THE UNIVERSITY OF MANITOBA

FACTORS AFFECTING COMMUNITY STRUCTURE, TRANSMISSION, AND REGULATION OF FISH-PARASITES IN DAUPHIN LAKE, MANITOBA.

BY

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ABSTRACT: Approximately 20,000 fish representing 23 species were collected from Dauphin Lake, Manitoba between May 1985 and December 1987. All of these were inspected for ectoparasites, and a subsample of fish (~15% of the total sample) were more closely examined. In total, 97,028 parasites comprising 51 species were found. The type and intensity of fish-parasites is influenced primarily by density-independent mechanisms such as temporal variation and host influences such as diet and sex. Each parasite metacommunity was characterized by one or two dominant species; whereas the component community was composed of a few important species, many species with intermediate importance, and many rare species. It appears that almost all transmission of ectoparasites to the fish hosts occurs in the littoral zone. Two host-parasite systems were examined specifically for evidence of regulation of parasite populations.

Neoechinorhynchus carpiodi infects quillback, Carpiodes cyprinus, and elicits the formation of intestinal nodules at the sites of attachment. The degree of pathology is density-dependent and affects the carrying capacity of the quillback gut. Infected quillback showed increased leakiness of the intestine at the sites of attachment by N. carpiodi, and their sera contained antibodies directed against N. carpiodi. The degree of intestinal leakage and the number of quillback producing antibodies increased with increasing

number of worms. The evidence suggests that host immune responses might be acting to regulate the N. carpiodi population.

Maturity, mass, and sex were determined for individual Raphidascaris acus from northern pike, Esox lucius. The number of R. acus in pike fluctuates seasonally due to changing patterns of predation, especially predation of yellow perch, Perca fluviatilus. Sub-lethal effects of parasite-density on worm size were translated into effects on fecundity of R. acus. However, the strength of the correlation between fecundity and mass for R. acus was influenced by host effects and differed between sample periods. Much of the variation in fecundity and mass could be attributed to continued growth of gravid worms after maturation, and inequalities in mass and fecundity varied seasonally. In northern pike, stochastic factors provide the dominant force effecting changes in numbers, growth, and fecundity of R. acus.

In yellow perch, most R. acus larvae are found in the liver as free (unencapsulated) and encapsulated worms, and the final stages of worm destruction (nodules). The number of free larvae, encapsulated larvae, and recently-formed nodules in the liver varied seasonally and between the sexes but was not correlated to water temperature or habitat of yellow perch. Mean intensity, but not recruitment, increased with age. Density of R. acus was highest in young perch and

decreased with age; the greatest decrease being concomitant with host maturity. Heavily infected perch were in poorer condition and exhibited reduced growth. Reduction in growth was most pronounced with high densities of unencapsulated larvae. These shifts in growth curves were interpreted as increased mortality of young, heavily infected yellow perch concomitant with maturation and delayed maturity for infected females.

Finally, I examined by simulation the consequences for parasites of skewed fecundity distributions, maintenance of this trait, and selection. Perhaps a high level of genetic variation can be maintained in populations of R. acus by *i*) quasi-fixation and quasi-loss of alleles, *ii*) increased numbers of recombinant progeny, *iii*) differences in selection pressures among infrapopulations, *iv*) mixing of eggs and larvae prior to each adult generation, and *v*) differences in selection between intermediate and definitive hosts. I suggest that maintenance of maximum genetic variation is more adaptive for unregulated parasites since it might serve to dampen population changes resulting from erratic fluctuations in environmental conditions. Furthermore, increased variation might decrease the likelihood of extinctions by ensuring that some larvae mature each generation.

ACKNOWLEDGEMENTS

The 'Acknowledgements' is one of the most important sections of any thesis, yet it receives the least amount of scrutiny by the editors' eyes. Consequently, the author's true writing abilities are often exposed. Recognizing this, I accept full responsibility for any 'Hungarianisms' that may appear in the text that follows, and hope they do not lead to any misinterpretations of my true intentions.

First and foremost I thank my supervisor, Dr. T. A. Dick. His unique blend of positive and negative reinforcement instilled in me what every scientist should have; the willingness to work a little harder. I can only hope that I have also gained some of his appetite for 'quality control' and his dogged determination to leave no problems unresolved. I know that in the future, Terry will continue to monitor my progress as a scientist. I'll do my best not to disappoint him.

I thank the members of my 'official' Advisory committee (Drs. T. A. Dick, A. N. Arnason, J. F. Craig, and A. O. Bush) for their time and efforts on my behalf, and for their many useful suggestions regarding the collection and analysis of data. I also thank my 'unofficial' advisors (Drs. L. C. Graham, G. F. Crow, and A. W. Shostak) for their many contributions, and my 'personal' advisor (T. deVos) for sharing many 'RH' and 'W' units with me.

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GENERAL INTRODUCTION

The theme of this thesis is the regulation of fish parasite populations. 'Regulation' refers to the ability of a population to attain stability through the operation of density-dependent mechanisms (Kennedy, 1977), and a population has stability if it can reach and maintain an equilibrium level (May, 1973; Anderson, 1974). It is generally accepted that density-dependent (deterministic) mechanisms (eg. intraspecific competition, host immune responses, and parasite-induced host mortality) operating at the population level can impart stability on a system, and that density-independent (stochastic) mechanisms (eg. temporal, spatial, and environmental heterogeneity) can have destabilizing influences that affect entire communities. Parasites are influenced to varying degrees by both types of mechanisms, so parasite populations have traditionally been regarded as either regulated and stable over long periods of time (Kennedy, 1977), or unregulated and in constant danger of extinction (Price, 1980).

The debate regarding whether or not parasite populations are regulated is further complicated by the complex nature of natural parasite infections. The latter has led to the development of a hierarchical scheme for the description of structure in populations and communities of parasites. Briefly, a population of parasites in an individual host is an 'infrapopulation'; the population of a parasite from all individuals of a given host species in an ecosystem is a 'metapopulation'; and the population of a parasite within

all hosts in an ecosystem and in all stages of development is a 'suprapopulation'(Esch et al., 1975; Riggs et al., 1987). At the community level, the parasites of an individual host (an 'infracommunity') constitute a sample of parasites that can infect that host species and are present in the environment (the 'component community') (Bush and Holmes, 1986).

This complex structure has led to the development of two schools of thought regarding fish parasite interactions. Some authorities (Wisniewski, 1958; Chubb, 1970; Esch, 1971) believe that fish parasites should be considered as comprising complex communities that are best studied using an ecosystem approach; while others (Halvorsen 1971; Wooten, 1973) argue that since fish parasites can be completely understood by studying individual host-parasite systems, ecosystem approaches are unnecessary. These opposing perspectives can lead to very different interpretations of fish parasite interactions. In an effort to resolve some of these conflicts I studied the fish parasite assemblage in Dauphin Lake from a number of perspectives. By comparing the results obtained from analyses at various community and population levels, I assess some of the confounding effects that changes in the scale of investigation can have on interpretations of data on fish parasite communities. Other objectives are *i*) to determine if fish parasite populations in Dauphin Lake are regulated, *ii*) to identify any existing mechanisms that could operate to regulate parasite numbers

in Dauphin Lake fishes, and *iii*) to evaluate the influence of stochastic factors on the long term stability of (apparently) unregulated parasite populations.

The organization of the thesis is as follows. First, the structure of the fish parasite assemblage in Dauphin Lake is characterized: Chapter 1 deals with similarities and differences in component community and infracommunity organization, and factors affecting infracommunity size; and in Chapter 2 ectoparasites, an integral part of any fish parasite community that has largely been ignored, are considered specifically. Because some parasites are highly host specific while others are capable of infecting numerous hosts (Holmes and Price, 1980), it is important to compare these types of parasites. I consider the roles played by parasite-induced pathology (Chapter 3-1) and host-immunity (Chapter 3-2) in the regulation of Neoechinorhynchus carpiodi, a host specific acanthocephalan found in quillback, Carpiodes cyprinus. The influence of deterministic and stochastic factors on populations of Raphidascaris acus, a nematode that infects many fish species but in Dauphin Lake uses mostly yellow perch (Perca flavescens) as an intermediate host and matures almost exclusively in northern pike (Esox lucius), is then evaluated. I propose that R. acus is responsible for parasite-induced mortality of yellow perch (Chapter 4-1), and that stochastic events are important in determining the structure of populations of R. acus in pike (Chapter 4-2).

