upon this analysis of the prevalence of Camallanus in 16

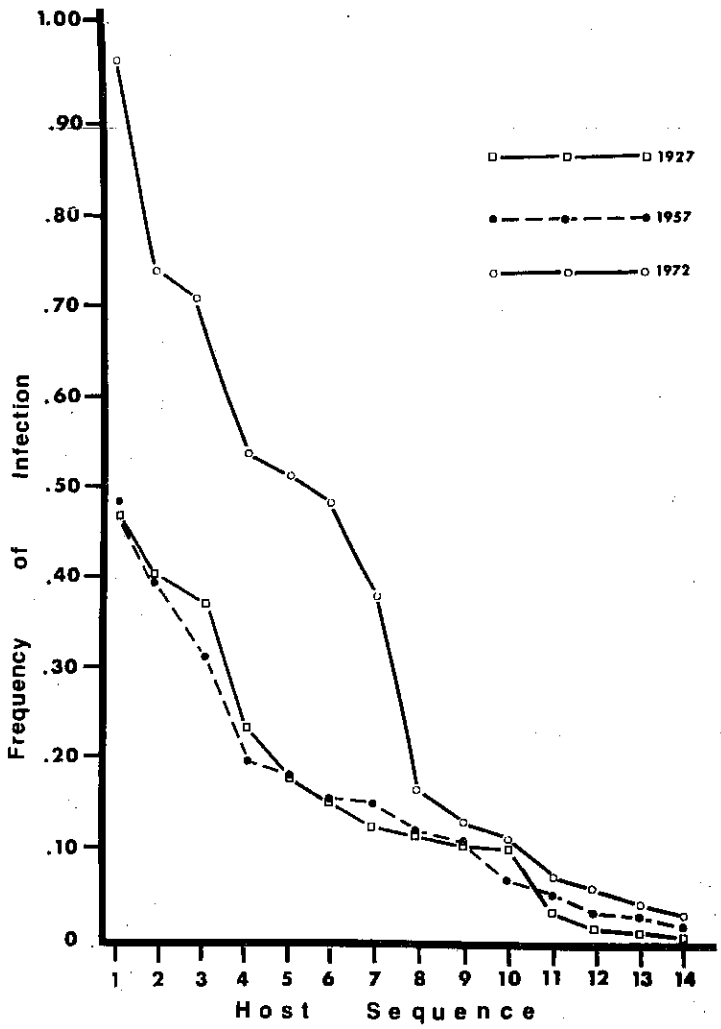

Figure 12. Comparison of the Prevalence of C. oxycephalus in Lake Erie in 1927, 1957 and 1972

species of Lake Erie fish, the parasite appears to be far more abundant than in past years. In addition, this analysis suggests that the shift upward has occurred since around 1957. Only troutperch of all the fish examined, had a lower frequency of infection with Camallanus in 1972 than in 1927. Nine of the 11 species with higher frequencies of infection are predatory species which include a significant portion of fish in their diet. All 6 species with unchanged infection frequencies are either plankton feeders or consume benthic arthropods.

Because the increase in the prevalence of C. oxycephalus in western Lake Erie has been largely in piscivorous fish, it seems likely that the increase has been caused by a rise in infected forage fish. The ability of C. oxycephalus to be carried from forage fish to predatory fish was demonstrated experimentally. The discovery of a high frequency of Camallanus in gizzard shad was a particularly significant finding in this regard. Although this fish was known from Lake Erie as early as 1848, Miller (1957) pointed out that it has only become abundant in Lake Erie since the 1950's. Bodola (1964) noted the rapid rise of gizzard shad especially in the western basin, since around 1950 and he stated that it has an important effect upon lake ecology. We frequently found shad in white bass and crappie stomachs indicating that the increase in shad has been exploited by some predatory fish. The rapid rise in a forage fish which becomes frequently infected and is preyed upon by many susceptible fish has increased the transmission of C. oxycephalus to its final host. This mechanism would also produce heavier infections in predatory fish and might be responsible for some fish mortality. The data available place the rise in gizzard shad stocks in the same time period as the increase in the prevalence of Camallanus, lending additional credibility to the hypothesis that shad are responsible, in part, for the abundance of the parasite.

Changes in the Lake Erie benthic fauna may have indirectly influenced the abundance of Camallanus. Britt et al (1973) documented the decline in abundance of the mayfly nymph Hexagenia since 1953. They noted a large decrease in Hexagenia after 1959. This reduction in food resource may have forced such generalized feeders as perch, drum, and catfish into eating greaters of forage. The concomitant rise of gizzard shad during this period increased the exposure of these fish to infection with Camallanus.

Ecology of Camallanus oxycephalus in western Lake Erie

To more fully understand the interaction of C. oxycephalus with its various life history components in the western Lake Erie system, an examination of certain host and parasite population phenomena was initiated. Such a study might help to determine how the abundance of

this parasite is maintained, what factors might cause fluctuations in the parasite population and finally, if any of these factors could be manipulated to reduce the abundance of the parasite.

The parasite occurred in 16 species of fish, but was most intensively investigated in white bass. All size classes of white bass were infected. The difference in incidence and intensity of infection between male and female fish was tested with the Mann-Whitney-U test and found to be not significant. Only a general relationship was evident between fish length and infection intensity (Figs. 13 and 14) because of the large degree of overdispersion in the sample. Infections from adult and young-of-the-year fish were fit to the Poisson series, but agreement was very poor. Because the ratio of variance to mean was high, the negative binomial distribution was fit to the data and agreement was good. The negative binomial, which is one of several overdispersed distributions, then is taken to be a satisfactory model of Camallanus in western Lake Erie fish. Data for freshwater drum, yellow perch and gizzard shad also approximate the negative binomial.

Table 5 illustrates the percentage infection and the mean intensity of infection in six size groups of fish. Adult fish one year or older (over 150 mm) generally had a higher incidence and higher mean worm burden than young-of-the-year fish. The heaviest worm burdens were always in large adult fish. The percentage of infection did not change with fish size in adult fish. These data indicate that white bass in western Lake Erie are frequently and heavily infected with Camallanus.

The structure of the parasite population varied considerably with the seasons. The population was composed entirely of adult worms during all months except July, August, and September (Table 6). During July and August, the population consisted mostly of third and fourth-stage larvae. This indicates that 1) the worms live for approximately one year, 2) worms die during early summer and 3) fish are re-infected in July and August. Old and new generations of the parasite can easily be differentiated by size and degree of maturation. Year old males and females average 4.57 mm and 18.18 mm in length respectively. The ratio of new males to new females is approximately 2:1 during August, but changes to 1:1 in September. This initial sex ratio is caused by the different molting times in the fourth-stage larvae. The sex ratio remains 1:1 until the following July, when females begin to die before males, producing a 2:1 ratio again.

Seasonal variation in the infection of adult white bass is evident in Table 7. While there is little change in the percentage of infection during the year, there is a marked decrease in the mean worm burden

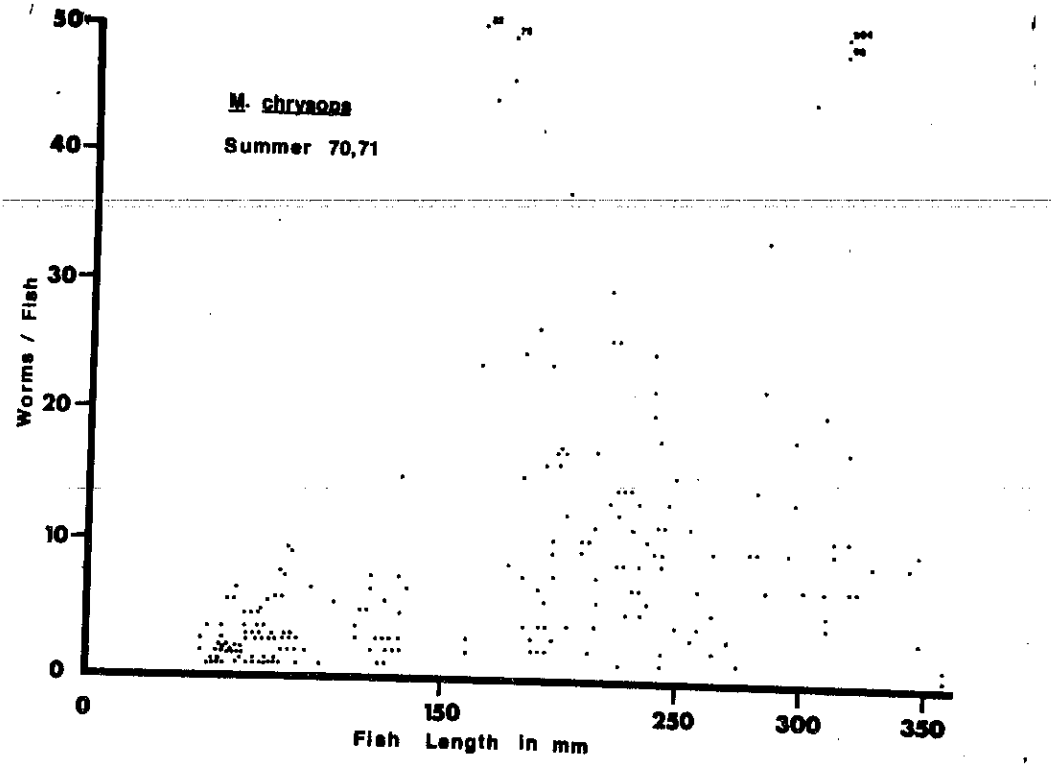

Figure 13. Relationship between infection intensity and fish length for white bass - Summer 1970, 1971

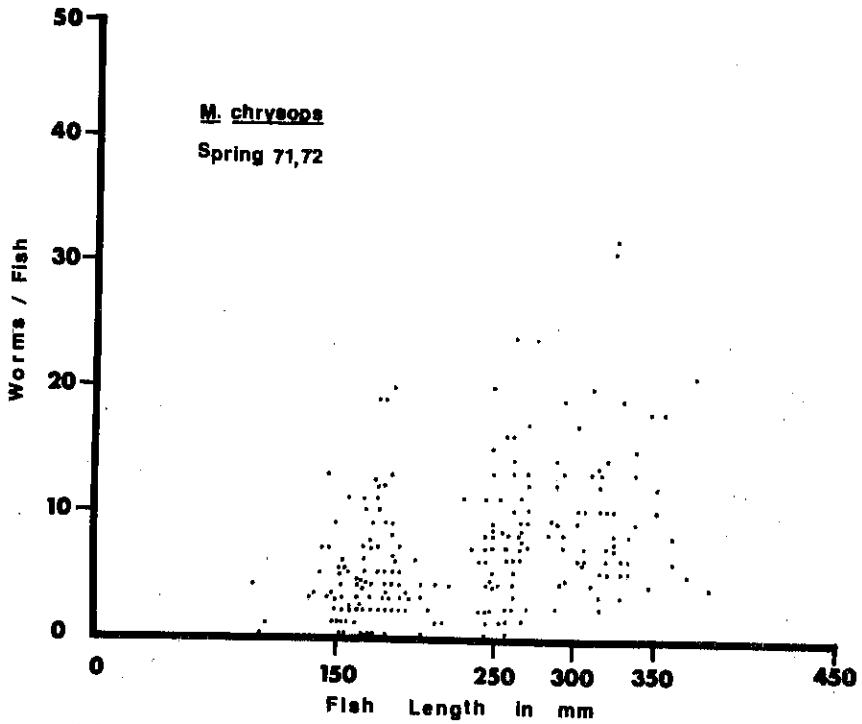

Figure 14. Relationship between infection intensity and fish length for white bass - Spring 1971, 1972

TABLE 5

RELATIONSHIP OF WHITE BASS LENGTH TO THE INCIDENCE AND INTENSITY OF INFECTION WITH *C. oxycephalus*

Length Group of Fish (mm)	No. Fish Examined	No. Fish Infected	% Infected	Mean Worm Burden
<100	254	182	71.7	1.97
101–150	93	80	86.0	4.14
151–200	158	134	84.8	8.04
201–250	132	107	81.1	8.78
251–300	110	100	90.9	7.54
300 <	102	93	91.2	11.11

TABLE 6

SEASONAL CHANGES IN THE POPULATION STRUCTURE OF *C. oxycephalus*

Month	% Males	% Females	% L_4	% L_3
June	48.2	51.8	0	0
July	11.6[a]	6.4[a]	58.4	14.3
	5.2[b]	3.9[b]		
August	1.0[a]	0.1[a]	43.1	9.2
	29.1[b]	17.5[b]		
September	51.6	45.6	3.2	0
October	51.2	48.8	0	0
November	50.5	49.5	0	0
April	49.2	50.8	0	0
May	47.9	52.1	0	0

a = year old adults

b = new adults

TABLE 7

SEASONAL CHANGES IN THE INFECTION OF ADULT WHITE BASS

Year and Month	Number of Fish Examined	Number of Fish Infected	Percent Infected	Mean Worm Burden
1970 November	15	14	93.3	10.9
1971 April	20	20	100.0	8.1
May	36	33	91.7	5.4
June	49	47	95.9	7.9
July	52	47	90.4	2.2 adults 13.5 juveniles
August	29	28	96.6	0.5 adults 14.6 juveniles
September	20	20	100.0	12.3
November	9	9	100.0	11.5
1972 April	23	22	95.7	8.4
May	43	43	100.0	7.5
June	40	36	90.0	5.7
July	69	43	62.3	2.7 adults 0.0 juveniles
August	89	70	78.6	0.3 adults 6.1 juveniles
October	31	29	93.4	4.6
November	16	15	93.8	2.8

between July and the following June. The infection increases rapidly in July, but a net loss of worms occurs as early as September. The decrease in the parasite population continues throughout the year so that the mean number of reproducing females in June is 30 - 60% less than the original number of immature females found the previous August. The 1970-71 and 1971-72 generations of C. oxycephalus were very similar in seasonal timing and size. The absence of larvae and lower incidence of the parasite in July 1972 indicates that the 1972-73 generation was delayed and not as effective in reaching the white bass population as in previous years. The November mean worm burden was markedly lower than the 1970 and 1971 values. The loss of worms from white bass appears partially related to fish size but may be directly related to the population density of worms within fish. Data from 1970-71 and 1971-72 were grouped and the mean worm burdens in four size groups of fish were plotted for six seasonal time periods (Fig. 15). It appears that the C. oxycephalus population remains

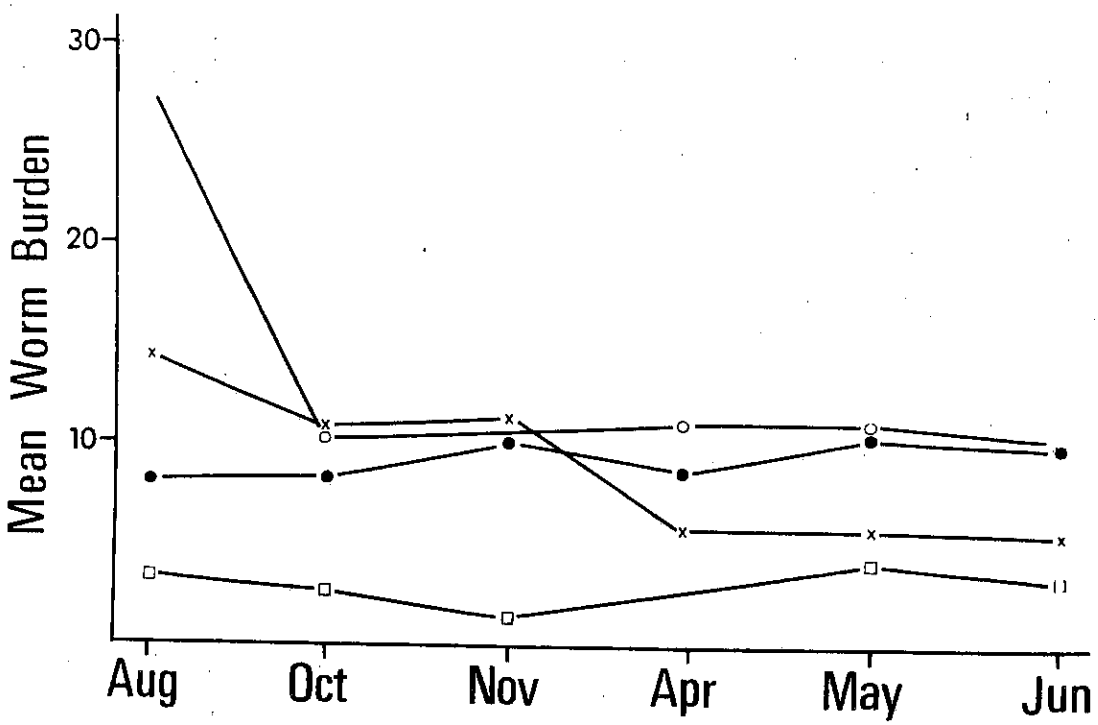

Figure 15. Seasonal variation in the mean worm burden of C. oxycephalus in different age groups of white bass. □ 0+ years (0-150 mm), x - 1+ years (151-250 mm), ● - 2+ years (251-300 mm), o - 3+ years (over 300 mm).

relatively stable in the fish with summer mean worm burdens of 10 worms or less. The two fish groups with high summer infections lost 50 - 60% of their nematodes. The initial worm burdens do not correlate with size completely since more worms occurred in the 1+ fish than in the 2+ fish. These data suggest the possibility of a density-dependent population regulation of C. oxycephalus in white bass. A density-dependent regulatory mechanism might be related to fish size because larger fish have more gut volume and therefore could support a higher density of parasites. This is suggested in Fig. 15, where 1+ fish had a greater initial worm density with greater worm mortality than the 2+ fish which had a lower worm density with no apparent worm mortality.

When a new generation of worms enters the white bass in July, they are distributed throughout the entire intestine. During July, when most worms are larvae, they occurred primarily in the intestine (Table 8). A maturational site selection occurs and by August, two thirds of the worms are found in the rectum. No worms occur anterior to the rectum between October and the following June. The worms remain in the rectum until death. Infected fish become obvious in April and May when growing female nematodes begin to protrude from the fish rectum and are readily observable with the unaided eye.

TABLE 8

SEASONAL CHANGES IN SITE SELECTION OF C. oxycephalus IN ADULT WHITE BASS

Month	Worms in Small Intestine No.	%	Worms in Large Intestine No.	%
June	0	0.0	386	100.0
July	458	66.5	231	33.5
August	210	36.1	370	63.9
September	42	18.3	188	81.7
October	0	0.0	34	100.0

The seasonal growth pattern of C. oxycephalus was established by plotting mean body length against time. Figure 16 reveals that the growth pattern for two generations was very similar and exhibits the following four characteristics: 1) the initial, autumn growth rate of female worms was more rapid than that of males; 2) female growth was arrested between November and April; 3) the difference in mean male body length between November and April was significant, ($t = 2.888$, $P < .005$) indicating that males continued to grow through the winter; 4) females resumed growth at an extremely rapid rate in April and continued growing until death, reaching a length 4 - 5 times longer than males. Sperm cells were found in the uteri of female worms in October and November and were carried through the winter, indicating copulation occurs in the autumn. Fertilization occurs in April when ova are shed into the uterus. Development of the embryos is relatively synchronous. Development is complete by mid-June and the female uterus is filled with active, first-stage larvae. Gravid females contain about 7,000 to 10,000 larvae. Most larvae are dispersed simultaneously when the female ruptures and prolapses the uterus through the body wall.

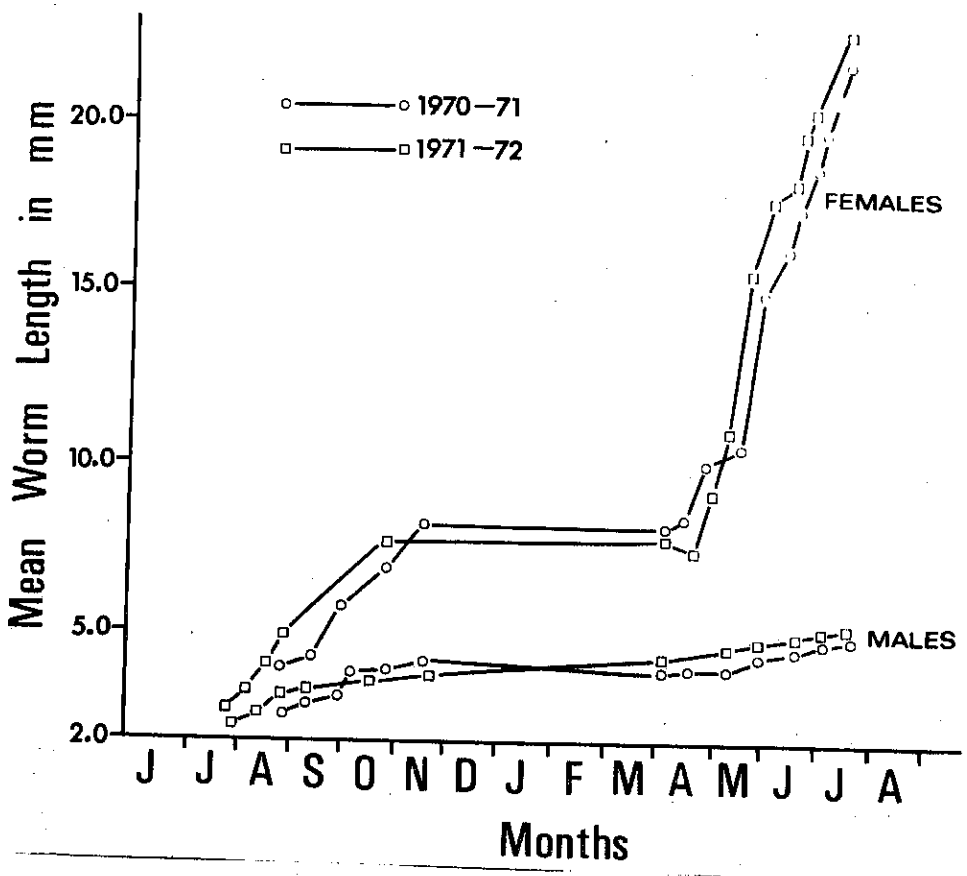

Figure 16. Seasonal growth pattern of C. oxycephalus in western Lake Erie

The cyclopoid copepod populations in western Lake Erie, composed predominantly of Cyclops bicuspidatus and Cyclops vernalis, reach an annual maximum density in June and July but are otherwise low throughout the rest of the year. Calanoid copepods are most dense in September (Britt et al, 1973). Our sampling determined that this pulse of cyclopoids in 1971, 1972 and 1973 was composed of two separate peaks. Figure 17 shows that the dispersal period for C. oxycephalus coincides with the annual maximum cyclopoid density. A close examination of Fig. 17 reveals some subtle differences in this synchrony among the three years. The summer of 1972 was unusually cold in northwestern Ohio. The abundance of cyclopoids was less than in 1971 and 1973. In addition, the two peak densities within the copepod pulse were 42 days apart, compared to the 10 - 20 day peak separation in 1971 and 1973. We arbitrarily designated the copepod populations to be "abundant" when they were at least as dense as 20/liter, since this density rarely occurs during non-pulse periods. The effectiveness of worm dispersal can be compared among the 3 years by dividing the number of dispersal days with abundant copepods by the total number of dispersal days (Table 9). It is clear from

TABLE 9

EFFECTIVENESS OF C. oxycephalus DISPERSAL

	Total Dispersal Days	"Abundant" Copepod Days	% Effectiveness
1971	38	19	50
1972	30	11	36
1973	29	26	89

Table 9 that the effectiveness of larval dispersal was much less than in 1971 or 1973.

Examination of forage fish revealed C. oxycephalus larvae in the following species: Alewife (Alosa pseudoharengus), gizzard shad, spotfin shiners (Notropis spilopterus), emerald shiners (N. atherinoides), and smelt (Osmerus mordax). Camallanus occurred most frequently in gizzard shad (71%) and with much less frequency in spotfins (16%), alewife (15%), smelt (4%) and emeralds (1%). Spottail shiners

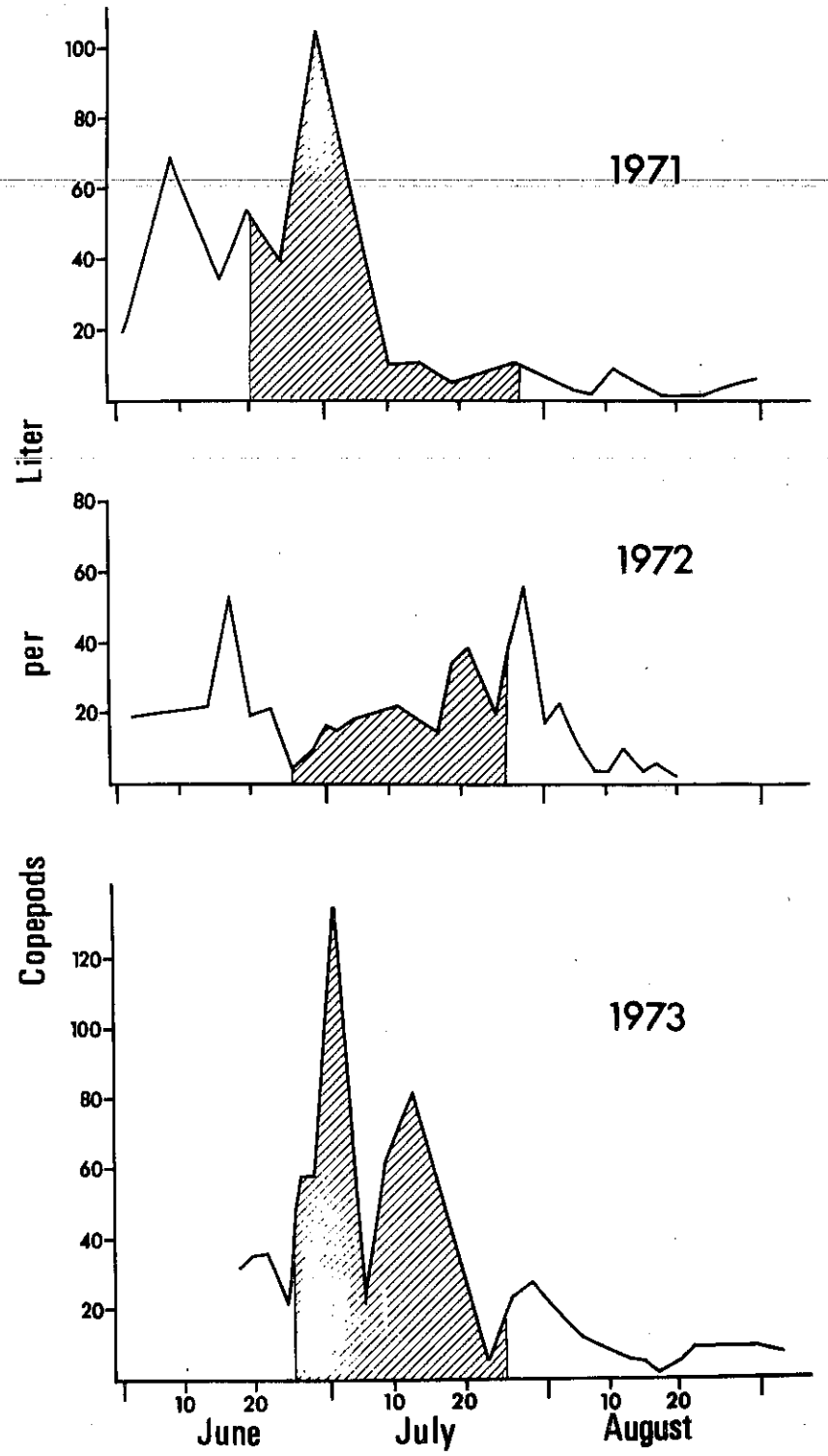

Figure 17. Density of cyclopoid copepods and the dispersal period for Camallanus oxycephalus in western Lake Erie. Shaded area is copepod density during dispersal of larvae.

(N. hudsonius), which are a very common forage species, were not infected. The distribution of Camallanus within these fish populations does not remain stable. Table 10 illustrates how the distribution in young gizzard shad changed over a 2 1/2 week period. The incidence of the parasite decreased by 50%. This was caused by parasite mortality. Most of the August 5 worms were third-stage larvae. Most of the August 23 worms were fourth-stage larvae, indicating that the worms undergo the third molt in shad. No adult worms were found, which suggests that the final molt does not occur in gizzard shad, and that fourth-stage larvae eventually die and pass out of the fish. Thus, C. oxycephalus larvae remain within the gizzard shad population for only several weeks and thus the period during which the parasite can be transferred to the white bass population is short.

Analysis of the total lengths of 256 young-of-the-year gizzard shad found in 67 white bass stomachs taken between August and November revealed a prey size selection by different size white bass (Fig. 18). No shad under 19 mm long was taken by any bass.

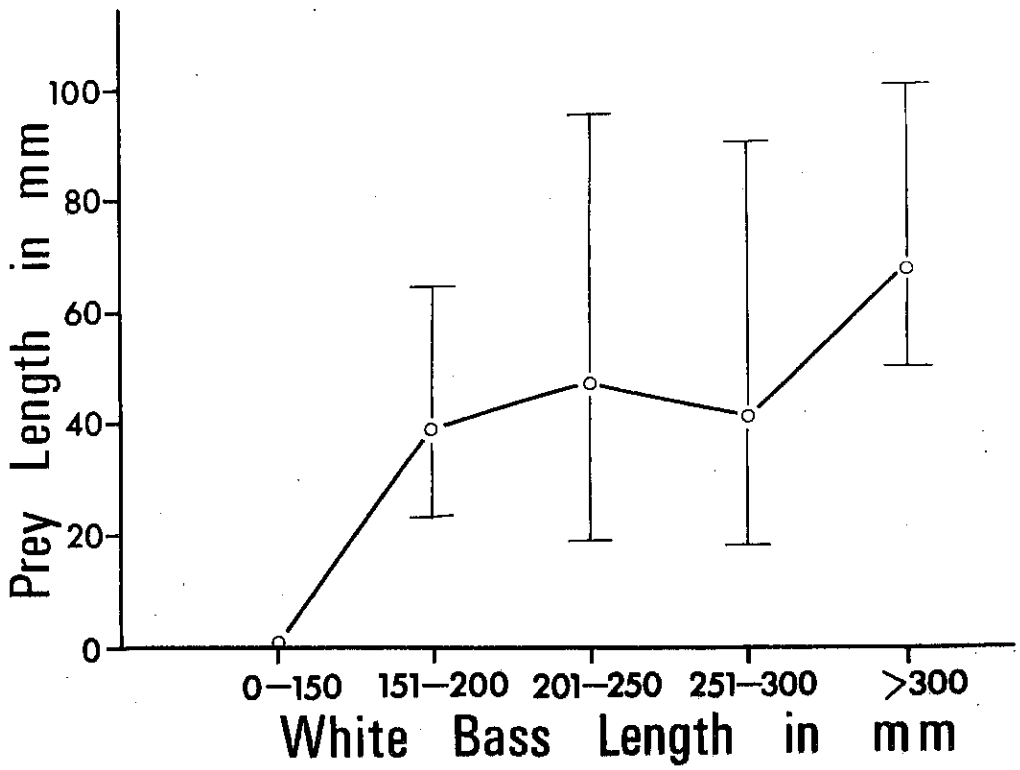

Figure 18. Food size selection on young-of-the-year gizzard shad by 5 size groups of white bass. o indicates mean prey length and vertical bars indicate selection range.

TABLE 10

DISTRIBUTION OF *C. oxycephalus* IN YOUNG GIZZARD SHAD

No. Worms X	Number of fish with x worms		
	August 5	August 13	August 23
0	23	34	46
1	27	23	21
2	18	15	10
3	8	5	1
4	2	0	1
5	1	1	0
6	0	2	0
No. Fish	80	80	79
Size range (mm)	30-45	34-67	41-60
% infected	71.2	57.9	42.5
% L_3	72.2	57.6	29.2
% L_4	27.8	42.4	70.8
% Adults	0	0	0
Total worms	108	85	48
Mean infection	1.9	1.8	1.4

Although maximum prey size was 100 mm, shad over 80 mm were rarely found in bass stomachs. White bass 201 - 300 mm in length selected shad throughout the entire 19 - 100 mm length interval, although the mean length was 40 mm. Large bass, however, showed a preference for large shad (mean length - 66.8 mm) and ate no fish less than 50 mm long. Small bass (151 - 200 mm) showed a preference for small shad (mean length - 39.4 mm) and ate no fish longer than 64 mm. Gizzard shad were not recovered from white bass less than 170 mm in length.

Young-of-the-year gizzard shad grow rapidly, reaching maximum prey size (100 mm) for white bass as early as September. If white bass feed only upon 19 - 100 mm shad, transmission of C. oxycephalus would be affected by a combination of 1) parasite distribution in the shad population, 2) growth rate of shad and 3) relative abundance of shad. Extreme water temperatures may alter the transmission by raising or lowering the availability of the parasite to predatory fish. Gizzard shad abundance may fluctuate from year to year. During years of relatively few shad, predators may select more shiners or alewife, which are not as frequently infected, resulting in few Camallanus reaching the final host and reproducing.

ANALYSIS

The abundance of Camallanus oxycephalus in western Lake Erie has been significantly affected by the following:

1. high infection efficiency of larvae in copepods
2. ability of infection to be passed from fish to fish
3. ability to reproduce in many species of Lake Erie fish
4. synchronization of dispersal with intermediate host abundance
5. rise in gizzard shad and alewife abundance
6. increase in copepod density in Lake Erie
7. production of many larvae by female worms

These factors are well inter-related in Lake Erie with the result that C. oxycephalus is very successful. The entire system depends upon the ecological overlap, both spatial and temporal, among the various life history components. Figure 19 is a general host-parasite interaction model. The frequency (F_{inf}) of any parasite is the net result of input and output in a host population. Input is a function of how often host and parasite contact (P - probability of contact). For endoparasitic species like C. oxycephalus, this probability is influenced by feeding habits, host size, behavior and distribution of the host. Output is related to how efficient the parasite is in invading the host (E), host resistance, longevity of the parasite and perhaps the density of parasites.

In some systems, the number of parasites is in constant equilibrium, while in others, input is seasonal and output occurs constantly.

This general model can be expanded to specifically fit C. oxycephalus in western Lake Erie (Fig. 20). In this systems model, there is a probability of interaction (P_x) associated with each of the possible links in the transmission pathways. There is also an efficiency of transfer (E_x) between each link. In addition, the mortality of parasites within each link (D_x). Since mortality occurs through time, this term should really be $\frac{dx}{t}$. Although the most efficient transmission route should be the shortest, for more worms reach the white bass population via the gizzard shad. This is due, in part, to the very close association between white bass and gizzard shad and gizzard shad and copepods. This results in higher incidence and heavier infections in adult white bass. This, in turn, means the production of many more infective larvae. Estimates of the mean larval production revealed about 14,800 larvae per young-of-the-year fish and 33,300 larvae per adult fish. Thus, the addition of the gizzard shad link has resulted in a great increase in the input of infective larvae back into the system. The raised input of larvae increases the parasite population density until equilibrium is established.

Not only is there great spatial overlap among the transmission components, but a fine degree of temporal synchronization as well. A general model of temperal relations in a host-parasite system is illustrated in Fig. 21. Generally, the number of adult parasites (N) is equal to the number of larvae (L_n) multiplied by their probability of success. If the larvae are distributed through time with the maximum probability of success, the density of adult parasites will be high. If the eggs or larvae are dispersed when the density of intermediate hosts is high, the parasite will be more abundant.

This model can also be expanded to fit the Camallanus - Lake Erie system (Fig. 22). The abundance of each component in the Camallanus transmission cycle is distributed normally with respect to time. The greater the temporal overlap between each component, the higher is the P of successful transfer. Camallanus disperses its larvae only once per year. This period coincides with the maximum copepod density. Infected copepods are in the plankton when young gizzard shad are eating copepods. Part of the time white bass feed upon gizzard shad, the shad contain Camallanus larvae. Thus, the total adult worms could be predicted from the number of larvae and the multiplicative probability of their reaching white bass.

The fine seasonal timing of parasite transmission can be disrupted, however. Fluctuations in environmental parameters such as temperature might alter parasite flow through the system and regulate

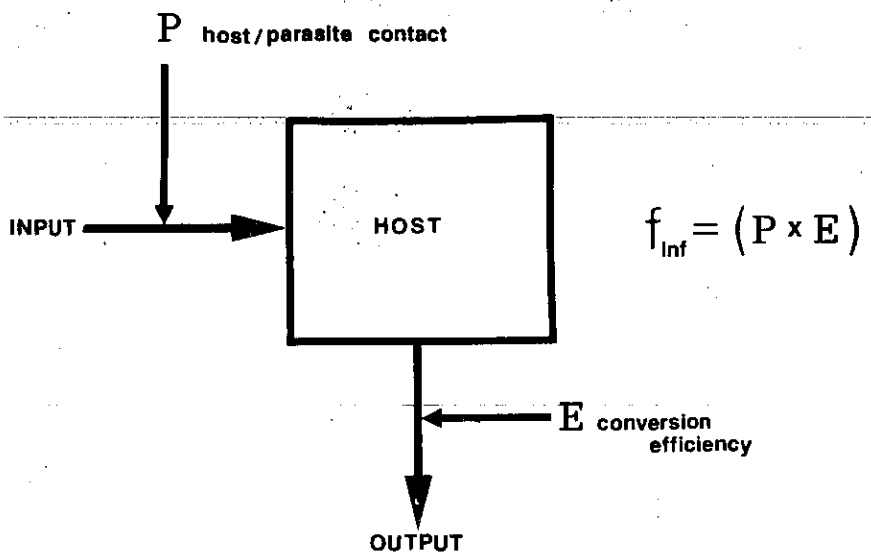

Figure 19. Host-Parasite Interaction Model

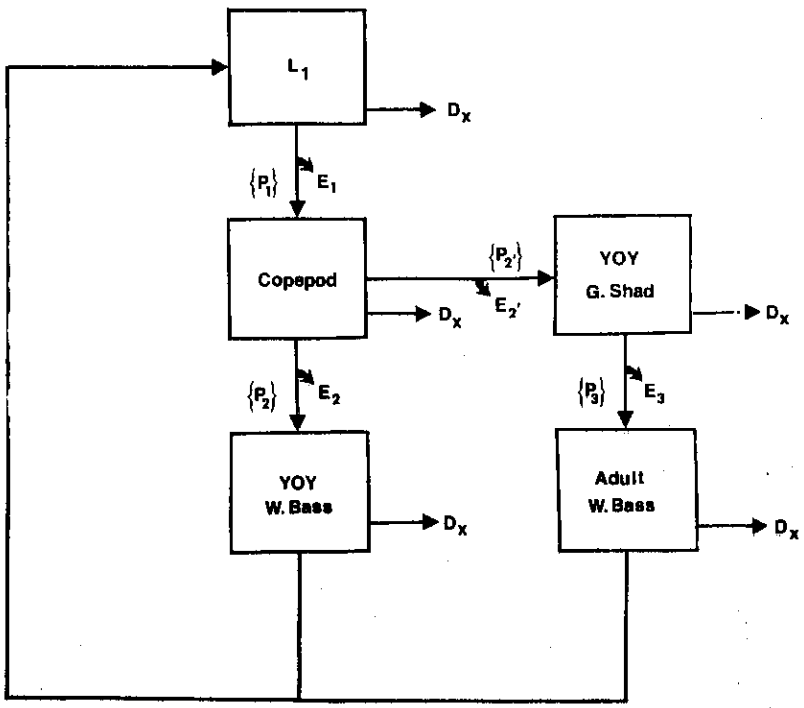

Figure 20. Camallanus - Lake Erie Systems Model

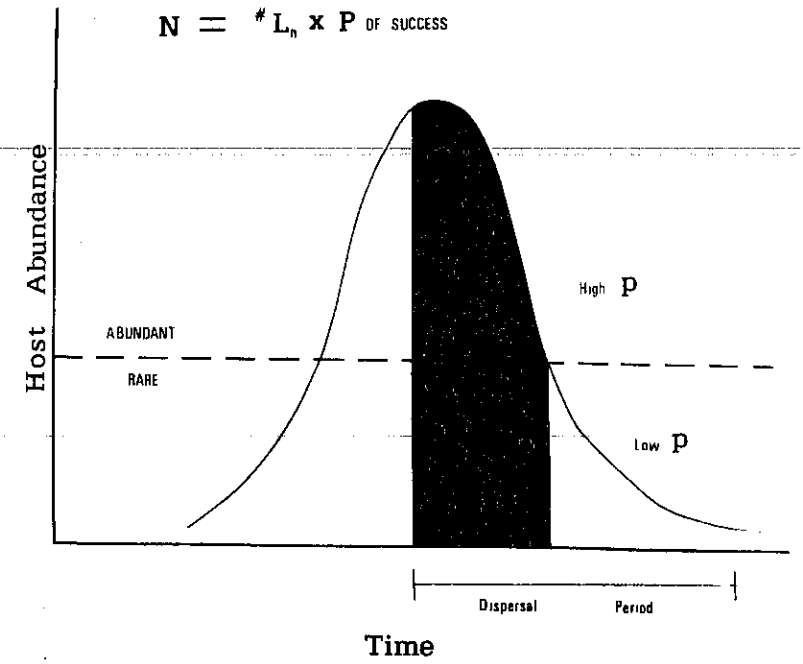

Figure 21. Temporal overlap model

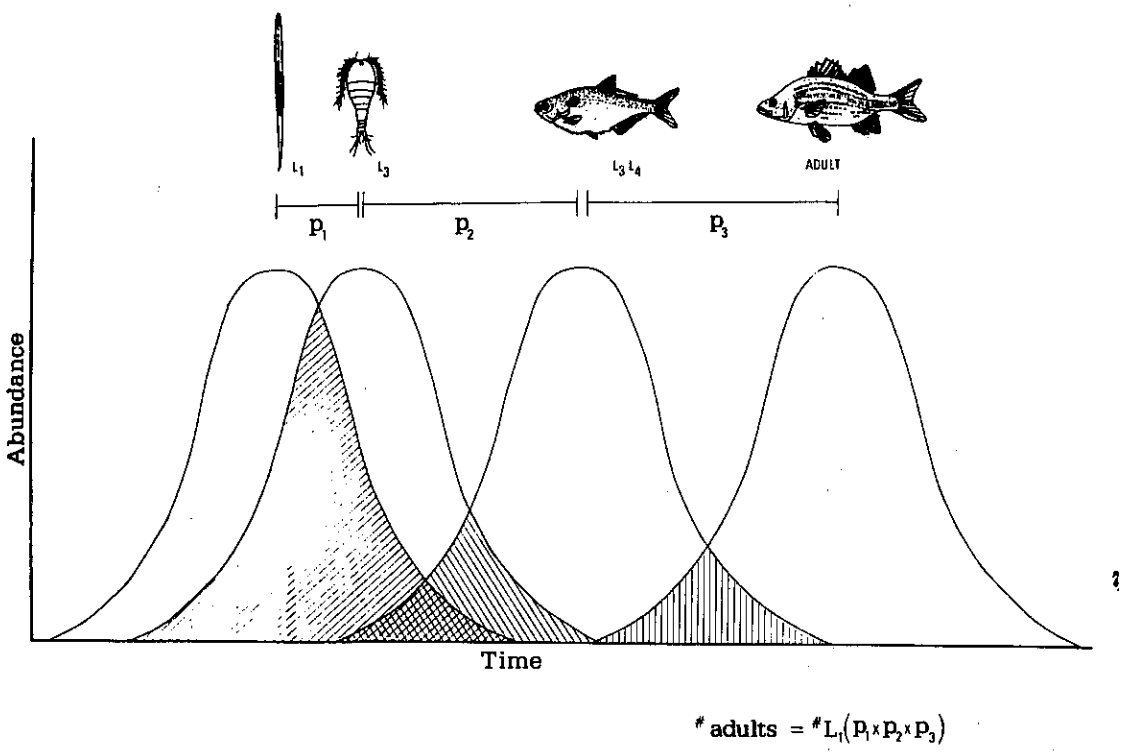

Figure 22. Temporal interactions of Camallanus - Lake Erie system

the number of adult worms. However, the Camallanus - Lake Erie system is very complex, involving as many as 15 final host species. Such complexity should contribute to the stability of this system by damping any tendency toward extreme fluctuations. The result should be a gradual increase in the parasite population until an equilibrium level is established.