Finally, I evaluate by computer simulation some of the roles played by stochastic factors in the maintenance of long term stability in unregulated populations of parasites (Chapter 4-3).

Dauphin Lake was selected as the study site because a large-scale biological survey initiated by the Canadian Department of Fisheries and Oceans (DFO)[1], provided an opportunity to obtain information on the relative numbers of fish hosts and their intensity and prevalence of infection by parasites. All three sources of information were considered in only one previous survey (Leong and Holmes, 1981), but Leong and Holmes lacked a proper assessment of the populations of fishes. Furthermore, by comparison with available historical information on the parasite fauna of Dauphin Lake (Stewart-Hay, 1951) some long-term changes in the fish parasite community could be documented.

[1] The DFO survey was designed to establish baseline information on fish populations and was part of a joint effort between the DFO and the Manitoba Department of Natural Resources to rehabilitate the Dauphin Lake fishery.

CHAPTER 1: THE FISH PARASITE COMMUNITY OF DAUPHIN LAKE.

INTRODUCTION

The tradition of teaching and research in Parasitology at the University of Manitoba ensured ample records of parasites of the more common fishes of Manitoba. However, there is little detailed information on the complexities and interactions of entire parasite-fish communities, here and elsewhere. The review by Lubinsky and Loch (1979) is the most comprehensive account of icthyoparasites for Manitoba (161 species of parasites from 50 species of fish), but provides no information on parasite community organization. Ichthyoparasites of medical or economical importance (eg. Diphyllobothrium latum and Triaenophorus crassus) have received more attention but there is still a paucity of information related to their interactions within the community.

Some suggest that since fishes harbour numerous species of parasites simultaneously and as parasites depend on interactions within ecosystems, the entire parasite community of a fish-host must be considered to properly interpret host-parasite relationships (Wisniewski, 1958; Noble et al., 1963; Chubb, 1970; Esch, 1971; Cloutman, 1975). Others believe that the relationships between fishes and their parasites are more important than remote interactions, and that traditional analyses based on surveys of infracommunities is adequate (Halvorsen, 1971; Wooten, 1973). Since any interpretation of fish parasite

interactions is dependent, to some extent, on which of these perspectives is taken, one of the major problems in parasite ecology is the choice of the appropriate scale for investigation. This choice is usually made *a priori* without any knowledge regarding large-scale or small-scale structure of the parasite community. Furthermore, although the relationship between types of parasites and community structure is of fundamental importance, this has received little attention as most field studies are of limited scope. Degrees of host-specificity and differences in the number of hosts and trophic levels utilized by parasites, and species-related differences or temporal changes in host behaviour, can effect changes in the parasite community.

This chapter presents the results of a survey of the ichthyoparasites of Dauphin Lake fishes. The occurrence of many new species of parasites and marked changes in the levels of some parasites since the biological investigation of Dauphin Lake by Stewart-Hay (1951), indicates the dynamic nature of the ichthyoparasite fauna. Changes in the aquatic environment and the bird and fish hosts have allowed these changes, so environmental and host influences which might effect changes in parasite numbers and community structure are analyzed. Patterns of association between fishes and their parasites revealed from analyses at the infra-, meta-, and component community levels are compared to determine what effect changing perspectives has on the interpretation of the results.

MATERIALS AND METHODS

Study site

Dauphin Lake (51°15'23" N, 99°46'12'' W; elevation 260 m) is approximately 42 km long and 20 km across (Fig. 1) with a mean depth of 2.1 m, a maximum depth of 3.5 m, and a total volume of 1.645×10^9 m^3. The lake has only a single island of negligible size (1 hectare) and a surface area of 700 km^2. A number of small rivers and streams empty into the southwest portion of the lake (Fig. 1), draining an area of 8,700 km^2. The Mossy River (Fig. 1) has been regulated since 1933 and is the lakes only outlet, flowing into Lake Winnipegosis to the east. The lake can be separated into two distinct habitat zones: the shallow southern basin which receives most of the inflowing water, has a sandy to muddy shoreline characterized by dense vegetation (mostly Phragmites spp., Scirpus spp., and Carex spp.) and a narrow littoral zone; the deeper northern basin has steep, rocky shorelines with sparse vegetation. Extreme fluctuations in water depth occur annually and seasonally (Fig.2), and wind-driven seiches can raise the water levels as much as 50 cm in a few days.

Figure 1. Map of Dauphin Lake, Manitoba showing: approximate locations of pound net sites (✱); areas sampled using beach seines and fyke nets (◯); and the boundaries of 38 sampling quadrats (horizontal and vertical lines) where gill nets were set.