RECOMMENDATIONS

At present, there appears to be little that fisheries biologists can do to reduce the abundance of Camallanus oxycephalus in Lake Erie. This system is simply too large for effective management. The spreading of this parasite, however, could be minimized if fish taken from the lake for fish-out ponds are captured during periods of low infection. This period extends from late June to late July. However, relatively low infections are found in April and May, so that fish can be taken and transported from Lake Erie from April to mid-July. No fish should be taken between mid-July and April. The control of this parasite any where depends upon elimination of forage fish which become infected and transmit the parasite to predatory species. It is recommended that gizzard shad and alewife not be used in ponds where Camallanus is an undesirable parasite.

Although no direct pathological effect is known to occur in connection with C. oxycephalus, some secondary bacterial, fungal or viral infection is possible, particularly in heavily infected fishes. It is apparent that gizzard shad abundance has been related to the increase of C. oxycephalus in Lake Erie fish. If this parasite continues to increase in abundance as a result of changes in the Lake environment, some mortality is likely. To this end, future investigations of white bass mortality, if any, should encompass an assessment of the role of C. oxycephalus as well as other parasites, in the health of the fish population.

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FEDERAL AID IN SPORT FISH RESTORATION

FINAL REPORT

STATE OF:	Ohio
PROJECT NO.:	F-48-R-1, 2, 3
PERIOD COVERED:	June 1, 1972 - May 31, 1975
PROJECT TITLE:	Impact of Parasitic Worms on Lake Erie Fishes
STUDY NO.:	III
STUDY TITLE:	Impact of Eustrongylides tubifex on Lake Erie Fishes
PREPARED BY:	C. Lawrence Cooper and John L. Crites
DATE:	31 July 1975

CONTENTS

ABSTRACT

Large, red worms, larval Eustrongylides tubifex (Nematoda: Dioctophymatoidea) have captured the attention and concern of sports and commercial fishermen as well as biologists. In Lake Erie fishes, the larval nematodes are found primarily in Yellow Perch (Perca flavescens.) The maintenance of this parasite is complex involving an avian definitive host, a piscine second intermediate host and an invertebrate first intermediate host.

Two developmental stages of this parasitic nematode are encountered in Lake Erie fishes. To determine the prevalence and intensity of infection of larvae, a total of 60 Smallmouth Blackbass (Micropterus dolomieui,) 198 Channel Catfish (Ictalurus punctatus,) 162 Freshwater-Drum (Aplodinotus grunniens,) and 3544 Yellow Perch (Perca flavescens) were examined. Analysis of larval incidence data reveals that older, larger fish are more heavily infected.

Both young-of-the-year and I year age-size Yellow Perch display an increasing larval worm burden from July through October. The II+ age-size class of Yellow Perch do not display an increasing worm burden during these months. The II+ age-size class perch harbor fourth-stage larvae entirely. Young-of-the-year perch harbor third-stage larvae only. Male and female perch are equally susceptible to infection. There is a 1:1 sex ratio of larvae in perch. Young-of-the-year perch acquire the infection in July and the late summer months. Recruitment of third-stage larvae is focused in young-of-the-year fish. The majority of larvae are recovered from yellowish-pink encapsulations in the mesenteries and fatty tissues near the small intestine of the fish.

Larvae emerge from their encapsulations and migrate through the flesh of dead fish in response to increasing temperature. This emergence behavior is an adaptation which facilitates transfer of the infection from intermediate to definitive host.

Examination of naturally infected fish-eating birds indicates that larvae E. tubifex successfully invade the proventriculus of a variety of birds. Most infections fail to develop and result in degenerating worm tracts in the serosal muscle of the proventriculus of the bird. The avian species primarily responsible for the maintenance of E. tubifex in the western basin region remains to be clearly identified. Preliminary examinations reveal that Red-breasted Mergansers (Mergus serrator) harbor the highest prevalence and intensity of mature infections.

Mature infections of E. tubifex have been experimentally established in hatchery-reared Mallards (Anas platyrhynchos.) The female worm matures in 16-23 days post infectionem. The adult male worm matures in 7-9 days post infectionem. The large number of negative infections in Mallards indicates that this species is largely refractory to infection. Experimental infections were attempted utilizing a Black-crowned Night Heron (Nycticorax nycticorax hoactli,) Herring Gulls (Larus argentatus,) and ring-necked Pheasants (Phasianus colchicus torquatus.) No mature infections resulted from the latter avian species.

Prior to this study, the specific identity of the larval Eustrongylides sp. infecting fishes in Lake Erie was not known. A complete description of both larval and adult stages of the species is included. Comparison of specimens recovered during this study with literature descriptions and museum specimens reveal the species refers to Eustrongylides tubifex (Nitzsch, 1819) Jagerskiold, 1909. The descriptions included in this report are the most complete ones available for this species.

BACKGROUND

Large, red worms, larval Eustrongylides sp. in Lake Erie fishes have captured the attention and concern of sports and commercial fishermen for some time. In Lake Erie fishes, these large, red worms are seen in the fillets and body cavity of yellow perch, Perca flavescens, predominantly. The maintenance of this parasite in the biological community is complex in that three hosts are required. An avian definitive host, a fish second intermediate host, and an invertebrate first intermediate host are essential for the maintenance of the worm population.

Bangham (1972) stated that Eustrongylides sp., a tissue invading nematode found in the body cavity and flesh of its piscine host is a seasonal economic factor in that its presence causes rejection of yellow perch fillets. Dechtiar (1972) in citing new host records for Lake Erie fish considered Eustrongylides sp. pathogenic to fish. Dogiel, Petrushevski, and Polyanski (1961) stated that larval Eustrongylides sp. parasitic in fish cause a hyperplasia of connective tissue resulting in the encapsulation of the larvae in somewhat round, flattened capsules. Paperna (1974) stated that larval Eustrongylides encyst in the mesenteries, spleen, and in the gonads of fish. In Lake Victoria, fishes of the genus Haplochromis harbor larvae in the gonads, particularly the ovaries. Unencysted larvae were observed in the body musculature as well. Paperna tentatively identified the larvae as Eustrongylides africanus.

In addition to being pathogenic in fish, Karmanova (1968) considered such larval forms capable of inflicting injury to fish-eating animals. Bowdish (1948) reported the mortality of a Newark, N.J. Great Blue Heron, Ardea herodias, from verminous peritonitis. The heron had severe perforations and adhesions of the intestines caused by several young worms tentatively identified as Eustrongylides ignotus. Locke (1961) recorded a second case of fatal verminous peritonitis in a Great Blue Heron plus a first report for the American Egret, Casmerodius albus egretta. In both cases a large shield-like mass of coiled fibrous tubes containing the large, red E. ignotus, adhered intestines, and ingesta filled the ventral portion of the abdominal cavity. A die-off of Red-breasted Mergansers, Mergus serrator, at Lake Holly, Virginia Beach, was attributed to massive tissue destruction and hemorrhage produced by migrations of larval Eustronylides sp. (Locke, 1964). Subsequent investigations revealed larval Eustrongylides sp. in mosquitofish and silversides upon which the mergansers had been

feeding. Shillinger (1936) attributed extensive loss of water fowl in British Columbia to the presence of great numbers of Eustrongylides mergorum in the walls of the proventriculus. Gibson and McKiel (1972) reported larval Eustrongylides sp. from the muscles of muskrat in Ontario, Canada. Morishita, Nishimura, and Kondo (1959) reported larval Eustrongylides sp. from man in Japan. Abram and Lichtenfels (1974) reported a single immature female Eustrongylides sp. was coughed by a man. They have also recovered specimens of Eustrongylides sp. from an otter collected in Maryland (1974).

The numerous reports of larval Eustrongylides sp. from many species of fish, some amphibians, and a few reptiles, plus the reports of adult and subadult infections in numerous aquatic birds, fish-eating mammals, and now man, reflects the broad range of infectivity expressed by worms of this genus. Although many organisms may become infected, few have been revealed as true hosts harboring mature, egg-producing infections. Eustrongylides infections in hosts which do not provide an adequate environment for full development of the worms result in a wide range of pathological conditions. In a number of instances, infection of a potential definitive host with larval Eustrongylides proves fatal to the host in question.

OBJECTIVES

Job No. III-a: Experimental Phase

1. Determination of the transmission cycle of Eustrongylides tubifex.
2. Demonstration of intermediate and definitive hosts involved in transmission process.
3. Demonstration of mechanism and seasonality of transmission of infective stages from one host to another.
4. Demonstration of development of different stages in intermediate and definitive host.

Job No. III-b: Descriptive Phase

1. Description of the parasitic stages of E. tubifex in the intermediate and definitive hosts.
2. Description and demonstration of routes of migration through tissues of hosts.
3. Description of pathology to hosts involved.

Job No. III-c: Population Phase

1. Determination of population biology of larval E. tubifex in fish hosts in western Lake Erie.
2. Determination of effect of season, locale, fish sex, fish age and size, tissue site, and larval sex on worm burden of larval E. tubifex in fish hosts in western Lake Erie.

PROCEDURES

During this study, prevalence and intensity of infection of larval Eustrongylides was recorded from 4 species of fish taken from the western basin of Lake Erie. Yellow perch, Perca flavescens, examined during this study were collected from two sites. During 1972, yellow perch were provided by a commercial fish corporation. These fish were collected with a 150 foot shore seine in the Sandusky Bay area. The fish were placed in ice chests and transported to South Bass Island for autopsy. Throughout the study, yellow perch were collected by otter trawl from the BioLab at a station between Green Island and Rattlesnake Island. After trawling, the fish were placed in ice chests and taken to The OSU Research Laboratory on South Bass Island for autopsy. During 1974, Smallmouth bass, Micropterus dolomieui, were collected by hook and line near South Bass Island.

Freshwater-drum, Aplodinotus grunniens, were collected with the otter trawl during 1973. During 1973 and 1974, channel catfish, Ictalurus punctatus, were collected with the otter trawl and by hook and line. In each of the latter instances, the fish were placed in ice chests and brought to the OSU Research Laboratory for immediate autopsy.

During 1972, yellow perch were autopsied using one of two techniques. Young-of-the-year perch, under 10 cm in length, were subjected to pepsin digest of the viscera. The pepsin digest solution consisted of 4 gms of pepsin, 7 ml of hydrochloric acid, and 1000 ml of distilled water. The mesenteric tissue of the fish was placed in pepsin digest fluid in one series of test tubes and the digestive tract and other visceral organs in another series. The tubes were manually agitated and placed in a constant temperature chamber at 25°C. The supernatant was periodically decanted and fresh pepsin digest solution added for a period ranging from 4-8 hours. At the termination of the digestion period, the contents of the tubes were examined under a binocular dissecting microscope utilizing both transmitted and reflected light.

The autopsy procedure for the remainder of the perch examined during 1972 can be summarized as follows: (1) measurement of total fish length, (2) notation of the sex of the fish, (3) thorough dissection of the fish. All other fish examinations conducted during this study utilized the latter procedure. Dissection of the fish consisted of initially searching the mesenteric tissue

for signs of yellow grandular encapsulations. Encapsulated larvae were plucked out with a needle point and placed in Ringer's 'Cold' solution. Next all mesenteric tissue was removed and examined under a binocular dissecting microscope. The intestine, stomach, heart, liver and gonads were subsequently examined. After examination of the viscera, the body wall and gas bladder were examined for evidence of migrating larvae and lesions. Finally, the external surface of the fish was examined for migrating larvae. The larval Eustrongylides removed were placed in Ringer's 'Cold' solution at 15°C. The larvae were then either used for experimental infections or relaxed and killed in heated Ringer's 'Cold' Solution and fixed in alcohol-formalin-acetic acid (AFA) solution for 24 hours. Larvae killed and fixed using the latter procedure were subsequently transferred to a solution of glycerin and 70% alcohol for storage.

Specimens of larvae and adult worms studied for descriptive purposes were cleared in a glycerine-alcohol solution. This technique involved evaporating the alcohol portion of the solution until the worms were suspended in pure glycerine. Internal structures of the study specimens were made apparent by use of this technique. The specimens were mounted in glycerin on glass slides for microscopic study.

The analysis of the population data of larval Eustrongylides sp. in yellow perch was facilitated by key punching the data onto computer cards. Several programs were run at Ohio State University's Instruction and Research Computer Center. OMNITAB, FORTRAN, SPSS, and PSTAT computer languages were used to obtain cross tabulations, scatter diagrams, and both parametric and nonparametric statistical parameters and tests.

Several species of birds were utilized as experimental definitive hosts for E. tubifex. The principal one among these was second generation flying Mallards, Anas platyrhynchos. From one to three dozen wild Mallards purchased from Whistling Wings, Hanover, Illinois, were utilized during each of the three years of this study. These birds were flown to Columbus at the age of one week and subsequently transported to OSU Research Laboratory on South Bass Island. Young ring-neck pheasants, Phasianus colchicus torquatus, obtained from the Ohio Department of Wildlife, a single nestling Black-crowned Night Heron, Nycticorax nycticorax hoactli, and six nestling Herring Gulls, Larus argentatus, obtained in the Bass Island region were also

utilized as experimental definitive hosts. A variety of species of fish taken by fyke net in Fishery Bay, South Bass Island, were utilized in larval transfer experiments. Specimens of several species of turtles taken in Licking County by Raymond Jezerinac were also utilized in larval transfer experiments. The Mallards and Pheasants were maintained on commercial poultry feeds. The Night-Heron and Gulls were maintained on uninfected fish fillets.

Large, red larval E. tubifex (fourth-stage larvae) were used to infect potential definitive hosts. The time of storage of the larvae from autopsy of the fish intermediate host to innoculation of the experimental avian host ranged from one to 12 hours. Innoculation of the avian host per os involved a plastic stomach tube or forced ingestion of a small fish containing a designated number of larvae. Autopsy of definitive hosts followed standard techniques.

All worms recovered from experimental infections were placed in Ringer's 'Warm' solution and relaxed by gradual heating of the solution. The worms were fixed in AFA and subsequently transferred to a glycerin-alcohol solution for storage. The larger gravid females were studied under a binocular scope while alive and teased apart for recovery of eggs. All eggs recovered from gravid female worms removed from experimental infections were placed in distilled water and incubated in constant temperature chambers at 15°C and 20°C.

All worms, both from fish and birds, were studied under a compound microscope with both phase-constrast and bright-field microscopy. Measurements were taken in millimeters. Scaled line drawings were produced with the aid of a Wild compound microscope equipped with drawing tubes.

In order to remedy taxonomic difficulties, specimens of nematodes of the genus Eustrongylides on file with the USNM Helminthological Collection were studied and compared with the specimens of Eustrongylides sp. recovered from the experimental Mallard infections. The specimens from the USNM were designated as representatives of E. ignotus and E. tubifex.

In cooperation with the Ohio Cooperative Wildlife Unit of the Department of Zoology at the Ohio State University, frozen proventriculi of three species of herons collected during 1972 and 1973 at Winous Point, Ottawa County, and West Sister Island, Ohio, were examined for natural infections. In addition a number

of herons and other aquatic birds were collected in the Bass Island region during 1974. The latter collections were made possible by scientific collection permits issued by the U.S. Fish and Wildlife Service. The proventriculi of these birds were thoroughly examined for natural infections. A number of Red-breasted Mergansers taken during the regular hunting season of 1974 were made available by personnel of the Fish Management section of the Ohio Division of Wildlife. The proventriculi of these birds were also examined for natural infections of E. tubifex.

In order to study the ability of larval Eustrongylides to effectively transfer from one piscine host to another and to transfer from piscine host to avian host, a number of transfer experiments were performed. To investigate the larval parasite's ability to successfully reinfect a predatory fish, larvae from yellow perch were intubated into a number of species of fish, and turtles. In each instance, the potential poikilothermous host was narcotized in an aqueous solution of Quinaldine. Larval Eustrongylides freshly removed from yellow perch were then intubated into the stomach by means of a plastic stomach tube. Experimental hosts were killed and autopsied at intervals ranging from 4 to 168 hours post infection. The body wall, body cavity, visceral organs, stomach and intestinal walls and lumen were thoroughly examined for recovery of larvae.

In order to investigate the larval worms behavior when transferring from a piscine intermediate host to a definitive host, a series of experiments utilizing yellow perch were performed. Yellow perch were collected by otter trawl and placed on ice. Upon arrival at The OSU Research Facility on South Bass Island, perch were wrapped in damp paper toweling and placed in an oven. A series of experiments utilizing temperatures ranging from 25°C to 70°C were conducted. Fish were placed in the oven for periods ranging from 2 to 4 hours. Upon removal of the fish from the oven, they were immediately placed in a freezer at -10°C to stop any further larval activity. The fish were subsequently autopsied and the number and position of the larvae infecting the fish were noted.

FINDINGS

Experimental Transmission Cycle Studies:

Larval Eustrongylides sp. from Yellow Perch were utilized in experimental infections of several avian hosts. Although previous attempts at infecting birds with larval Eustrongylides sp. have been made in North America, none were successful in obtaining mature adult forms (Von Brand and Cullinan, 1943.) In Eurasia, experimental infections of cormorants have yielded mature forms of E. excisus (Iksanov, 1958; Ciurea, 1924 in Karmanova, 1968; Dubinin, 1949; and Karmanova, 1968.) This study is the first to successfully recover mature forms from birds experimentally infected in North America. Table 1 summarizes the results of experimental infections conducted during this study.

In all the experimental infections attempted during this study, few patent (egg-producing) worms have been recovered. In most instances, infections result in degenerating worms found in the muscular serosa of the proventriculus. Degenerating worms do not appear to have molted to the adult form prior to death. In those instances in which the worms develop to maturity, the adult nematodes are localized in coiled, tubular connective tissue encapsulations encompassing the glands and serosa of the proventriculus. A few larvae burrowed completely into the abdominal cavity, but soon died. The majority of worms survived the host's digestive processes and burrowed into the proventricular tissue. Specifically, they located on the serosal side of the proventriculus within encapsulated tubes. The anterior and posterior ends of the worms protrude from the tubes, through the glandular portion of the proventriculus, into the lumen of the organ.

The one Black-crowned Night Heron innoculated with larval Eustrongylides sp. died 42 hours post infectionem of verminous peritonitis. Following infection, the bird's appetite abated. Prior to death, the progressive etiology involved a failure to raise the head, hyperventilation and convulsions. Autopsy of the bird revealed 3 larvae in the body cavity and 2 in the proventricular region. Hemorrage in the lower abdomen was evident.

To test the behavior of the larval nematodes in avian hosts which do not harbor this parasite, i.e. an abnormal host, 5 juvenile Ring-neck Pheasants were innoculated with larval Eustrongylides. One of the 5 Pheasants died 12 hours post infectionem. Verminous peritonitis produced symptoms similar to

TABLE 1

EXPERIMENTAL AVIAN INFECTIONS WITH LARVAL *EUSTRONGYLIDES* SP.

Year	Host	N	% Infected	Total # Larvae Innoculated	Total # Larvae Recovered	% Recovered
1971	*Anas platyrhynchos*	15	73.3	71	25	35.2
1972	*Anas platyrhynchos*	14	57.1	94	14	14.9
	Larus argentatus	4	0.0	4	0	0.0
	Nycticorax nycticorax	1	100.0	4	4	100.0
	Phasianus colchicus	5	80.0	16	11	68.7
1973	*Anas platyrhynchos*	11	81.8	232	26	11.2
1974	*Anas platyrhynchos*	34	73.5	340	84	24.7
	Larus argentatus	2	50.0	50	2	4.0

those exhibited by the experimental Black-crown Night Heron. Larvae were recovered from tissue sites in the pectoral muscles, neck muscles, proventriculus and liver, mesenteries, and from the wall of the lower intestine. Considerable tissue damage and hemorrhage was evident. Two Pheasants exhibited the described symptoms but did not die. Autopsied at 68 hours and 13 days post infectionem, respectively, degenerating larval worms were recovered from the proventricular walls and the liver. The larvae failed to establish infections in the remaining two birds.

Herring Gulls proved largely refractory to infection. A total of two degenerating worms were removed from one of the six gulls innoculated.

Of all the experimental infections attempted in avian hosts, mature and patent infections were established only in Mallards. Male worms underwent their last molt 7-9 days post infectionem. After 16 days, all male worms recovered were in a state of degeneration. Female worms molted 7-16 days post infectionem. In patent infections, fertilized eggs were voided by gravid females 16-24 days post infectionem. The thick-shelled oval eggs, voided with the feces, were in the one cell stage of development. Polar thinnings and numerous depressions were noted on the shell surface.

Overall, Mallards were refractory to infection. In most instances where infections were established, the worms died and degenerated. Mature and patent infections were established in 3 of the 9 birds infected during 1973. One gravid worm was recovered from the mallards infected during 1974. In an attempt to obtain more adult males and gravid females, 10 male and 10 female worms were innoculated into the mallards utilized during 1973 and 1974. In 1973, only 2 males and 2 females were used. Increasing the larval dosage resulted in a lower recovery rate. No visible clinical symptoms of peritonitis or other complications of parasitism were noticeable in the Mallards prior to sacrifice and necropsy.

Egg cultures were started from those eggs recovered from gravid female worms. Eggs were taken from the uterus and vagina and placed in distilled water at 15, 20, and 25°C. In the egg culture maintained at 25°C, development of the embryonic nematode reached the tadpole stage in most eggs 20 days post necropsy. Twenty days post necropsy, a few eggs remained in the morula stage of development. After 61 days, development ranged from the tadpole stage to the fully larvated stage within

the egg shell. Most eggs were fully larvated. A few eggs failed to develop and died. Eggs removed from one gravid worm failed to develop. In the egg culture maintained at 20°C, development at 134 days or 4-1/2 months post necropsy still ranged from morula to fully larvated eggs. The majority of eggs were dead after 161 days. In the egg culture maintained at 15°C, development was much slower. At 214 days or 7 months post necropsy, no eggs had become fully larvated. Instead, they appeared to have ceased development at the morula to the tadpole stage. Figures 1 and 2 show fully larvated eggs at 61 days post necropsy.

When fish-eating birds ingest fish infected with larval Eustrongylides sp., a mature infection occassionally results. To test the behavior of the larvae in small perch that are ingested by predators other than fish-eating birds and to observe the nature of the infection established, larvae from perch encapsulations were intubated into a variety of predators. The results of these experimental infections are presented in Table 2.

A few larvae are found free in the mesenteric tissue or in the body musculature of Yellow perch. Occassionally bright, red fourth-stage larvae were found burrowing through the body wall from their capsules in the mesenteries. This behavior suggested that burrowing might be a response to a temperature cue. A temperature increase might trigger the larvae to exit their capsules. Table 3 summarizes the results of this series of experiments.

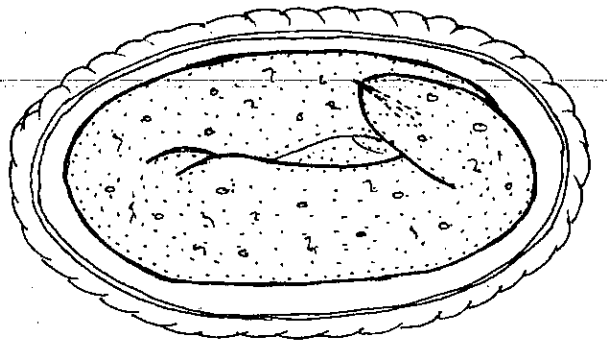


Figure 1

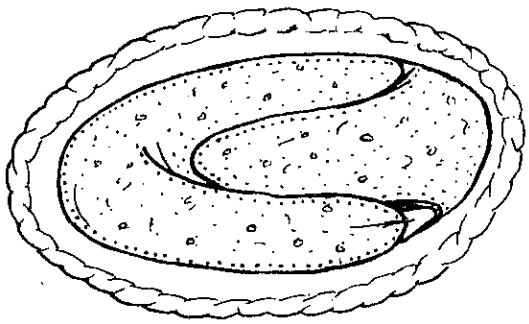


Figure 2

TABLE 2

BEHAVIOR OF LARVAL EUSTRONGYLIDES TUBIFEX IN POIKILOTHERMOUS PREDATORS

Experimental Host	N	No. Larvae Innoculated	No. Larvae Recovered	Site of Reinfection
Fish				
Ambloplites ruprestris Rockbass	1	5	2	Myomeres (1); intestine (1)
Ictalurus punctatus Channel Catfish	5	24	19	body cavity (12); intestine (1); liver (1); stomach wall (5)
Lepomis gibbosus Pumkinseed Sunfish	3	20	9	body cavity (3); body wall (5); stomach (1)
Lepomis macrochirus Bluegill Sunfish	2	13	8	myomeres (6); heart (1); stomach (1)
Micropterus dolomieui Smallmouth Blackbass	2	10	2	mesenteries (2)
Pomoxis nigromaculatus Black Crappie	2	10	0	
Amphibians				
Rana pipiens Grass Frog	7	26	17	body cavity (17)
Reptiles				
Chelydra serpentina Snapping Turtle	2	20	7	stomach wall (3); stomach lumen (4)
Emys blandingi Blanding's Turtle	1	10	6	liver (1); stomach wall (1); stomach lumen (4)
Trionyx spinifer Spiny Soft-shelled Turtle	4	40	23	body cavity (11); liver (9); stomach wall (3)
Terrepene carolina Box Turtle	1	10	7	liver (2); intestinal wall (1); stomach wall (4)

TABLE 3

EMERGENCE BEHAVIOR OF LARVAL *Eustrongylides* IN RELATION TO TEMPERATURE

Temperature	Length of Time	Worms Encapsulated	Worms Initiating Emergence from Encapsulations	Worms Penetrating Body Wall	Worms Emerging on Surface of Fish	Total Number of Worms	Total Number of Infected Fish
25° C	4 hr	18	00	13	01	32	11
33° C	3 hr	15	04	00	00	19	11
33° C	4 hr	11	01	06	00	18	11
36° C	4 hr	02	08	16	00	26	10
40° C	3 hr	00	02	23	03	28	10
44° C	3 hr	00	05	12	27	44	17
50° C	3 hr	00	00	6	42	48	14
60° C	3 hr	00	01	13	06	20	10

Descriptive Studies:

Prior to this study, the specific identity of the larval Eustrongylides sp. infecting fish in Lake Erie was unknown. Study of specimens removed from experimentally infected Mallards and naturally infected aquatic birds collected in the western basin region of Lake Erie indicates these organisms refer to Eustrongylides tubifex (Nitzsch, 1819) Jagerskiold, 1909. The adult male and female of the species are redescribed on the basis of specimens from experimental infections in Mallards. Specimens have been compared with specimens of other species on file in the USNM Helminthological Collection. Representative specimens collected during this study have been filed in the Collection.

Jagerskiold redescribed the type species, E. tubifex, from loons (Gavia sp.) on the basis of one male and five females in his monograph in 1909. Russian investigators described the species from loons. A more complete redescription, based on American specimens, of both larval and adult specimens is presented.

Description of Larval Eustrongylides tubifex: Larval Eustrongylides were recovered from Yellow Perch autopsied for the population phase of this study in the Western Basin of Lake Erie. Upon investigation it was found that the larvae for descriptive purposes could be separated into two distinct morphological groups. Karmanova (1965) in experimental studies of the life cycle of E. excisus on the Volga delta of the U.S.S.R. had infected fish with third-stage larvae from freshwater oligochaetes. In 1968, Karmanova claimed a molt to the fourth-stage occurred in larvae between 30.10-31.69 mm long within thin encapsulations or in the free condition in the body cavity of the fish. She (1968) asserted that early fourth-stage larvae either migrated to the stomach musculature or remained in the body cavity in flat spirals within encapsulations of host fish tissue. Third-stage larval E. excisus, 7.99-30.00 mm long, were described by Karmanova (1968) from Volga delta fish Neogobuis and Rutilus. Though fourth-stage larval E. excisus were reported by Karmanova from the fish, they were not, however, described.

In view of the findings of Karmanova (1965, 1968), the two larval groups of E. tubifex recovered from Perca flavescens in Lake Erie are arbitrarily considered to be third- and fourth-stage larvae through no molt was observed between the stages. The molting process appears complex and requires further study. Whereas, the larvae designated third-stage have one cuticle the larvae designated fourth-stage gradually acquire a double cuticle toward the end of their size range, the characters of the adult

stage seen within the last cuticle. The two larval groups of E. tubifex from yellow perch are here described as third-stage and fourth-stage larvae.

Description: Third-stage larval E. tubifex are localized in the body cavity of yellow perch and freshwater drum either lying free in the mesentery usually near the intestine or coiled up in a delicate thin walled capsule approximately 1.5-3.0 mm in diameter attached to the outside wall of the intestine. The larvae range in color from white to pale pink. All measurements were taken in millimeters.

Third-stage Larvae (Figures 3, 4, 6 and 7) (Based on 17 specimens): Length 9.18-32.19 mm (average 17.20 mm), maximum width 0.120-0.426 (0.216). Width at level of outer circle of papillae 0.048-0.015 (0.062). Width at posterior end 0.078-0.237 (0.115). Twelve head papillae arranged in two circles of six each, two lateral and four submedian. Papillae digitiform. Outer circle papillae 0.006-0.015 (0.009) wide by 0.006-0.009 (0.007) long. Inner circle papillae 0.003-0.006 (0.004) wide and 0.003-0.009 (0.006) long. Nerve ring 0.081-0.120 (0.104) from anterior end. Mouth cavity 0.054-0.096 (0.079) long. Two rows of lateral papillae prominent at anterior and posterior ends, not always encountered in middle of worm. Esophagus 3.36-8.70 (5.32) long. Junction of esophagus with intestine by three lipped cardial valve. Mesenteric connectives join body wall to intestine and rectum. Anterior of rectum to posterior end 0.195-0.525 (0.297) long. Single terminal opening in both sexes. One layer of cuticle. Sexual dimorphism present. Female tail symmetrical. Male tail asymmetrical, curved ventrally. Genital tube single. Female genital tube, in form of loop, arises and terminates at posterior end. Rudiments of male genital tube ventral, extends anteriorly. Spicule sheath dorsal. Spicule not yet formed. Rectum, genital tube, and spicule sheath empty into short cuticular cloaca.

Description: Fourth-stage larval E. tubifex are usually localized in the body cavity of yellow perch within yellowish brown granular mesentery capsules ranging in diameter from 4-15 mm (average 7 mm). Usually 1-2 larvae are found lying coiled in a flat spiral within these capsules. Up to 7 larvae were found in a single huge granular encapsulation 30 mm in diameter. Larvae on occasion are encountered burrowing into the body musculature or through the abdominal viscera. The number of larvae encountered burrowing increases as the time since death of the fish to time of autopsy increases. The larvae range in color from dark pink to dark red. Occasional aberrant white larvae are encountered. All measurements are in millimeters.

EXPLANATION OF PLATE 1

Figure 3. Third-stage larva, anterior end, ventral view, showing nerve ring, head papillae, and lateral papillae.

Figure 4. Third-stage larva female, posterior end, lateral view, showing looped genital tube, rectum, and mesenteric connectives attaching intestine and rectum to body wall.

Figure 5. Fourth-stage larva, en face view, showing head papillae and mouth aperture. (en face oriented with a lateral papilla uppermost).

Figure 6. Third-stage larva, lateral view, showing junction of esophagus and intestine with three lipped cardial valve, and beginning of mesenteric connectives.

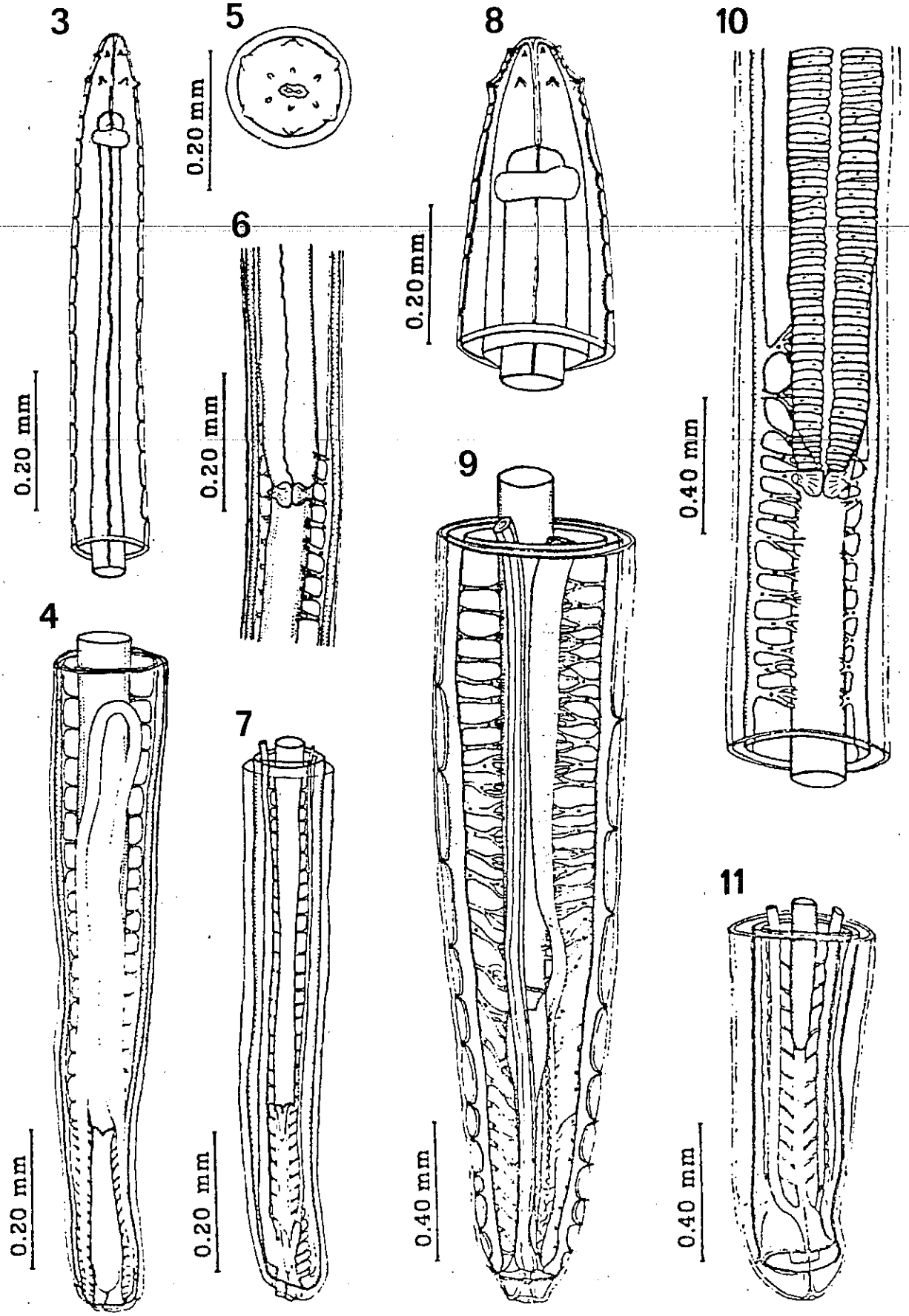
Figure 7. Third-stage larva male, posterior end, lateral view, showing rudiments of dorsal spicule sheath and ventral genital tube emptying with rectum into cloaca.

Figure 8. Fourth-stage larva, anterior end, ventral view, showing lateral papillae extending anterior to outer circle lateral head papillae.

Figure 9. Late fourth-stage larva female, posterior end, ventral view, showing double cuticle, lateral papillae, mesenteric connectives, ventral vagina with terminal vulva, and cuticular posterior extension.

Figure 10. Fourth-stage larva, lateral view, showing junction of esophagus and intestine by three lipped cardial valve, and glandular cells of posterior esophagus.

Figure 11. Late fourth-stage larva male, posterior end, lateral view, showing mesenteric connectives to intestine and rectum, dorsal spicule sheath, ventral genital tube, forming bursa with cuticular fringed border, and double cuticle.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Fourth-stage Larvae (Figures 5, 8-11) (38 specimens): Length 32.50-93.37 mm (average 63.45 mm), maximum width 0.337-0.668 (0.502). Width at level of outer circle of papillae 0.098-0.154 (0.123). Width at posterior end 0.147-0.334 (0.290). Mouth cavity 0.082-0.196 (0.136) long. Nerve ringe 0.131-0.214 (0.176) from anterior end. Head papillae in two circles of six each, two lateral and four submedian. Outer circle head papillae 0.008-0.023 (0.018) wide by 0.005-0.020 (0.013) long. Inner circle papillae 0.005-0.016 (0.010) wide by 0.008-0.016 (0.011) long. Two rows of lateral papillae on anterior and posterior ends prominent, not always encountered in middle of worm. Esophagus 7.02-26.01 (15.99) long. Grandular cells of esophagus notably large near junction of esophagus with intestine. Cardial valve three lipped. Mesenteric connectives attach intestine and rectum to body wall. Anterior of rectum to posterior end 0.804-0.891 (0.848) long in male larva. Anterior of rectum to posterior end 0.871-0.988 (0.935) long in female larva. Sexual dimorphism present. Female tail symmetrical, truncated. Male tail asymmetrical, curved ventrally. Beginnings of cup shaped muscular bursa with cuticular fringed border and internal genital "cone" evident within outer cuticle. Both sexes of fourth-stage larvae with double cuticle, especially evident at anterior and posterior ends. Genital tube single. Female genital tube arises from attachment to rectum wall, extends anteriorly, loops, and eventually gives rise to a posteriorly directed thick walled vagina, 5.34-10.68 (6.71) long. Vagina terminates with vulva at beginning of cuticular extension of molting cuticle at posterior end. Male genital tube arises blindly in posterior half of worm, convolutes, ends in ventral tube emptying into cloaca. Muscular-like genital "cone" noted deep in the bursal cup. Occasional papillae encountered on inner surface of bursa.

Description of Adult Male and Female Eustrongylides tubifex (Nitzsch, 1819) Jagerskiold, 1909. Eustrongylides tubifex (Nitzsch, 1819) Jagerskiold, 1909. Jagerskiold (1909) in his monograph described the new genus Eustrongylides. Utilizing five females and one male from the Wein Museum of Natural History taken from Colymbus septentrionalis (=Gavia stelata), the red-throated loon, and one male specimen from his personal Finland collection taken from C. arcticus (=G. arctica), Jagerskiold redescribed Strongylus tubifex Nitzsch, 1819 and placed it in the new genus Eustrongylides. Eustrongylides tubifex had appeared under various generic names. Jagerskiold in studying material from various museums created partial synonymies of different species and considered some of the past descriptions to be actually assembly species in their composition.

The adult forms of Eustrongylides recovered from my experimental infections of mallards, Anas platyrhynchos, were compared with the U.S. Museum Helminthological Collection specimens of E. tubifex and E. ignotus and also with the descriptions of the different species of Eustrongylides as written by Jagerskiold (1909) and discussed by Karmanova (1968). The species of nematode found in the experimental mallard infections was established to be E. tubifex (Nitzsch, 1819) Jagerskiold, 1909. Key characteristics used to elucidate the species were: (1) 12 papillae about the mouth, (2) the lack of a deep cleft in the male bursa, (3) the larger size of the outer circle papillae, (4) the fringed cuticular border of the male bursa, and (5) the straight spine-like structures terminating the inner circle papillae. This combination of characters distinguishes E. tubifex from the other species of the genus Eustrongylides.

In as much as the adult E. tubifex recovered from the experimental mallard infections were much larger in size (males 56.62-86.64 mm long and females 65.00-120.00 mm long) than those of Jagerskiold (1909) (male 34.0 mm long and females 35.0-44.0 mm long) a description of the adult male and female E. tubifex taken from Anas platyrhynchos appears desirable.

Description: Diotophymatida, Dioctophymatina, Dioctophymatoidea, Dioctophymatidae, Eustrongylidinae. Nematodes of large size. Body spindle shaped, gradually widening to middle. Narrower anterior and posterior ends hang free into lumen of proventriculus. Thick middle part of worm located within connective tissue tubular capsules on sides of serosal layer of proventriculus. Live nematodes a rose-beige color. Cuticle of anterior and posterior end thicker than cuticle of middle part of worm. Transverse striations of cuticle most prominent on anterior and posterior ends, that covering middle part of body appears smooth.

Mouth opening, lacking lips, of variable form from triangular to hexagonal to round depending on condition of nematode. Head with 12 papillae arranged in two concentric circles. Each circle consists of 6 papillae, two lateral and four submedian. Papillae of inner circle much smaller than huge papillae of outer circle. Each papilla of inner circle provided with straight spine-like structure at its summit. This spine comparatively long, with straight lateral walls in shape of cone. Each prominent papilla of outer circle, having wider base as well as greater height, provided at summit with button-like formation.

Two rows of lateral papillae arranged lengthwise along body of nematode, especially prominent on anterior and posterior ends of body. At head end lateral papillae often begin just behind laterally positioned papillae of outer circle of head papillae, but sometimes before.

Large nerve rings located near anterior end, just behind start of esophagus. Mouth capsule, cuticular and narrow, opens into esophagus. Esophagael lumen triangular in section. Esophagus widens posteriorly somewhat, sometimes anterior portion of esophagus winds or loops. At junction of esophagus into intestine, three lipped cardial valve. Intestine opens into cuticular rectum. Rectum terminated by anus in females. Rectum opens into cloaca in males. Numerous mesenteric connectives clearly visible adjoining body wall to intestine and rectum.

Male (Figures 12-15)(6 specimens): Length 56.62-86.84 mm (80.73), maximum width 0.97-1.07 (1.00). Esophagus 11.80-15.40 (13.59) long. Nerve ring 0.134-0.214 (0.172) from anterior end. Mouth cavity 0.066-0.134 (0.116) long. Width of body at level of outer circle of papillae 0.167-0.267 (0.200). Width of body at posterior end (bursal width) 0.400-0.467 (0.421). Outer circle head papilla width 0.042-0.050 (0.045). Outer circle papilla height 0.032-0.040 (0.036). Inner circle head papilla width 0.015-0.032 (0.025). Inner circle papilla height 0.016-0.026 (0.023)(including spine-like structure). Genital apparatus consists of genital tube, spicule with spicule sheath, and copulative bursa. Genital tube hologonic, single. Blind end of testes arises in posterior half of worm, highly convoluted. Genital tube opens into cloaca below rectum. Spicule very long, 8.6-10.2 (9.15), thin, ending in sharp needle-like point. Spicule located in muscular sheath. Proximal end of hollow spicule wider. Muscular sheath of spicule extends forward past proximal end of spicule. Copulative muscular bursa terminates at tail end. Inner surface of bursa with very small sensitive papillae. Bursal edge in form of delicate cuticular fringe. Cone-shaped structure or genital "cone" deep within bursal cup. Rectum plus cloaca length 0.817-1.340 (1.128).

Female (Figures 12, 16) (10 specimens): Length 65.0-120.0 mm (87.9), maximum width 1.65-4.30 (2.29). Esophagus 8.0-22.5 (18.8) long. Nerve ring 0.167-0.335 (0.221) from anterior end. Mouth cavity 0.067-0.134 (0.113) long. Width of body at level of outer circle of papillae 0.200-0.267(0.223). Width of body at posterior end 0.267-0.536 (0.394). Outer circle head papilla width 0.047-0.067 (0.054). Outer circle papilla height 0.020-0.048 (0.031). Inner circle head papilla width

EXPLANATION OF PLATE 2

Figure 12. Adult male, *en face* view, showing round mouth aperture and 12 head papillae arranged in 2 circles of 6 papillae each. (*en face* oriented with lateral papilla uppermost).

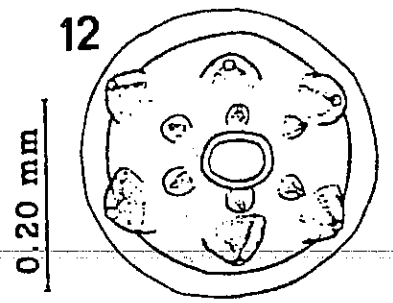
Figure 13. Adult male, lateral view, showing mesenteric connectives, dorsal spicular sheath with spicule, ventral genital tube, bursa with cuticular fringed border, and genital "cone" within bursal cup.

Figure 14. Adult male, anterior end, lateral view, showing head papillae, nerve ring, and body wall extensions to mouth cavity.

Figure 15. Late fourth-stage male molting to adult male 9 days *post infectionem*, lateral view, showing molted cuticle, extended spicule, muscular contraction of bursa, and looped genital tube over intestine and rectum.

Figure 16. Adult female, posterior end, ventral view, showing muscular vagina with eggs, terminal vulva ventral and terminal anus at end of rectum.

Figure 17. Egg, voided 16 days *post infectionem* in one cell stage, showing thick shell with surface depressions, and polar thinnings.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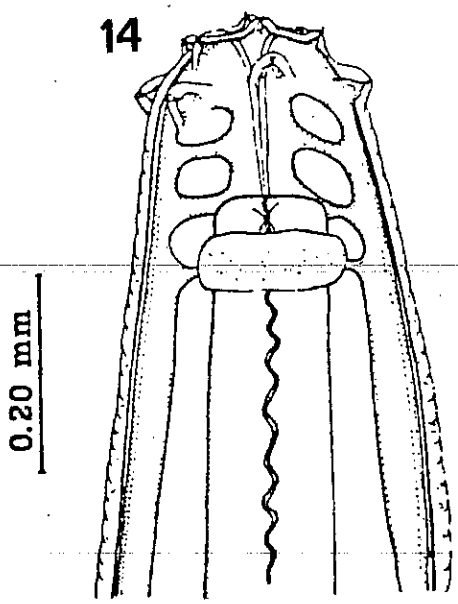
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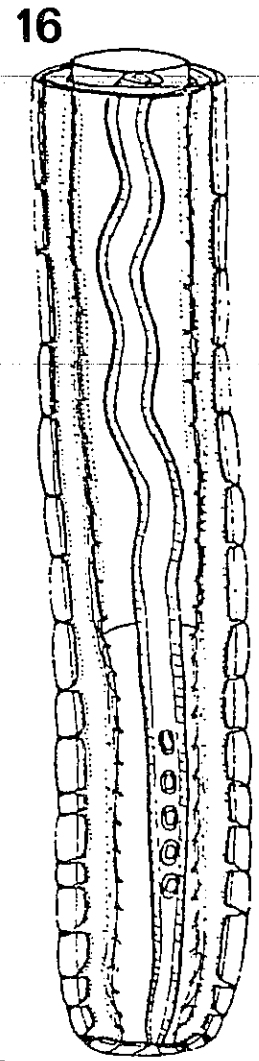
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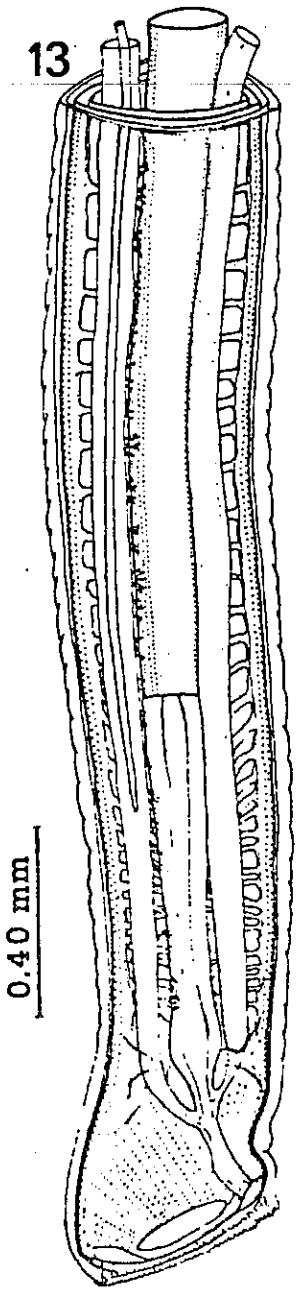
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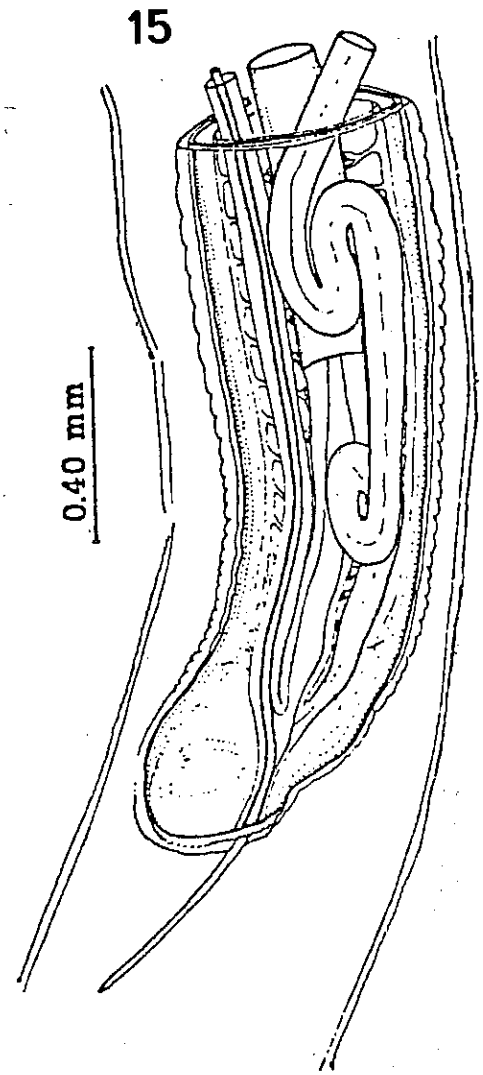
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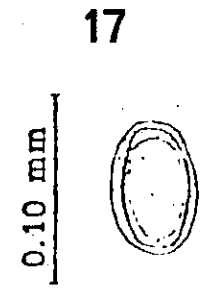

17
0.10 mm

0.023-0.054 (0.032). Inner circle papilla height 0.013-0.040 (0.021) (includes spine-like structure). Rectum length 0.17-1.27 (0.99). Posterior end blunt, rounded. Anus terminal. Genital tube single, hologonic. Vulva positioned terminally. Muscular vagina long, 18.76-21.43 (20.10).

Eggs of oval form with polar thinnings, thick shelled, surface with numerous depressions. Dimensions 0.030-0.038 (0.036) wide by 0.050-0.063 (0.059) long.

HOST: Anas platyrhynchos (experimental)

SITE OF INFECTION: Proventriculus

LOCALITY: Lake Erie

Pathology Studies:

Studies on the pathology induced in piscine intermediate hosts by larval worms and in avian definitive hosts by adult worms remain in preliminary stages. A series of tissue samples have been fixed in Bouins (alcoholic) fixative for later study. These samples include infected tissues from naturally and experimentally infected fishes and birds. A number of tissue samples have been prepared for microscopic examination. Detailed diagnosis of the tissue pathology exhibited by these samples has been curtailed by the termination of support for this effort.

Population Studies:

In order to understand the maintenance cycle of the parasitic nematode Eustrongylides tubifex, one needs to investigate the natural hosts harboring the infection and the degree of parasitism in these hosts through time. To effect this end, our investigations have focused on a variety of fish intermediate hosts and bird definitive hosts.

In a 1957 survey of fish parasites from the western basin of Lake Erie, Bangham (1972) found five species of fish harboring larval stages of Eustrongylides. Bangham reported 8 of 93 yellow perch (Perca flavescens), 2 of 88 freshwater drum (Aplodinotus grunniens), one of 58 logperch darters (Percina caprodes semi-fasciata), 2 of 3 mottled sculpins (Cottus bairdi), and 1 of 39 channel catfish (Ictalurus punctatus) infected. Much earlier, Bangham and

Hunter (1939) in an extensive survey of fish parasites in Lake Erie failed to note the presence of this parasite. Vendeland (1968) in a survey of Fresh-water Drum from the Bass Island region found 18 of 33 fish infected with larval Eustrongylides sp. Dechtiar (1972) surveyed 150 Yellow Perch from Lake Erie between 1961 and 1969. Three of the 150 fish examined were infected with larval Eustrongylides.

During this study, larval Eustrongylides tubifex have been removed from naturally infected Channel Catfish, Fresh-water Drum, Smallmouth Blackbass (Micropterus dolomieui), and Yellow Perch. Literature reports, especially from Eurasia (Karmanova, 1968), indicate Yellow Perch play the major role in the maintenance of Eustrongylides larvae in fish populations. The results of our examiniations reveal a similar pattern. The prevalence and intensity of infection in Yellow Perch exceeds that of the other species of fish found to harbor larvae. In addition, our investigations indicate the prevalence and intensity of infection of Yellow Perch currently exceeds that previously reported in Lake Erie.

The overall incidence of larval Eustrongylides tubifex in the 178 Channel Catfish examined as a part of this study was 14.6%. Channel Catfish were collected from June through September during 1973 and 1974. Table 4 summarizes the incidence and mean worm burden according to 8 arbitrarily designated size classes of catfish. Graph 1 depicts the incidence of parasitism in the 8 size classes. Larval E. tubifex were observed burrowing through the liver, kidneys, and ovaries. Larvae were not observed in the smallest size class of catfish. The highest incidence occurred in the two largest size divisions. Fresh encapsulations and burrowing third or fourth stage larvae were observed in Channel Catfish from size classes 7 and 8. Some encapsulations were sclerotized and contained dead worms.

The incidence of larval E. tubifex in 162 Fresh-water Drum examined during 1973 is summarized in Table 5. Age classes were derived from Herdendorf and Hair (1972). Due to a considerable amount of overlap, these designations are approximate. The incidence of infection increases with the size class of fish, as do the other parameters of infection. In the fish examined, the majority of E. tubifex were small third-stage larvae from hardened mesenteric encapsulations. Larvae from hardened encapsulations were invariably dead. Of 31 YOY examined, only 1 viable larvae was encountered. Of 87 I-year class examined, only 9 viable third-stage and 5 viable fourth-stage larvae were removed. Of 44 II+ year class examined, 2 viable third-stage larvae were encountered.

TABLE 4

INCIDENCE AND INTENSITY OF *Eustrongylides tubifex* CORRELATED WITH SIZE OF 178 CHANNEL CATFISH, (*Ictalurus punctatus*) DURING THE 1973 AND 1974 COLLECTION PERIODS

Size	Incidence	Mean Worm Burden	Standard Deviation
1) 0-10 cm	-	-	-
2) 10.1-15 cm	4.2%	1.00	-
3) 15.1-20 cm	17.9%	1.00	-
4) 20.1-25 cm	16.7%	1.00	-
5) 25.1-30 cm	5.9%	1.00	-
6) 30.1-35 cm	15.0%	1.00	-
7) 35.1-40 cm	25.0%	1.75	.96
8) 40.1-60 cm	27.3%	9.83	7.68

Graph 1. Incidence of Eustrongylides tubifex correlated to size of channel catfish ,

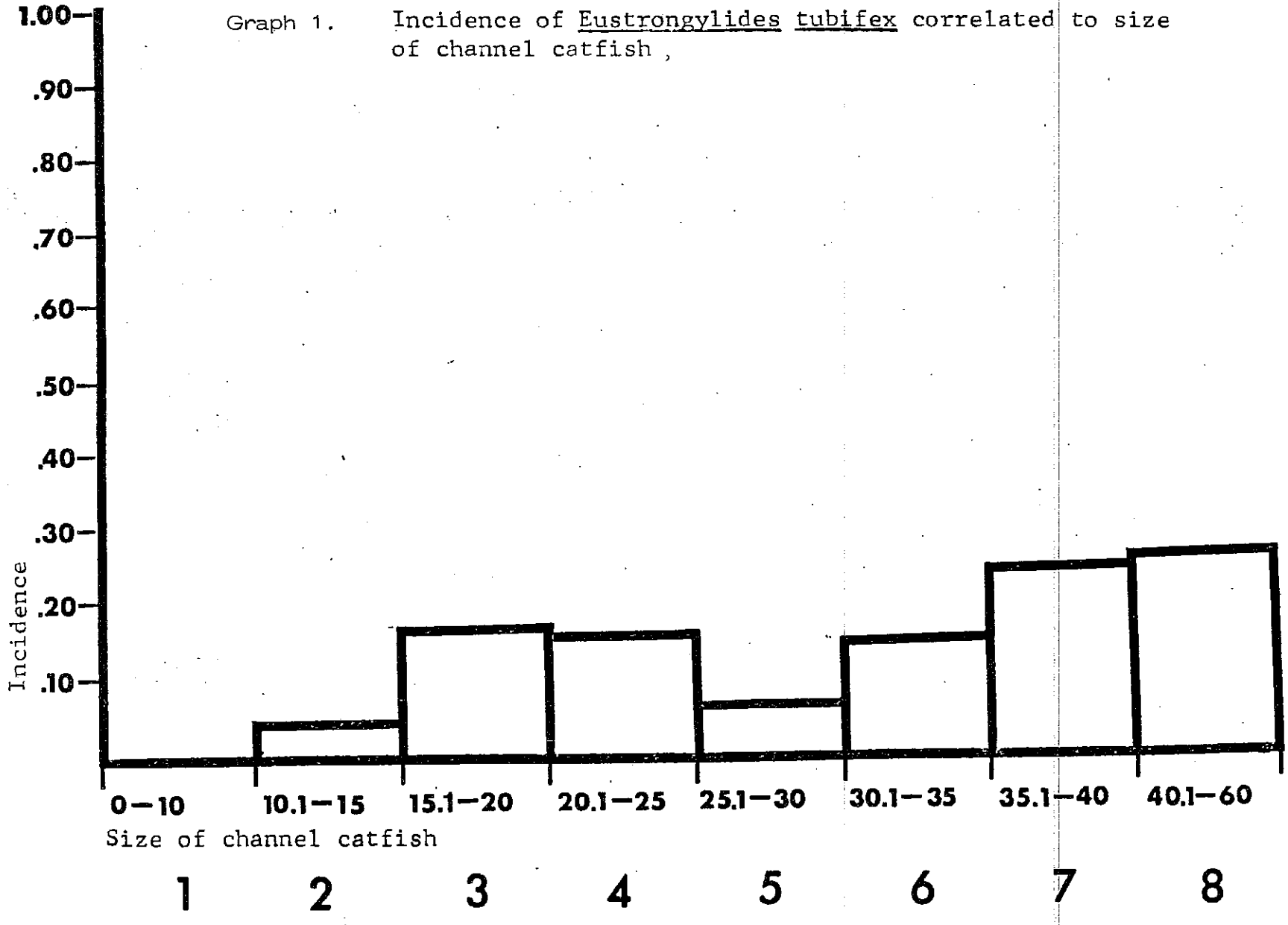


TABLE 5

SUMMARY POPULATION DATA OF *Eustrongylides tubifex** FROM THE FRESH-WATER DRUM (*Aplodinotus grunniens*) FROM JUNE, JULY, AUGUST, 1973

Age-Size Class of Fish	# Drum Necropsied	Incidence (%)	$\bar{X}_{inf}$	$\bar{X}_{pop}$	Range
YOY (3.8-13.0)	31	3.2	.03	1.0	0-1
	(1 larva recovered alive - third-stage)				
I (13.1-19.0)	87	54.0	1.3	2.4	0-7
	(9 larvae recovered alive - third-stage)				
	(5 larvae recovered alive - fourth-stage)				
II+ (19.1 and up)	44	97.7	6.6	6.7	0-33
	(2 larvae recovered alive - third-stage)				

* Majority of larvae recovered dead in hardened encapsulation, larvae appear to have been third-stage.

The incidence of larval E. tubifex in 60 Smallmouth Blackbass examined during 1973 is summarized in Table 6. The fish were arbitrarily divided into 3 size classes. The number of larvae recovered increased with the length of the fish. Only one of the 36 Blackbass under 30 cm long was infected with E. tubifex larvae. The majority of E. tubifex removed were fourth-stage larvae. Most were encapsulated in the serosal portion of the stomach wall. Some larvae were encapsulated in the mesentery and fatty tissue.

Yellow Perch were found to be the most extensively infected species of fish examined for larval E. tubifex during this study. As a result, efforts were concentrated on this species. The incidence and intensity of larval E. tubifex infections in Yellow Perch examined during the 1971-72, 1973 and 1974 collection periods is summarized in Table 7. Tables 8 and 9 present the monthly incidence and intensity of infection levels for the 1973 and 1974 collection periods respectively. Graphs 2, 3 and 4 depict the percentage infection of larval E. tubifex in each respective age-size class by month during the 1973 and 1974 collection periods. Graph 5 summarizes the percentage infection in the entire Yellow Perch population during the 1973 and 1974 collection periods. Graphs 6, 7 and 8 depict the mean worm burden of larval E. tubifex in each respective age-size class of fish by month during the 1972, 1973 and 1974 collection periods. Graph 9 summarizes the mean worm burden of larval E. tubifex by month in infected Yellow Perch during the 1973 and 1974 collection periods. Graph 10 summarizes the mean worm burden of larvae by month in the entire Yellow Perch sample during the 1973 and 1974 collection periods. The age-size classes were derived from Herdendorf and Hair (1972).

In order to ascertain the avian species which serve as definitive hosts in the western basin region of Lake Erie, a number of birds were collected and examined for E. tubifex infections. With the exception of a single Red-breasted Merganser (Mergus serrator) taken in 1972, all were collected in 1974. The results of these examinations are summarized in Table 10. In addition, 90 proventriculi from a total of 3 species of herons collected during 1972 and 1973 at West Sister Island and Winous Point were made available for examination through the courtesy of Dr. R. D. Curnow and Mr. R. Hoffman of the Ohio Cooperative Wildlife Unit. The extent and diversity of helminth parasitism in the proventriculi of these birds is summarized in Tables 11 and 12.

TABLE 6

SUMMARY POPULATION DATA OF *Eustrongylides tubifex* FROM SMALLMOUTH BLACKBASS (*Micropterus dolomieui*) FROM JUNE, JULY, AUGUST, 1973

Size Class	# Bass Necropsied	% Infection	$\bar{X}_{inf}$	$\bar{X}_{pop}$	Range
14.8-30.0 cm	36	2.78	*7.00	.19	0-7
30.0 - 40.0 cm	18	38.89	1.71	.67	0-4
40.1-50.0 cm	6	100.0	7.83	7.83	1-26

* One bass (23.6 cm long) had 7 L_3 *E. tubifex* in mesentery encapsulation. Rest of fish negative.

TABLE 7

SUMMARY POPULATION DATA OF LARVAL *Eustrongylides tubifex* FROM YELLOW PERCH (*Perca flavescens*) DURING THE 1971-72, 1973, 1974 COLLECTION PERIODS

Entire Sample	1971-1972	1973	1974
# Perch	1056	1413	1075
% Infection	37.0	32.27	45.61
$\bar{X}_{inf}$	1.879	1.789	2.235
$\bar{X}_{pop}$	0.696	0.577	1.012
Range	0-15	0-11	0-21
S.D. of $\bar{X}_{pop}$	1.352	1.183	1.805
Y-O-Y (4.4-11.9 cm)			
# Perch	239	315	121
% Infection	10.0	11.75	9.25
$\bar{X}_{inf}$	1.167	1.297	1.048
$\bar{X}_{pop}$	0.117	0.152	0.099
Range	0-4	0-3	0-2
S.D. of $\bar{X}_{pop}$	0.403	0.461	-

TABLE 7 (CON'T.)

SUMMARY POPULATION DATA OF LARVAL *Eustrongylides tubifex* FROM YELLOW PERCH (*Perca flavescens*) DURING THE 1971-72, 1973, 1974 COLLECTION PERIODS

I+ (12.0-17.5 cm)	1971-1972	1973	1974
# Perch	373	539	441
% Infection	41.8	32.23	54.37
$\overline{X}$ inf	1.589	1.672	2.259
$\overline{X}$ pop	0.665	0.540	1.237
Range	0-7	0-10	0-20
S.D. of $\overline{X}$	1.007	1.051	-
II+ (17.6-34.6 cm)			
# Perch	444	559	513
% Infection	47.8	43.83	50.24
$\overline{X}$ inf	2.175	1.947	2.361
$\overline{X}$ pop	1.034	0.853	1.159
Range	0-15	0-11	0-21
S.D. of $\overline{X}$ pop	1.765	1.476	-

TABLE 8

POPULATION DATA STATISTICS OF LARVAL E.tubifex INFECTION IN YELLOW PERCH, 1973

Entire Sample	March	May	June	July	Aug	Sept	Oct
$\bar{X}_{pop}$	1.760	0.400	0.869	0.455	0.461	0.751	0.812
$\bar{X}_{inf}$	3.385	1.200	1.893	1.699	1.610	2.131	1.673
% inf	52.00	33.33	45.90	26.78	28.65	35.26	48.51
S. D.	2.891	0.632	1.437	1.009	0.891	1.611	1.164
# Perch	25	* 15	122	422	555	173	101
YOY							
$\bar{X}_{pop}$	-	-	-	0.061	0.105	0.205	0.800
$\bar{X}_{inf}$	-	-	-	1.143	1.000	1.286	1.667
% inf	-	-	-	5.34	10.48	15.91	48.00
# Perch	-	-	-	131	105	44	25
I							
$\bar{X}_{pop}$	-	-	0.950	0.456	0.432	0.754	0.853
$\bar{X}_{inf}$	-	-	2.235	1.523	1.609	1.704	1.706
% inf	-	-	42.50	29.93	26.85	44.26	50.00
# Perch	-	-	40	147	257	61	34

Continues

TABLE 8 (CON'T.)

POPULATION DATA STATISTICS OF LARVAL E. tubifex INFECTION IN YELLOW PERCH

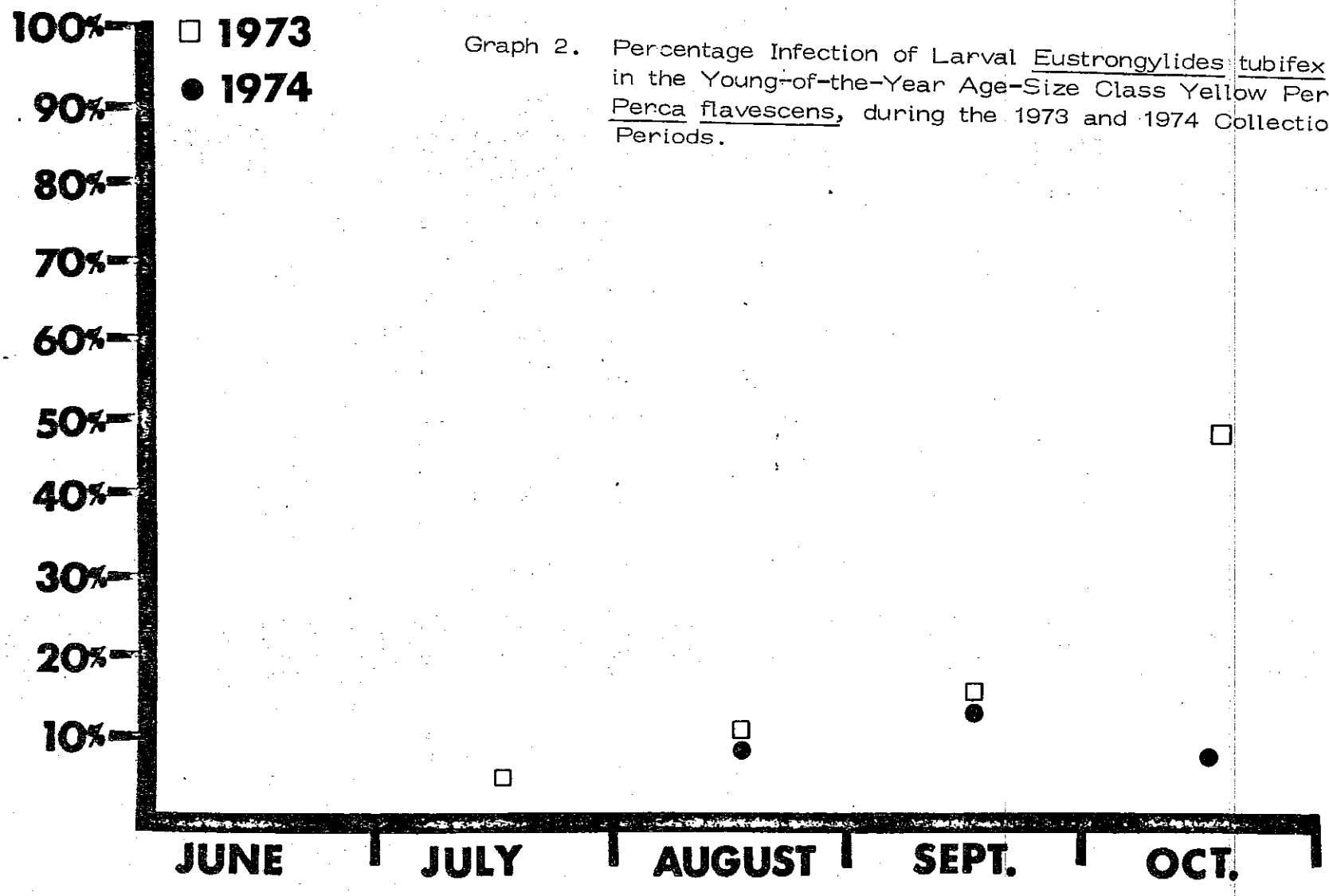
II+	March	May	June	July	Aug	Sept	Oct
$\bar{X}_{pop}$	1.760	0.400	0.944	0.813	0.694	1.103	0.786
$\bar{X}_{inf}$	3.385	1.200	1.744	1.887	1.696	2.778	1.650
% inf	52.00	33.33	54.17	43.06	40.93	39.71	47.62
# Perch	25	* 15	72	144	193	68	42

* May Sample of 15 yellow perch too small to compare with values obtained in remaining months.

TABLE 9

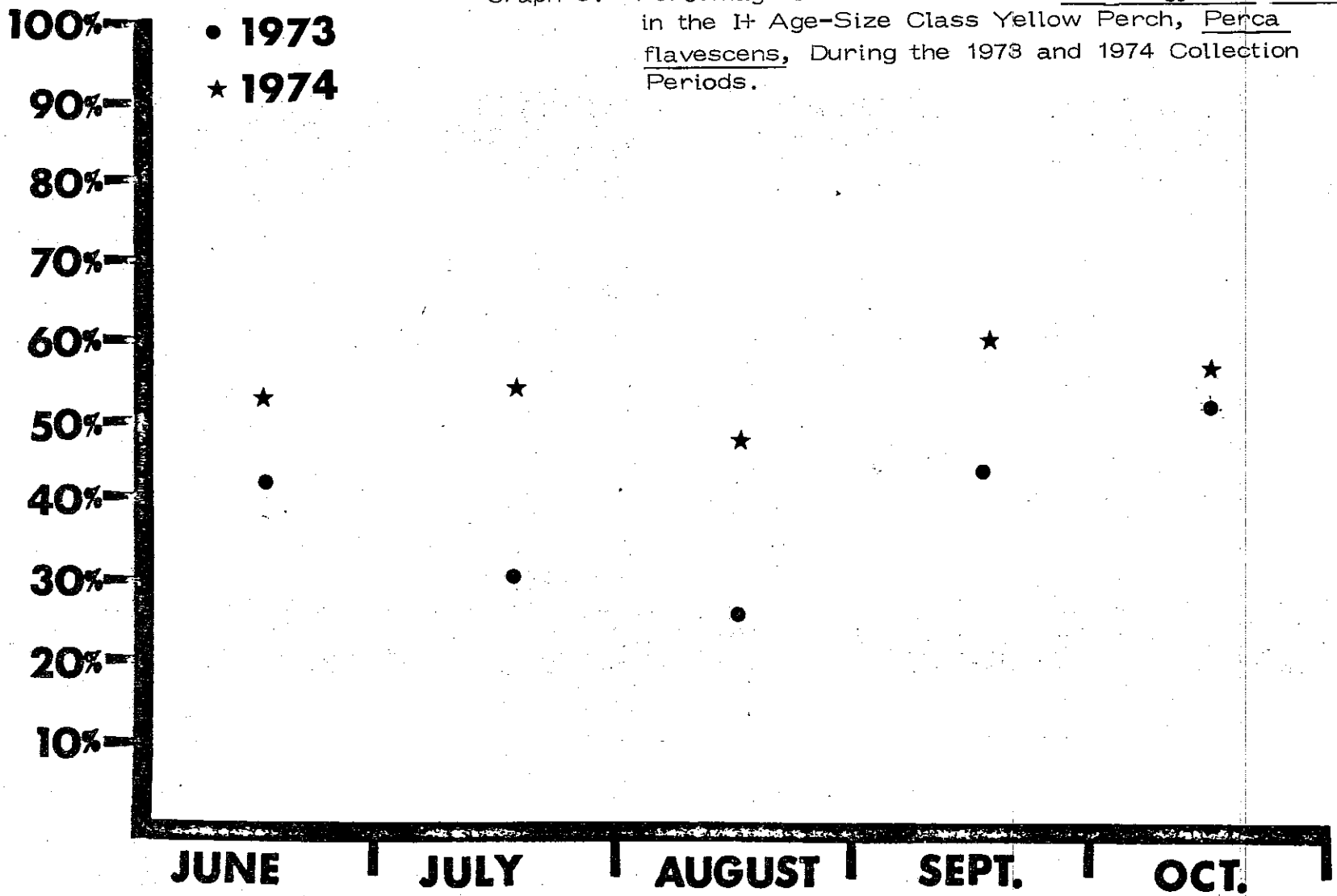
POPULATION DATA STATISTICS OF LARVAL *E. tubifex* INFECTION IN YELLOW PERCH, 1974

Entire Sample	June	July	August	September	October
$\bar{X}$ pop.	0.761	0.967	1.119	1.054	1.157
$\bar{X}$ inf.	1.591	2.008	2.536	2.440	2.600
% inf.	47.83	48.15	44.07	43.60%	44.44
S.D.	1.075	1.628	2.009	2.027	2.285
# Perch	184	270	295	250	135
0+ Age Class					
$\bar{X}$ pop.	0.478	0.250	0.081	0.146	0.069
$\bar{X}$ inf.	2.20	1.125	1.000	1.143	1.00
% inf.	21.74	22.22	9.11	12.73	6.90
# Perch	23	36	37	55	29
I+ Age Class					
$\bar{X}$ pop.	0.798	1.145	1.135	1.589	1.517
$\bar{X}$ inf.	1.512	2.088	2.368	2.581	2.750
% inf.	52.53	54.84	47.90	61.43	55.17
# Perch	99	124	119	70	29
II+ Age Class					
$\bar{X}$ pop.	0.807	1.000	1.381	1.176	1.429
$\bar{X}$ inf.	1.613	2.037	2.743	2.496	2.619
% inf.	50.00	49.09	50.36	47.20	54.55
# Perch	62	110	139	125	77

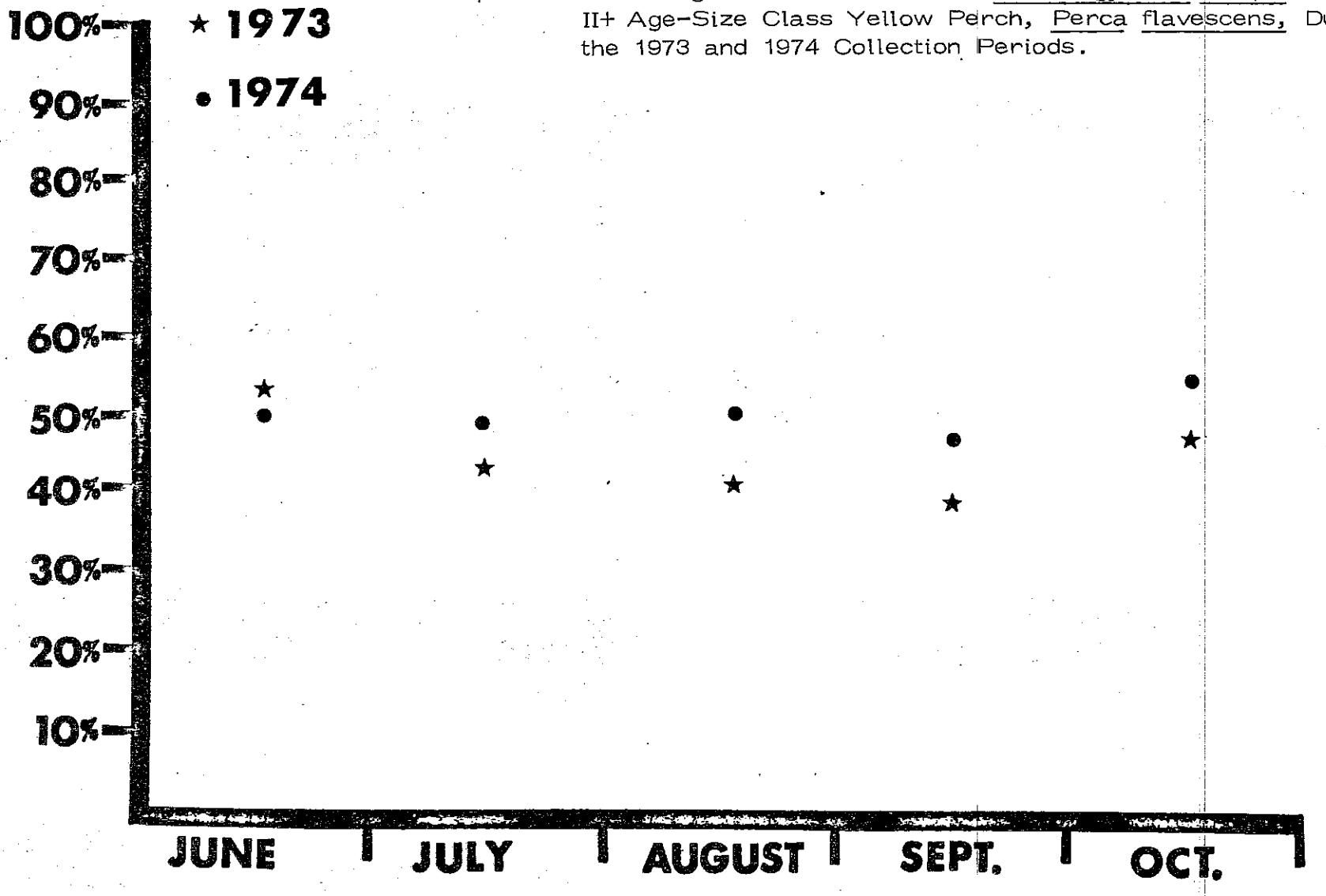


Graph 2. Percentage Infection of Larval Eustrongylides tubifex in the Young-of-the-Year Age-Size Class Yellow Perch, Perca flavescens, during the 1973 and 1974 Collection Periods.

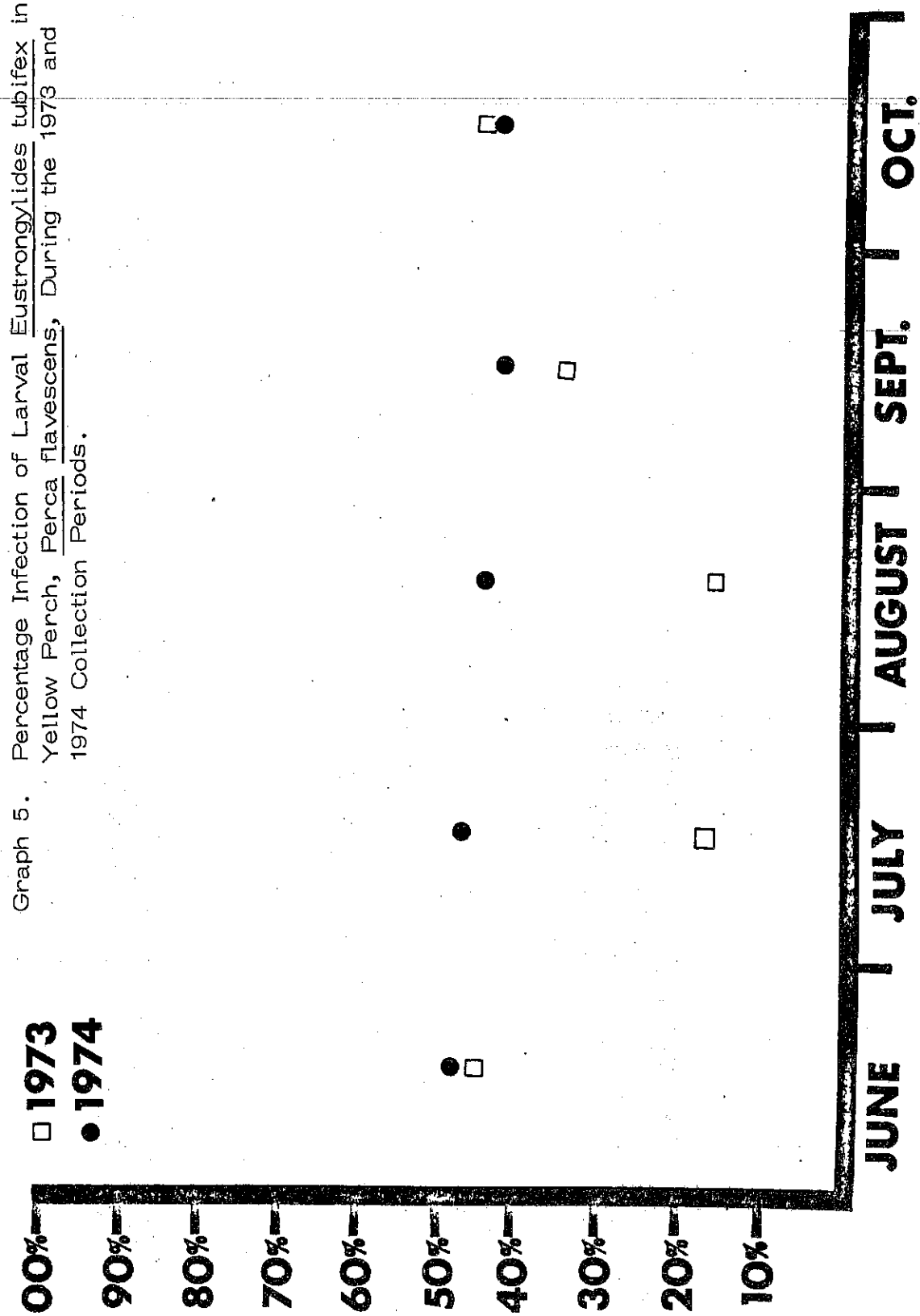
Graph 3. Percentage Infection of Larval Eustrongylides tubifex in the I+ Age-Size Class Yellow Perch, Perca flavescens, During the 1973 and 1974 Collection Periods.



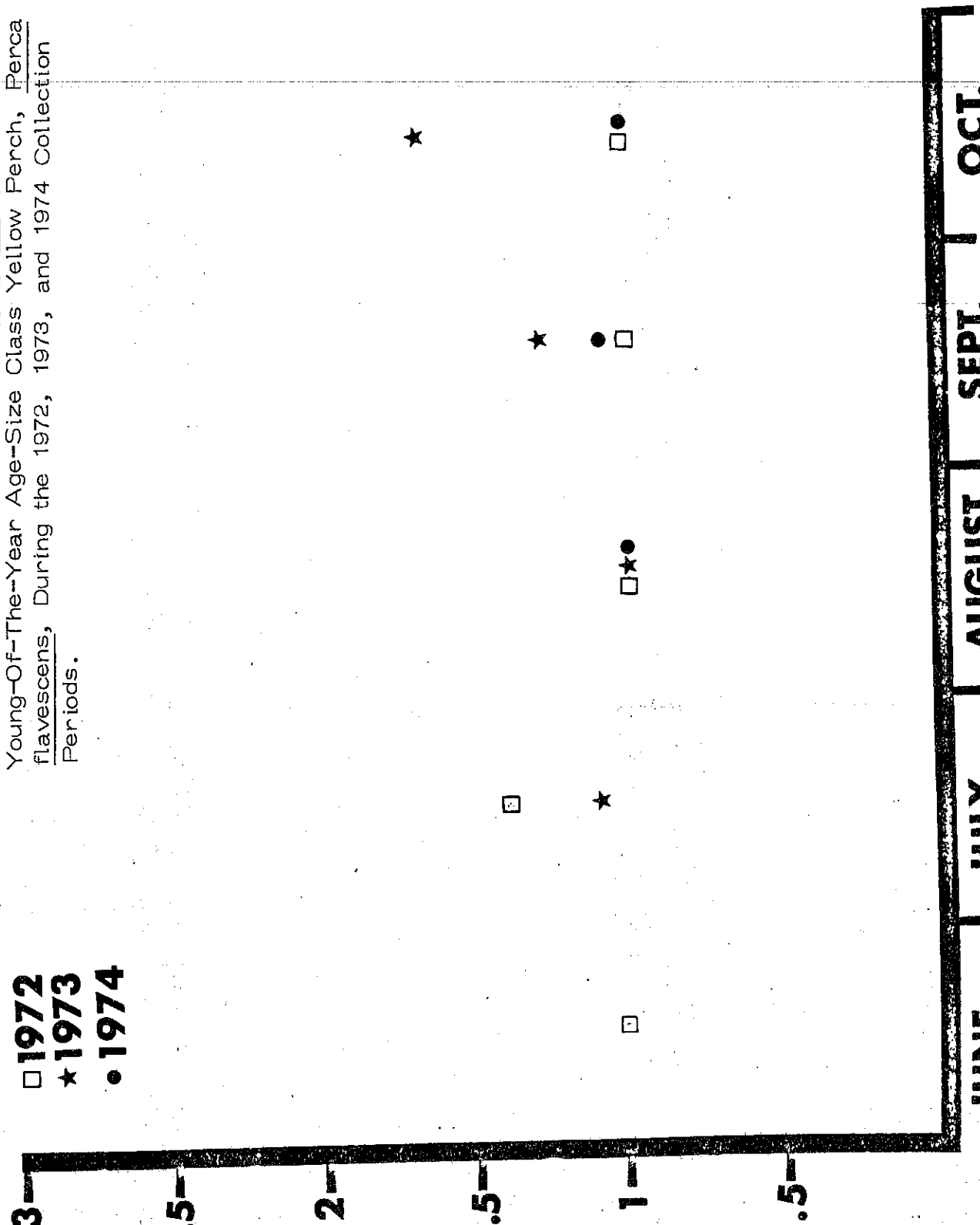
Graph 4. Percentage Infection of Larval Eustrongylides tubifex in the II+ Age-Size Class Yellow Perch, Perca flavescens, During the 1973 and 1974 Collection Periods.



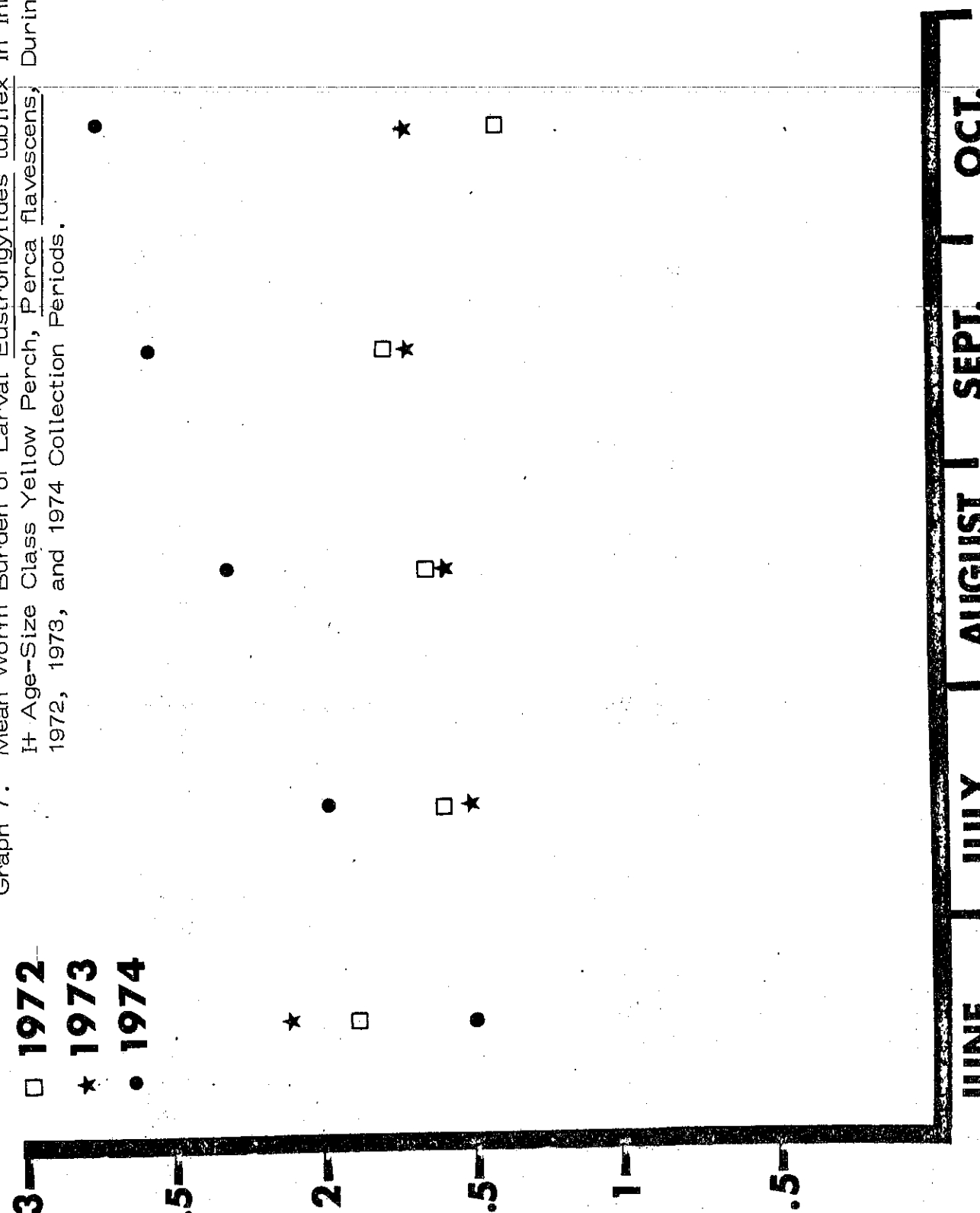
Graph 5. Percentage Infection of Larval Eustrongylides tubifex in Yellow Perch, Perca flavescens, During the 1973 and 1974 Collection Periods.