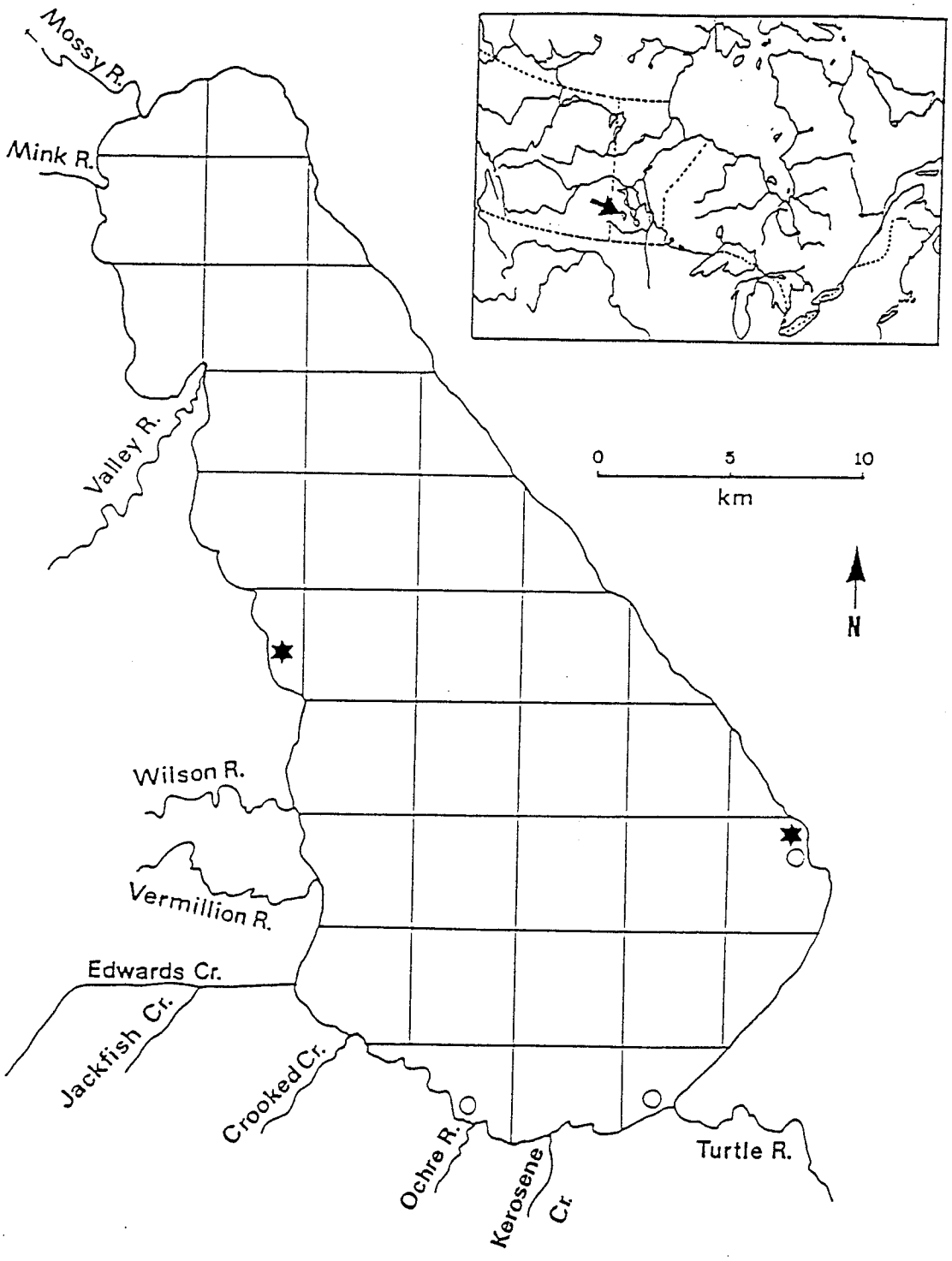
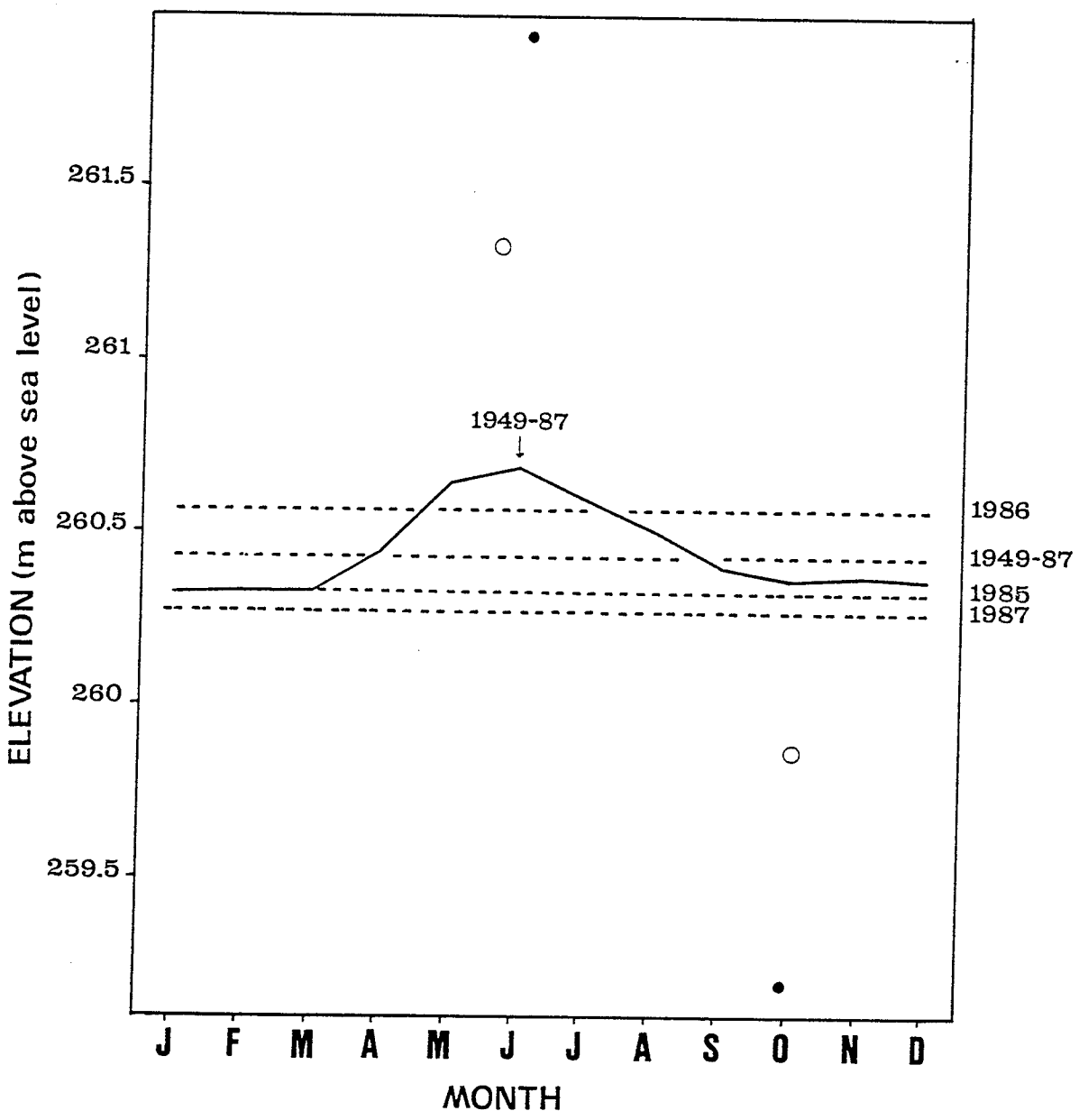

Mossy R.
Mink R.
Valley R.
Wilson R.
Vermillion R.
Edwards Cr.
Jackfish Cr.
Crooked Cr.
Ochre R.
Kerosene Cr.
Turtle R.
0
5
10
km
N

Figure 2. Annual (- - -) and monthly (———) mean water levels (m above sea level) at the Mossy River outlet on Dauphin Lake, Manitoba[2]. Daily extremes for 1985-1987 (○) and 1949-1987 (●) are indicated.

[2] Based on the Historical Water Levels Summary: Manitoba. Inland Waters Directorate, Water Resources Branch; Water Survey of Canada; Ottawa, Canada. 1988.


261.5
261
260.5
260
259.5
ELEVATION (m above sea level)
1949-87
1986
1949-87
1985
1987
J F M A M J J A S O N D
MONTH

Average daily temperature ranges from -20°C in January to 17.5°C in July; freeze-over occurs on about 15 November, and ice-off on about 7 May[3].

Dauphin Lake has a diverse fish fauna; 23 species of fish were collected during the course of this study. Based on the survey by Stewart-Hay (1951) four of these (sauger, Stizostedion canadense; silver redhorse, Moxostoma anisurum; freshwater drum, Aplodinotus grunniens; and smallmouth bass, Micropterus dolomieui) were not present in Dauphin Lake in 1951. Very little angling takes place and most of this is confined to river mouths, and commercial harvests are limited to the winter season. The lake supports resident colonies of American white pelicans (Pelecanus erythrorhynchus), double-crested cormorants (Phalacrocorax auritus), and gulls and terns (Laridae); a few grebes (Podicipedidae) and great blue herons (Ardea herodias) nest in the area; and large numbers of ducks and geese (Anseriformes) are present during the spring and fall migrations. No fish eating mammals were observed during the period of study.

Sampling procedures

Fish were collected in 1985, 1986, and 1987 using gill nets, pound nets, fyke nets, and beach seines. These

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[3]statistics from the Hydrological Atlas of Canada, Fisheries and Environment Canada, 1987.

sampling gear are described fully in Everhart et al. (1976). A brief description of each sampling procedure follows.

From 1985 to 1987, 135 gill net samples were collected during the spring (26 May to 6 June), summer (21 July to 1 August), and fall (22 September to 2 October) using variable mesh size gill nets (15 nets per season, 45 per annum). Gill nets consisted of gangs of six adjacent 20 x 2 m panels of 38, 64, 89, 101, 108, and 127 mm stretched mesh, and each of these was set in one of 38 randomly chosen quadrats (4 x 4 km) on the lake (Fig. 1). Each year, 46 to 56% of the nets were set in quadrats that intersected the shoreline. Winter samples were collected annually from the commercial harvest (November to December); the latter used 108 mm stretched mesh gill nets.

In total 14,737 fish were collected with gill nets. All of these fish were inspected for external parasites, tumors, or lesions; the fork length (FL; mm) and round weight (W; g) were measured; ageing structures were collected; and the sex and maturity of each fish was recorded. A random subsample of fish (N=2,865) was more thoroughly examined for ectoparasites and a complete necropsy was performed on 1,864 of these to provide estimates of internal parasites.

Annually from 7 August to 12 September, fish were live-trapped using pound nets installed at two inshore stations on the lake (Fig. 1). A total of 3,869 fish were captured, examined, and their ectoparasites were removed. Three-thousand four-hundred and seventy-eight (90%) of these were

weighed, measured, marked with individually numbered jaw or spaghetti tags (Everhart et al., 1976), and released. The left pectoral fin of each tagged fish was clipped to identify any losses of tags.

Small (age-0) fish and minnows (N=2,153) were collected using fyke nets and seines (Fig. 1) in 1986 and 1987. All of these were examined for ectoparasites and a complete necropsy was performed on 123 of them. The remaining fish were inspected for plerocercoids of Ligula intestinalis but data on these are not included in this thesis.