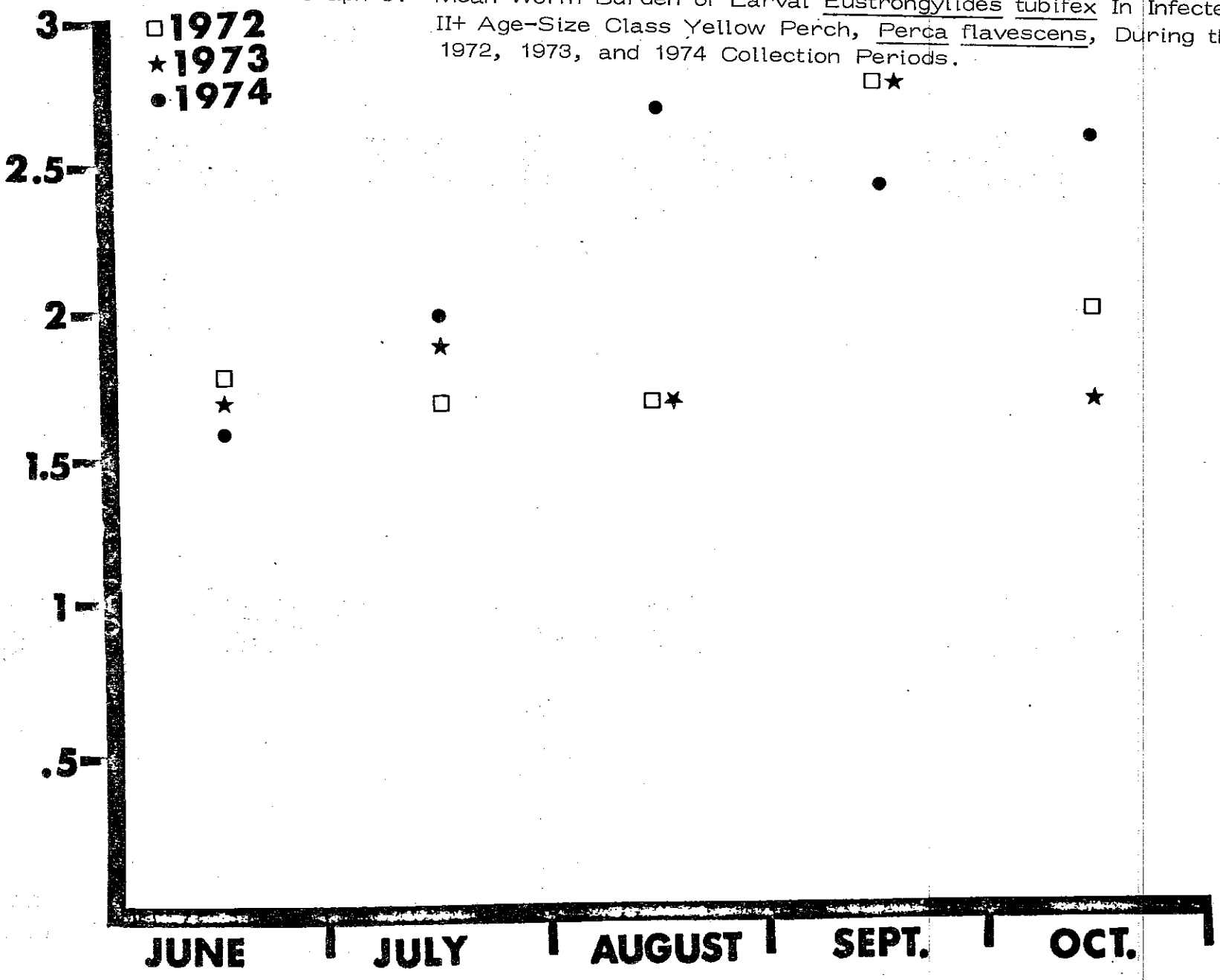
Graph 6. Mean Worm Burden of Eustrongylides tubifex in Infected Young-Of-The-Year Age-Size Class Yellow Perch, Perca flavescens, During the 1972, 1973, and 1974 Collection Periods.



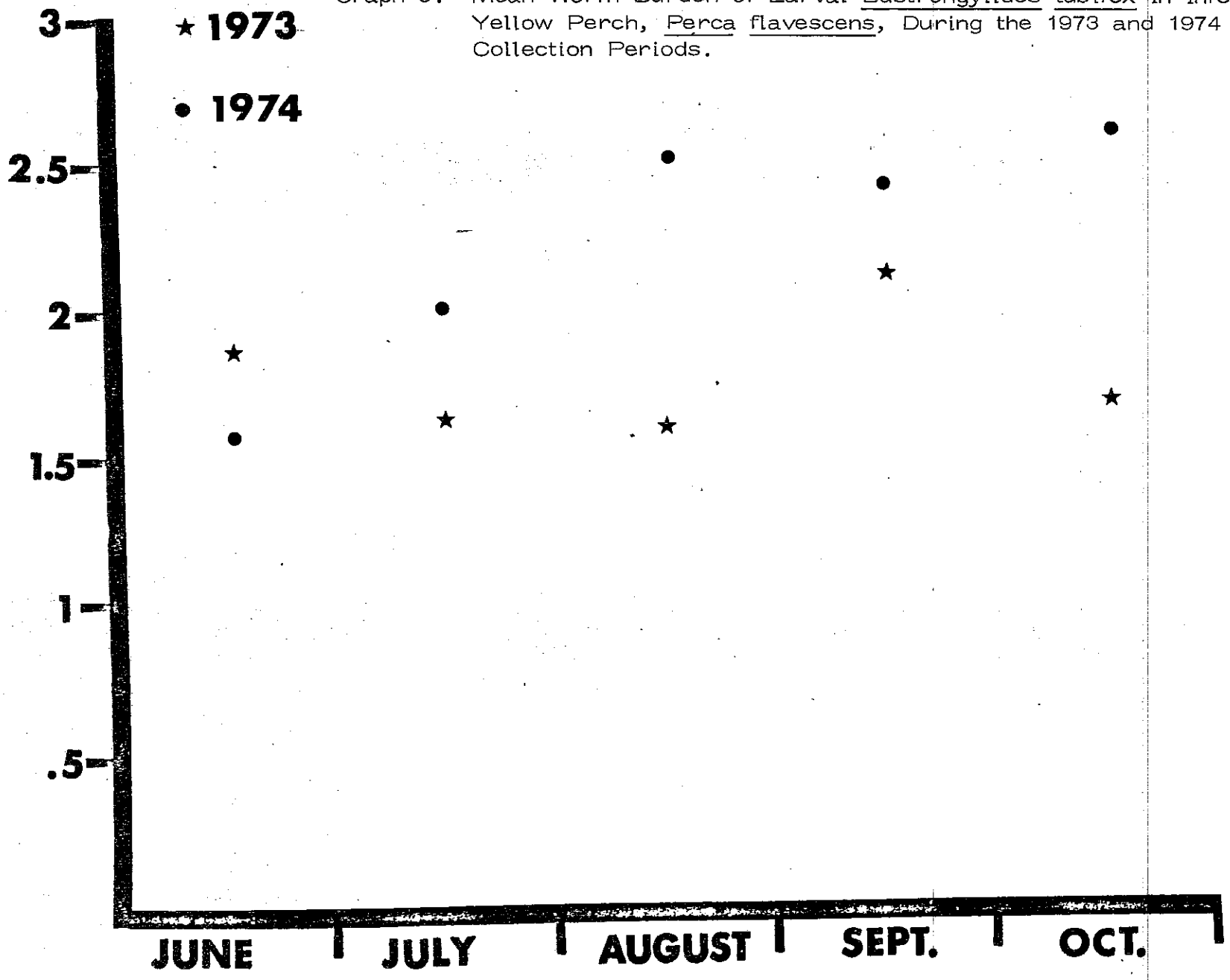
Graph 7. Mean Worm Burden of Larval Eustrongylides tubifex In Infected 1+ Age-Size Class Yellow Perch, Perca flavescens, During the 1972, 1973, and 1974 Collection Periods.



Graph 8. Mean Worm Burden of Larval Eustrongylides tubifex In Infected II+ Age-Size Class Yellow Perch, Perca flavescens, During the 1972, 1973, and 1974 Collection Periods.



Graph 9. Mean Worm Burden of Larval Eustrongylides tubifex In Infected Yellow Perch, Perca flavescens, During the 1973 and 1974 Collection Periods.



Graph 10. Mean Worm Burden of Larval Eustrongylides tubifex In The Entire Yellow Perch, Perca flavescens, Population During the 1973 and 1974 Collection Periods.

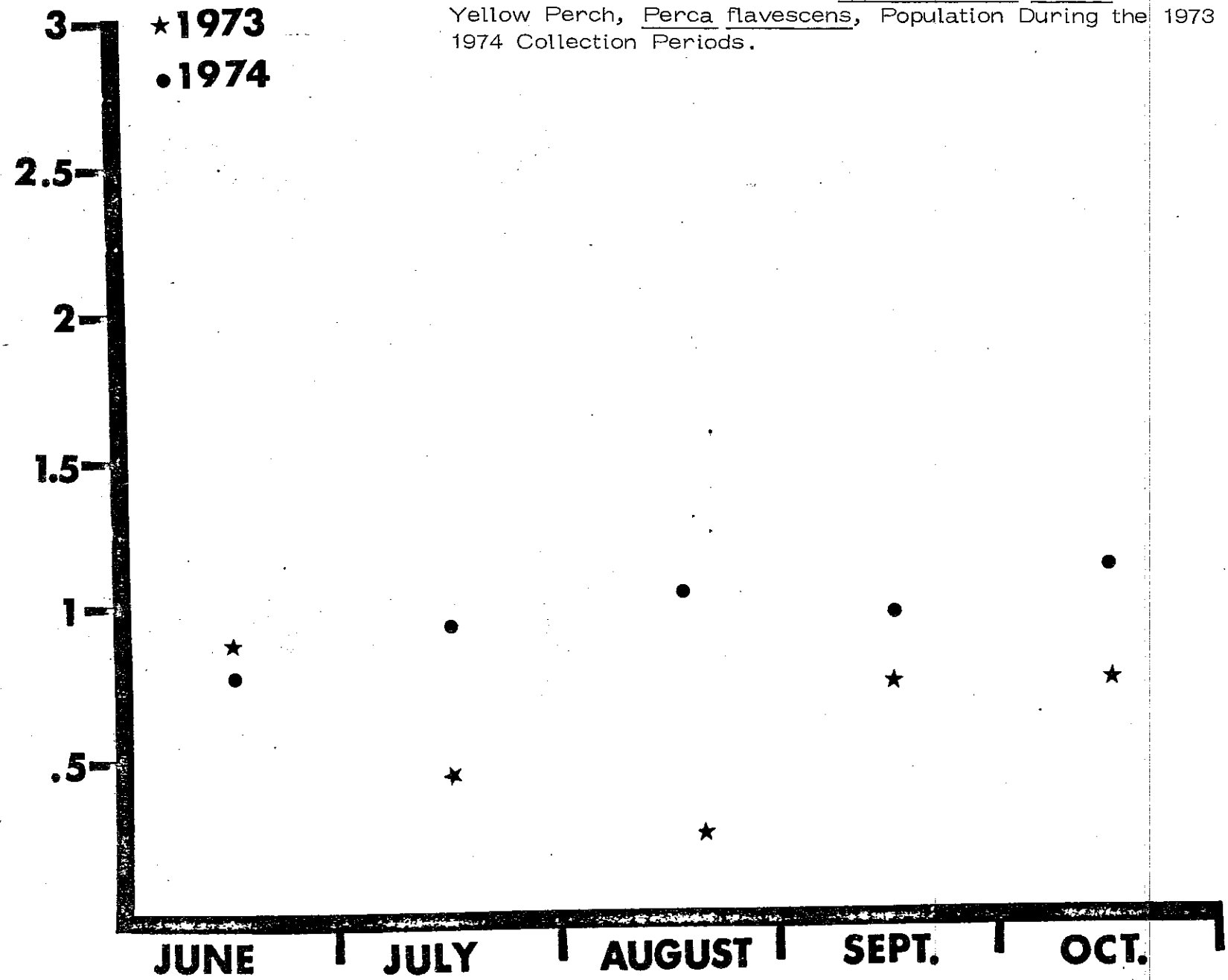


TABLE 10

PREVALENCE AND INTENSITY OF *Eustrongylides tubifex* IN NATURALLY INFECTED AVIAN HOSTS COLLECTED FROM THE WESTERN BASIN REGION OF LAKE ERIE

Avian Host	N	Prevalence (%)	Range of Infection	Mean Intensity of Infection
Anas platyrhynchos Mallards	11	0.0	-	-
Botaurus lentiginosus American Bittern	1	100	-	1
Fulica americana American Coot	1	0.0	-	-
Larus argentatus Herring Gull	3	39	1-7	2.7
Larus philadelphia Bonapartes Gull	1	0.0	-	-
Larus pipixcan Franklin's Gull	1	0.0	-	-
Mergus serrator Red-breasted Merganser	6	100	1-20	6.33
Nycticorax nycticorax Black-crowned Night Heron	4	50.0	4-6	5.0
Podilymbus podiceps Pied-billed Grebe	4	0.0	-	-
Sterna hirundo Common Tern	6	0.0	-	-

TABLE 11

PREVALENCE OF HELMINTH PARASITES OF THE PROVENTRICULUS
IN THREE SPECIES OF HERONS
FROM THE WESTERN BASIN REGION OF LAKE ERIE, 1973

Parasite	Black-Crowned Night Heron		Common Egret		Great Blue Heron	
	Nestling N = 8	Adult N = 22	Nestling N = 13	Adult N = 23	Nestling N = 6	Adult N = 17
Contracaecum sp.	0.00	23.63	0.00	0.00	0.00	11.76
Eustrongylides sp.	62.8	68.18	7.69	13.04	16.66	47.06
Tetrameres sp.	0.00	45.45	7.69	17.39	16.66	11.76

TABLE 12

DISTRIBUTION OF PROVENTRICULAR HELMINTH PARASITES
IN THREE SPECIES OF HERONS
FROM THE WESTERN BASIN REGION OF LAKE ERIE, 1973

Parasite	Black-Crowned Night Heron				Common Egret				Great Blue Heron			
	N = 30				N = 36				N = 23			
	Nestling N = 8		Adult N = 22		Nestling N = 13		Adult N = 23		Nestling N = 6		Adult N = 17	
	* Inf	* Uninf	Inf	Uninf	Inf	Uninf	Inf	Uninf	Inf	Uninf	Inf	Uninf
Contracaecum sp.	0	8	5	18	0	13	0	23	0	6	2	15
Eustrongylides sp.	5	3	15	7	1	12	3	20	1	5	8	9
Tetrameres sp.	0	8	10	12	1	12	4	19	1	5	2	15

* Inf = Infected
Uninf = Uninfected

Examiniation of heron tissue revealed considerable levels of parasitism. However, no patent infections were observed. In almost every instance of parasitism by E. tubifex in these birds, the proventriculus was invaded but the larvae died and degenerated prior to maturity. Only one mature infection was observed in the Herring Gulls examined. All other infections in Herring Gulls were degenerating larval forms. The single infection observed in an American Bittern (Botaurus lentiginosus) was a recently acquired one. The larval parasite in this instance was quite viable and potentially could have established a mature infection. Mature infections were observed in all of the Red-breasted Mergansers examined. At least two of the Mergansers examined exhibited patent infections as well. This preliminary evidence indicates that migratory mergansers most frequently harbor mature E. tubifex infections. Most of the summer resident populations of avian hosts appear to be refractory to the establishment of mature and patent infections.

ANALYSIS

A rough analysis of larval E. tubifex infections in Channel Catfish, Fresh-water Drum, and Smallmouth Blackbass according to size classes was conducted. These analyses are presented for each respective fish in Tables 4, 5 and 6. A more rigorous analysis of the infections in the Yellow Perch was conducted with the aid of the Ohio State University's Computer Center.

Inspection of Tables 4, 5 and 6 indicates that the percentage infection of E. tubifex in catfish, Drum and Blackbass, respectively, increases greatly with size class, as do the other parameters of infection. Inspection of Table 7 reveals that the older, larger perch are more highly infected than younger, smaller perch. This is bourne out in the parameters listed, $\bar{X}_{inf}$, $\bar{X}_{pop}$, and range. The increase in infection levels with the size of fish probably reflects both a change in feeding habits of the fish and an accumulation of worms through time. Price's (1963) study of the food habits of Lake Erie fishes stated that as perch size increased their feeding on zooplankton decreased. Greater age was also correlated with increased benthic feeding habits. In as much as the first intermediate host for nematodes of the genus Eustrongylides is thought to be freshwater oligochaetes (Karmanova, 1968), transmission of infective E. tubifex larvae from oligochates to perch would have a higher probability in older, larger perch. The same is true for Fresh-water Drum.

Another factor operating in the increased infection levels in older, larger blackbass, catfish and perch, is the increased carnivorus habits of the larger fish. Von Brand (1944) successfully transmitted larval Eustrongylides ignotus from one fish to another. To test this hypothesis, the large series of transfer experiments were performed, Table 2. In almost every experimental infection, larval E. tubifex from Yellow Perch were able to successfully reinfect another poikilothermous host. In most instances, the larvae reestablished the infection in the body cavity of the experimental host. The diversity of experimental hosts in which larvae were able to reestablish infections was considerable. Experimental infections occurred in piscine, amphibian and reptilian hosts. The high incidence of larval E. tubifex infections in the largest age-size classes of Channel Catfish, Smallmouth Blackbass, and Yellow Perch undoubtedly is a result of their predation on smaller, infected Yellow Perch. Overall, the gradual increase in the mean number of worms in infected larger fish ($\bar{X}_{inf}$) reflects an accumulation of the worms throughout the life of the fish. In catfish and blackbass, the

accumulation is probably principally through predation. In Freshwater Drum and Yellow Perch, the accumulation is probably a result of feeding on oligochaetes and secondarily a result of predation on infected fish. The accumulation hypothesis is reinforced by the observation of the wider range of worms in older fish; for example, YOY = 0-4 while II+ Yellow Perch = 0-21 worms. The longevity of the larval worms in fish is indicated by Von Brand's (1943) report of *in vitro* culturing of *E. ignotus* from *Fundulus diaphanus* for four years.

One-half or more of the Yellow Perch examined were not infected with larval *E. tubifex*. The majority of infected Yellow Perch harbored 1 or 2 worms. Few perch exhibited high numbers of larvae. The amount of dispersion in range of infection increases as the length of the perch increases. In addition, one notes that the larval *E. tubifex* infections in perch are not distributed normally. If the distribution of the number of larvae is plotted against the numbers of infected perch, the distribution would have a heavy tail and be heavily skewed. For statistical analyses, only zero or positive values for larval infection can be observed, no negative values for larval infection can be observed, no negative values are possible. Therefore, non-parametric statistical tests which do not assume the population to be normally distributed were used. Only data from Yellow Perch examined during the 1973 collection period were utilized for extensive population analyses. Procedures for the population analyses utilized in this study are described in Non-parametric Statistical Methods by M. Hollander and D. A. Wolfe. Computer programs outlined by D. A. Wolfe were utilized.

A breakdown of the initial statistical parameters and results of cross tabulations for larval *E. tubifex* in Yellow Perch is presented in Table 7. The percentage infection increases with the size of the fish. Likewise, the parameters $\bar{X}_{inf}$ and $\bar{X}_{pop}$ increase with the size of the fish. The range in number of larvae per fish also widens with the size of the fish. One notes that the standard deviation of the mean number of larvae in the perch population ($\bar{X}_{pop}$) increases. The variability increases, i.e., the worms are more dispersed. Generally, there is little difference in the values from year to year.

To test whether there was a significant difference in the worm burden among the three age-size classes of perch, the Jonckheere test for a one way layout of samples (k=3) was used. The July, August, and September, 1973, samples of Yellow Perch were used as a data base. The null hypothesis states that the three samples are from the same population against the alternative hypothesis of an increasing ordered difference with at least one of the inequalities strict. The

Large Sample Approximation (L.S.A.) statistic, J*, equals 7.364. The smallest significance level (α) for rejection of the null hypothesis, H_o, in favor of the ordered alternative of the worm burden increasing with each of the perch size classes is <0002. Therefore, the H_o is rejected in favor of the ordered alternative. The confidence coefficient for this ordered difference is >99.98%.

Tables 8 and 9 summarize initial statistics by collection month for the entire population and by age-size class of yellow perch during 1973 and 1974 respectively. The young-of-the-year (YOY) age class were not large enough to be collected by otter trawl until July and August. One can note that larval E. tubifex is present in the fish throughout the collection period. In addition, the relative stability of the mean number of larvae per infected fish from month to month varies.

The Kruskal-Wallis test was used to determine if there was a difference in the worm burden within perch size class with the month sampled. For the young-of-the-year class, the results of examinations of Yellow Perch conducted during the months of July through October, 1973, were utilized. The test statistic H equals 39.39 and is asymptotically chi-square with 3 degrees of freedom. The smallest significance level for rejection of H_o is less than 0.0001. Therefore, the null hypothesis is rejected. The alternative hypothesis that not all the worm burdens within this age-size class are equal in each of the collection months is accepted. The confidence coefficient is 99.9%.

Having established that a significant difference exists in the young-of-the-year age class, an increasing ordered difference for the months July through October, 1973, was tested using the Jonckheere test. The L.S.A. test statistic, J*, equaled 2.666. The smallest significance level for rejection of the null hypothesis is 0.0038. Therefore, the null hypothesis was rejected in favor of the alternative hypothesis that an increasing ordered difference exists in the effect of the sample month on the worm burden of this age-size class. The confidence coefficient is 99.6%. Hence, the larval worm burden increases for the young-of-the-year age size class from July up to October.

For the 1 year age-size class of Yellow Perch, the Kruskal-Wallis test was used to detect a difference in worm burden with month sampled. Data from examinations during June through October, 1973, were used. The test statistic H equaled 16.19 and is asymptotically chi-square with 4 degrees of freedom. The smallest significance level for rejection of the null hypothesis equals 0.0003.

TABLE 15

MEAN NO. WORMS ($\bar{X}_{pop}$) OF THIRD AND FOURTH STAGE LARVAE IN THREE AGE-SIZE CLASSES OF YELLOW PERCH, 1973

	Entire Population	YOY	I	II+
Third-Stage Larvae	0.067	0.121	0.078	0.027
Fourth-Stage Larvae	0.510	0.032	0.462	0.826

TABLE 16

STATISTICS OF LARVAL Eustrongylides tubifex IN MALE AND FEMALE YELLOW PERCH, 1973

	Mean	S. D.	# Yellow Perch
Male	0.740	1.374	604
Female	0.538	1.127	540

To determine whether the number of female larvae differed from the number of male larvae in each infected fish, a Fisher SIGN test for paired replicates was performed. The actual purpose for performing this test was to determine if there was a 1:1 sex ratio of larvae in Yellow Perch. The results of examinations of 96 Yellow Perch collected during July and August, 1973, were utilized. The L.S.A. sign statistic, B^*, equals -0.54882. The estimate of the effect of sex, $\hat{\theta}$, equals 0.0. The null hypothesis of no difference in the number of female and male larvae was tested against the alternative hypothesis of (1) more female than male larval worms and (2) more male than female larval worms. The test for $H_{(1)}$ gave a significance level for rejection of H_o of 0.5. The test for $H_{(2)}$ gave a significance level of 0.2912. Both significance values are too high to reject the null hypothesis. The confidence coefficient for there being more male than female larvae is 71%. The confidence coefficient for there being more female than male larvae is 50%. In view of the results that the estimator of the treatment effect of worm sex is 0.0 and that a 95% confidence interval of treatment effect is (-1, 1), one must conclude that there is no significant difference in the number of female and male larval E. tubifex.

Overall, a number of conclusions covering larval E. tubifex in Yellow Perch are possible. The older the fish the more heavily infected it is with larvae. Yellow perch seem to be least infected in late summer, specifically, in late July and in August. Young-of-the-year perch pick up the infection in July. Recruitment of third-stage larvae is focused in the young-of-the-year fish and concentrated in late summer and fall.

Tables 17 and 18 compare site selection by larval E. tubifex in Yellow Perch examined during 1973 and the combined examinations performed during 1971 and 1972. The majority of larvae are recovered from yellowish-pink encapsulations in the mesentery and fatty tissue near the small intestine, most often near the spleen. Fewer larvae were found free in the mesenteric tissue and in the body musculature. Larvae recovered lying free from encapsulation in the mesenteries were always third-stage larvae. Very few larvae were removed from encapsulations on the gonads. Larvae recovered in the body musculature were in the process of burrowing through the abdominal wall from their capsules in the mesentery. These larvae were always bright red fourth-stage larvae. The burrowing fourth-stage larvae were most often noted in fish that were necropsied several hours after death. If the fish were kept on ice after collection and examined within a few hours of collection, very few, if any, burrowing larvae were recovered.

TABLE 17

SITE SELECTION BY LARVAL *E. tubifex* IN *Perca flavescens* FOR ENTIRE SAMPLE MARCH, 1973 TO OCTOBER, 1973 **

Tissue Site	# Fish	Total # *E. tubifex*	($\bar{X}_{inf}$) Mean # Worms	Range	S. D.
Mesentery & Fatty Tissue Encapsulation	432	761	1.762	1 - 11	1.149
Free in Mesentery	22	26	1.182	1 - 3	0.501
Burrowing Thru Vicera Or Musculature	18	27	1.500	1 - 3	0.786
Gonad Encapsulation	1	1	1.000	1	-

** Total number of fish in sample = 1413
Mean worm burden of population ($\bar{X}_{pop}$) = 0.577

TABLE 18

SITE SELECTION BY LARVAL *E. tubifex* IN *Perca flavescens* FOR ENTIRE SAMPLE (JUNE - AUG., 1971, MAY - OCT., 1972) **

Tissue Site	# Fish	Total # *E. tubifex*	Mean (X_{inf}) # Worms	Range	S. D.
Mesentery & Fatty Tissue Encapsulation	359	662	1.844	1 - 14	1.571
Free in Mesentery	13	13	1.000	1	-
Burrowing Thru Viscera Or Musculature	32	46	1.438	1 - 4	0.759
Gonad Encapsulation	2	2	1.000	1	-

** Total number of fish in sample = 1056
Mean worm burden of population = 0.696

The latter observation lead to the hypothesis that the burrowing larvae were migrating from the encapsulations in response to an environmental cue. The most obvious cue appeared to be temperature. The conditions under which E. tubifex larvae establish an infection in the definitive avian host following the ingestion of an infected fish was not known. We hypothesized that the larvae in mesenteric encapsulations are activated by the high body temperature of the bird following ingestion of an infected fish. A series of experiments were performed with freshly trawled fish. The results of this series of emergence experiments are summarized in Table 3. At temperatures of 40°C and higher, the E. tubifex larvae migrated out of their encapsulations onto the surface of the fish. Larvae emerged from the bodies of the infected fishes most often in the region immediately behind the pectoral fin where the body wall musculature is thinnest. Soon after these experiments were initiated, a Herring Gull was collected which had recently ingested an infected Yellow Perch. The fish removed from the proventriculus of this bird displayed an emergent larval E. tubifex. The larvae in question was migrating out of the body of the fish immediately behind the pectoral fin. This is the region of the fish where the greatest number of encapsulations are encountered in the viscera, near the spleen. At temperatures below 40°C (approximately body temperature of most birds) larvae initiated emergence from their encapsulations and/or penetration of the body wall of the fish very slowly or did not initiate emergence at all. It appears that the emergence behavior demonstrated by these experiments represents an adaptation to facilitate rapid infection of a definitive warm-blooded host upon ingestion of an infected fish.

A single experiment was conducted to determine the behavior, if any, of larval E. tubifex in perch that are quick frozen at -08°C soon after being caught. The fish in this experiment were placed on ice as soon as they were caught and subsequently quick frozen for 24 hours. The frozen fish were then placed in a 70°C oven for 25 hours. Although a number of worms survived the freezing and heating regimens, none were able to penetrate the body wall into the musculature of the fish. At best, the surviving worms succeeded in initiating emergence from the mesenteric encapsulations.

Based on our examinations of fish-eating birds in the western basin region of Lake Erie, one is lead to conclude that E. tubifex larvae possess adaptations which permit initial infection, i.e., emergence from the ingested fish and penetration of the proventriculus of the avian host, of a wide range of bird species. However, most initial infections do not develop. Instead, most initial infections result in degenerating worm tracts in the serosal muscle of the

proventriculus. In experimentally infected Mallards, the life span of the worm is no more than 30 days. In fact, 24 days post-infection, patent infections are essentially spent. This indicates that E. tubifex may be an organism which matures quickly and produces a finite number of eggs within a relatively short time span. One is tempted to speculate that this is an adaptation for maintaining the population of parasites by utilizing migratory birds which are present on the lake for relatively short periods of time. This speculation is reinforced by the observation that Red-breasted Mergansers exhibited the highest prevalence and intensity of mature infections of all the naturally infected birds examined during this study.

RECOMMENDATIONS

Several recommendations for future management can be made on the basis of our research efforts. In regards to stocking ponds and lakes with Lake Erie Yellow Perch, our studies indicate the most appropriate time to stock and the appropriate size of fish to stock to avoid transferring eustrongylidosis. Young-of-the-year fish are the least infected of the perch stock in the Lake. Transferring Y-O-Y fish as soon after hatching as possible would be advised. Y-O-Y fish should definitely be transferred before September or October. If the I year or II+ year age-size classes are to be stocked, the most advisable time to transfer them would be July and August. Once ponds have been stocked, it is advisable to keep fish-eating birds away as they could bring the infective stages of E. tubifex to the area.

The first step of the transmission cycle of this parasite needs to be elucidated. Since freshwater oligochaetes are believed to be the sole first intermediate hosts, measures aimed at pollution abatement would reduce the rich, organic mud utilized by these organisms. If the numbers of oligochaetes are reduced, logically the transmission from oligochaete to fish should decrease.

Sport and commercial fishermen continue to express concern with larvae they observe migrating through the body musculature, i.e. the fillet portion, of their catch. Our investigations reveal that keeping the fish cold or eviscerating the fish upon landing will eliminate the possible activation and emergence of the larvae from its encapsulation and its subsequent migration into the body musculature. Allowing fish to lie on the hot deck of a boat in the summer simply guarantees invasion of the fillet portion of the fish by larval E. tubifex. The latter circumstance allows for potential infection of humans with E. tubifex. A recent report of human infection with Eustrongylides sp. would suggest thorough cooking of the fillets before consumption. Simply 'smoking' Yellow Perch actually invites infection with a pathogenic organism.

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Center for Lake Erie
Area Research

Date 31 July 1975

FEDERAL AID IN SPORT FISH RESTORATION

FINAL REPORT

STATE OF:	Ohio
PROJECT NO.:	F-48-R-3
PERIOD COVERED:	June 1, 1974 - May 31, 1975
PROJECT TITLE:	Impact of Parasitic Worms on Lake Erie Fishes
STUDY NO.:	IV
STUDY TITLE:	Impact of the Cestode Parasite Triaenophorus nodulosus on Lake Erie Fishes
PREPARED BY:	Owen D. Buck and John L. Crites
DATE:	31 July 1975

ABSTRACT

White bass (Morone chrysops) collected in the vicinity of South Bass Island, Ohio, during the summer of 1974 were found to be heavily infected with the plerocercoid stage of the tapeworm Triaenophorus nodulosus. Investigations of the impact of this parasite on its host concentrated on three topics: 1) descriptive pathology of the liver, 2) experimental determination of pathology manifesting in blood chemistry, and 3) population interactions between white bass and T. nodulosus in the Bass Island area.

The first intermediate host of T. nodulosus has been shown to be the copepod Cyclops bicuspidatus; white bass become infected by feeding upon infected copepods. When infected white bass are eaten by the northern pike (Esox lucius) the parasite develops to the adult stage. The eggs of T. nodulosus are released in the spring. The emergent larva, or coracidium, hatches in the water and is eaten by a copepod.

The plerocercoid stage of T. nodulosus in white bass was found primarily in the liver, but occasionally occurred in mesenteric encapsulations. Gross characteristics of triaenophoriasis include pea-sized cysts, reddish-brown tracks through the liver tissue, and extensive areas of hepatic hemorrhage and necrosis. Microscopic examination of infected livers revealed an acute cellular response, metaplasia, fibrosis, and displacement of functional parenchyma.

Blood collected from white bass was assayed for hemoglobin,

hematocrit, plasma protein, total bilirubin, and blood glucose. A statistically signifigant negative correlation was found between fish size and hematocrit values. The liver necrosis resulting from infections of T. nodulosus was statistically associated with lowered plasma protein content.

Prevalence and intensity of triaenophoriasis in white bass increased signifigantly with fish size. Triaenophoriasis was very rare in young-of-the-year white bass; one fish of 121 examined was infected. The adult stage of T. nodulosus, which occurs in the northern pike, was not found in the study area. A survey of fish parasites indicated that the yellow perch (Perca flavescens) is the only fish species other than white bass to be heavily infected with plerocercoids of T. nodulosus in the Bass Island area.

CONTENTS

BACKGROUND

Triaenophoriasis is a disease of fishes caused by plerocercoids and adult cestodes belonging to the genus Triaenophorus Rudolphi, 1873. There are three species, all occurring in North America: T. nodulosus (Pallas, 1760); T. crassus (Forel, 1868); and T. stizostedionis (Miller, 1945). The plerocercoid stage of T. nodulosus has been reported from many fish species in Lake Erie (Bangham, 1972; Bangham and Hunter, 1939; Dechtiar, 1972), but is abundant only in white bass (Morone chrysops) and yellow perch (Perca flavescens). The adult stage of T. nodulosus has been reported only from the northern pike (Esox lucius). The plerocercoid of T. stizostedionis has been reported only from the trout perch (Percopsis omiscomaycus) in Lake Erie (Dechtiar, 1972), and the adult only from the walleye (Stizostedion vitreum) (Bangham, 1972; Dechtiar, 1972). T. crassus has not been recorded from Lake Erie. The adult of this species occurs in northern pike, as does T. nodulosus, but T. crassus utilizes different second intermediate hosts: Leucicthys sp., Coregonus clupeaformis, and Prosopium oregonium. Plerocercoids of T. crassus are found in intra muscular encapsulations, while T. nodulosus encapsulates in the viscera (Miller, 1945b).

The life cycle of T. nodulosus has been studied by Miller (1943a, 1943b, 1945a) in Alberta. Adult worms occur in the intestines of

northern pike, the only known definitive host. Eggs are released in the spring and coracidia, the first larval stage, hatch in the water in 15-16 days. The coracidia are eaten by many species of copepods, but only Cyclops bicuspidatus and C. vernalis are suitable first intermediate hosts in which development of the parasite will continue. The ingested coracidium penetrates through the gut wall into the copepod hemocoel, where it develops to the procercoid stage. If the infected copepod is consumed and digested by a fish suitable as a second intermediate host, the liberated procercoid migrates to the liver, develops to the plerocercoid stage, and encapsulates. The cycle is completed when an infected fish is eaten by a northern pike and the parasite matures in the upper intestine.

Plerocercoids of T. nodulosus cause extensive hemorrhaging and necrosis in the livers of white bass, particularly in larger fish. The surface of the liver may support numerous pea-sized cysts, and in heavy infections many worms may be observed actively burrowing through the liver tissue. The organ may take on a yellowish hue. In some cases the lobes of the liver are bleeding distally. The effects of triaenophoriasis on the physiology of the fish had not been investigated prior to the present study.

OBJECTIVES

1) Experimental investigation of blood chemistry alterations associated with infections of *Triaenophorus nodulosus* in white bass (*Morone chrysops*)

2) Description of gross and microscopic histopathology of the liver associated with *T. nodulosus*.

3) Investigation of population interactions between *T. nodulosus* and white bass in Lake Erie.

4) Characterization of the life cycle of *T. nodulosus* specifically as it occurs in Lake Erie

5) Assessment of the impact of *T. nodulosus* on fish species other than white bass.

PROCEDURES

Experimental work, collection of specimens, and compilation of data out at the Franz Theodore Stone Laboratory, The Ohio State University, Put-In-Bay, Ohio, from June through August, 1974. Subsequent work and statistical data analyses were performed in the parasitology laboratory of the Department of Zoology, The Ohio State University, Columbus, Ohio.

Yearling and older white bass (Morone chrysops) were taken by angling. These fish were kept alive by immediately placing them in containers of water; upon return to the laboratory they were transferred to large holding tanks. After a fasting period of at least 24 hours, to avoid alteration of blood values by feeding and associated metabolic activity, fish were anesthetized with quinaldine, blood was drawn from the caudal vein, and autopsy was performed.

Blood drawing was accomplished with a syringe and 18 gauge needle. Sodium heparin was used as an anticoagulant. Approximately one ml. of blood was removed from each fish, and then transferred to a culture tube. A 100 lambda capillary tube was filled with whole blood; 13 lambda were removed from this for hemglobin assay with the Bio-Dynamics Unitest System, and the remainder was centrifuged for hematocrit determination. The whole blood remaining in the culture tube was centrifuged, and the plasma decanted for the following tests: plasma protein content determination, with the American

Optical Model 10400 TS Meter; total bilirubin testing, with the Bio-Dynamics Unitest System; and blood glucose evaluation, by the General Diagnostics Gluco Strate method.

Size, weight, and sex of the fish were recorded. The liver was removed and evaluated for three aspects of triaenophoriasis: number of encapsulations or cysts present, number of active unencapsulated plerocercoids present, and extent of old lesions or necrosis in the liver (evaluated visually on a scale of 0 to 3). The liver was weighed, preserved with Bouin's fixative, and stored in 70% ethanol. Livers were mounted in parrafin and sectioned; mounted sections were stained with Mallory's triple stain. Individual plerocercoids were preserved in AFA, stained with Semichon's carmine, and mounted in piccolyte.

Young-of-the-year white bass and yellow perch were taken by otter trawl from the Research Vessel Biolab and placed in ice for autopsy in the laboratory. Centrarchid fishes were taken by angling or by trap nets; these were maintained alive in the laboratory until time of autopsy.

Because white bass became available only at very sporadic intervals during the collection period, the application of a time factor to the data would not have been statistically meaningful.

Data collected were key punched onto computer cards at the Instruction and Research Computer Center (IRCC) of the Ohio State University. Programs run were extracted from the Statistical Package for Social Sciences (SPSS) battery maintained at the center.

FINDINGS

Population Aspects of Triaenophoriasis in White Bass

Ninety-eight white bass of age one year and older were examined for triaenophoriasis during the summer of 1974. On the bases of both length and weight, fish fell into two distinct groups; these groups correspond to yearling and 2+ age classes. Specifications for assignment to age class are listed in Table 1. Because age class composition differed slightly, depending upon whether length or weight was used as the determinant, all statistical tests in this study were performed once with each mode of classification.

Initial statistical tests were designed to investigate size differences between male and female white bass in the study sample. Results of "t" tests are presented in Table 2. The "t" test is for the null hypothesis (H_o) that no significant difference exists between the mean values of the groups in question, in this case, that the mean value of size of male fish in the population from which the sample was drawn is the same as the mean value for female fish, the observed difference being due to the chance associated with sample selection. Adopting the conventional 95% level of confidence, a probability greater than .05 would mandate retention of the null hypothesis. All probabilities for the "t" values obtained exceed .05; therefore H_o is retained, and it is assumed that there is no sex differential in length, weight, or liver weight.

"F" values were also calculated and tested (Table 2). H_o in this case is that the variance of one sex does not differ from the variance of the other. In testing the total weight of the fish, a probability of 0.016 for H_o was obtained. Because this probability is less than .05, the null hypothesis is rejected, and it is assumed that there is a sex differential in the variability of weight. Comparison of statistics for the two sexes (Table 3) reveals that females (standard deviation = 152.783) are more variable in total body weight than are males (standard deviation = 105.844).

A chi-square test was conducted to discover any significant differences in the sex ratios between the two age classes. Results are given in Table 4. The significance of H_o in each case exceeds .05; therefore, the null hypothesis, that there is no difference in the proportion of males to females in each age class, is retained.

Incidence and intensity data are listed in Table 5 for each of the three aspects of triaenophoriasis studied: number of encapsulations or cysts, number of unencapsulated plerocercoids, and extent of liver necrosis (old lesions). Young-of-the-year white bass will be considered separately. Other statistics for triaenophoriasis in white bass are given in Tables 6 and 7.

Correlations between fish size and triaenophoriasis are presented in Table 8. H_o in this test is that there is no correlation between the two variables being tested, or, in other words, that one variable could not be predicted at all, given the other. Correlations between size

and number of encapsulations, and between size and extent of necrosis, were highly significant, the significance of H_o being much less than .05. The number of plerocercoids correlated significantly with liver weight, but not with fish length or fish weight. Regression equations, graphical representations of how much increase or decrease in one factor is associated with a unit increase in the other, are plotted in Graphs 1-9.

For the purpose of "t" testing, fish were assigned to age classes. Results of these tests (Tables 9 and 10) indicate that there is a highly significant difference between the age classes in the mean number of encapsulations present, and in the average extent of liver necrosis. The difference in mean number of unencapsulated plerocercoids was not significant. "F" tests disclosed significant differences in the variances for number of encapsulations, number of plerocercoids, and necrosis. Referring to Tables 6 and 7, it can be seen that, in each case, the 2+ age class has higher mean values and standard deviations than does the yearling class.

Test results suggest that triaenophoriasis in white bass is a cumulative disease, with necrosis and number of encapsulations increasing as the fish grows. The correlation of number of unencapsulated plerocercoids (representing the initial hepatic infection) with liver weight implies that the rate at which white bass accumulate the disease is determined in part by the amount of liver tissue

Information concerning this problem has been received from the Nanticoke Project, Ministry of Natural Resources, Ontario, Canada. Findings of tagging studies on white bass indicate that these fish are highly migratory between the eastern basin of Lake Erie and the island region of the western basin. Also, northern pike infected with adult T. nodulosus are reported to be common in the vicinity of Port Dover, Ontario. It is likely that white bass acquire the infection at some other locality of the lake, possibly the eastern basin, carrying the parasite into other parts of their range.

TABLE 1

CHARACTERISTICS OF YEARLING AND 2+ AGE CLASSES OF WHITE BASS COLLECTED AT PUT-IN-BAY, OHIO, SUMMER, 1974

	Determination by Fish Length	Determination by Fish Weight
Yearling Class--		
Range	170-256 mm	51-267 g
N	59	61
Sex Ratio	27 male: 32 female	28 male: 33 female
2+ Class--		
Range	278-345 mm	304-580 g
N	39	37
Sex Ratio	14 male: 25 female	13 male: 24 female

TABLE 2

T-TEST FOR SEX DIFFERENTIAL IN WHITE BASS SIZE

	By total length	By total weight	By liver weight
Pooled Variance Estimate--			
t value	-1.13	-1.51	-1.30
Degrees of Freedom	96	96	93
2-tail Probability	0.263	0.134	0.195
Separate Variance Estimate			
t value	-1.17	-1.60	-1.37
Degrees of Freedom	94.48	95.89	92.41
2-tail Probability	0.247	0.113	0.174
F value	1.51	2.08	1.77
2-tail Probability	0.172	0.016	0.065

TABLE 3

MEANS, STANDARD DEVIATIONS, AND STANDARD ERRORS OF SIZE IN MALE AND FEMALE WHITE BASS

	Mean	Std. Deviation	Std. Error
Male--			
Fish Length (mm.)	246.8293	41.357	6.459
Fish Weight (g.)	215.2683	105.844	16.530
Liver Weight (g.)	3.5795	2.245	0.359
Female-			
Fish Length (mm.)	257.7017	50.830	6.733
Fish Weight (g.)	257.1052	152.783	20.237
Liver Weight (g.)	4.3161	2.987	0.399
Total-			
Fish Length (mm.)	253.153	47.180	4.766
Fish Weight (g.)	239.602	136.111	13.749
Liver Weight (g.)	4.014	2.718	0.279

TABLE 4

CHI-SQUARE TEST OF SEX BY AGE CLASS FOR WHITE BASS

	By total length	By total weight
Chi-square	0.57742	3.16615
Degrees of Freedom	1	3
Significance	0.4473	0.3667

TABLE 5

INCIDENCE AND INTENSITY OF TRIAENOPHORIASIS IN WHITE BASS, 1974:
AGE CLASS DETERMINATION BY FISH LENGTH

	Yearling (N=59)		2+ Year (N=39)		Total (N=98)	
	Percent Incidence	Average Intensity	Percent Incidence	Average Intensity	Percent Incidence	Average Intensity
Encapsulations	18.64	1.36	74.36	2.34	39.80	2.08
Plerocercoids	16.95	1.50	30.77	1.25	22.45	1.36
Necrosis	27.12	1.06	94.87	1.19	54.08	1.15

TABLE 6

MEANS, STANDARD DEVIATIONS, AND STANDARD ERRORS OF TRIAENOPHORIASIS IN WHITE BASS:
BREAKDOWN BY AGE CLASS AS DETERMINED BY FISH LENGTH

	Yearling	2+ Year	Total
Number of Encapsulations-			
Mean	0.2586	5.4483	1.989
Std. Deviation	0.579	10.425	6.455
Std. Error	0.076	1.936	0.692
Number of Plerocercoids-			
Mean	0.2542	0.4412	0.323
Std. Deviation	0.659	0.894	0.754
Std. Error	0.086	0.153	0.078
Necrosis-			
Mean	0.2931	1.7600	0.735
Std. Deviation	0.496	0.831	0.912
Std. Error	0.065	0.166	0.100

TABLE 7

MEANS, STANDARD DEVIATIONS, AND STANDARD ERRORS OF TRIAENOPHORIASIS IN WHITE BASS:
BREAKDOWN BY AGE CLASS AS DETERMINED BY FISH WEIGHT

	Yearling	2+ Year	Total
Number of Encapsulations-			
Mean	0.2586	5.6429	1.989
Std. Deviation	0.579	10.563	6.455
Std. Error	0.076	1.996	0.692
Number of Plerocercoids-			
Mean	0.2542	0.4063	0.323
Std. Deviation	0.685	0.875	0.754
Std. Error	0.089	0.155	0.078
Necrosis-			
Mean	0.3103	1.7917	0.735
Std. Deviation	0.503	0.833	0.912
Std. Error	0.066	0.170	0.100

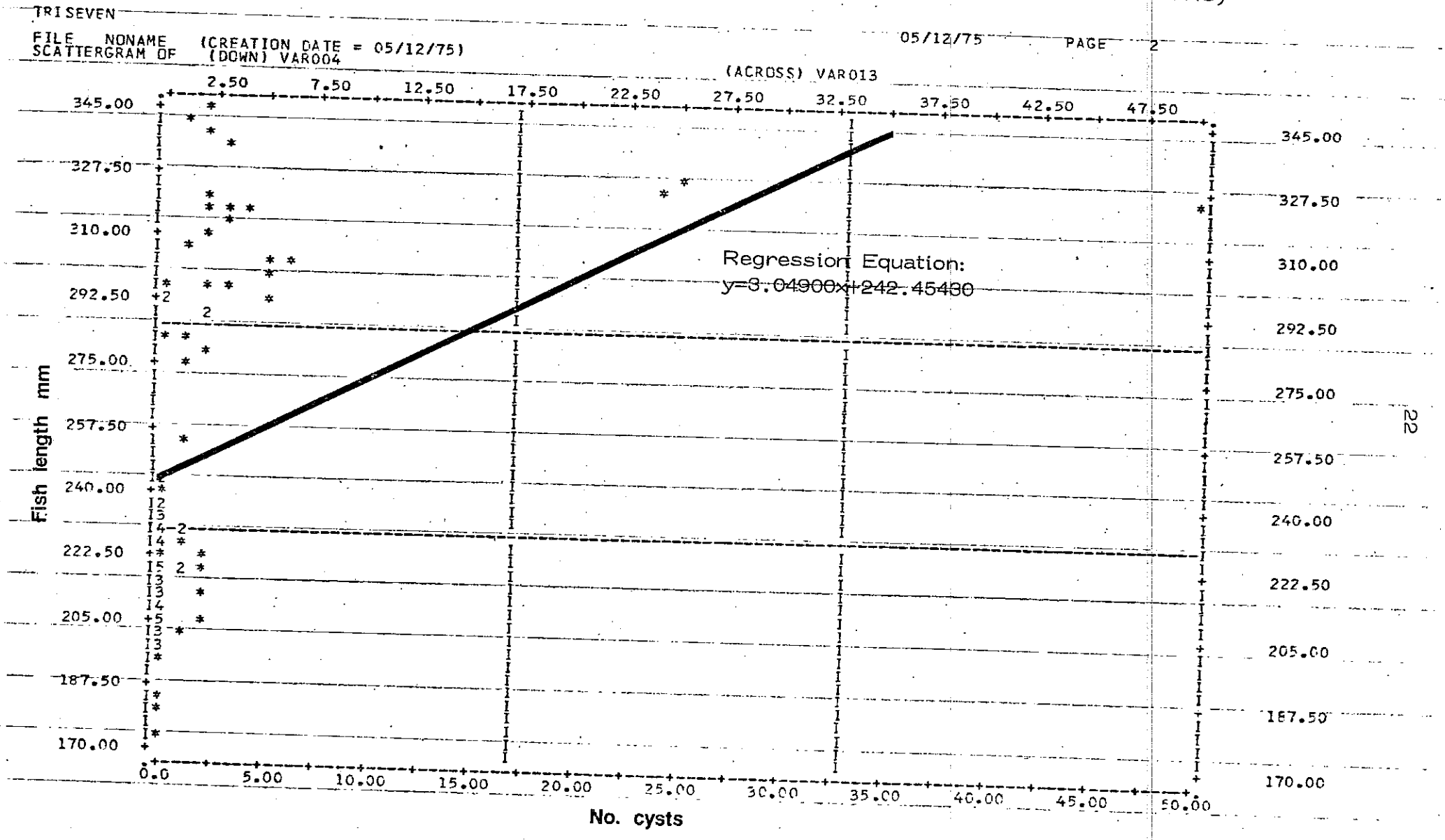
TABLE 8

CORRELATIONS BETWEEN WHITE BASS SIZE AND EXTENT OF TRIAENOPHORIASIS

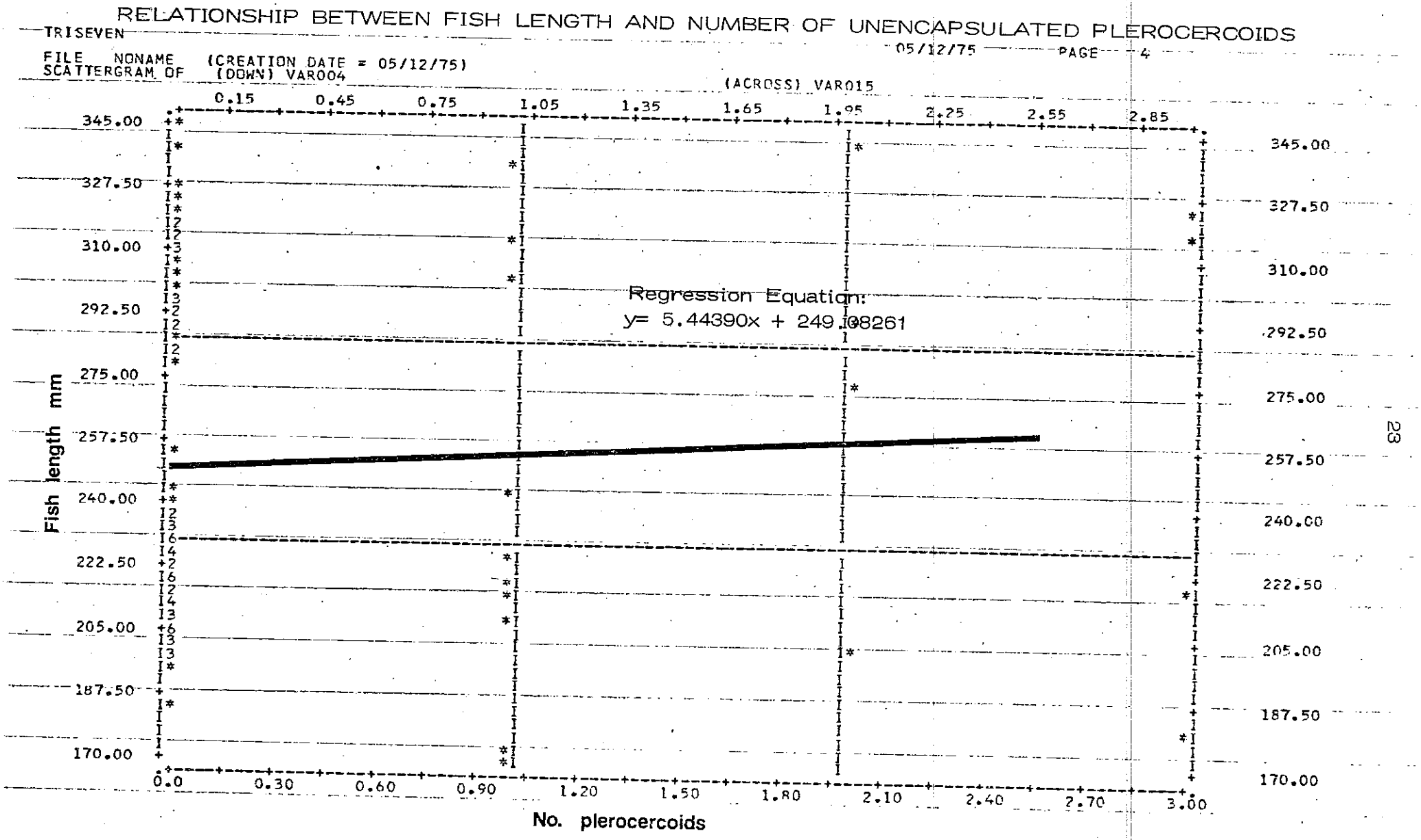
Fish Size	Triaenophoriasis	Correlation (R)	R^2	Significance
Fish length	× No. encapsulations	0.42804	0.18322	0.00002
	× No. plerocercoids	0.08687	0.00755	0.20383
	× Necrosis	0.80030	0.64048	0.00001
Fish weight	× No. encapsulations	0.49468	0.24471	0.00001
	× No. plerocercoids	0.12827	0.01645	0.11023
	× Necrosis	0.82141	0.67471	0.00001
Liver weight	× No. encapsulations	0.70611	0.49859	0.00001
	× No. plerocercoids	0.28456	0.08097	0.00328
	× Necrosis	0.76546	0.58593	0.00001

GRAPH 1

RELATIONSHIP BETWEEN FISH LENGTH AND NUMBER OF CYSTS (ENCAPSULATIONS)

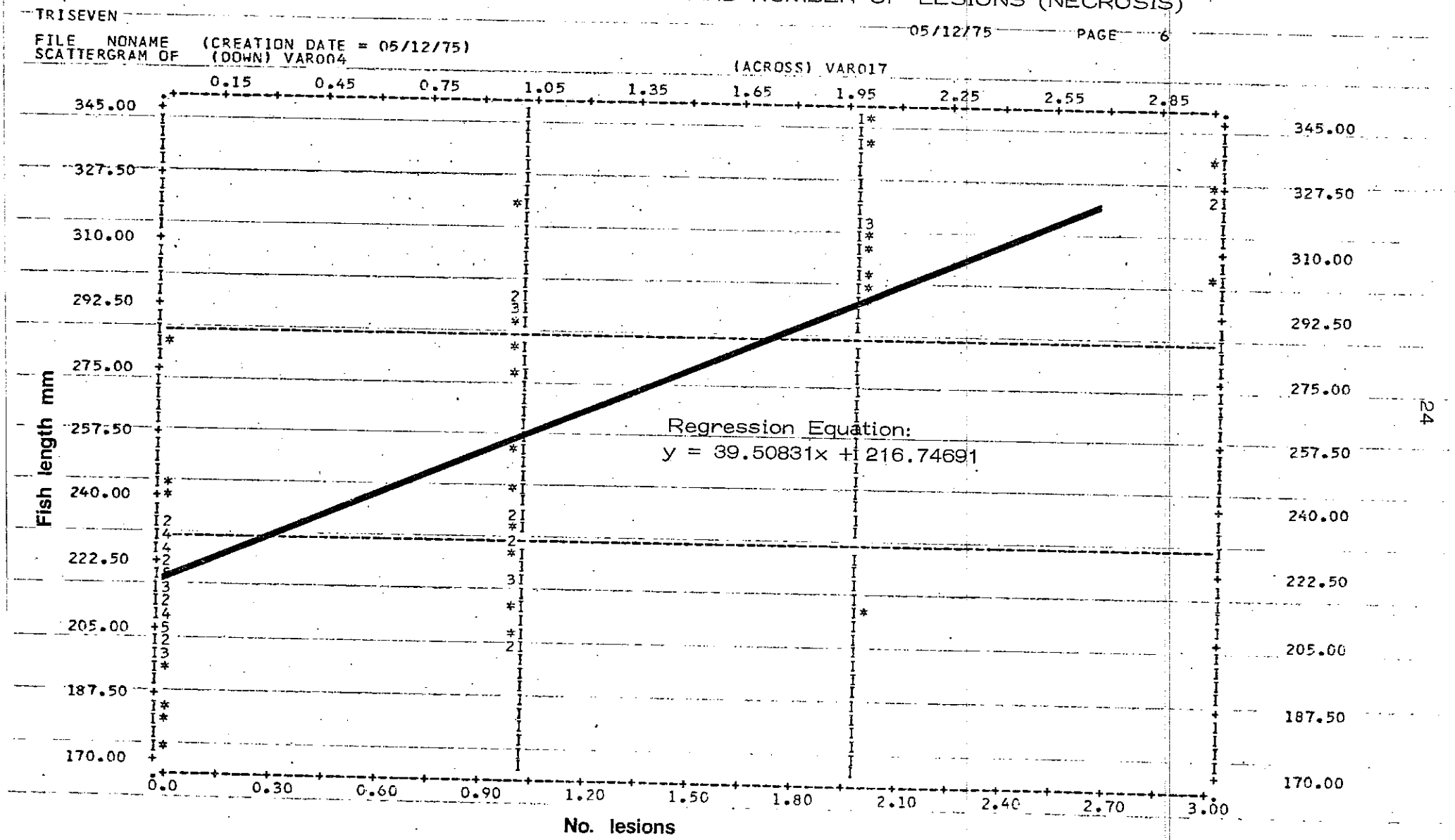


GRAPH 2


RELATIONSHIP BETWEEN FISH LENGTH AND NUMBER OF UNENCAPSULATED PLEROCERCOIDS
TRISEVEN
05/12/75 PAGE 4
FILE NONAME (CREATION DATE = 05/12/75)
SCATTERGRAM OF (DOWN) VAR004
(ACROSS) VAR015
Regression Equation:
y= 5.44390x + 249.98261
Fish length mm
No. plerocercoids
345.00
327.50
310.00
292.50
275.00
257.50
240.00
222.50
205.00
187.50
170.00
0.15
0.45
0.75
1.05
1.35
1.65
1.95
2.25
2.55
2.85
0.0
0.30
0.60
0.90
1.20
1.50
1.80
2.10
2.40
2.70
3.00

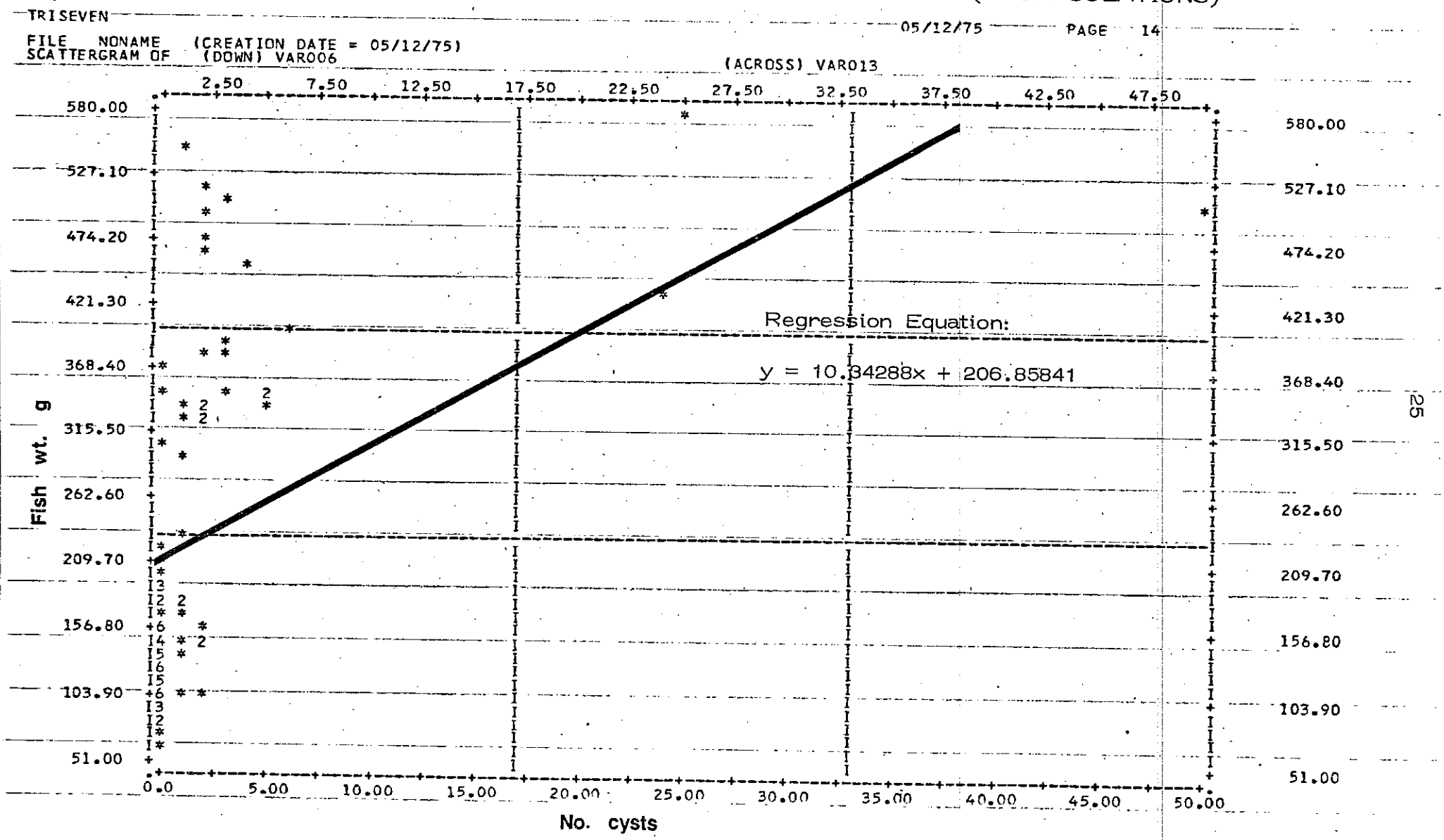
GRAPH 3

RELATIONSHIP BETWEEN FISH LENGTH AND NUMBER OF LESIONS (NECROSIS)



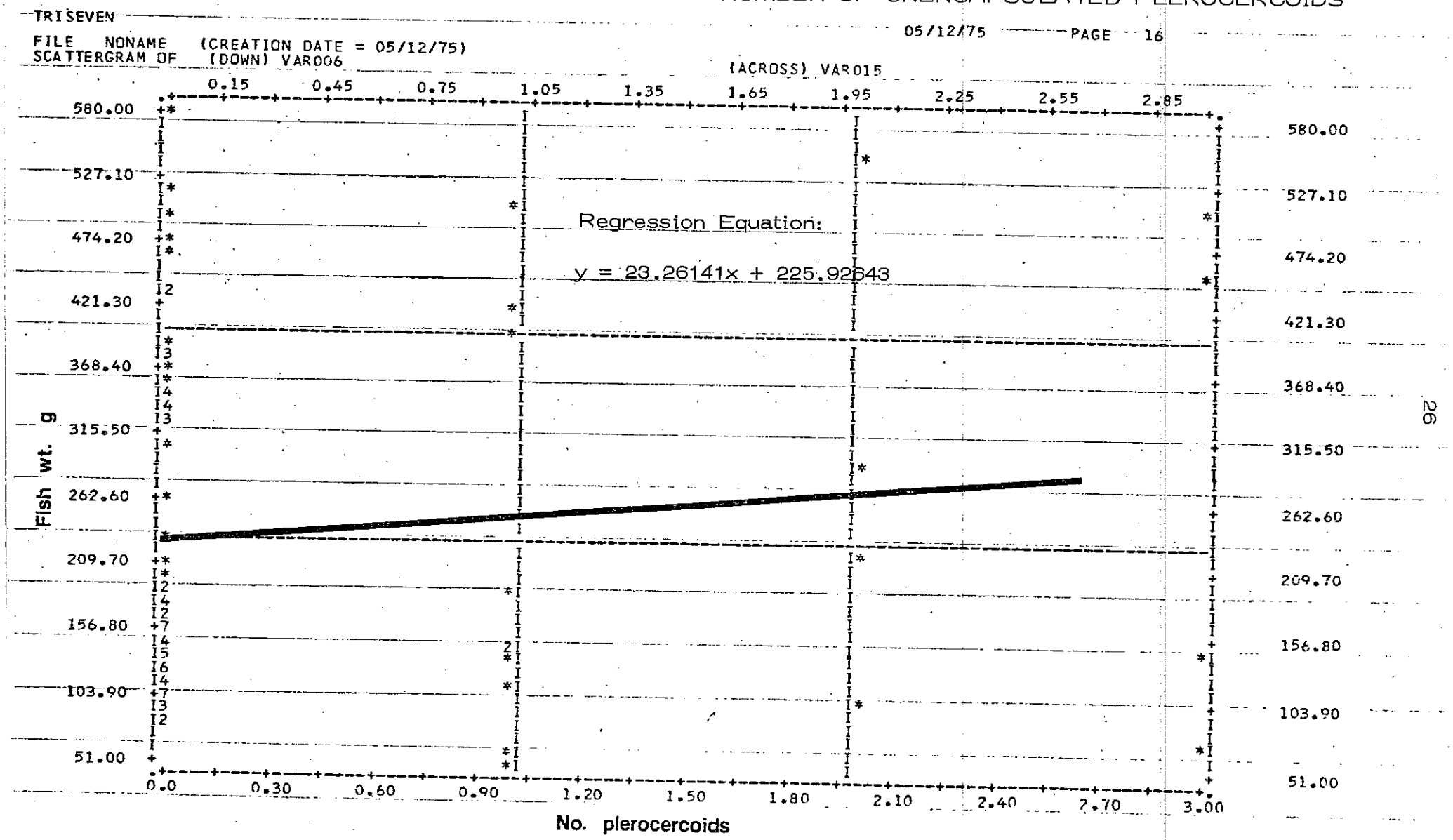
GRAPH 4

RELATIONSHIP BETWEEN FISH WEIGHT AND NUMBER OF CYSTS (ENCAPSULATIONS)

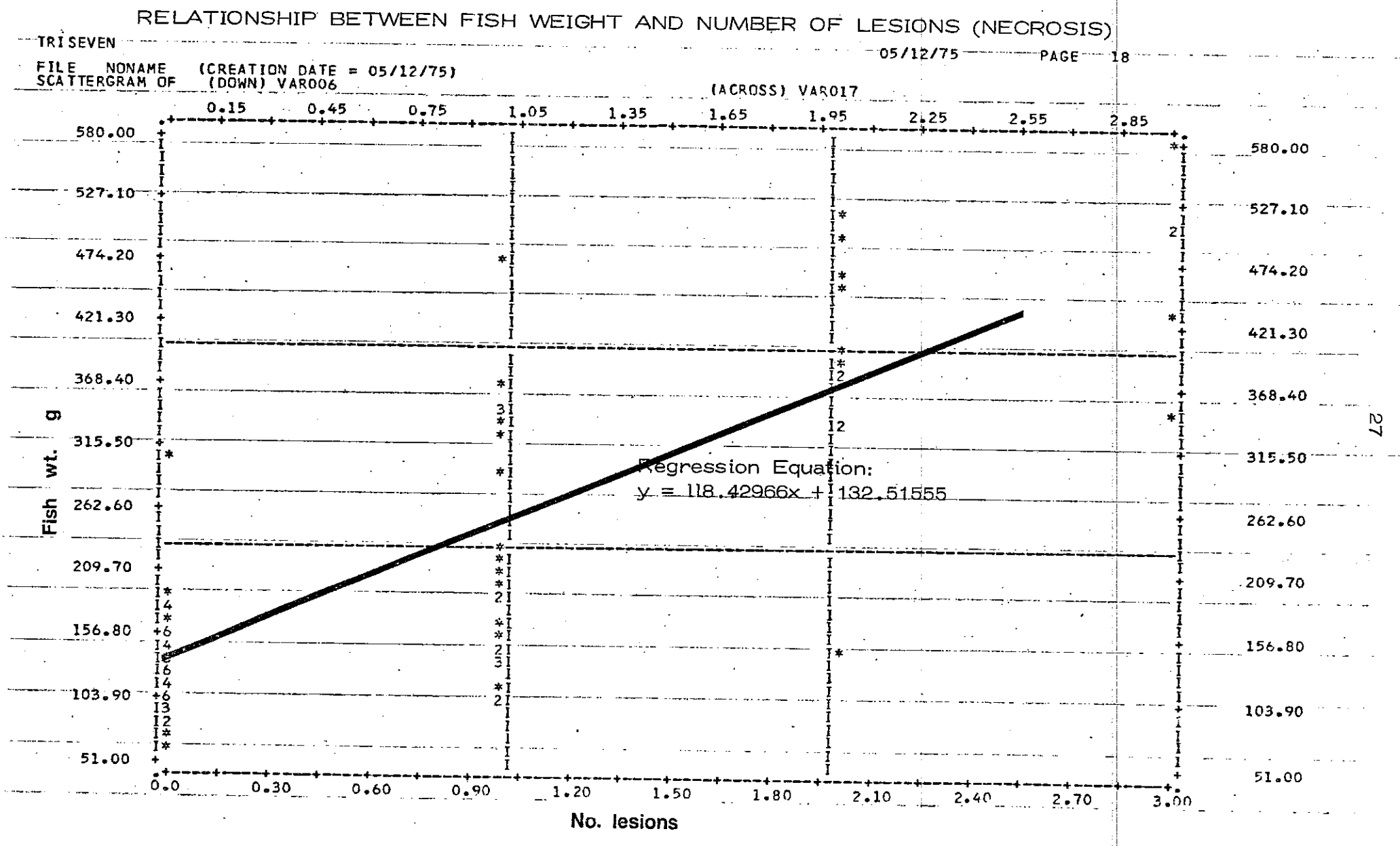


GRAPH 5

RELATIONSHIP BETWEEN FISH WEIGHT AND NUMBER OF UNENCAPSULATED PLEROCERCOIDS

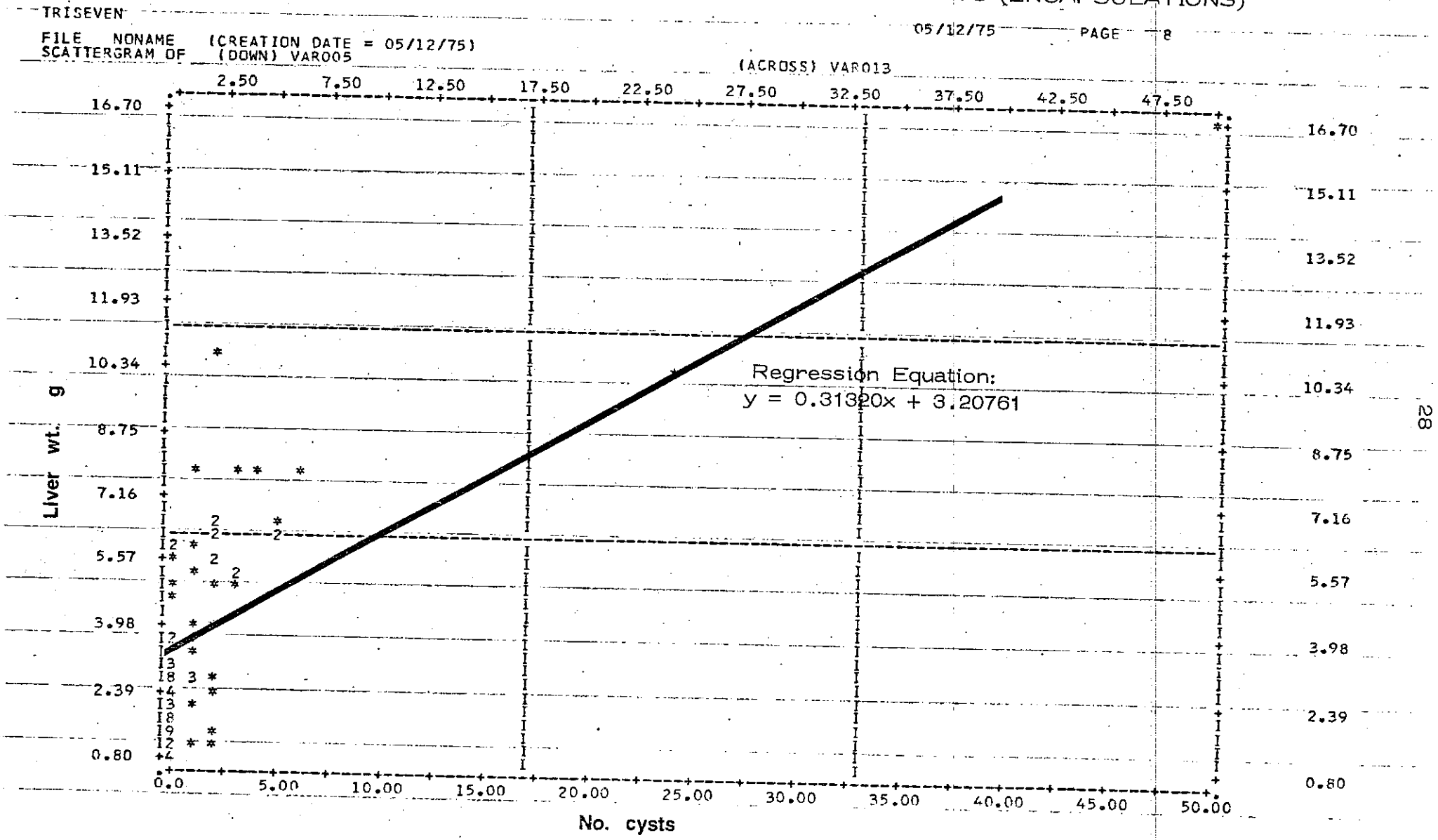


GRAPH 6


RELATIONSHIP BETWEEN FISH WEIGHT AND NUMBER OF LESIONS (NECROSIS)
TRISEVEN
05/12/75 PAGE 18
FILE NONAME (CREATION DATE = 05/12/75)
SCATTERGRAM OF (DOWN) VAR006
(ACROSS) VAR017
0.15 0.45 0.75 1.05 1.35 1.65 1.95 2.25 2.55 2.85
580.00
527.10
474.20
421.30
368.40
315.50
262.60
209.70
156.80
103.90
51.00
Fish wt. g
Regression Equation:
y = 118.42966x + 132.51555
0.0 0.30 0.60 0.90 1.20 1.50 1.80 2.10 2.40 2.70 3.00
No. lesions

GRAPH 7

RELATIONSHIP BETWEEN LIVER WEIGHT AND NUMBER OF CYSTS (ENCAPSULATIONS)



GRAPH 9

RELATIONSHIP BETWEEN LIVER WEIGHT AND NUMBER OF LESIONS (NECROSIS)

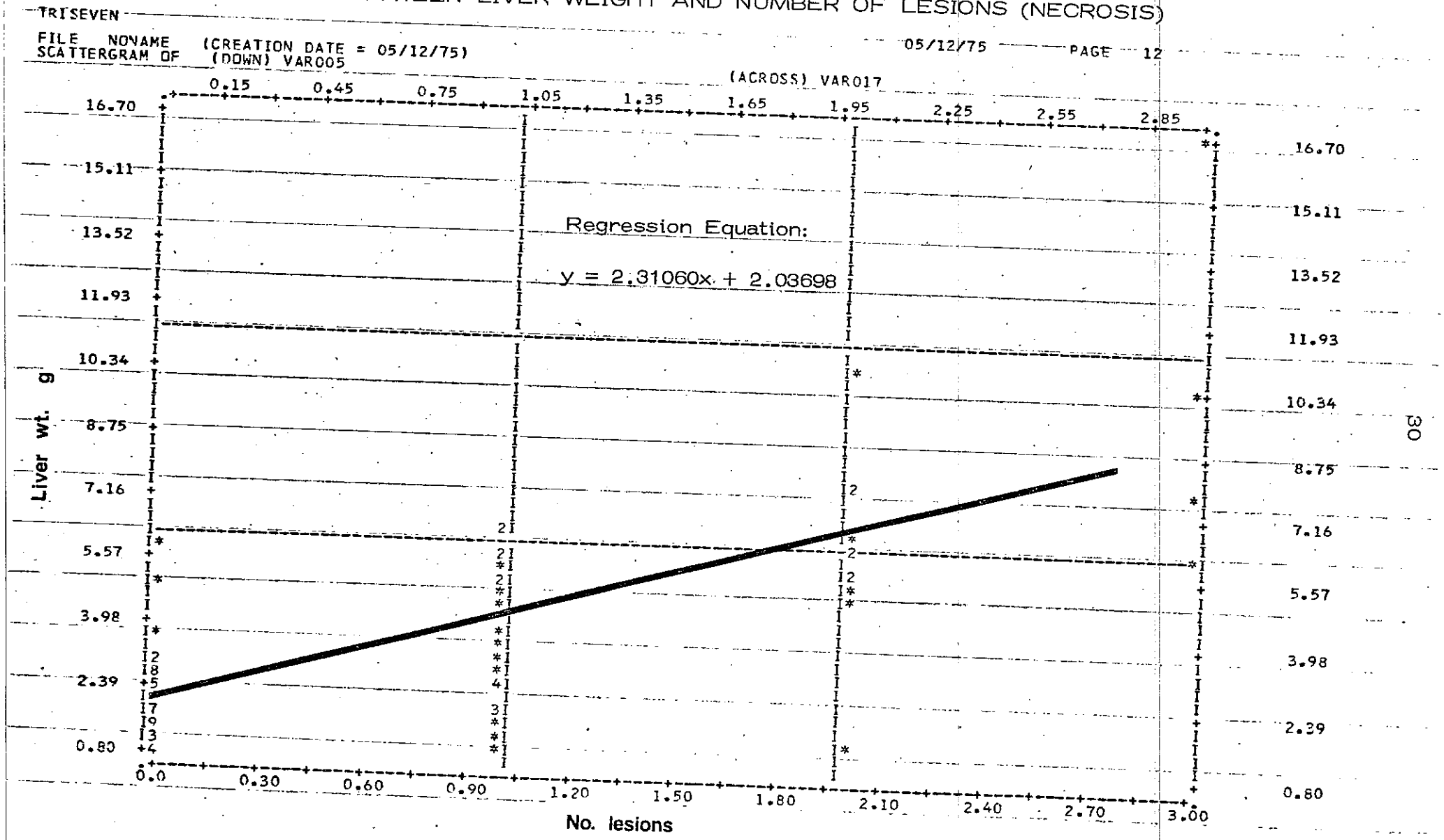


TABLE 9

T-TEST FOR AGE CLASS DIFFERENTIAL IN EXTENT OF TRIAENOPHORIASIS IN WHITE BASS: AGE DETERMINATION BY FISH LENGTH

	No. Encapsulations	No. Plerocercoids	Necrosis
Pooled Variance Estimate-			
t Value	-3.80	-1.15	-9.98
Degrees of Freedom	85	91	81
2-tail Probability	0.000	0.252	0.000
Separate Variance Estimate-			
t Value	-2.68	-1.06	-8.22
Degrees of Freedom	28.09	53.89	31.63
2-tail Probability	0.012	0.292	0.000
F Value	324.00	1.84	2.81
2-tail Probability	0.000	0.041	0.002

TABLE 10

T-TEST FOR AGE CLASS DIFFERENTIAL IN EXTENT OF TRIAENOPHORIASIS IN WHITE BASS: AGE DETERMINATION BY FISH WEIGHT

	No. Encapsulations	No. Plerocercoids	Necrosis
Pooled Variance Estimate-			
t Value	-3.89	-0.92	-9.91
Degrees of Freedom	84	89	80
2-tail Probability	0.000	0.362	0.000
Separate Variance Estimate-			
t Value	-2.70	-0.85	-8.12
Degrees of Freedom	27.08	51.96	30.18
2-tail Probability	0.012	0.398	0.000
F Value	332.60	1.63	2.74
2-tail Probability	0.000	0.107	0.003

TABLE 11

T-TEST FOR SEX DIFFERENTIAL IN EXTENT OF TRIAENOPHORIASIS IN WHITE BAS

	No. Encapsulations	No. Plerocercoids	Necrosis
Pooled Variance Estimate-			
t Value	-1.42	-2.37	-1.81
Degrees of Freedom	85	91	81
2-tail Probability	0.158	0.020	0.074
Separate Variance Estimate-			
t value	-1.76	-2.74	-1.91
Degrees of Freedom	57.27	70.52	79.03
2-tail Probability	0.084	0.008	0.060
F value	30.16	8.75	1.69
2-tail Probability	0.000	0.000	0.117

TABLE 12

MEANS, STANDARD DEVIATIONS, AND STANDARD ERRORS FOR EXTENT OF TRIAENOPHORIASIS IN WHITE BASS: BREAKDOWN BY FISH SEX

	Male	Female	Total Population
Number of Encapsulations-			
Mean	0.7647	2.7736	1.989
Std. Deviation	1.478	8.118	6.455
Std. Error	0.254	1.115	0.692
Number of Plerocercoids-			
Mean	0.1053	0.4727	0.323
Std. Deviation	0.311	0.920	0.754
Std. Error	0.050	0.124	0.078
Necrosis-			
Mean	0.5152	0.8800	0.735
Std. Deviation	0.755	0.982	0.912
Std. Error	0.131	0.139	0.100

TABLE 13

CHI-SQUARE TEST OF SEX BY EXTENT OF TRIAENOPHORIASIS FOR WHITE BASS

	No. Encapsulations	No. Plerocercoids	Necrosis
Chi-square	12.81197	6.15134	3.80835
Degrees of Freedom	9	3	3
Significance	0.1713	0.1045	0.2829

TABLE 14

INCIDENCE OF TRIAENOPHORIASIS IN 121 YOUNG-OF-THE-YEAR WHITE BASS, 1974

Collection Date	N	Average Length mm.	Incidence of Triaenophoriasis %
29 July 1974	30	33.3	0
5 August 1974	23	49.9	0
12 August 1974	23	57.3	0
19 August 1974	24	61.3	4.2
29 August 1974	15	65.6	0
4 September 1974	4	96.2	0
7 October 1974	2	135.0	0
Total	121	54.3	0.83

Chemical Characterization of White Bass Blood

Statistical tests were performed to investigate the chemical properties of white bass blood, and to discover the relationships of blood chemistry to fish size and sex. Five blood factors were investigated: hemoglobin, hematocrit, plasma proteins, total bilirubin, and blood glucose. Statistics describing the data obtained are presented in Table 15.

For each blood factor studied, a "t" test was conducted in order to disclose any difference in blood values between male and female white bass. Results are given in Table 16. In no case was there a significant difference between the sexes in the mean blood values obtained. An "F" test revealed a significant difference in the variablility of total bilirubin; with the males (standard deviation = 1.074) being more variable than the females (standard deviation = 0.654).

Correlations between blood values and fish size are listed in Table 17. Only hematocrit correlated significantly with fish size at the 95% level. Plasma proteins correlated significantly with fish length at the 90% level. Regression relationships are presented in Graphs 10-24.

Fish were assigned to age classes and "t" tests were performed (Tables 18 and 19). Hematocrit was the only factor for which the difference in mean values was significant. Statistical characteristics for hematocrit values in each age class are listed in Tables 20 and 21.

Both age classes exhibited essentially the same variability for each chemical factor.