Parasite enumeration and identification

For complete necropsies the eyes, gills, heart, swimbladder, and viscera of fish were removed, sealed in leak-proof plastic bags, and transported to the laboratory for immediate inspection or frozen (-20°C). The coelom of eviscerated fish was inspected and the fish filleted. The presence of intramuscular parasites was checked by slicing (transversely) through the coelomic side of the fillet to the skin and inspecting the exposed surfaces of the musculature. This process was repeated along the full length of both fillets with parallel slices ~1 cm apart. Thawed or fresh samples were examined in the laboratory using a Wild M3 dissecting microscope. Each organ was isolated, slit open longitudinally, and the contents removed by scraping the inner surface with a blunt probe. All organs were examined under tap water (frozen specimens) or physiological saline

(fresh specimens). The number and types of parasites found, their sites of infection, and the stomach contents were recorded for each fish.

Monogeneans, digeneans, cestodes, acanthocephalans, and leeches were fixed overnight in formalin-acetic acid-alcohol (FAA); copepods and nematodes were fixed in 70% ethanol; and cysts containing myxosporan trophozoites were punctured, and their contents smeared onto glass slides and air dried. Leeches and parasitic crustaceans collected from fish captured in pound nets were temporarily stored in lake water, relaxed at 4°C overnight, and narcotized in a dilute solution of chloroform in distilled water (three drops chloroform per 10 ml water). Parasitic crustaceans were killed by immersion in hot (35°C) ethanol (70%) and leeches were fixed in FAA. All specimens were stored in ethanol. Monogeneans, digeneans, cestodes, acanthocephalans, and leeches were stained with dilute (1%) Semichon's acetocarmine in water. All metazoans were dehydrated, cleared in xylene, and mounted in Permount. Myxosporan trophozoites were stained with Wright's stain (Humason, 1979), air dried, and mounted in Permount. Stomach contents were identified according to Ward and Whipple (1966) and seasonal data on these were summarized for each fish species (Appendix I). Parasites were identified with the aid of various keys (Appendix II). The presence of lymphocystis, identified by characteristic wart-like growths on the skin of afflicted fish (Ribelin and Migaki, 1975), was recorded

and these are referred to as tumors throughout the text. A complete set of representative specimens of all parasites is available from the author and T. A. Dick (University of Manitoba, Department of Zoology).

Statistical analysis

Differences in infracommunity size among years, seasons, and biological strata (littoral versus limnetic) occupied by fish were tested for using ANOVA, Pearson's correlation coefficient (r), simple linear regression, and Students' t-tests, respectively. The influence of host age and sex on infracommunity size was examined using linear regression and with t-tests. Community organization was examined at the level of the component community (all icthyoparasites present in the environment; Bush and Holmes, 1986) and for metacommunities (all parasites within a host population; Riggs et al., 1987) by plotting dominance-diversity curves (Southwood, 1978; Whittaker, 1975). Briefly, a dominance-diversity curve is a bivariate plot of the relative 'importance' of each species in a community (ordinate; logarithmic scale) in sequence from the most dominant to the least dominant species (abscissa; normal scale). A variety of measures can be used to measure importance. For metacommunities parasite abundance was used as the measure of importance. Ideally, the importance of parasites in the component community should take into account differences in the sizes of different host populations. However, attempts

to estimate the sizes of fish populations based on analysis of mark-recapture data using POPAN (Arnason and Baniuk, 1978) were hampered by low numbers of recaptured fish. Where the number of recaptures was sufficient to allow estimates of fish population sizes to be made, these were very imprecise (coefficient of variation ≥ 0.28) and were rejected. Therefore, the importance of each parasite species in the component community was estimated by summing (across all host species) the products of parasite abundance and the relative proportion of each fish species in the lake.

Relative proportions of fish hosts were estimated based on the proportions of fish caught in gill nets (excluding winter samples). An effort was made to minimize capture bias (a wide range of mesh sizes was used, nets were set at random throughout the lake, and in many cases the entire water column was being sampled) and since the relative proportions of each fish species caught in gill nets was highly correlated between years (Spearman's coefficient of rank correlation, $\underline{r}_r$=0.786-0.929), the combined (1985-1987) open-water catch is believed to accurately represent the true proportions of fish species in the lake. Further support is the high correlation between the composition of samples collected with gill nets and pound nets; the correlation between proportions of fish caught with these two sampling gears was positive but lower ($\underline{r}_r$=0·53) due mainly to higher proportions of cisco (Coregonus artedii) and lower proportions of quillback (Carpiodes cyprinus)

caught by gill nets versus pound nets (34% versus 1% for cisco, and 1% versus 22% for quillback, respectively). Cisco prefer deeper water (Scott and Crossman, 1973) and quillback are difficult to capture in gill nets, accounting for these differences. Furthermore, comparison of frequency distributions for fork length of fish caught by gill nets and pound nets (data not included) revealed that distributions for fish caught by the two gear were nearly identical with coincident modal frequencies. Since pound net samples indicated that quillback (Carpiodes cyprinus) were rarely caught by gill nets, their relative numbers were estimated subjectively as equal to the number of shorthead redhorses (Moxostoma macrolepidotum). The estimated relative proportions of each fish species (N) comprising the fish community in Dauphin Lake are given in Appendix III.

Since most measures of species diversity are influenced by the number of species (S) and the shape of the underlying dominance-diversity distribution (May, 1975), and since both of these were variable only the simplest and most basic indices were used to estimate within-community diversity (*a*-diversity). The number of species, bias corrected for small sample size ($S^* = S/log N$; N= total number of parasites) (Whittaker, 1975) and the Berger-Parker index, $D = N_{max}/N$ (N_{max}= number of individuals of the most numerous species) (Southwood, 1978) were employed. Diversity between communities (*b*-diversity) was measured using Jaccard's index, J (Southwood, 1978).

Throughout the text a probability of $P<0.05$ was considered significant. The terms 'infracommunity' and 'parasite numbers' are used synonymously. Mean intensity, prevalence, and abundance were calculated according to Margolis et al. (1982). All analyses were done using the Statistical Analysis Systems (SAS[4]) package or the A Programming Language (APL[5]) package as implemented by the University of Manitoba Computer Services Center.

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[4]SAS Institute Inc.; Box 8000, Cary, NC, USA.

[5]Allen Rose Associates; Suite N1405, 1200 Old Georgetown Road, Rockville MD, USA.

RESULTS

Parasite survey

Twenty-three species of fish were examined and 20 of these carried parasites. Burbot (Lota lota; N=1), carp (Cyprinus carpio, N=3), and fathead minnows (Pimephales promelas, N=14) were not infected. In total, 97,028 parasites were found and 91% (N=1,680) of the necropsied fish had at least one parasite. The number of parasite species (S) found was positively correlated to the number of hosts examined (r_r=0.92), indicating that rarer species were recovered as the number of fish examined increased. A total of 51 species of parasites spanning 39 genera and 30 families, plus viral lymphocystis and one plant-parasitic nematode (O. Dorylaimida) was found (Appendix II). Only six parasite species found mature in vertebrates other than fish and all of these use piscivorous birds as the definitive host (Table 1). The remaining species use fish as the definitive host; 33% (N=15) are directly transmitted and 62% (N=28) are transmitted via food-web relationships (Table 1). The life-cycles of two species (Paurorhynchus hiodontis and Creptotrema funduli) are unknown (Table 1). Detailed host-parasite lists are given in Appendix III; 15 parasite species were not previously reported from Manitoba and three species (Lissorchis crassicrurum, Neoechinoryhynchus distractus, and Rowardleus pennensis) are new for Canada.

Table 1. Parasites of Dauphin Lake fishes.

family	species	life cycle
Myxosomatidae		
	Myxosoma sp.	direct
Dactylogyridae		
	Anoncohaptor anomalum	direct
	Urocleidus adspectus	direct
Tetraonchidae		
	Tetraonchus monenteron	direct
Diplostomatidae		
	Diplostomulum spp.	snail-fish-bird
	Neascus spp.	snail-fish-bird
	Posthodiplostomum minimum	Physa-fish-bird
Strigeidae		
	Tetracotyle spp.	fish-bird
Azygiidae		
	Azygia longa	snail-fish
Bucephalidae		
	Paurorhynchus hiodontis	unknown
Lissorchiidae		
	Lissorchis crassicrurum[a]	snail-oligochaete-fish
	L. gullaris	snail-oligochaete-fish
Allocreadiidae		
	Crepidostomum cooperi	clams-mayfly-fish
	C. illinoiense	clams-mayfly-fish
	Creptotrema funduli	unknown
Cryptogonimidae		
	Caecincola sp.	snail-fish-fish
	Centrovarium lobotes	snail-fish-fish
Caryophyllaeidae		
	Biacetabulum infrequens	Tubifex-fish
	Biacetabulum sp.	oligochaete-fish
	Hunterella nodulosa	oligochaete-fish
	Monobothrium hunteri	oligochaete-fish
	Rowardleus pennensis[a]	oligochaete-fish
Lytocestidae		
	Khawia iowensis	oligochaete-fish
Bothriocephalidae		
	Bothriocephalus cuspidatus	Cyclops-fish-fish
Ligulidae		
	Ligula intestinalis	copepod-fish-bird
Triaenophoridae		
	Triaenophorus nodulosus	Cyclops-fish-fish
Proteocephalidae		
	Proteocephalus luciopercae	Cyclops-fish
	P. pearsei	Cyclops-fish
	P. pinguis	Cyclops-fish
	P. wickliffi	Cyclops-fish

Table 1. Parasites of Dauphin Lake fishes (continued).

family	species	life cycle
Anisakidae		
	Contracaecum sp.	fish-fish-bird
	Raphidascaris acus	*Cyclops*-fish-fish
Philometridae		
	Philometroides nodulosa	*Cyclops*-fish
Cystidicolidae		
	Spinitectus gracilis	mayfly-fish
Thelaziidae		
	Rhabdochona canadensis	mayfly-fish
Dorylaimidae[a]		plant-parasitic
Neoechynorhynchidae		
	Neoechinorhynchus carpiodi	crustacean-fish
	N. crassus	crustacean-fish
	N. cristatus	crustacean-fish
	N. distractus[a]	crustacean-fish
	N. tennelus	crustacean-fish
Pomphoryhynchidae		
	Pomphorhynchus bulbocolli	*Hyallela*-fish
Glossiphoniidae		
	Placobdella montifera	direct
Piscicolidae		
	Cystobranchus verilli	direct
	Myzobdella moorei	direct
Unionidae		
	glochidium	direct
Argulidae		
	Argulus appendiculosus	direct
Lernaeidae		
	Lernaea cyprinacea	direct
Ergasilidae		
	Ergasilus lizae	direct
	E. luciopercarum	direct
	E. nerkae	direct
	E. versicolor	direct

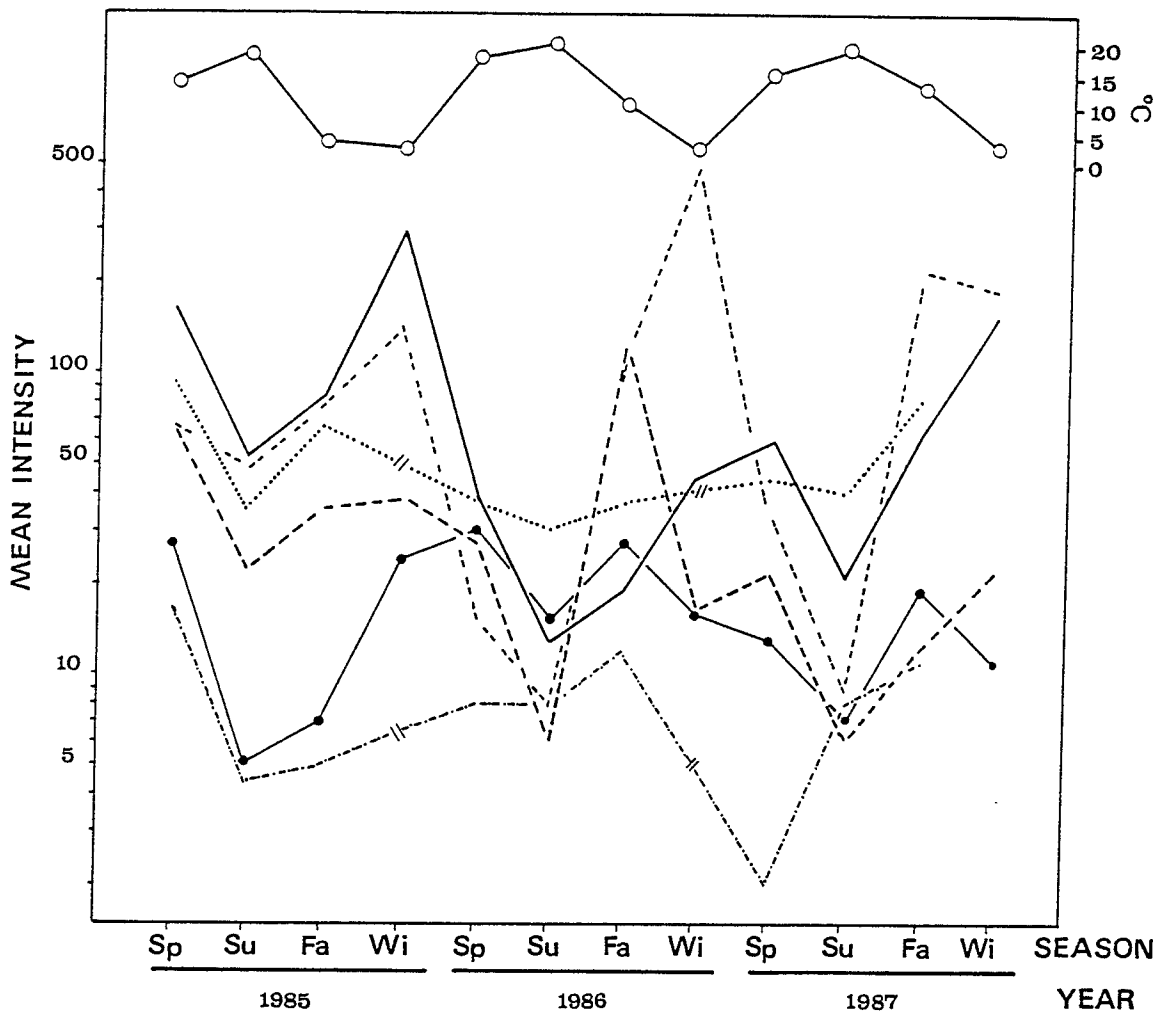
[a] new record for Canada according to Margolis and Arthur, 1979.

The diets of Dauphin Lake fishes are summarized in Appendix I; these will be dealt with here only briefly. Northern pike (Esox lucius), walleye (Stizostedion vitreum vitreum), and sauger (S. canadense) are piscivores and their prey consists mainly of yellow perch (Perca flavescens), cisco (Coregonus artedii), and forage-fishes (mostly troutperch; Percopsis omiscomaycus). Yellow perch feed on a wide variety of fish and invertebrates, while cisco and goldeye (Hiodon alosoides) feed primarily on invertebrates. The catostomids (silver redhorses, Moxostoma anisurum; shorthead redhorses, M. macrolepidotum; and white suckers, Catostomus commersoni) and quillback (Carpiodes cyprinus) feed on a variety of invertebrate fauna with molluscs, trichoptera, and ephemeroptera predominating.

Parasite numbers

Infracommunity size was variable between years for walleye (F=16.95, P=0.0001, N=329), northern pike (F=7.2, P=0.04, N=356), yellow perch (F=3.16, P=0.021, N=292) and cisco (F=4.45, P=0.03, N=286) but differences were not significant for all other species (ANOVAs). The number of parasites in walleye, pike, perch, cisco, white suckers, and shorthead redhorses fluctuated seasonally (Fig. 3); parasite numbers declined from a high level in spring to a minimum in mid-summer, and increased to a maximum in winter. Walleye and cisco had the greatest seasonal fluctuations (Fig. 3).

Figure 3. Seasonal changes in mean intensity (number of parasites/infected host) for walleye (——), northern pike (-----), yellow perch (·····), cisco (-- --), white suckers (—•—), and shorthead redhorses (--·--); and average water temperature (○; °C at ca. 1 m depth) for Dauphin Lake, Manitoba.