TABLE 15

MEANS, STANDARD ERRORS, STANDARD DEVIATIONS, VARIANCES, AND RANGES OF WHITE BASS BLOOD VALUES

	Hemoglobin (g%)	Hematocrit	Plasma Protein (g%)	Total Bilirubin(mg%)	Blood Glucose (mg%)
Mean	7.486	45.323	7.150	0.603	242.750
Std. Error	0.130	0.952	0.128	0.092	17.282
Std. Deviation	1.284	9.323	1.263	0.837	91.446
Variance	1.649	86.916	1.594	0.701	8362.414
Range	5.700	47.000	6.600	5.760	318.000
Minimum	4.700	24.000	4.400	0.0	82.000
Maximum	10.400	71.000	11.000	5.760	400.000

TABLE 16

T-TEST FOR SEX DIFFERENTIAL IN BLOOD VALUES IN WHITE BASS

	Hemoglobin	Hematocrit	Plasma Protein	Total Bilirubin	Blood Glucose
Pooled Variance Estimate-					
t value	0.06	0.73	1.28	0.32	1.50
Degrees of Freedom	96	94	96	80	26
2-tail Probability	0.956	0.465	0.205	0.753	0.147
Separate Variance Estimate-					
t value	0.06	0.74	1.28	0.29	1.52
Degrees of Freedom	88.20	87.26	86.09	45.84	22.46
2-tail Probability	0.955	0.461	0.205	0.777	0.144
F Value	1.08	1.14	1.01	2.69	1.13
2-tail Probability	0.812	0.679	0.962	0.002	0.869

TABLE 17

CORRELATIONS BETWEEN SIZE AND BLOOD VALUES IN WHITE BASS

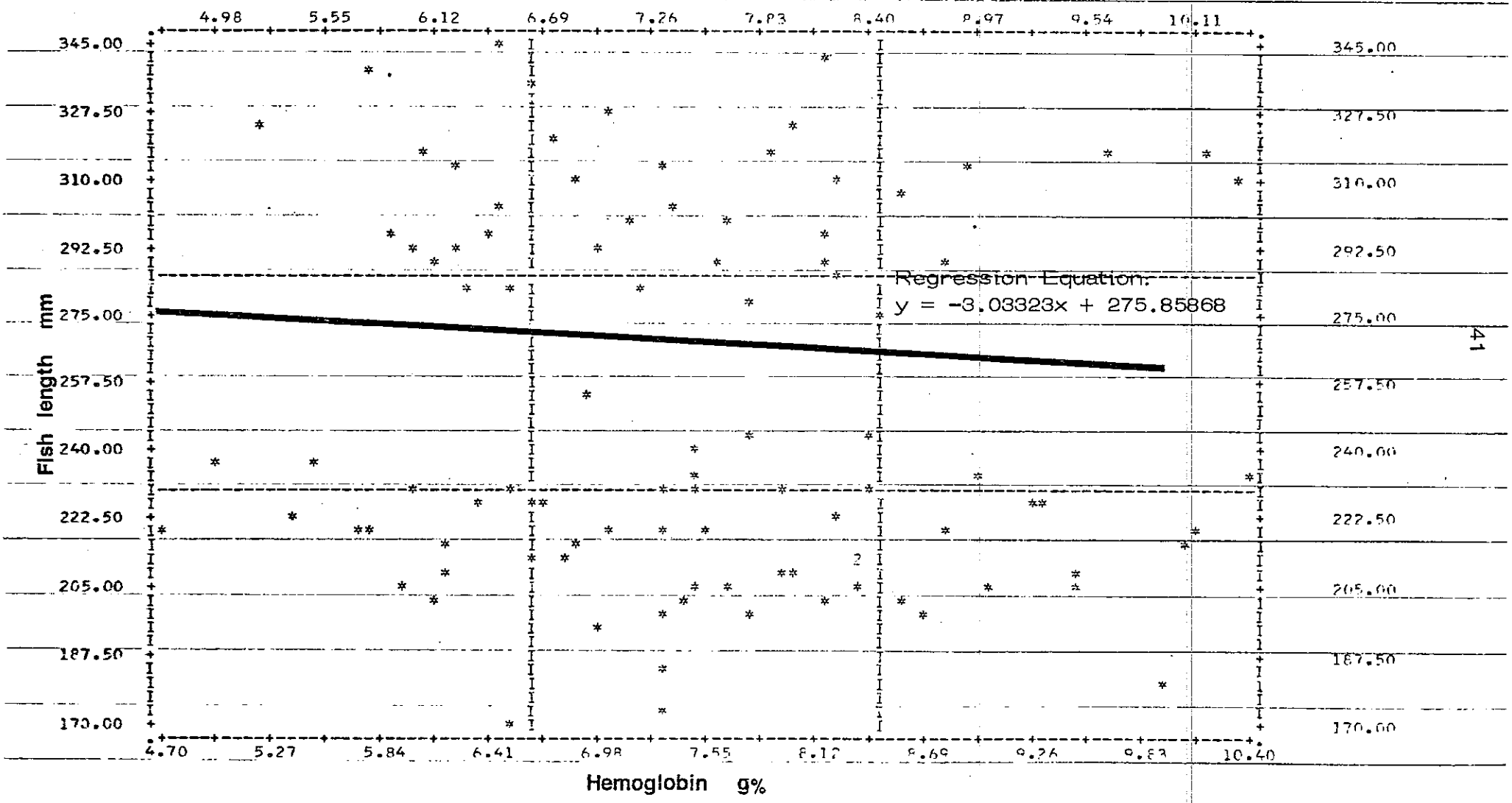
Fish Size	Blood Parameter	Correlation (R)	R^2	Significance
Fish length	x Hemoglobin	-0.08255	0.00682	0.20950
	x Hematocrit	-0.28854	0.08326	0.00218
	x Plasma Protein	-0.13414	0.01799	0.09395
	x Total Bilirubin	0.07998	0.00640	0.23752
	x Blood Glucose	-0.13725	0.01884	0.24307
Fish weight	x Hemoglobin	-0.05797	0.00336	0.28536
	x Hematocrit	-0.28701	0.08238	0.00229
	x Plasma Protein	-0.11176	0.01249	0.13662
	x Total Bilirubin	0.09662	0.00933	0.19394
	x Blood Glucose	-0.06049	0.00366	0.37988
Liver weight	x Hemoglobin	-0.08447	0.00714	0.20785
	x Hematocrit	-0.29314	0.08593	0.00217
	x Plasma Protein	-0.08418	0.00709	0.20866
	x Total Bilirubin	0.03463	0.00120	0.38021
	x Blood Glucose	-0.12261	0.01503	0.27117

GRAPH 10

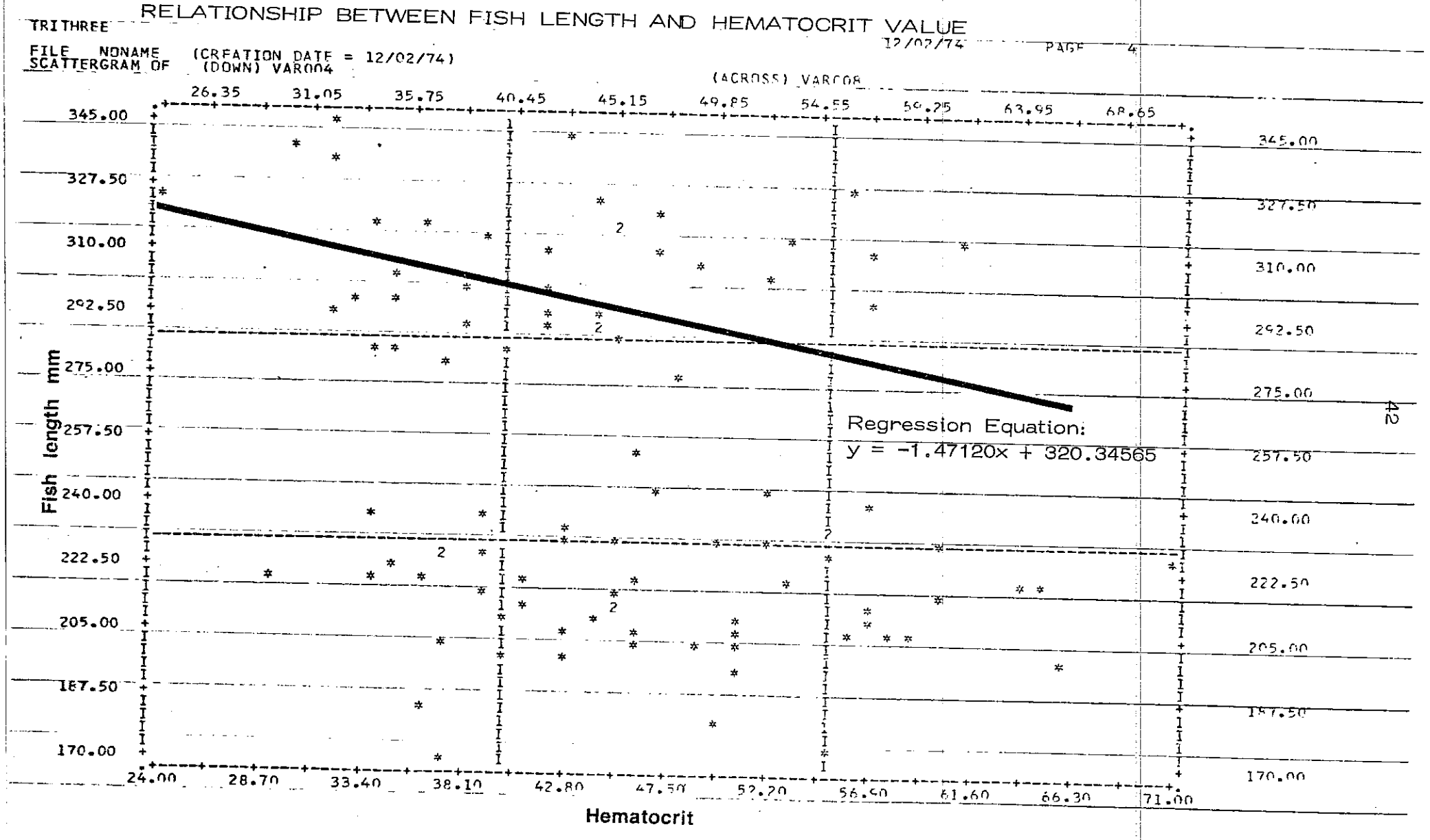
RELATIONSHIP BETWEEN FISH LENGTH AND HEMOGLOBIN LEVEL

TRITHREE 12/02/74 PAGE 2

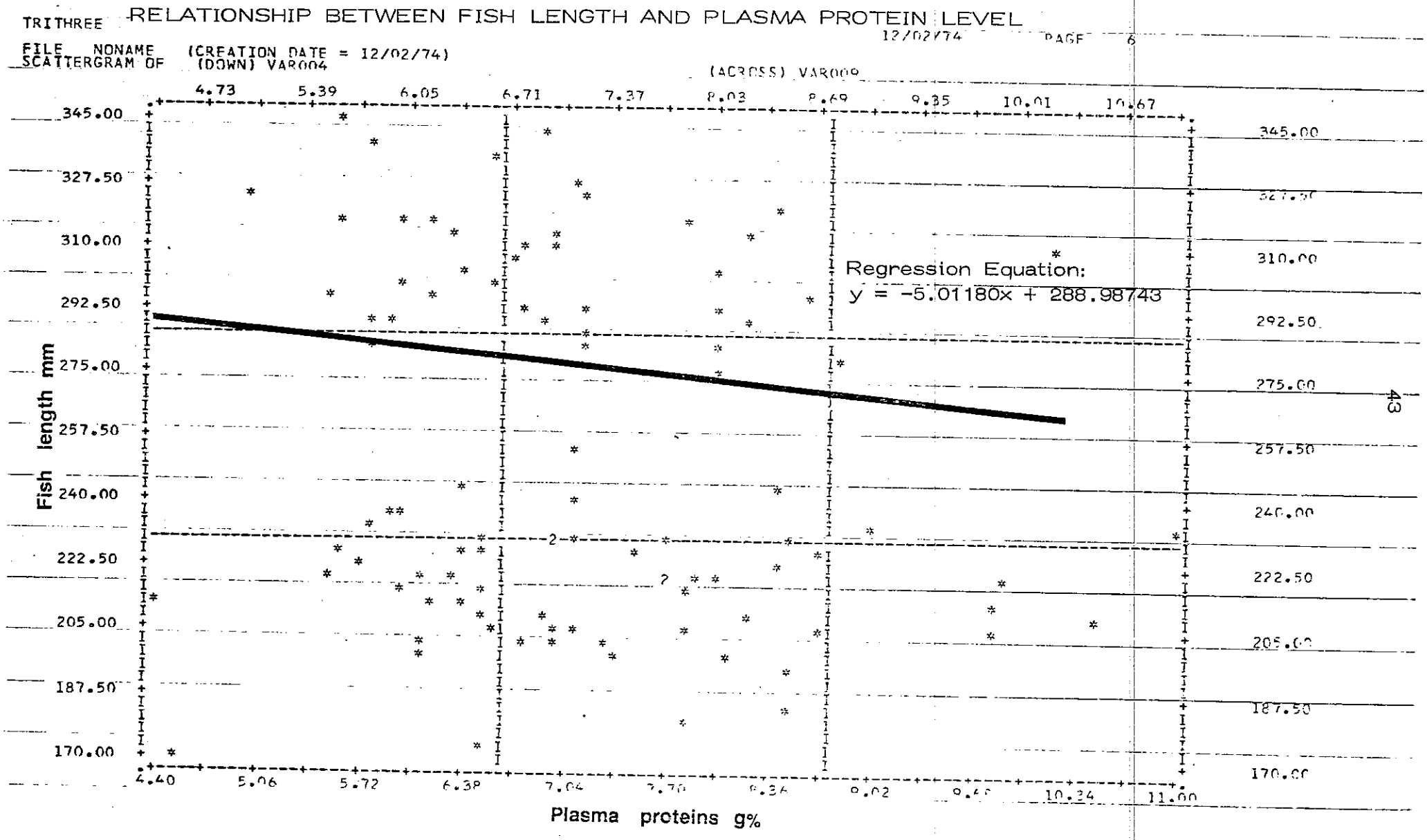
FILE NONAME (CREATION DATE = 12/02/74)
SCATTERGRAM OF (DOWN) VAR004 (ACROSS) VAR007



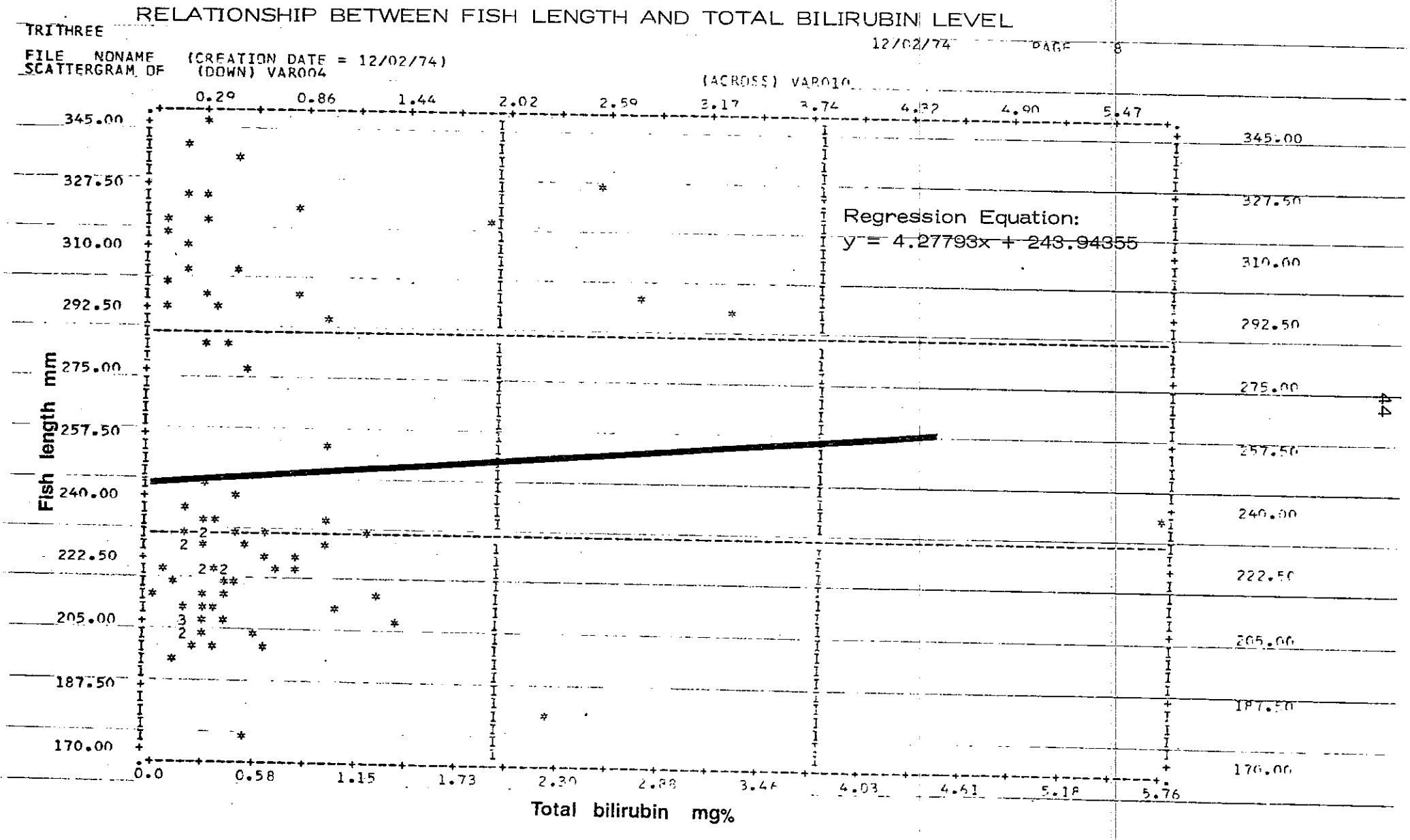
GRAPH 11


RELATIONSHIP BETWEEN FISH LENGTH AND HEMATOCRIT VALUE
TRITHREE
FILE NONAME (CREATION DATE = 12/02/74)
SCATTERGRAM OF (DOWN) VAR004
12/02/74 PAGE 4
(ACROSS) VAR008
26.35 31.05 35.75 40.45 45.15 49.85 54.55 59.25 63.95 68.65
345.00
327.50
310.00
292.50
275.00
257.50
240.00
222.50
205.00
187.50
170.00
Fish length mm
Regression Equation:
y = -1.47120x + 320.34565
24.00 28.70 33.40 38.10 42.80 47.50 52.20 56.90 61.60 66.30 71.00
Hematocrit
42

GRAPH 12

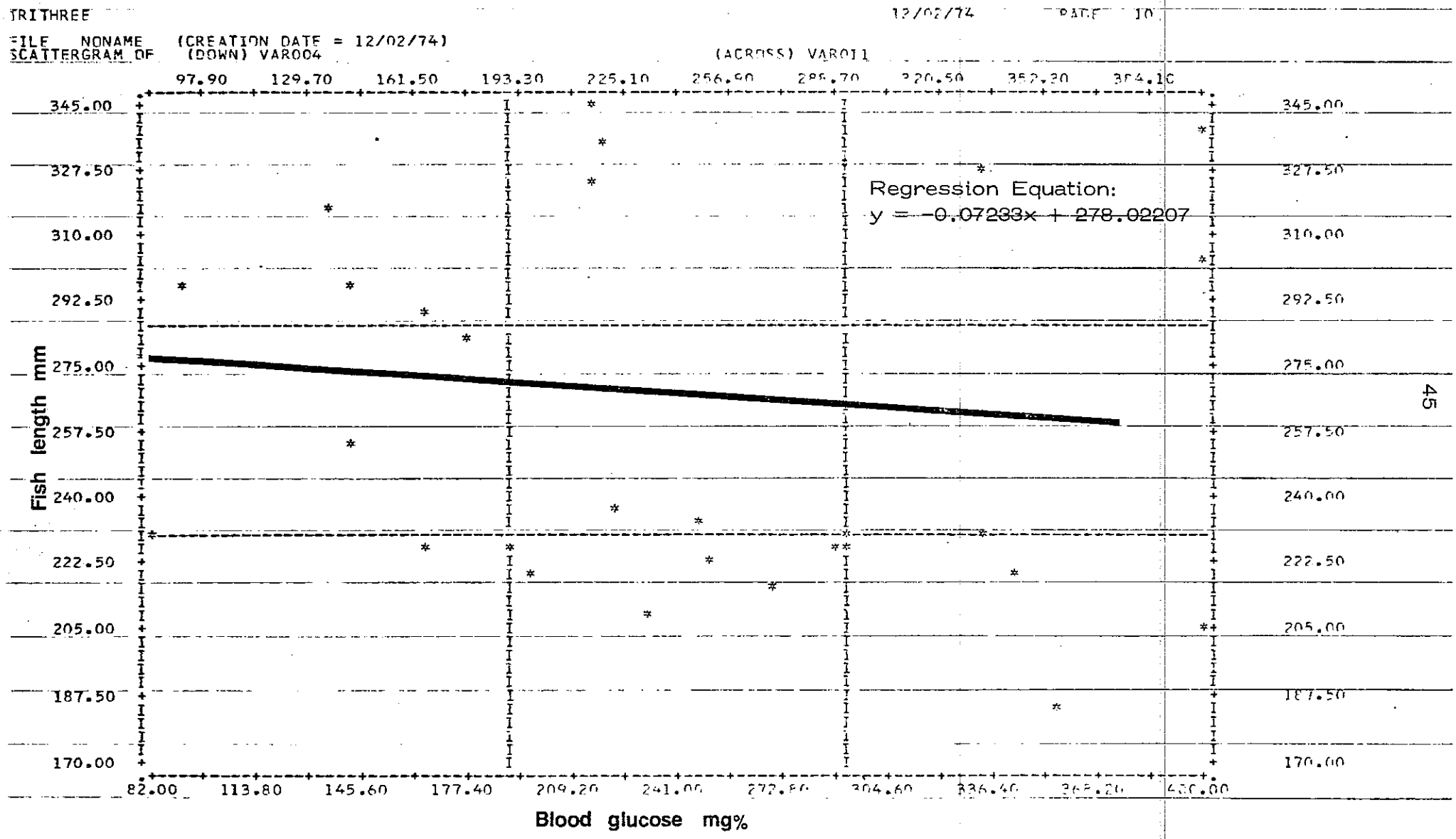

TRITHREE RELATIONSHIP BETWEEN FISH LENGTH AND PLASMA PROTEIN LEVEL
12/02/74 PAGE 6
FILE NONAME (CREATION DATE = 12/02/74)
SCATTERGRAM OF (DOWN) VAR004
(ACROSS) VAR009
4.73 5.39 6.05 6.71 7.37 8.03 8.69 9.35 10.01 10.67
345.00
327.50
310.00
292.50
275.00
257.50
240.00
222.50
205.00
187.50
170.00
Regression Equation:
y = -5.01180x + 288.98743
Fish length mm
4.40 5.06 5.72 6.38 7.04 7.70 8.36 9.02 9.68 10.34 11.00
Plasma proteins g%
43

GRAPH 13

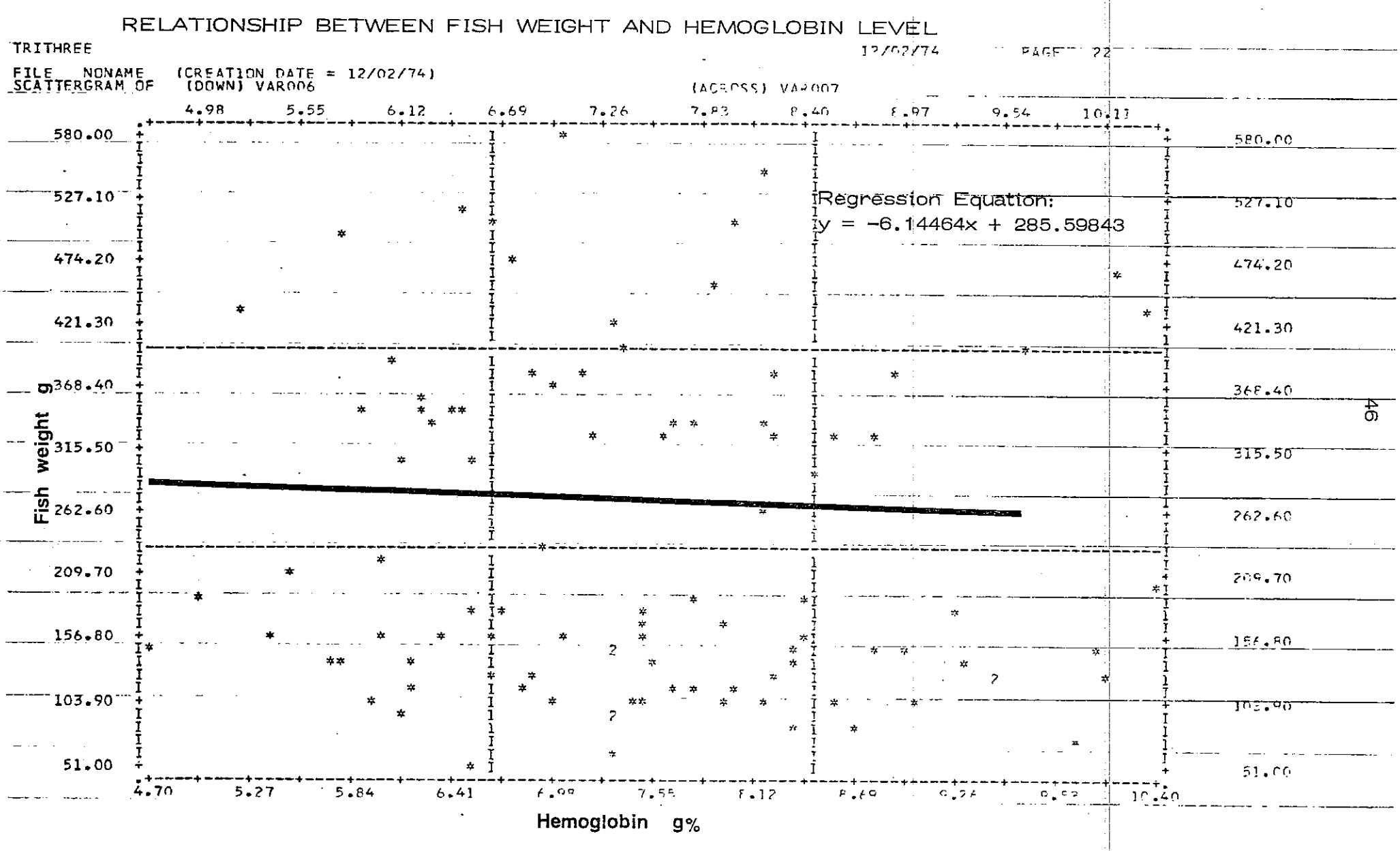

RELATIONSHIP BETWEEN FISH LENGTH AND TOTAL BILIRUBIN LEVEL
TRITHREE
12/02/74 PAGE 8
FILE NONAME (CREATION DATE = 12/02/74)
SCATTERGRAM OF (DOWN) VAR004
(ACROSS) VAR010
0.29 0.86 1.44 2.02 2.59 3.17 3.74 4.32 4.90 5.47
345.00
327.50
310.00
292.50
275.00
257.50
240.00
222.50
205.00
187.50
170.00
Regression Equation:
y = 4.27793x + 243.94355
Fish length mm
0.0 0.58 1.15 1.73 2.30 2.88 3.46 4.03 4.61 5.18 5.76
Total bilirubin mg%

GRAPH 14

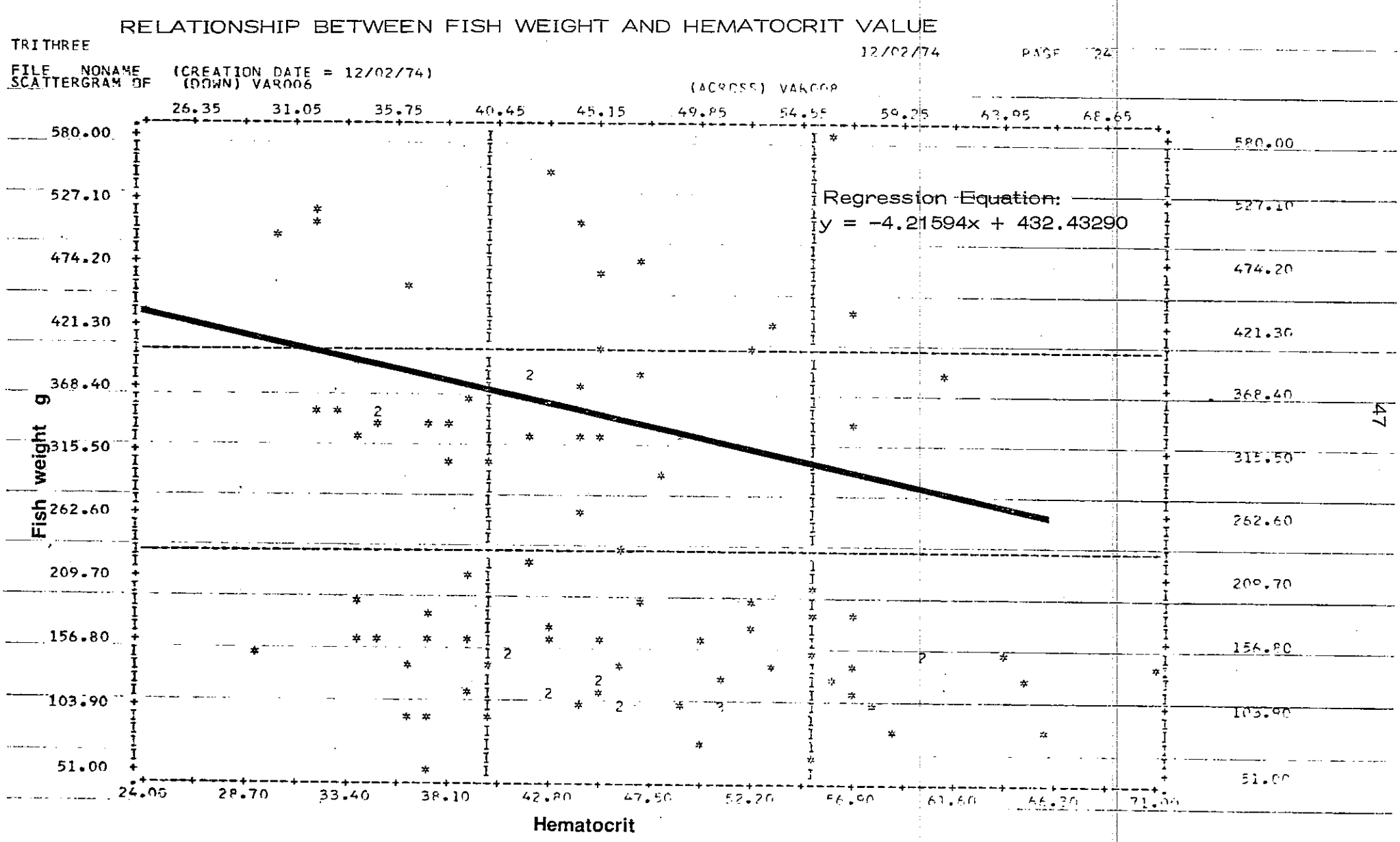
RELATIONSHIP BETWEEN FISH LENGTH AND BLOOD GLUCOSE LEVEL



GRAPH 15

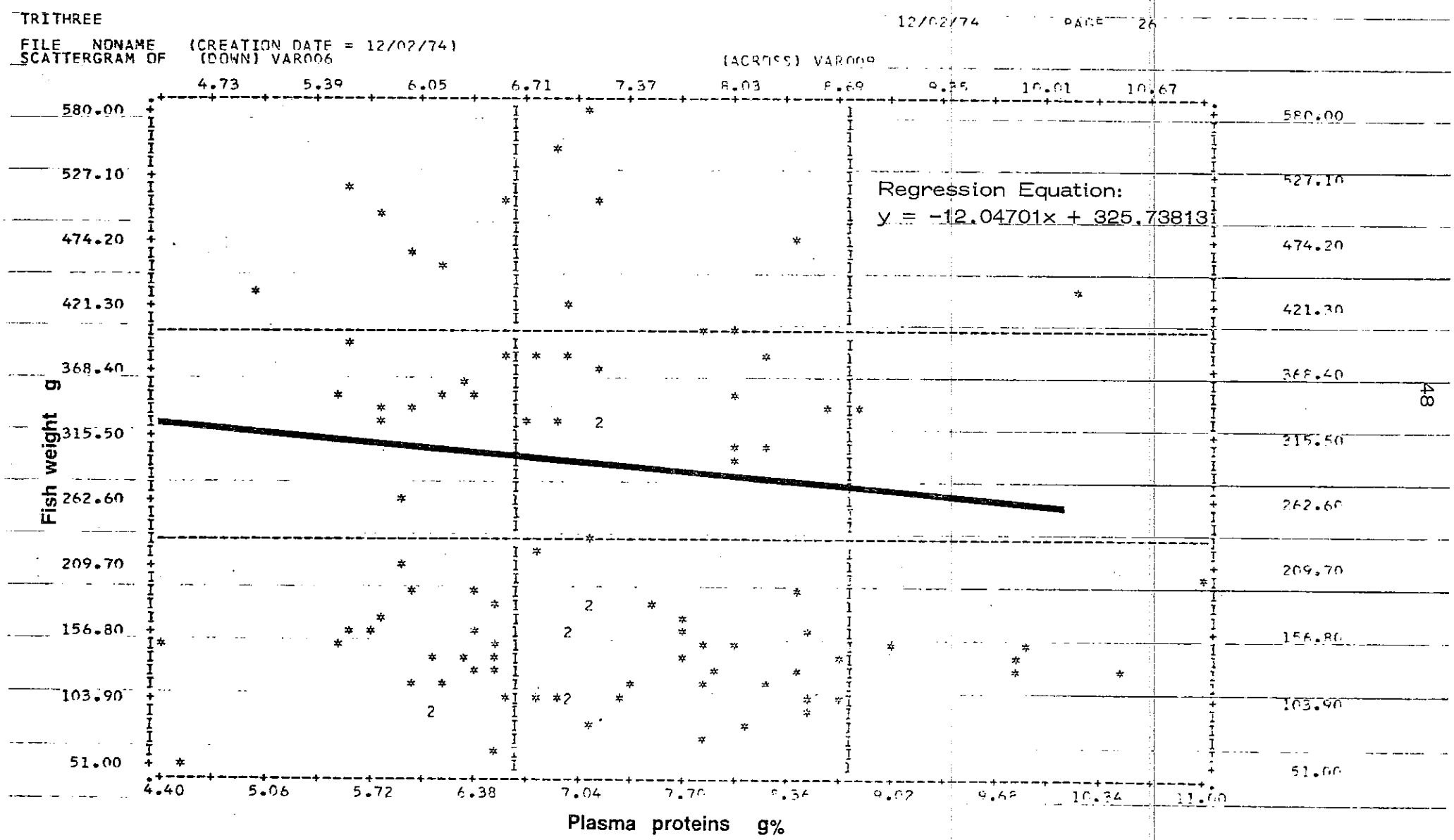

RELATIONSHIP BETWEEN FISH WEIGHT AND HEMOGLOBIN LEVEL
TRITHREE
12/02/74
PAGE 22
FILE NONAME (CREATION DATE = 12/02/74)
SCATTERGRAM OF (DOWN) VAR006
(ACROSS) VAR007
4.98
5.55
6.12
6.69
7.26
7.83
8.40
8.97
9.54
10.11
580.00
527.10
474.20
421.30
368.40
315.50
262.60
209.70
156.80
103.90
51.00
Regression Equation:
y = -6.14464x + 285.59843
Fish weight g
4.70
5.27
5.84
6.41
6.98
7.55
8.12
8.69
9.26
9.83
10.40
Hemoglobin g%

GRAPH 16


RELATIONSHIP BETWEEN FISH WEIGHT AND HEMATOCRIT VALUE
TRITHREE
12/02/74
PAGE 24
FILE NONAME (CREATION DATE = 12/02/74)
SCATTERGRAM OF (DOWN) VAR006
(ACROSS) VAR008
26.35
31.05
35.75
40.45
45.15
49.85
54.55
59.25
63.95
68.65
580.00
527.10
474.20
421.30
368.40
315.50
262.60
209.70
156.80
103.90
51.00
Regression Equation:
y = -4.21594x + 432.43290
Fish weight g
24.05
28.70
33.40
38.10
42.80
47.50
52.20
56.90
61.60
66.30
71.00
Hematocrit
47