MEAN INTENSITY
500
100
50
10
5
20
15
10
5
0
C°
Sp Su Fa Wi Sp Su Fa Wi Sp Su Fa Wi
SEASON
1985
1986
1987
YEAR

Infracommunity size was negatively correlated to water temperature (Fig. 3) for walleye (r=-0.313, P=0.0001, N=303), yellow perch (r=-0.15, P=0.015, N=236) and cisco (r=-0.195, P=0.004, N=215); and parasite numbers increased with decreasing water depth at capture for cisco (r=-0.184, P=0.007, N=213) and yellow perch (r=-0.183, P=0.003, N=259). Furthermore, cisco captured by gill nets set in the littoral zone (>1 m depth) had fewer parasites (68.9 ± 109.7, N=206) than their benthic (≤1 m depth) counterparts (196.7 ± 266.7, N=78) (t-test, P=0.0001). Average infracommunity size increased with age for cisco, yellow perch, and shorthead redhorses (Fig. 4). Differences in infracommunity size between the sexes were apparent only for yellow perch, with females (N=234) harbouring more parasites (60.1 ± 73.3) than males (38 ± 40.2) (t-test, P=0.0042). Further analyses showed that male yellow perch (N=47) were significantly smaller (154.2±59.4 g) and younger (3.5±2.1 years) than females (209.7±66.1 and 4.7±2.4, respectively; N=234) (t-tests, P=0.0001 and P=0.007, respectively).

Community structure

Although the number of parasite species per metacommunity (S^*) was variable (Fig. 5) there was only a weak correlation (r_r=0.374) between the number of parasite species and mean intensity (Fig. 5). On average, infracommunities were composed of two parasite species (Figs. 5, 6) and the frequency histogram for the number of parasite species per

Figure 4. Mean intensity versus age for yellow perch (●), cisco (△), and shorthead redhorses (○). Symbols and bars represent means and standard errors, respectively. Sample size (number of fish) is given above each error bar. Pearson's correlation coefficient (r) for the regression of *log* mean intensity versus age is given for each host. $P \leq 0.0001$ for all regressions.

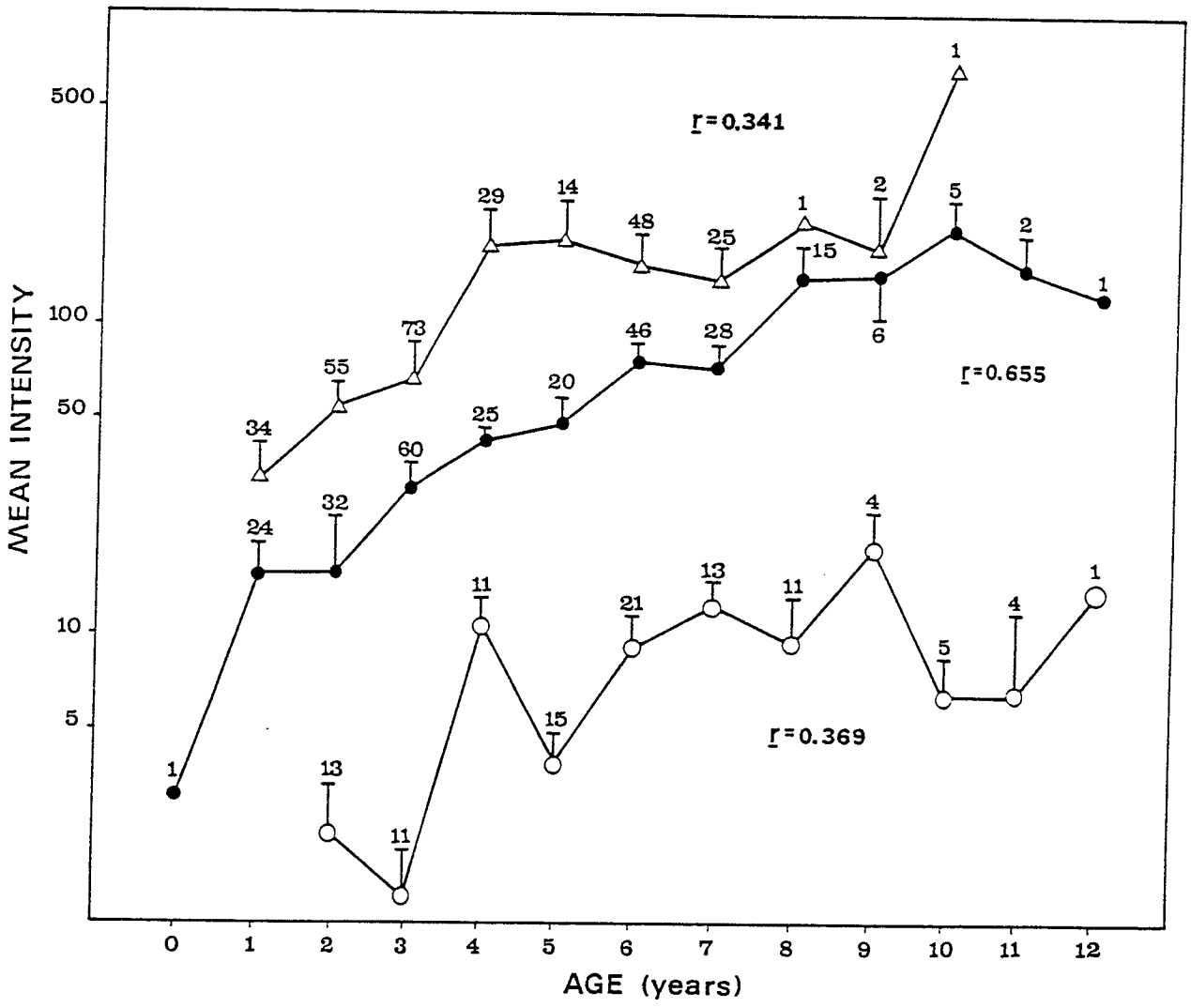

MEAN INTENSITY
500
100
50
10
5
r=0.341
r=0.655
r=0.369
AGE (years)
0 1 2 3 4 5 6 7 8 9 10 11 12

Figure 5. Mean intensity (●), number of parasite species, (S^*;△), and the Berger-Parker index, (D; ○) for parasite metacommunities from Dauphin Lake fishes. Bars and numbers indicate one standard error and sample size (number of fish), respectively. Fish hosts are in sequence from lowest to highest mean intensity (longnose dace to cisco, respectively). In all calculations lymphocystis, Myxosoma and Dorylaimida and parasite species reported as incidental sightings (eg. Ligula intestinalis in quillback) were not included. Data where the entire infracommunity was not considered were also eliminated. Burbot, carp, and fathead minnows were uninfected and these are not included on the figure. Note change in scale for mean intensity.

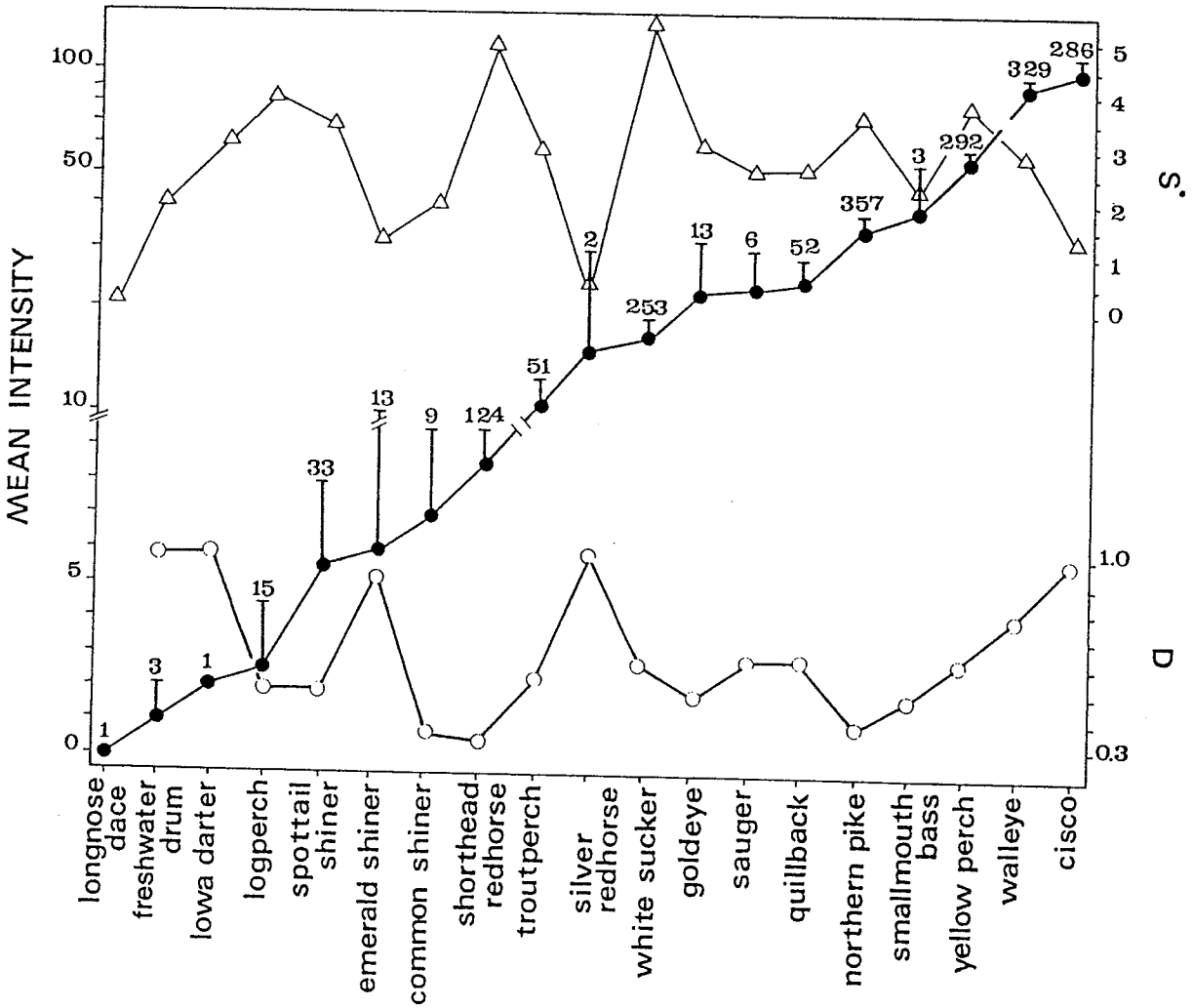
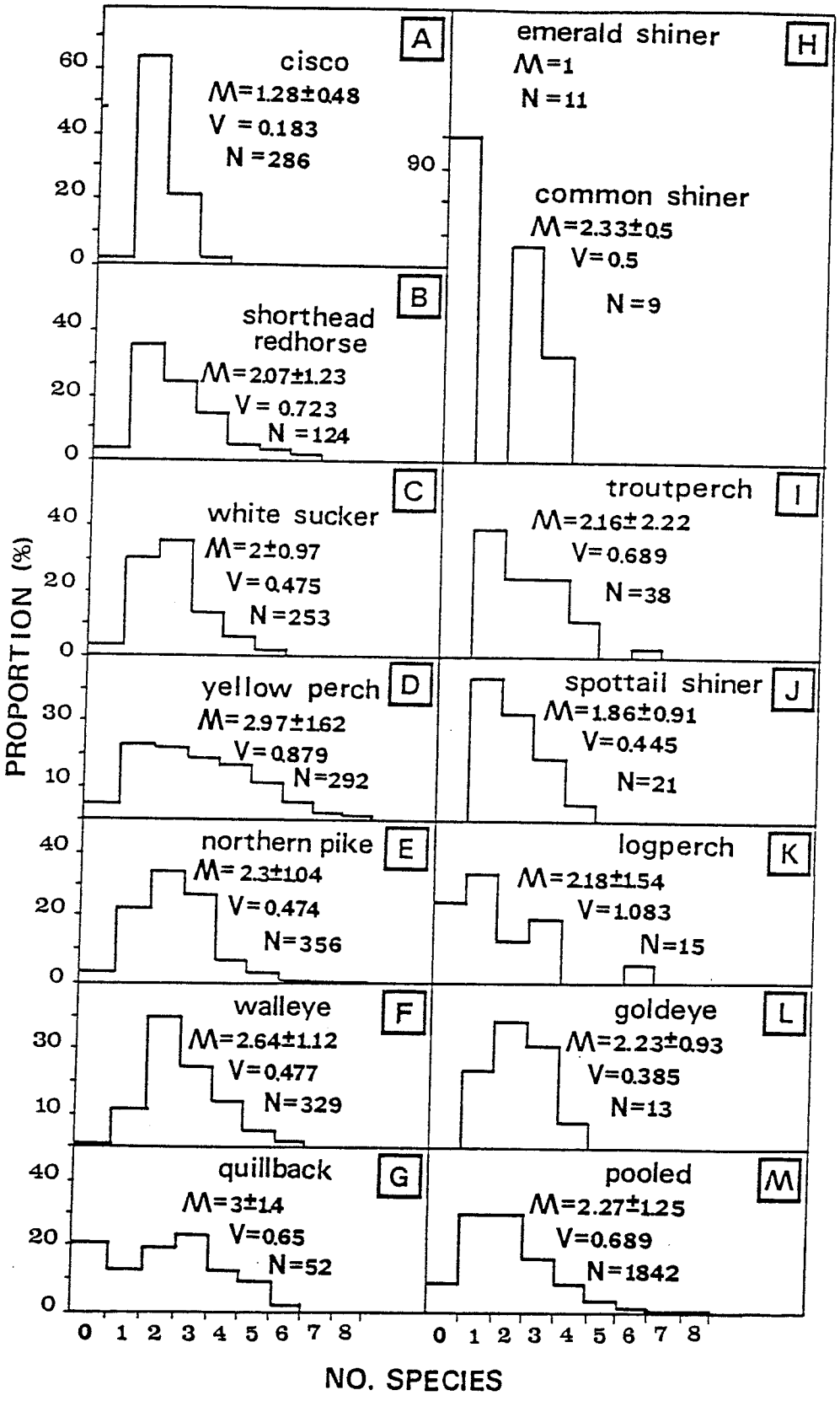

MEAN INTENSITY
100
50
10
5
0
S.
D
5
4
3
2
1
0
1.0
0.3
1
3
1
15
33
13
9
124
51
2
253
13
6
52
357
3
292
329
286
longnose dace
freshwater drum
lowa darter
logperch
spottail shiner
emerald shiner
common shiner
shorthead redhorse
troutperch
silver redhorse
white sucker
goldeye
sauger
quillback
northern pike
smallmouth bass
yellow perch
walleye
cisco

Figure 6. A-M; relative proportions (%) of fish hosts harbouring 0 to 8 species of parasites. Mean intensity (M; mean ± standard deviation), variance to mean ratio (V), and sample size (N; number of fish) are given for 13 fish species from Dauphin Lake. Histograms for smallmouth bass (M=3.33±1.15, V=0.4, N=3), sauger (1.5±0.55, 0.545, 6), silver redhorse (M=1, N=1), johnny darter (M=1, N=1), and freshwater drum (M=1, N=1) are not presented due to small N, but these species were included in the pooled distribution.


A
cisco
M=1.28±0.48
V = 0.183
N =286
B
shorthead redhorse
M=2.07±1.23
V = 0.723
N =124
C
white sucker
M=2±0.97
V=0.475
N =253
D
yellow perch
M=2.97±1.62
V=0.879
N=292
E
northern pike
M= 2.3±1.04
V=0.474
N=356
F
walleye
M=2.64±1.12
V=0.477
N=329
G
quillback
M=3±1.4
V=0.65
N=52
H
emerald shiner
M=1
N =11
common shiner
M=2.33±0.5
V=0.5
N=9
I
troutperch
M=2.16±2.22
V=0.689
N=38
J
spottail shiner
M=1.86±0.91
V=0.445
N=21
K
logperch
M=2.18±1.54
V=1.083
N=15
L
goldeye
M=2.23±0.93
V=0.385
N=13
M
pooled
M=2.27±1.25
V=0.689
N=1842
PROPORTION (%)
NO. SPECIES

metacommunity (Fig. 6) was positively skewed with a variance to mean ratio (V) less than one.