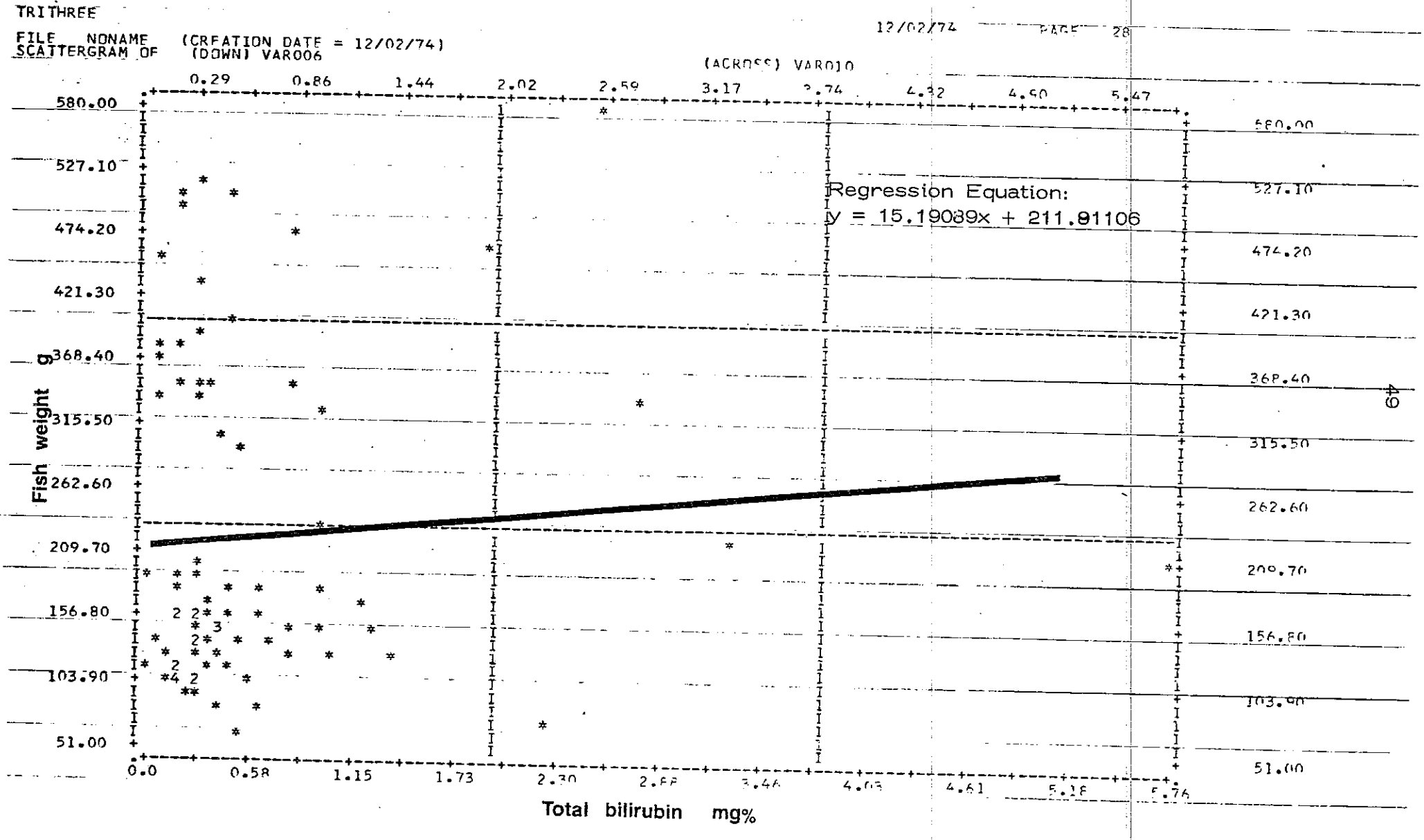
GRAPH 17

RELATIONSHIP BETWEEN FISH WEIGHT AND PLASMA PROTEIN LEVEL



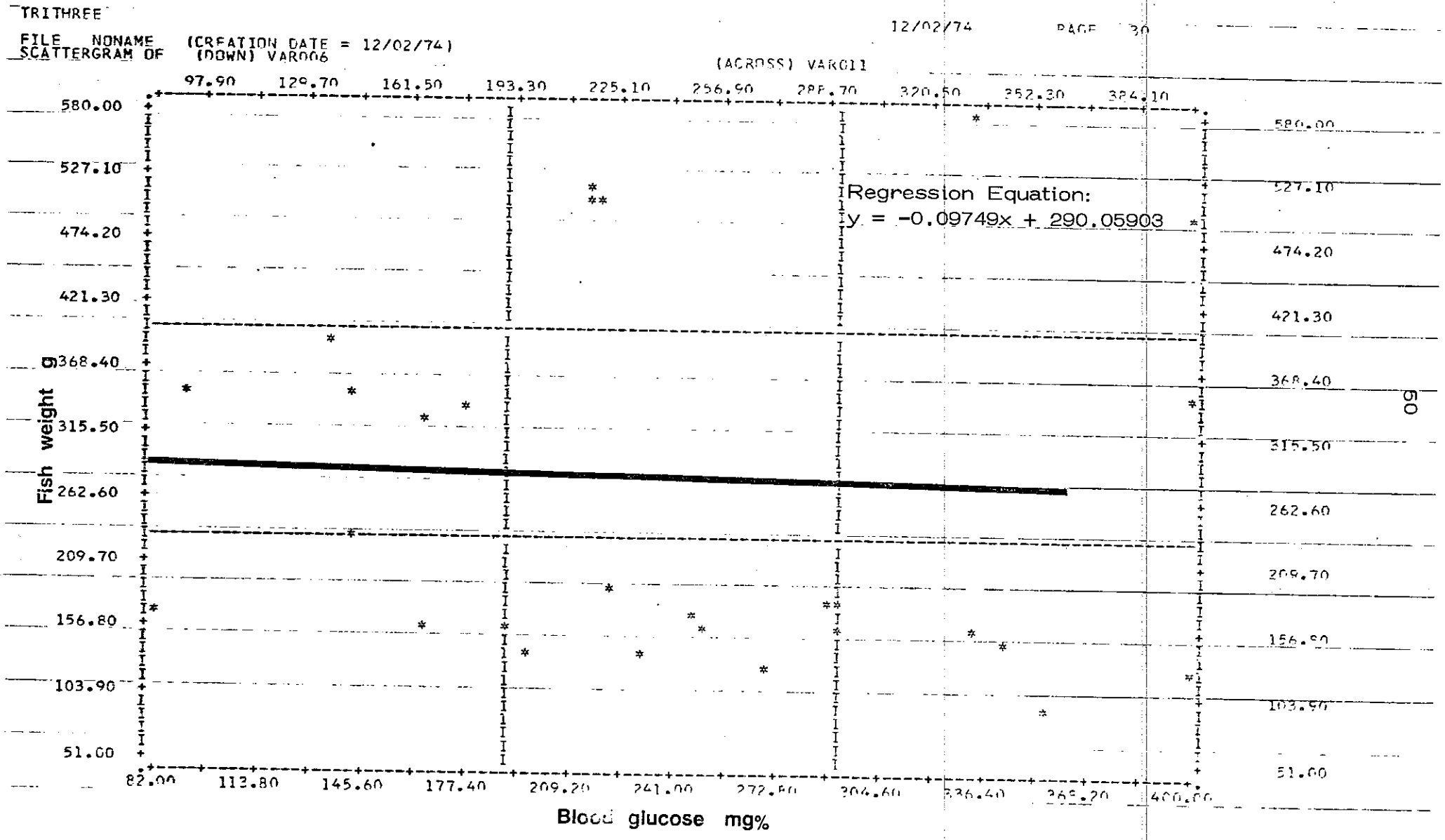
GRAPH 18

RELATIONSHIP BETWEEN FISH WEIGHT AND TOTAL BILIRUBIN LEVEL



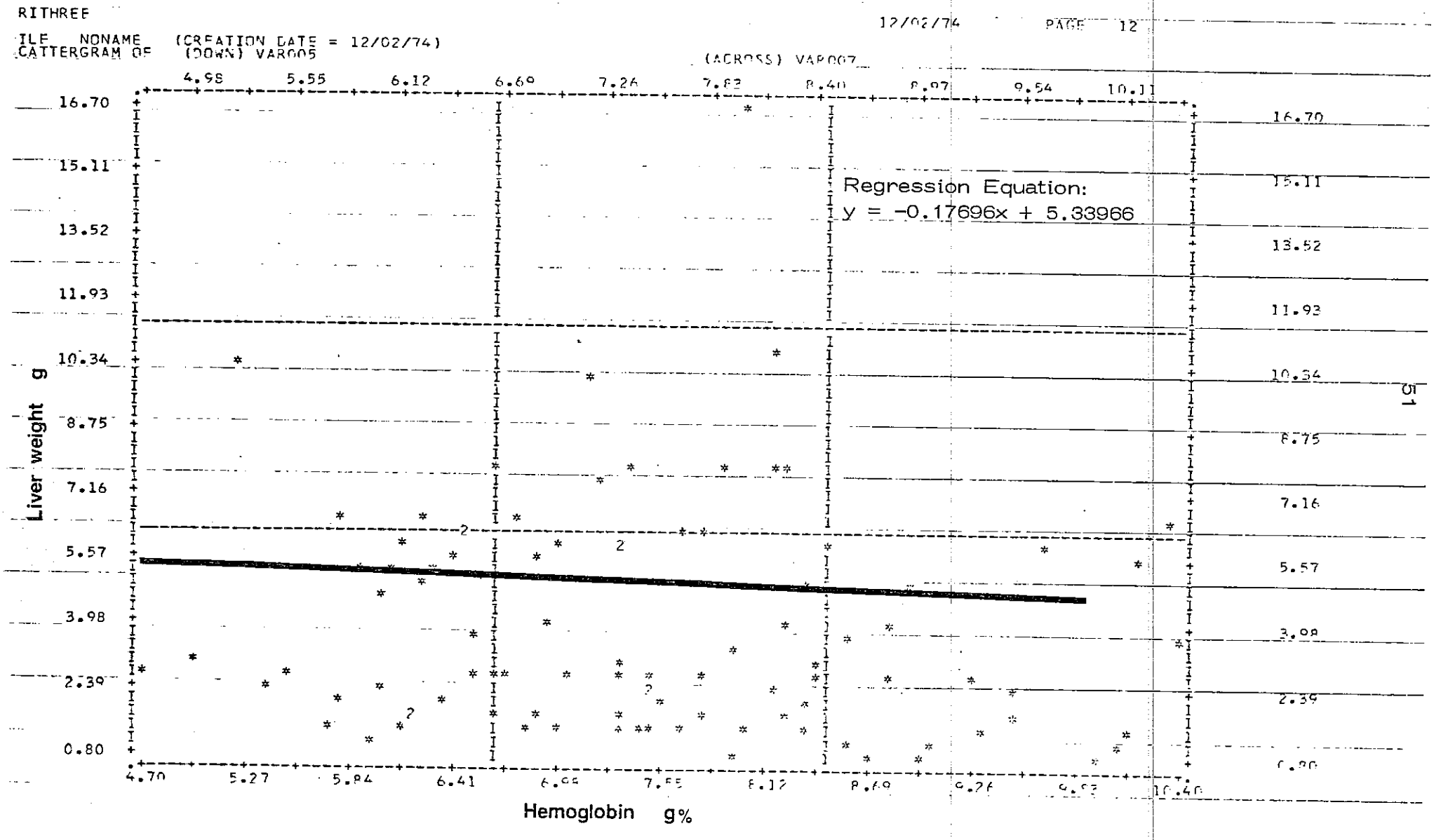
GRAPH 19

RELATIONSHIP BETWEEN FISH WEIGHT AND BLOOD GLUCOSE LEVEL



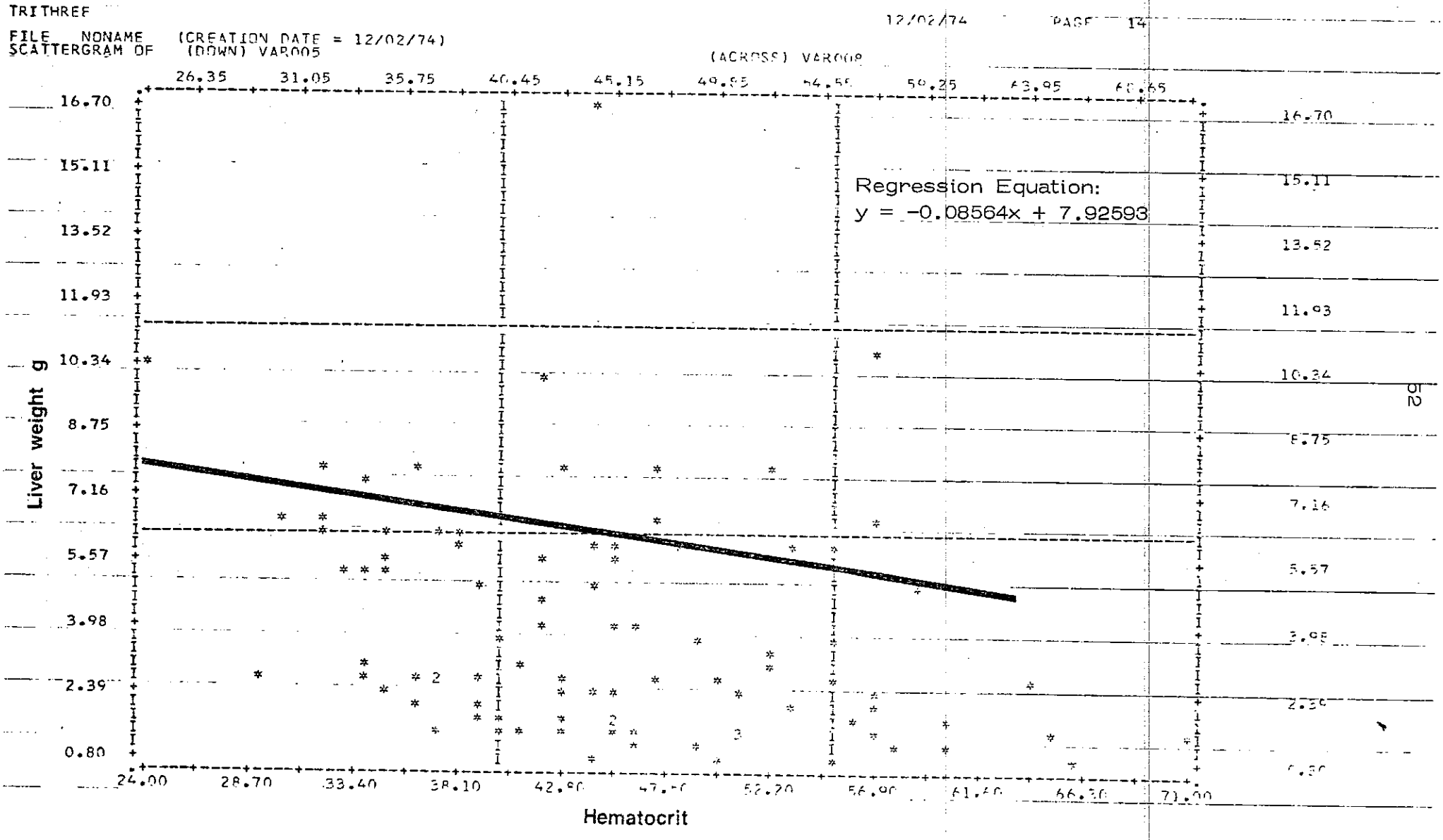
GRAPH 20

RELATIONSHIP BETWEEN LIVER WEIGHT AND HEMOGLOBIN LEVEL

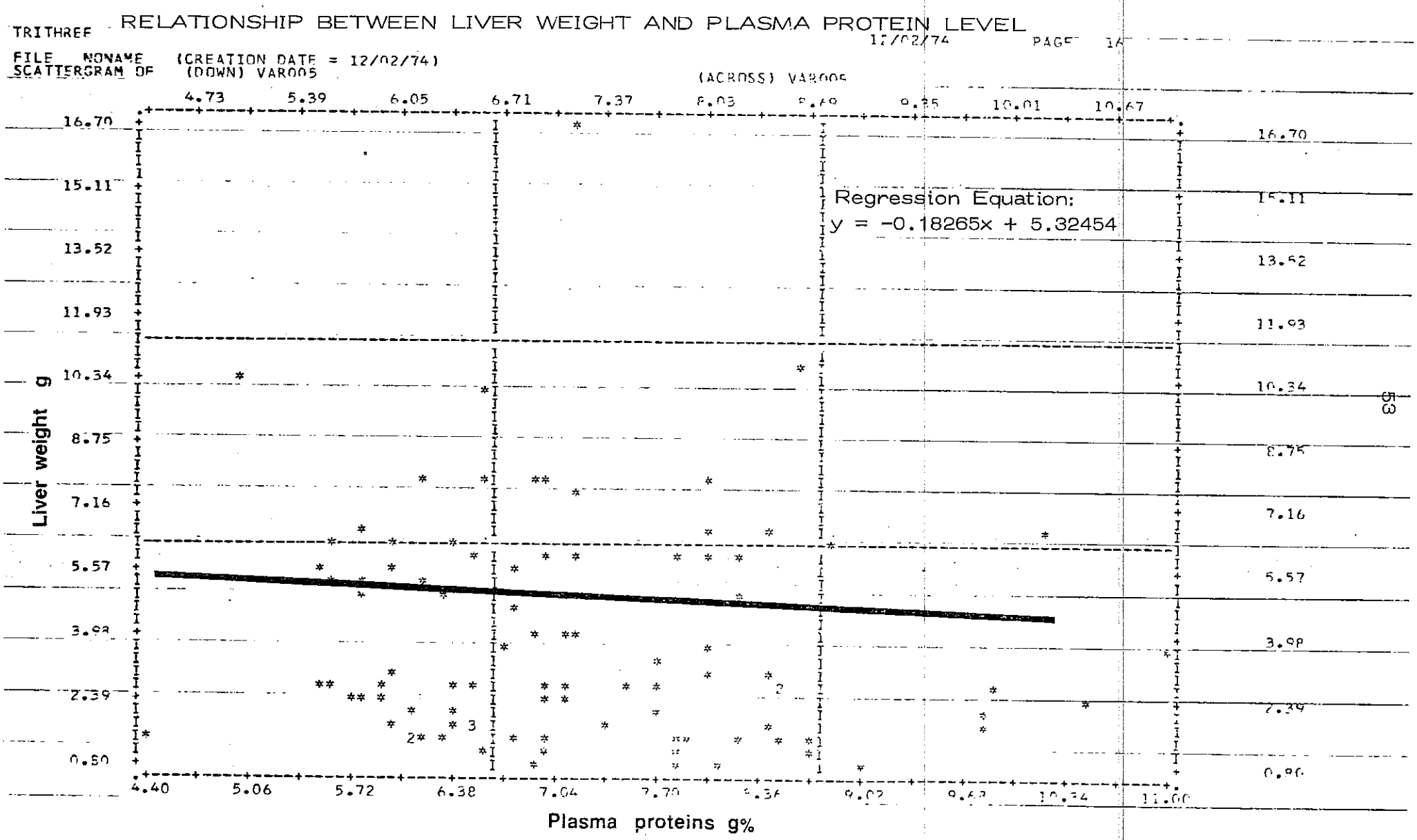


GRAPH 21

RELATIONSHIP BETWEEN LIVER WEIGHT AND HEMATOCRIT VALUE

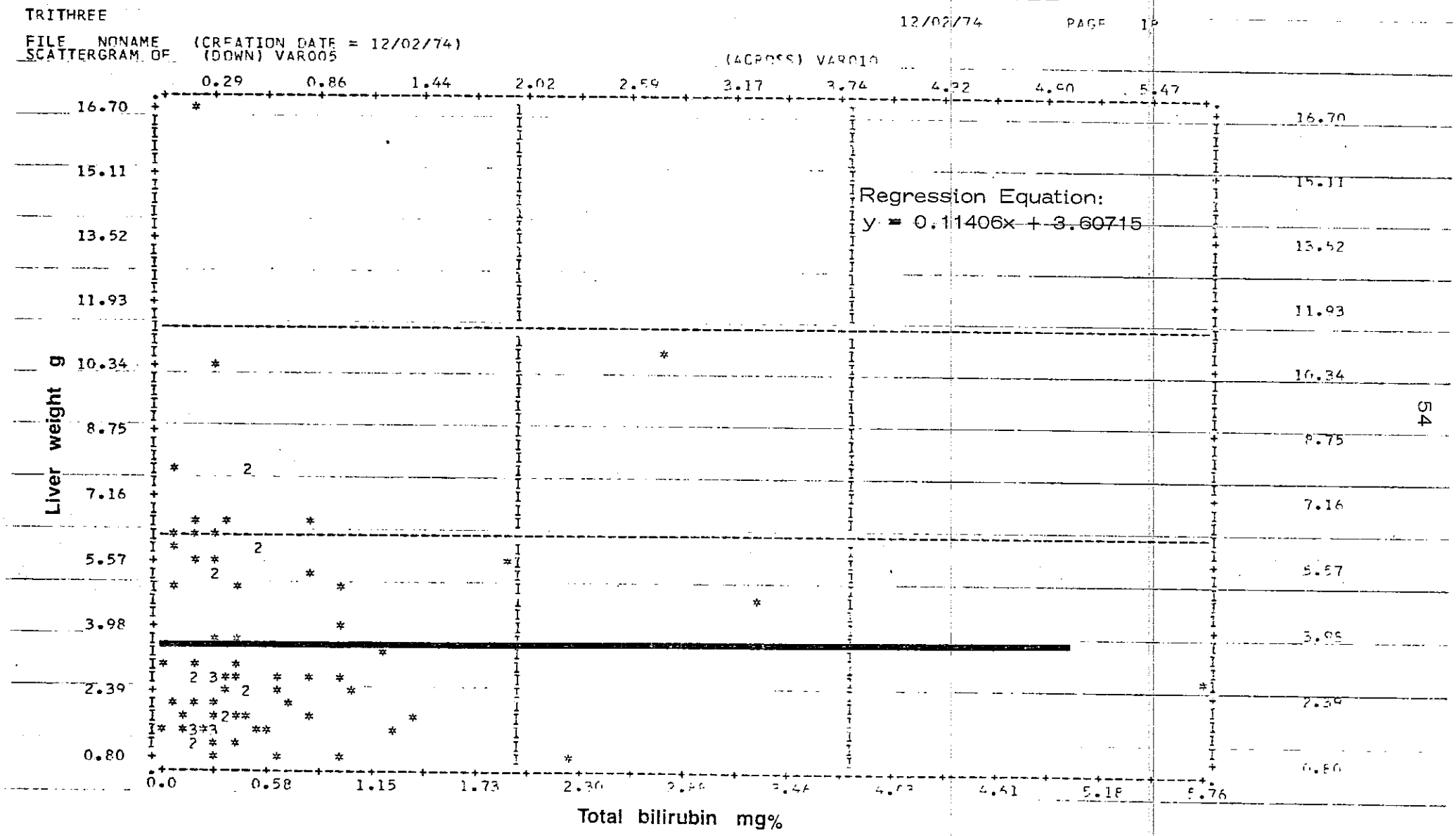


GRAPH 22


TRITHREE
RELATIONSHIP BETWEEN LIVER WEIGHT AND PLASMA PROTEIN LEVEL
FILE NONAME (CREATION DATE = 12/02/74)
SCATTERGRAM OF (DOWN) VAR005
(ACROSS) VAR006
Regression Equation:
y = -0.18265x + 5.32454
Liver weight g
Plasma proteins g%
16.70
15.11
13.52
11.93
10.34
8.75
7.16
5.57
3.98
2.39
0.80
4.40
5.06
5.72
6.38
7.04
7.70
8.36
9.02
9.68
10.34
11.00

GRAPH 23

RELATIONSHIP BETWEEN LIVER WEIGHT AND TOTAL BILIRUBIN LEVEL



GRAPH 24

RELATIONSHIP BETWEEN LIVER WEIGHT AND BLOOD GLUCOSE LEVEL

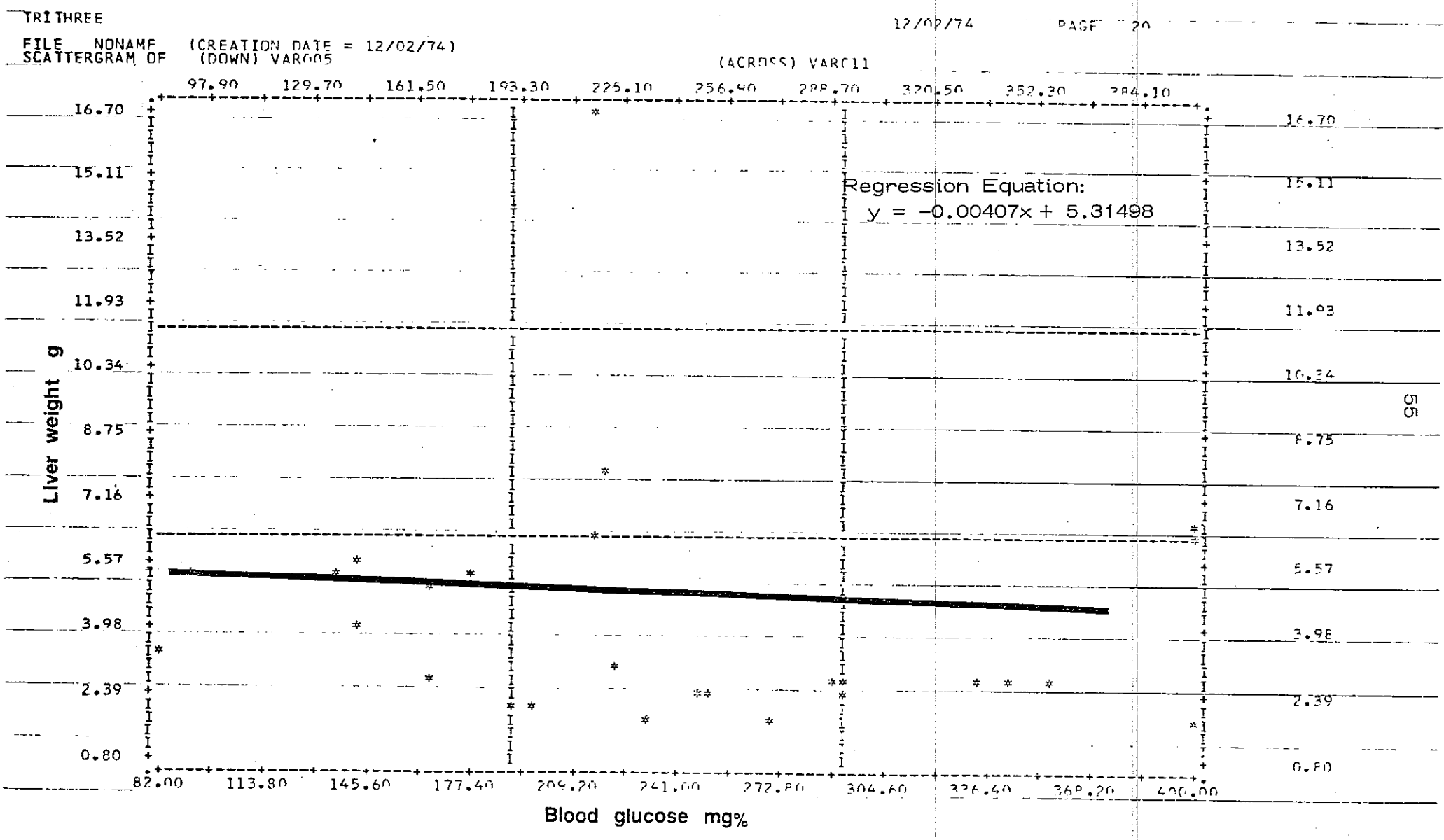


TABLE 18

T-TEST FOR AGE CLASS DIFFERENTIAL IN BLOOD VALUES OF WHITE BASS: AGE DETERMINATION BY FISH LENGTH

	Hemoglobin	Hematocrit	Plasma Protein	Total Bilirubin	Blood Glucose
Pooled Variance Estimate-					
t value	0.62	3.14	1.20	-0.73	0.74
Degrees of Freedom	96	94	96	80	26
2-tail Probability	0.538	0.002	0.232	0.467	0.466
Separate Variance Estimate					
t value	0.63	3.20	1.26	-0.70	0.70
Degrees of Freedom	86.26	87.32	91.88	41.21	17.66
2-tail Probability	0.531	0.002	0.212	0.490	0.493
F value	1.19	1.25	1.52	1.28	1.66
2-tail Probability	0.578	0.478	0.174	0.444	0.356

TABLE 19

T-TEST FOR AGE CLASS DIFFERENTIAL IN BLOOD VALUES OF WHITE BASS: AGE DETERMINATION BY FISH WEIGHT

	Hemoglobin	Hematocrit	Plasma Protein	Total Bilirubin	Blood Glucose
Pooled Variance Estimate-					
t value	0.56	3.06	1.19	0.05	0.74
Degrees of Freedom	94	92	94	79	26
2-tail Probability	0.574	0.003	0.239	0.963	0.466
Separate Variance Estimate-					
t value	0.58	3.10	1.23	0.05	0.70
Degrees of Freedom	81.72	80.89	85.68	50.66	17.66
2-tail Probability	0.567	0.003	0.222	0.961	0.493
F value	1.20	1.15	1.39	1.39	1.66
2-tail Probability	0.571	0.658	0.294	0.389	0.356

TABLE 20

MEANS, STANDARD DEVIATIONS, AND STANDARD ERRORS OF HEMATOCRIT VALUES IN WHITE BASS:
BREAKDOWN BY AGE CLASS AS DETERMINED BY FISH LENGTH

	Yearling	2+ Year	Total Population
Mean	47.6842	41.8718	45.323
Std. Deviation	9.295	8.329	9.323
Std. Error	1.231	1.334	0.952

TABLE 21

MEANS, STANDARD DEVIATIONS, AND STANDARD ERRORS OF HEMATOCRIT VALUES IN WHITE BASS:
BREAKDOWN BY AGE CLASS AS DETERMINED BY FISH WEIGHT

	Yearling	2+ Year	Total Population
Mean	47.5789	41.8108	45.323
Std. Deviation	9.177	8.550	9.323
Std. Error	1.215	1.406	0.952

Effects of Triaenophoriasis on White Bass Blood Chemistry

Triaenophoriasis in each study fish was evaluated in three categories: number of encapsulations, number of unencapsulated plerocercoids, and extent of liver necrosis. Correlations between the five blood factors and these three aspects of triaenophoriasis are presented in Table 22; correlations signifigant at the 95% level were found between hematocrit and necrosis, and between plasma protein and necrosis. Regression equations are plotted in Graphs 25-39.

For the purpose of "t" tests, fish were divided into groups triaenophoriasis positive and triaenophoriasis negative for each of the three categories. Results are presented in Tables 23-25. Probabilities signifigant at the 90% level were found for a difference in mean hematocrit values between fish with and without encapsulations, and between those with and without necrosis. Specific statistics are given in Tables 26 and 27, respectively. Also, a significant difference in mean values of blood glucose was found between fish with and without necrosis; statistics are given in Table 28. In view of the lack of correlation between blood glucose and necrosis (Table 22), it seems likely that this is a spurious difference, possibly caused by the relatively small "N" and the relatively high variance (Table 28) for this character.

Among all the correlation coefficients which were calculated, significant correlations were found between the following pairs of characters: hematocrit and necrosis, hematocrit and fish size,

plasma proteins and necrosis, plasma proteins and fish size (at the 90% level), encapsulations and fish size, and necrosis and fish size. Correlations of fish weight were sufficiently close to those of fish length that they may be considered equivalent; correlations of fish length were used in the following computations.

Partial correlation coefficients, designed to reveal the true correlation between two factors excluding the effects of a third factor, were calculated. Partial correlations for the factors hematocrit, necrosis, and size are given in Table 29. The strongest correlation was between necrosis and size, the second strongest (a negative correlation) between hematocrit and size. These results indicate that hematocrit values are normally lower in larger fish, that larger fish have accumulated more liver necrosis, and that necrosis itself does not effect hematocrit values. Partial correlations for the factors plasma protein, necrosis, and size are presented in Table 30. In this case, plasma proteins correlated more strongly with necrosis than with fish size, indicating that liver necrosis resulting from triaenophoriasis is directly related to a lowered plasma protein level. No direct relationships between number of encapsulations and altered blood values were found.

TABLE 22

CORRELATIONS BETWEEN BLOOD VALUES AND TRIAENOPHORIASIS IN WHITE BASS

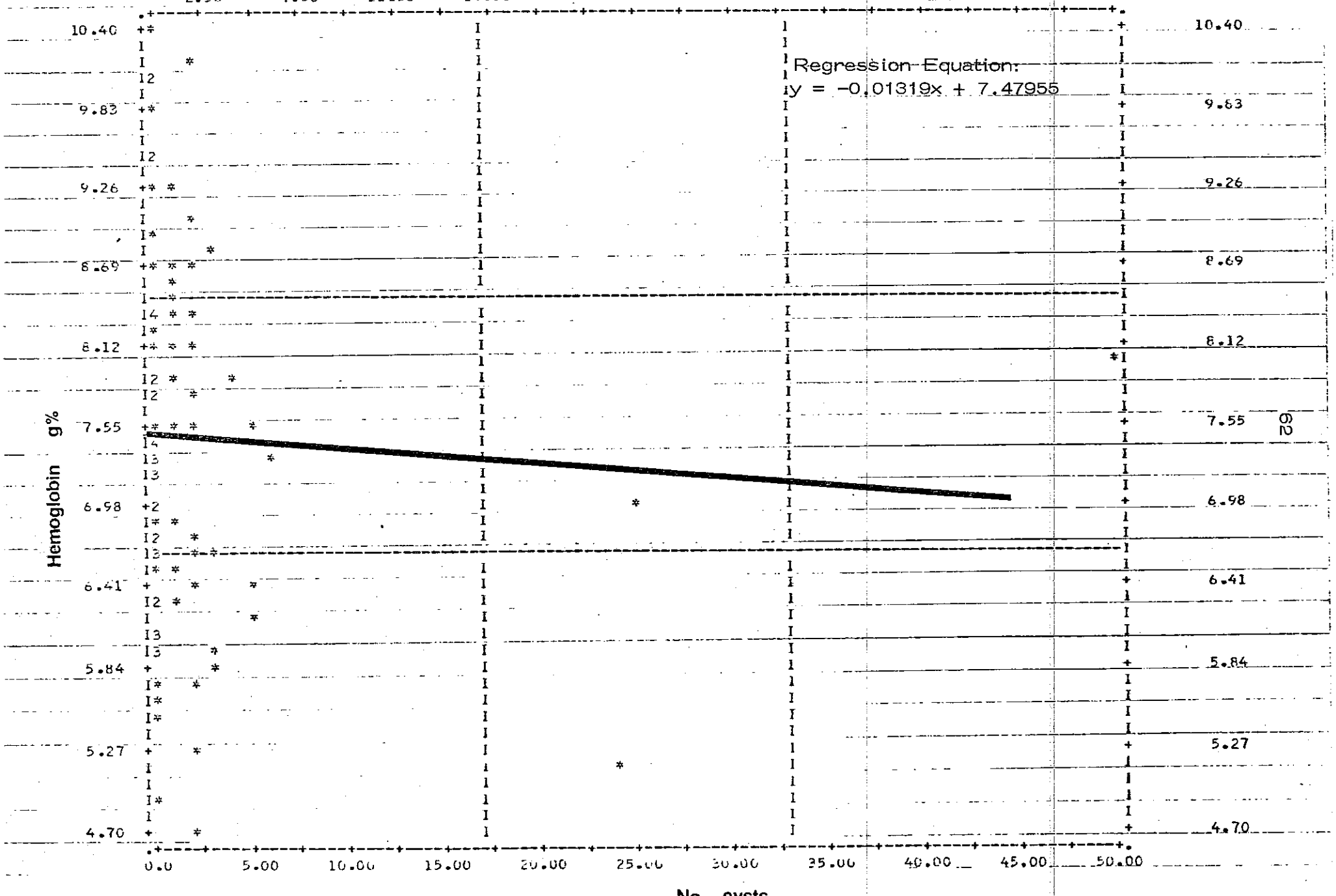
Parameter	Triaenophoriasis	Correlation (R)	R^2	Significance
Hemoglobin	× No. encapsulations	-0.06654	0.00443	0.27015
	× No. plerocercoids	0.10232	0.01047	0.16455
	× Necrosis	-0.11525	0.01328	0.14975
Hematocrit	× No. encapsulations	-0.11812	0.01395	0.14081
	× No. plerocercoids	0.00134	0.00000	0.49497
	× Necrosis	-0.27744	0.07697	0.00608
Plasma Protein	× No. encapsulations	-0.10232	0.01047	6.17283
	× No. plerocercoids	-0.03460	0.00120	0.37097
	× Necrosis	-0.24736	0.06119	0.01208
Total Bilirubin	× No. encapsulations	0.03151	0.00099	0.38933
	× No. plerocercoids	0.08536	0.00729	0.22288
	× Necrosis	0.12992	0.01688	0.12235
Blood Glucose	× No. encapsulations	0.03648	0.00133	0.42690
	× No. plerocercoids	0.09868	0.00974	0.30868
	× Necrosis	-0.06023	0.00363	0.38039

GRAPH 25

TRIONE 11/07/74 PAGE 4

FILE NONAME (CREATION DATE = 11/07/74)
SCATTERGRAM.OF (DOWN) VAR007 (ACROSS) VAR013

RELATIONSHIP BETWEEN HEMOGLOBIN LEVEL AND NUMBER OF CYSTS (ENCAPSULATIONS)



TRIONE

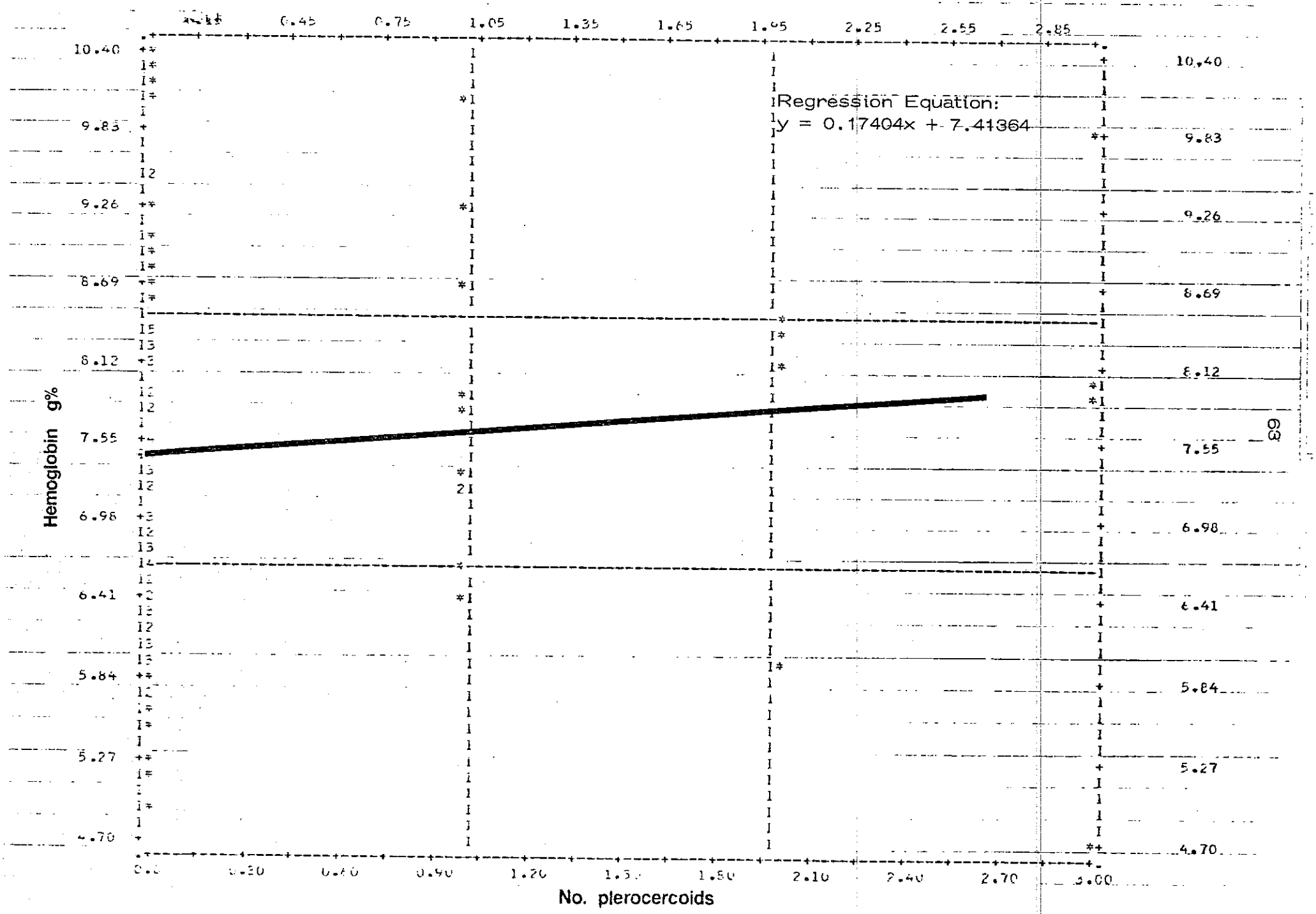
GRAPH 26

11/07/74 PAGE 6

RELATIONSHIP BETWEEN HEMOGLOBIN LEVEL AND NUMBER OF UNENCAPSULATED PLEROCERCOIDS

FILE NONAME (CREATION DATE = 11/07/74)

SCATTERGRAM OF (DOWN) VAR007 (ACROSS) VAR015



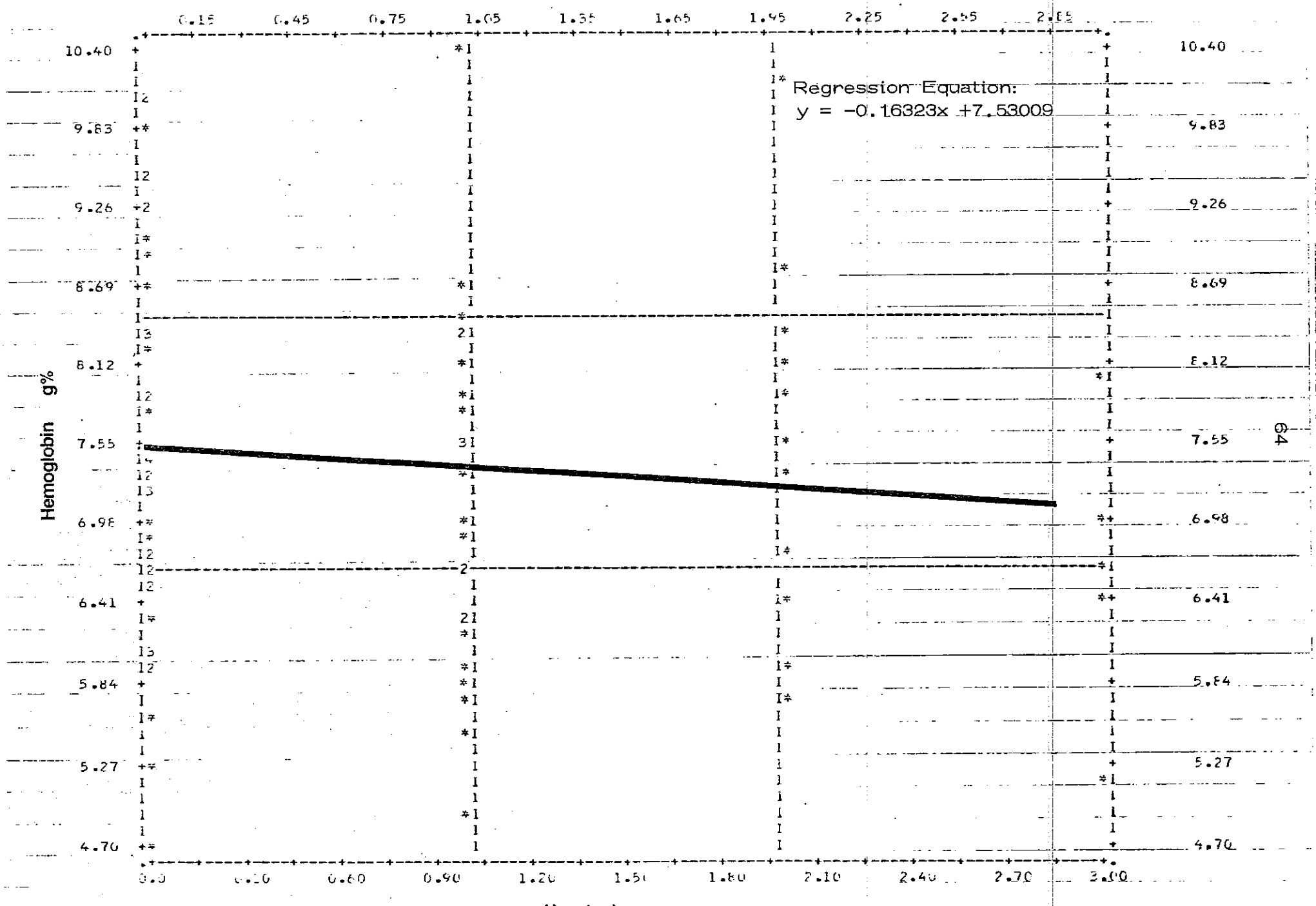
GRAPH 27

TRIONE 11/07/74 PAGE 8

RELATIONSHIP BETWEEN HEMOGLOBIN LEVEL AND NUMBER OF LESIONS(NECROSIS)

FILE NONAME (CREATION DATE = 11/07/74)

SCATTERGRAM OF (DOWN) VAR007 (ACROSS) VAR017



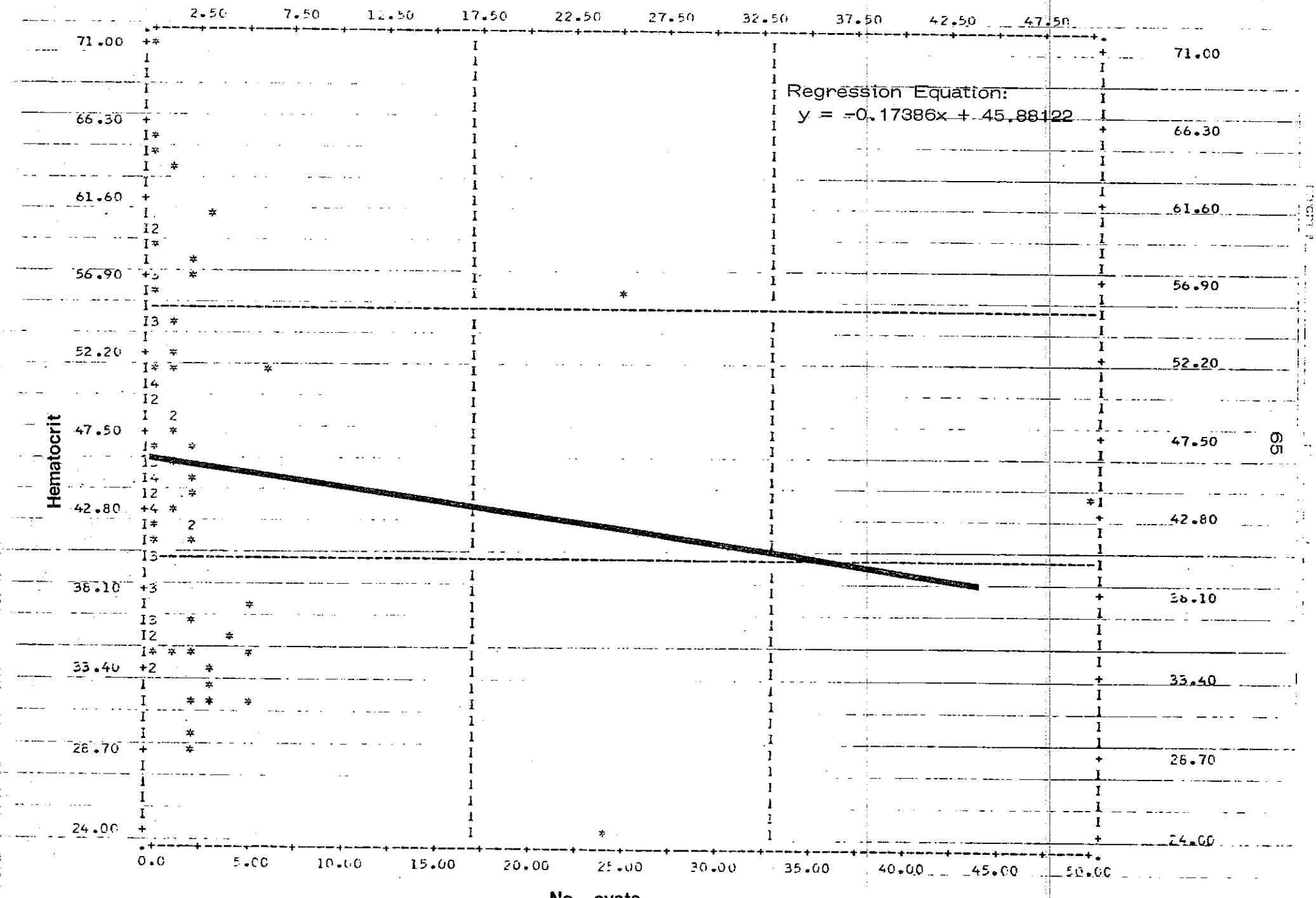
GRAPH 20

TRIONE

11/07/74 PAGE 10

RELATIONSHIP BETWEEN HEMATOCRIT VALUE AND NUMBER OF CYSTS (ENCAPSULATIONS)

FILE NONAME (CREATION DATE = 11/07/74)
SCATTERGRAM OF (DOWN) VAR008 (ACROSS) VAR012



65

GRAPH 29

TRIONE

RELATIONSHIP BETWEEN HEMATOCRIT VALUE AND NUMBER OF UNENCAPSULATED PLEROCERCOIDS

11/07/74 PAGE 12

FILE NONAME (CREATION DATE = 11/07/74)
SCATTERGRAM OF (DOWN) VAR00P (ACROSS) VAR015

Regression Equation:
y = 0.01666x + 45.59890

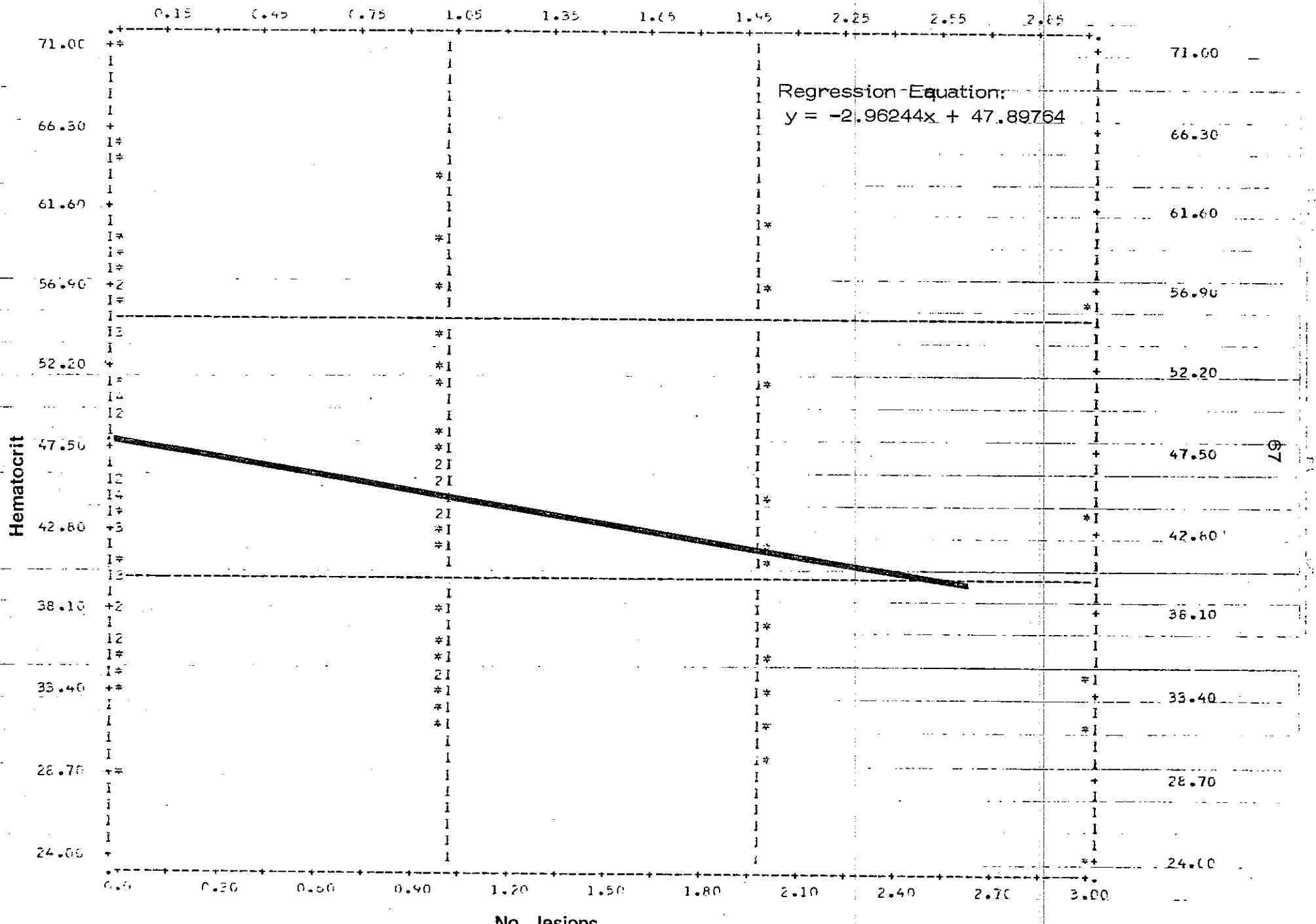
Hematocrit

No. plerocercoids

TRIONE 11/07/74 PAGE 14

RELATIONSHIP BETWEEN HEMATOCRIT VALUE AND NUMBER OF LESIONS (NECROSIS)

FILE NONAME (CREATION DATE = 11/07/74)
SCATTERGRAM OF (DOWN) VAR008 (ACROSS) VAR017

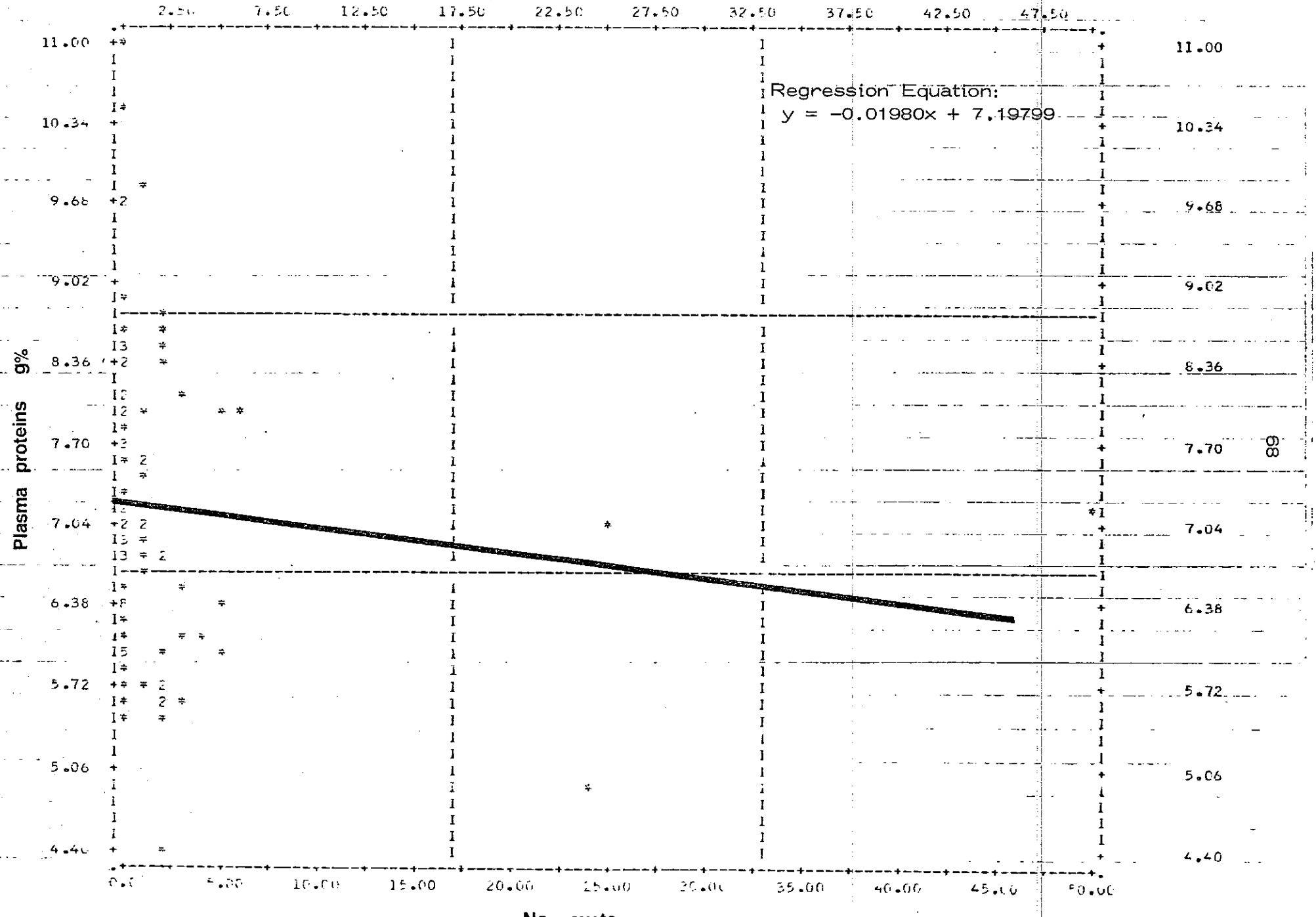


TF1ONE 11/07/74 PAGE 16

RELATIONSHIP BETWEEN PLASMA PROTEIN LEVEL AND NUMBER OF CYSTS (ENCAPSULATIONS)

FILE NOVAME (CREATION DATE = 11/07/74)

SCATTERGRAM OF (DOWN) VAR009 (ACROSS) VAR013

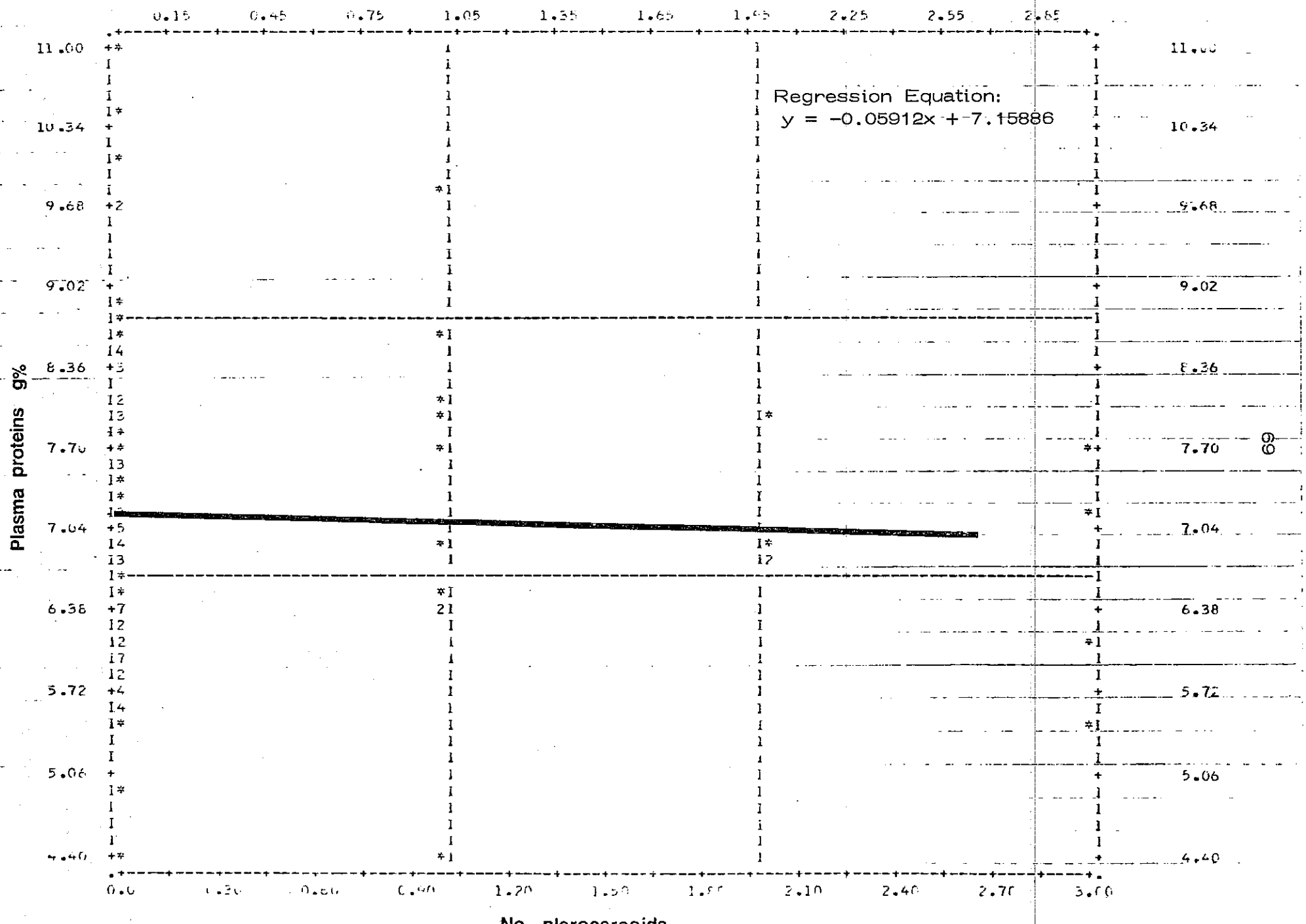


GRAPH 32

TRICKE 11/07/74 PAGE 18

RELATIONSHIP BETWEEN PLASMA PROTEIN LEVEL AND NUMBER OF UNENCAPSULATED PLEROCERCOIDS

FILE NONAME (CREATION DATE = 11/07/74)
SCATTERGRAM OF (DOWN) VAR009 (ACROSS) VAR015



TRIONE

GRAPH 33

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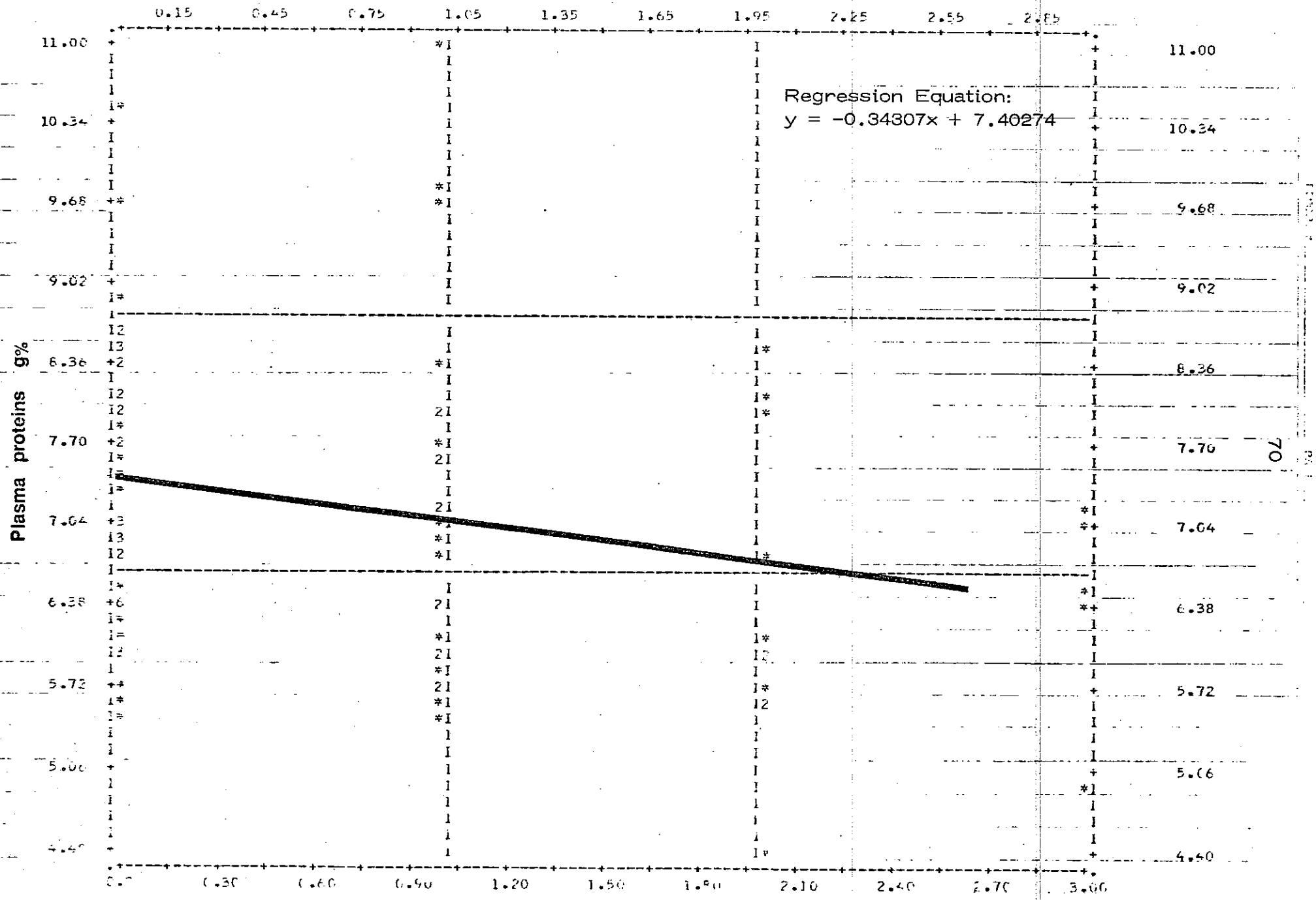
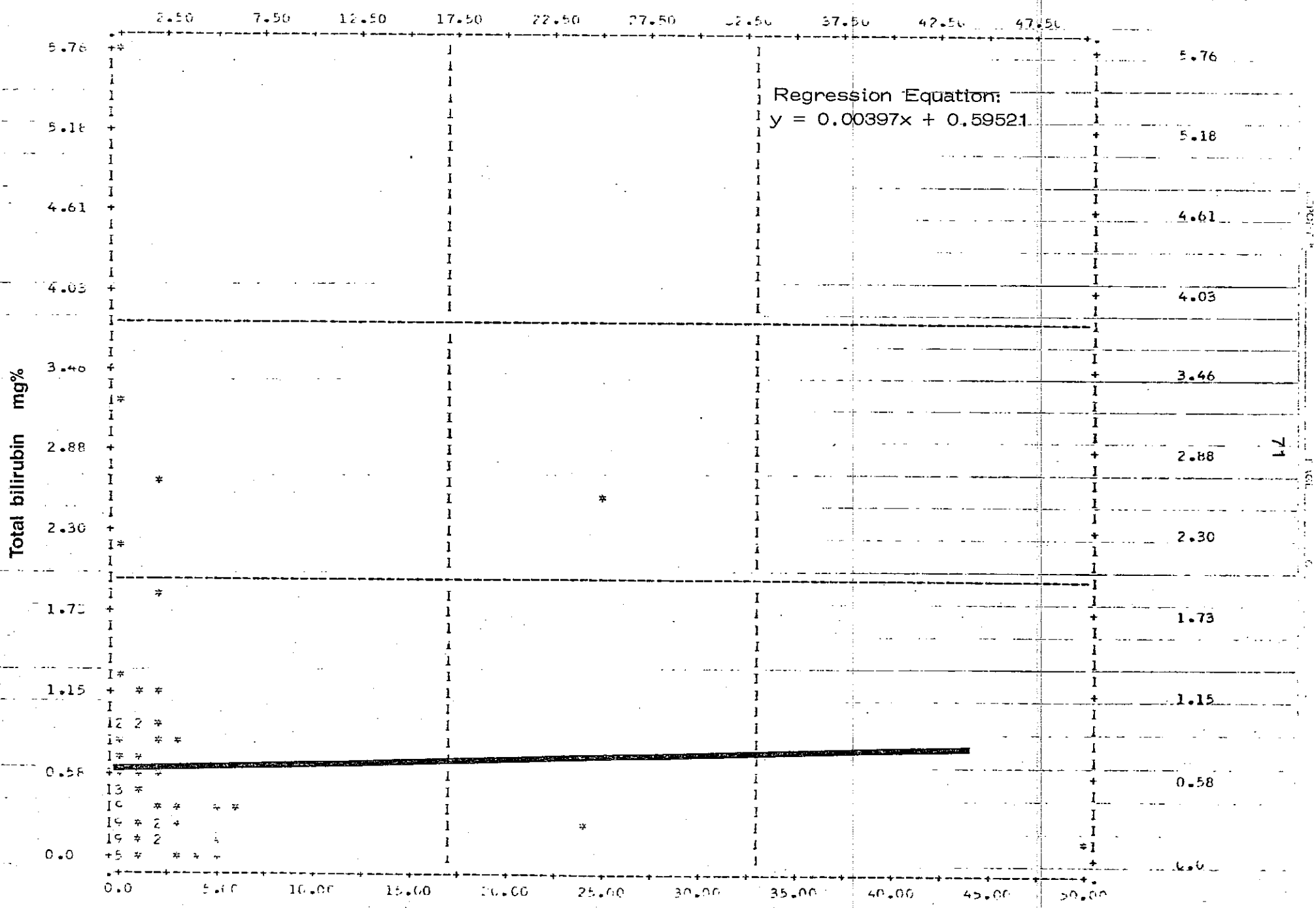
RELATIONSHIP BETWEEN PLASMA PROTEIN LEVEL AND NUMBER OF LESIONS (NECROSIS)

FILE NONAME (CREATION DATE = 11/07/74)
SCATTERGRAM OF (DOWN) VAR009 (ACROSS) VAR017



RELATIONSHIP BETWEEN TOTAL BILIRUBIN LEVEL AND NUMBER OF CYSTS (ENCAPSULATIONS)

FILE NONAME (CREATION DATE = 11/07/74)
SCATTERGRAM OF (DOWN) VAR010 (ACROSS) VAR015



GRAPH 35

TRIONE 11/07/74 PAGE 24

FILE NONAME (CREATION DATE = 11/07/74)

RELATIONSHIP BETWEEN TOTAL BILIRUBIN LEVEL AND NUMBER OF UNENCAPSULATED PLEROCERCOIDS

SCATTERGRAM OF (DOWN) VAR016 (ACROSS) VAR015

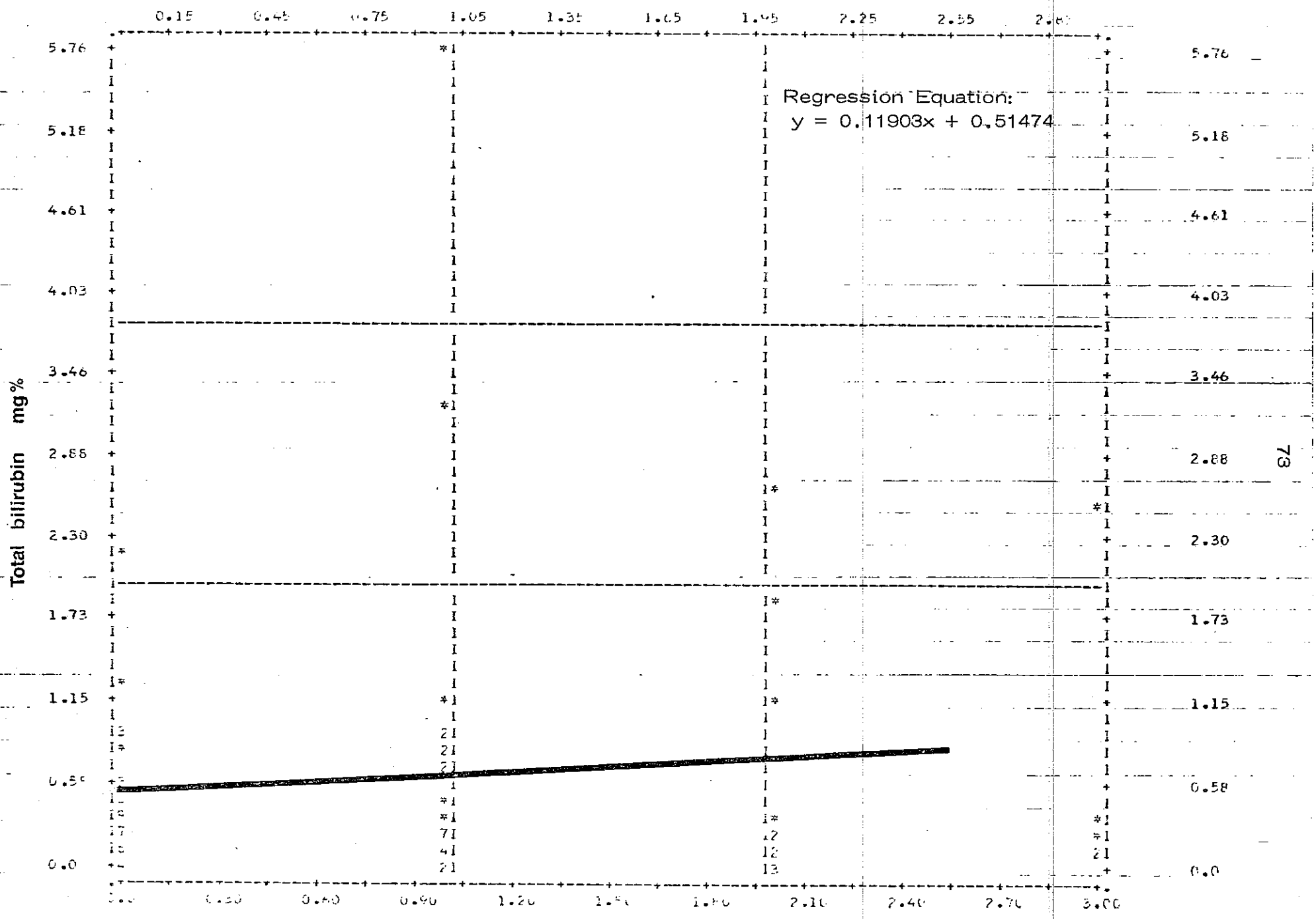
Regression Equation:
y = 0.09306x + 0.57378

Total bilirubin mg%

No. pierocercoids

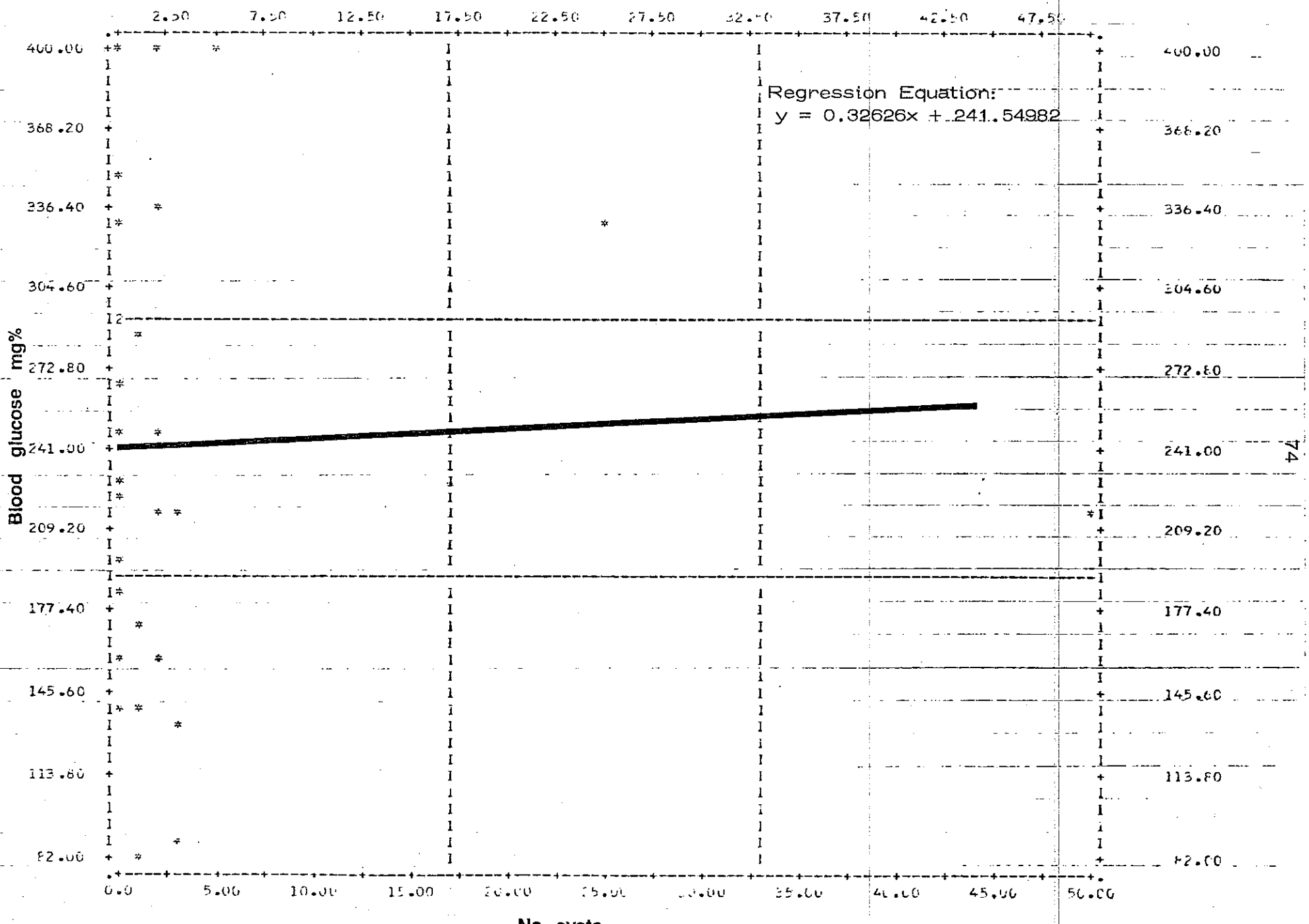
RELATIONSHIP BETWEEN TOTAL BILIRUBIN LEVEL AND NUMBER OF LESIONS (NECROSIS)

FILE NONAME (CREATION DATE = 11/07/74)
SCATTERGRAM OF (DOWN) VAR010 (ACROSS) VAR017



RELATIONSHIP BETWEEN BLOOD GLUCOSE LEVEL AND NUMBER OF CYSTS (ENCAPSULATIONS)

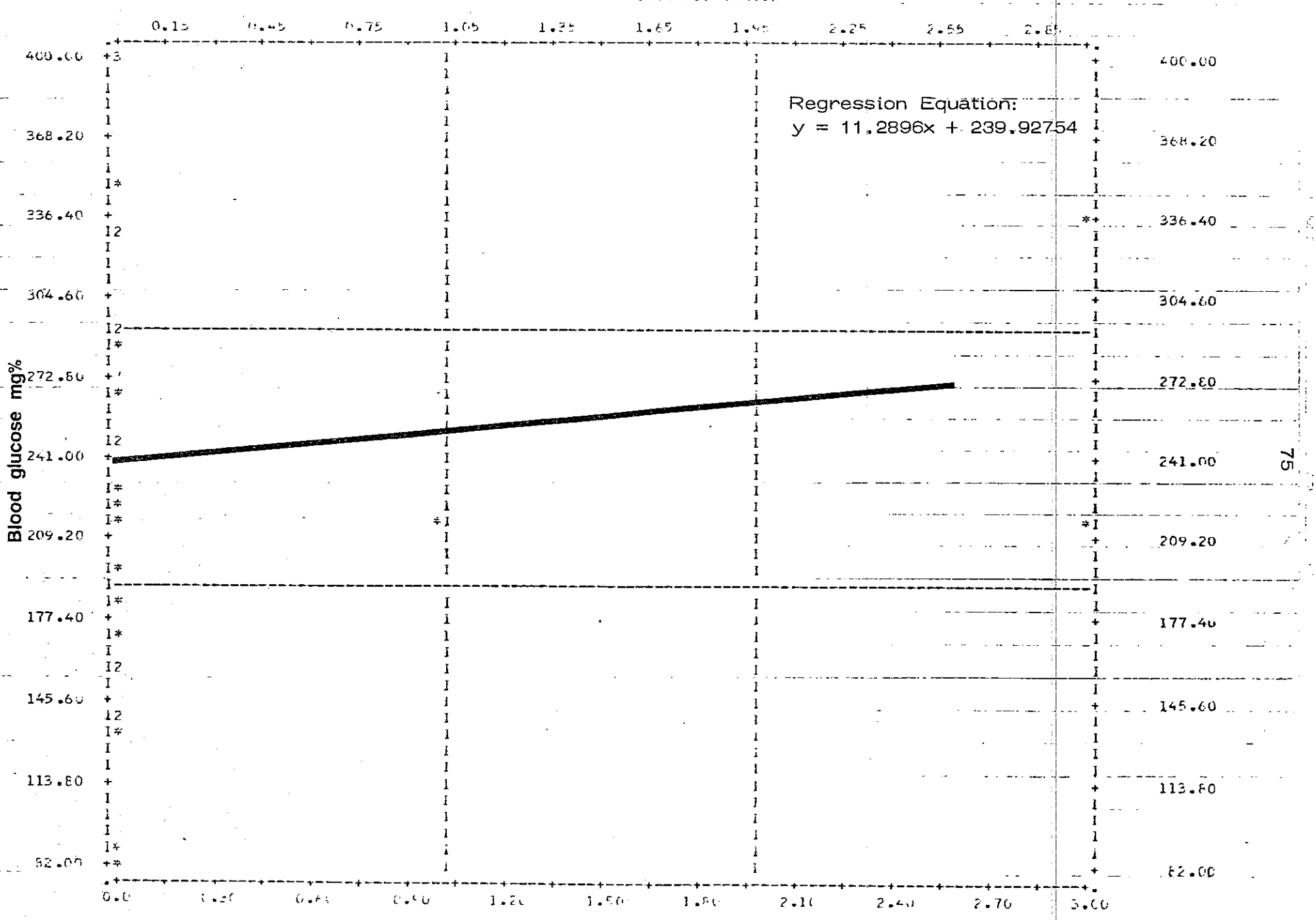
FILE NONAME (CREATION DATE = 11/07/74)
SCATTERGRAM OF (DOWN) VAR011 (ACROSS) VAR013



GRAPH 38

RELATIONSHIP BETWEEN BLOOD GLUCOSE LEVEL AND NUMBER OF UNENCAPSULATED PLEROCERCOIDS

FILE NONAME (CREATION DATE = 11/07/74)
SCATTERGRAM OF (DOWN) VAR011 (ACROSS) VAR015



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GRAPH 39

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RELATIONSHIP BETWEEN BLOOD GLUCOSE LEVEL AND NUMBER OF LESIONS (NECROSIS)

FILE NONAME (CREATION DATE = 11/07/74)

SCATTERGRAM OF (DOWN) VAR011 (ACROSS) VAR017

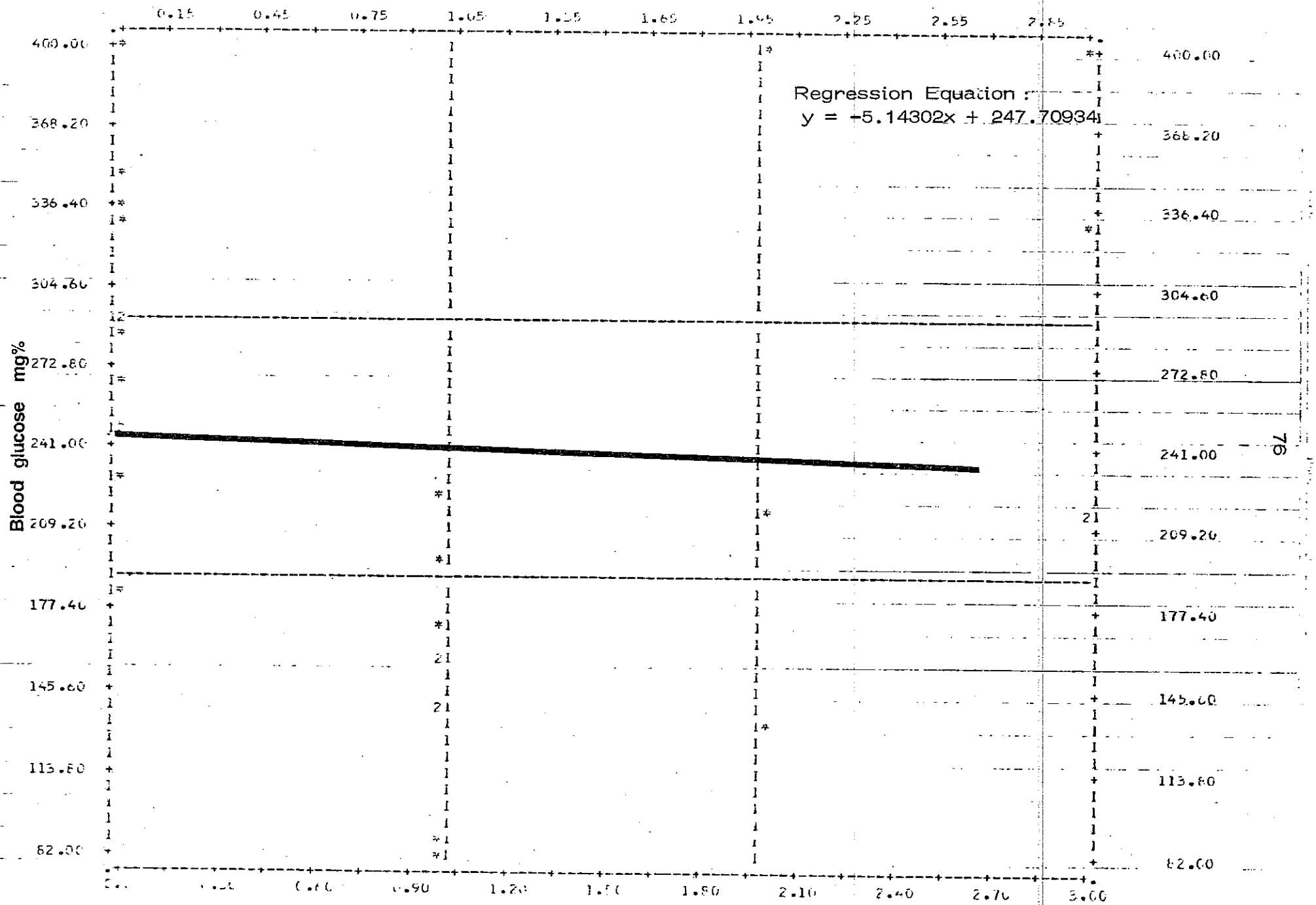

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TABLE 23

T-TEST FOR BLOOD CHEMISTRY DIFFERENTIAL ASSOCIATED WITH TRIAENOPHORIASIS IN WHITE BASS: ENCAPSULATIONS (CYSTS) IN LIVER PRESENT/ABSENT

	Hemoglobin	Hematocrit	Plasma Protein	Total Bilirubin	Blood Glucose
Pooled Variance Estimate-					
t value	-0.18	1.77	1.32	-0.46	0.75
Degrees of Freedom	89	87	89	80	26
2-tail Probability	0.858	0.081	0.192	0.650	0.458
Separate Variance Estimate-					
t value	-0.18	1.75	1.31	-0.49	0.77
Degrees of Freedom	83.31	77.96	83.44	78.62	25.40
2-tail Probability	0.858	0.084	0.192	0.627	0.449
F value	1.03	1.20	1.02	1.89	1.84
2-tail Probability	0.911	0.546	0.928	0.061	0.294

TABLE 24

T-TEST FOR BLOOD CHEMISTRY DIFFERENTIAL ASSOCIATED WITH TRIAENOPHORIASIS IN WHITE BASS: UNENCAPSULATED PLEROCERCOIDS PRESENT/ABSENT IN LIVER

	Hemoglobin	Hematocrit	Plasma Protein	Total Bilirubin	Blood Glucose
Pooled Variance Estimate-					
t value	-1.29	-0.73	-0.07	-0.51	-0.33
Degrees of Freedom	95	93	95	79	26
2-tail Probability	0.199	0.464	0.941	0.614	0.746
Separate Variance Estimate-					
t value	-1.25	-0.66	-0.08	-0.47	-0.41
Degrees of Freedom	32.56	30.22	39.34	17.70	2.91
2-tail Probability	0.221	0.512	0.936	0.642	0.710
F Value	1.14	1.45	1.36	1.23	1.74
2-tail Probability	0.653	0.246	0.431	0.559	0.848

TABLE 25

T-TEST FOR BLOOD CHEMISTRY DIFFERENTIAL ASSOCIATED WITH TRIAENOPHORIASIS IN WHITE BASS: OLD LESIONS (NECROSIS) IN LIVER PRESENT/ABSENT

	Hemoglobin	Hematocrit	Plasma Protein	Total Bilirubin	Blood Glucose
Pooled Variance Estimate-					
t value	0.43	1.91	0.99	-1.54	2.69
Degrees of Freedom	95	93	95	79	26
2-tail Probability	0.667	0.059	0.324	0.124	0.012
Separate Variance Estimate-					
t value	0.43	1.90	1.00	-1.54	2.88
Degrees of Freedom	89.46	86.20	94.27	48.92	25.13
2-tail Probability	0.669	0.061	0.319	0.130	0.008
F value	1.13	1.10	1.22	7.56	2.69
2-tail Probability	0.669	0.748	0.502	0.000	0.103

TABLE 26

MEANS, STANDARD DEVIATIONS, AND STANDARD ERRORS OF HEMATOCRIT VALUES IN WHITE BASS:
BREAKDOWN BY ENCAPSULATIONS (CYSTS) IN LIVER PRESENT/ABSENT

	Encapsulations Absent	Encapsulations Present	Total Population
Mean	47.2000	43.6667	45.323
Std. Deviation	8.974	9.823	9.323
Std. Error	1.269	1.573	0.952

TABLE 27

MEANS, STANDARD DEVIATIONS, AND STANDARD ERRORS OF HEMATOCRIT VALUES IN WHITE BASS:
BREAKDOWN BY OLD LESIONS (NECROSIS) IN LIVER PRESENT/ABSENT

	Necrosis Absent	Necrosis Present	Total Population
Mean	47.1905	43.5849	45.323
Std. Deviation	9.368	8.950	9.323
Std. Error	1.446	1.229	0.952

TABLE 28

MEANS, STANDARD DEVIATIONS, AND STANDARD ERRORS OF BLOOD GLUCOSE LEVELS IN WHITE BASS:
BREAKDOWN BY OLD LESIONS (NECROSIS) IN LIVER PRESENT/ABSENT

	Necrosis Absent	Necrosis Present	Total Population
Mean	291.1665	206.4375	242.750
Std. Deviation	58.623	96.178	91.446
Std. Error	16.923	24.045	17.282

TABLE 29

CORRELATIONS AMONG FISH SIZE, LIVER NECROSIS AND HEMATOCRIT VALUES IN WHITE BASS

	Correlation (R)	Significance	Partial Correlation
Size x Necrosis	0.80030	0.00001	0.78306
Size x Hematocrit	-0.28854	0.00218	-0.11524
Necrosis x Hematocrit	-0.27744	0.00608	-0.08098

TABLE 30

CORRELATIONS AMONG FISH SIZE, LIVER NECROSIS, AND PLASMA PROTEIN LEVELS IN WHITE BASS

	Correlation (R)	Significance	Partial Correlation
Size x Necrosis	0.80030	0.00001	0.78306
Size x Plasma Protein	-0.13414	0.09395	0.10977
Necrosis x Plasma Protein	-0.24736	0.01208	-0.23565

Life Cycle Investigations

The adult stage of Triaenophorus nodulosus was never encountered during the course of this study. Therefore, it was not possible to conduct infection experiments of the first intermediate host, Cyclops bicuspidatus, with the eggs of T. nodulosus.

Plerocercoids removed from hepatic cysts of white bass were introduced via plastic tubing into the stomachs of a walleye, a smallmouth bass, and a pumpkinseed. Fish were autopsied six days postinfection and examined for triaenophoriasis. One plerocercoid was recovered from the liver of the pumpkinseed; the walleye and smallmouth bass were triaenophoriasis negative. T. nodulosus was found to occur naturally in pumpkinseed at a low level of incidence; therefore, it cannot be concluded from this work that successful re-establishment of the plerocercoid stage was demonstrated.

Triaenophoriasis in Other Fish Species

The plerocercoid stage of *Triaenophorus nodulosus* has been recorded from several species of fish in Lake Erie. Bangham and Hunter (1939) found the parasite in *Percopsis omiscomaycus*, *Perca flavescens*, and *Micropterus dolomieui*. Bangham (1972) resurveyed the fish parasites of Lake Erie in 1957 and found the plerocercoid of *T. nodulosus* in the following species: *P. flavescens*, *Stizostedion vitreum*, *S. canadense*, *Pomoxis nigromaculatus*, *Ambloplites rupestris*, *Micropterus salmoides*, and *Morone chrysops*. Dechtiar (1972) was the first to report the adult stage of *T. nodulosus* in Lake Erie, from the northern pike (*Esox lucius*). He also reported plerocercoids from the following fish: *Moxostoma anisurum*, *M. erythrurum*, *Carassius auratus*, *Notropis cornutus*, and *Morone chrysops*.

In an investigation of triaenophoriasis in fish species other than white bass, a survey was conducted of yellow perch (*Perca flavescens*) and six species of the family Centrarchidae. Perch data were collected by Dr. C. Lawrence Cooper and Mr. Robert R. Ashmead. Summary incidence and intensity data are presented in Table 31.

TABLE 31

INCIDENCE AND INTENSITY OF *Triaenophorus nodulosus* IN FISH EXAMINED AT SONE LABORATORY, SUMMER, 1974

Species	N	Percent Incidence	Average Intensity
White Crappie (*Pomoxis annularis*)	5	0	
Black Crappie (*Pomoxis nigromaculatus*)	19	0	
Rockbass (*Ambloplites rupestris*)	18	0	
Smallmouth Bass (*Micropterus dolomieui*)	6	0	
Bluegill Sunfish (*Lepomis macrochirus*)	2	0	
Pumpkinseed Sunfish (*Lepomis gibbosus*)	18	1	1.00
Yellow Perch (*Perca flavescens*)	735	12	1.56

Gross Pathology Associated with Triaenophoriasis

Triaenophorus nodulosus is the cause of very striking pathology in the liver of white bass. The damage is due to plerocercoids migrating through the liver where they are acitve for a time, ultimately either successfully encapsulating or being killed by the host response.

The external surface of the diseased liver is characterized by whitish, pea-sized encapsulations, and by dark, reddish-brown streaks which are the remnants of unencapsulated plerocercoids which failed to survive the host response and have become sclerotized. In heavier infections, large areas of hemorrhage and necrotic tissue are evident. Active, unencapsulated plerocercoids are often present.

Upon dissection of the liver, active plerocercoids were frequently found in the sinusoids and venous circulation. It was also demonstrated that the dark streaks, caused by dead plerocercoids, are found throughout the liver substance. Encapsulations occurred on, or just beneath, the liver surface.

Microscopic Pathology Associated with Triaenophoriasis

Observations on microscopic histopathology made during the course of this study have been published (Stromberg and Crites, 1974).

A distinct cellular response was observed around some plerocercoids, but not all; these cells were primarily lumphocytes and macrophages. Frequently areas of fibrotic and necrotic liver tissue were observed surrounding the plerocercoid. Formation of new tissue, in this case due to fibroblasts, is often indicative of the presence of some irritant, suggesting the activity of a histolytic secretion to provide nutrients for the parasite. Such a secretion associated with the T. nodulosus plerocercoid has been previously suggested by Scheuring (1922).

Some of the worms grow to a large size and encapsulate. These are the whitish cysts seen in gross examination, and are the infective plerocercoids which will mature if the white bass is eaten by a northern pike. When a number or these encapsulations form, pressure is exerted on the surrounding tissue, causing compression atrophy and avascular necrosis by constricting the blood passageways to the adjacent liver cells.

Not all of the plerocercoids are able to survive the host response. Some are killed and become sclerotized. A chemical alteration occurs in the cuticles of these worms, which gives a bright red reaction with Mallory's triple stain. The worm tissue within the

cuticle appears necrotic. Associated with these dead plerocercoids is a squamous metaplasia, and sometimes an extensive fibrosis. It is these dead parasites, and the dense tissue surrounding them, which produce the dark tracks running through the liver tissue. The result is a destruction and displacement of functional liver parenchyma which may be more extensive than is apparent in gross examination.

Petichial hemorrhages, caused by damage to capillaries, is also associated with triaenophoriasis in the liver. Also, areas of dead blood cells, plssible a result of portal hypertension, have been observed.

ANALYSIS

It seems probable, on the basis of our work, that the Bass Island region is not a significant focus of infection for Triaenophorus nodulosus in white bass, as no infected definitive hosts and only one infected young-of-the-year white bass was found. The presence of the disease in pumpkinseed demonstrates that it does occur in our study area, although at a low level. White bass are a migratory species, and it is possible that triaenophoriasis is established in the white bass population at other localities in Lake Erie. The disease is an accumulation of encapsulations and liver necrosis, the plerocercoids being continually acquired.

Descriptions of gross and microscopic histopathology were made. Study of blood chemistry revealed a negative correlation between plasma protein level and liver necrosis due to triaenophoriasis. Hematocrit value was found to be negatively contingent upon fish size.

White bass and yellow perch were shown to be the major second intermediate hosts of T. nodulosus in the study area. Incidental infections have been encountered in other fish species.

No clear-cut impact of T. nodulosus on white bass population dynamics was demonstrated. Life cycle studies were limited by the unavailability of adult T. nodulosus and its eggs.

RECOMMENDATIONS

Our work points to a lowering of the plasma protein level in the blood of white bass associated with the liver necrosis due to triaenophoriasis. We believe that more specific testing of this particular factor, considering especially albumin and serum glogulins, would further elucidate the effects of Triaenophorus nodulosus on white bass. Antigen studies would provide useful knowledge concerning the host response.

White bass appear to continually become infected with T. nodulosus, beyond the age at which the copepods, which serve as the first intermediate hosts for this parasite, would be replaced in the diet by forage fishes. This observation suggests a secondary transfer of plerocercoids from forage fish to white bass. Artificial infection experiments of uninfected young-of-the-year white bass would be necessary to bear this out.

Control of triaenophoriasis has been attempted in Heming Lake, Manitoba, by eradication of the northern pike, the definitive host (Lawler, 1961). This program met with some success, but would not be practical in a body of water as large as Lake Erie.

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