Similarity between metacommunities measured using Jaccard's index (J) was unaffected by metacommunity size ($\underline{r}_r$=-0.04) but was positively correlated to S^* ($\underline{r}_r$=0.34). Jaccard's index was greatest for metacommunities from closely related hosts (e.g. white suckers and shorthead redhorses; J=55.2), predators and their prey (eg. northern pike and yellow perch; J=44.1), and for fish occupying similar niches (eg. quillback and white suckers; J=20.1) (Table 2). Overall, the catostomids had the most species rich parasite communities (24 species) and shared at least one parasite with all other hosts in the lake (Table 2; Appendix III). Parasite metacommunities from logperch (Percina caprodes) and northern pike had the highest average J (Table 2).

The dominance-diversity curve for the component community (Fig. 7) approaches the log-normal distribution of Preston (1948). Proteocephalus wickliffi, Raphidascaris acus, and Bothriocephalus cuspidatus together accounted for 74% of all parasites found. Parasites that rely on piscivory for transmission (Table 1) to their definitive hosts (R. acus, B. cuspidatus, Triaenophorus nodulosus , Centrovarium lobotes, and Contracaecum sp.) are relatively common (0.75% to 16% for Contracaecum sp. and R. acus, respectively), while bird-transmitted types (Table 1) are rare ($\leq$0.14%) (Fig. 7). Dominance-diversity curves for metapopulations

Figure 7. Dominance-diversity curve for the component community of fish parasites from Dauphin Lake. Along the abscissa parasites are arranged in a sequence from the most dominant species (nearest the origin) to the least dominant species (furthest from the origin). Where relative dominance of parasites is greater than or equal to 1%, parasite species are identified. The curve was fitted by eye.

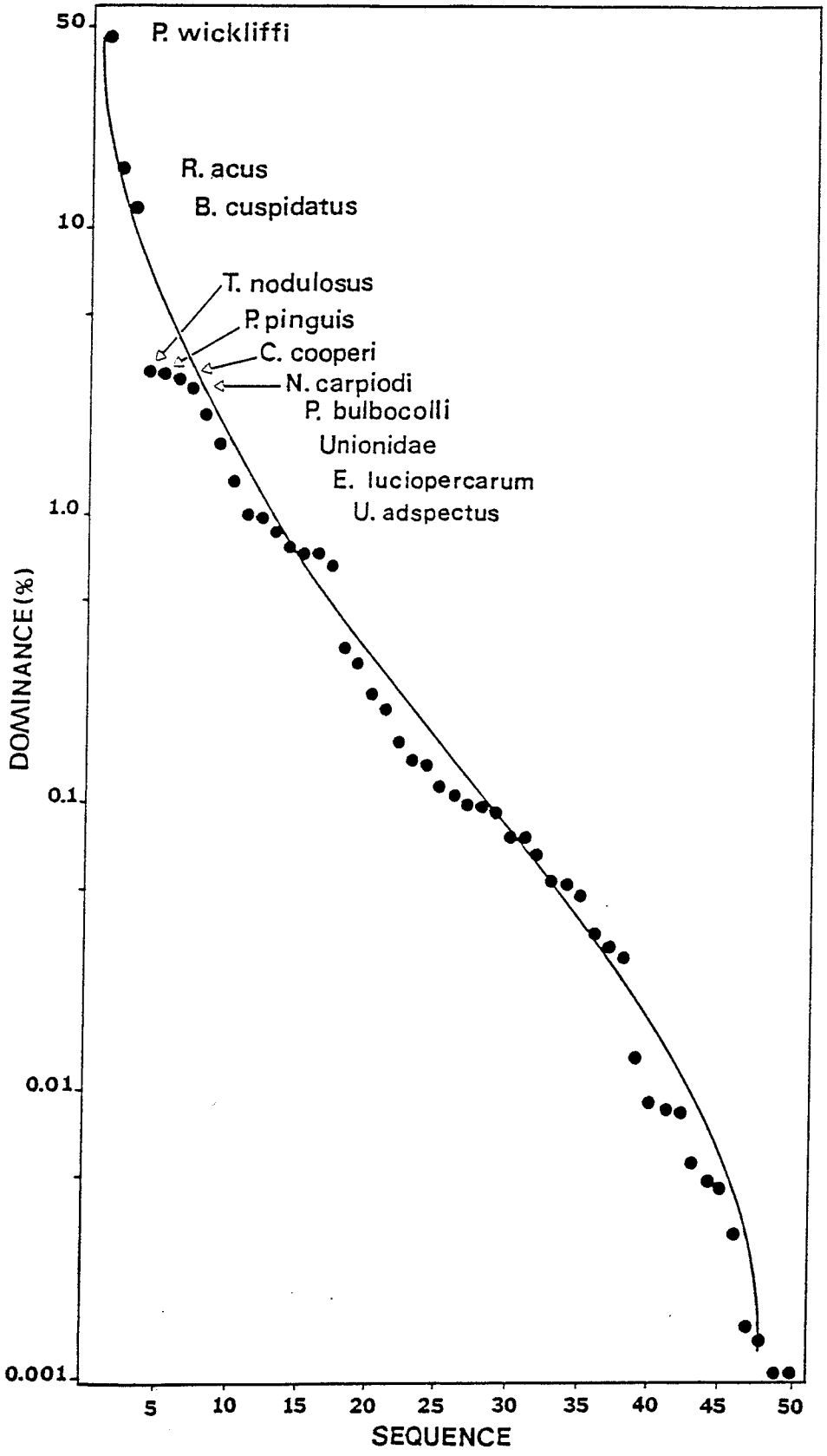

P. wickliffi
R. acus
B. cuspidatus
T. nodulosus
P. pinguis
C. cooperi
N. carpiodi
P. bulbocolli
Unionidae
E. luciopercarum
U. adspectus
50
10
1.0
0.1
0.01
0.001
DOMINANCE (%)
5
10
15
20
25
30
35
40
45
50
SEQUENCE

Table 2. Degree of similarity of parasite communities (measured using Jaccard's index, J) in each of 17 fish species found in Dauphin Lake.

fish host (code)	S[a]	%[b]	J[c]	most similar host, (J)
walleye (we)	16	87.5	18.1 ±13.4	yp (45.8)
northern pike (np)	17	93.4	23.0 ± 11.8	yp (44.1)
sauger (sa)	6	81.3	12.1 ± 10.6	we (37.5)
yellow perch (yp)	19	87.5	21.0 ± 13.8	we (45.8)
smallmouth bass (sb)	5	87.5	10.7 ± 7.8	lp (27.3)
freshwater drum (fd)	1	3.4	3.5 ± 5.4	sb (16.7)
white sucker (ws)	24	100	15.4 ± 13.1	sh (55.2)
shorthead redhorse (sh)	21	100	14.4 ± 12.5	ws (55.2)
silver redhorse (si)	1	1.9	0.6 ± 1.5	sh (4.8)
quillback (qb)	12	87.5	9.2 ± 5.7	ws (20.1)
cisco (ci)	8	87.5	11.4 ± 8.2	tp (28.6)
goldeye (ge)	9	75	7.8 ± 6.1	st (20)
troutperch (tp)	10	81.3	19.1 ± 14.3	lp (41.7)
logperch (lp)	7	87.5	23.4 ± 14	cs (57.1)
spottail shiner (st)	9	81.3	19.3 ± 11.9	np (42.1)
common shiner (cs)	4	81.3	15.6 ± 15	lp (57.1)
emerald shiner (es)	4	81.3	13.7 ± 10.9	cs (33.3)

[a]number of parasite species
[b]percent of all host species sharing at least one parasite species.
[c]mean ± standard deviation over all host pairs.

from most of the larger fish species (Fig. 8) and from all of the forage-fishes (Fig. 9) approached the geometric distribution (Whitaker, 1975). The curves for metacommunities from cisco, northern pike, and white suckers had obvious deviations from linearity (Fig. 8) not expected for the geometric series. For the latter, metacommunities were dominated by one (cisco and quillback) or two (northern pike) parasite species (P. wickliffi and Neoechinorhynchus carpiodi, and P. pinguis and T. nodulosus, respectively) (Fig. 8). Metacommunities of parasites from these hosts also had high values for the Berger-Parker index (D; Fig. 5).

Figure 8. Dominance-diversity curves for parasite metacommunities from 10 larger sized fish species from Dauphin Lake. Along the abscissa parasites are arranged in a sequence from the most dominant species (nearest the origin) to the least dominant species (furthest from the origin). The species sequences (scale bars) are shown separately for each host. Regression lines were fitted by eye.

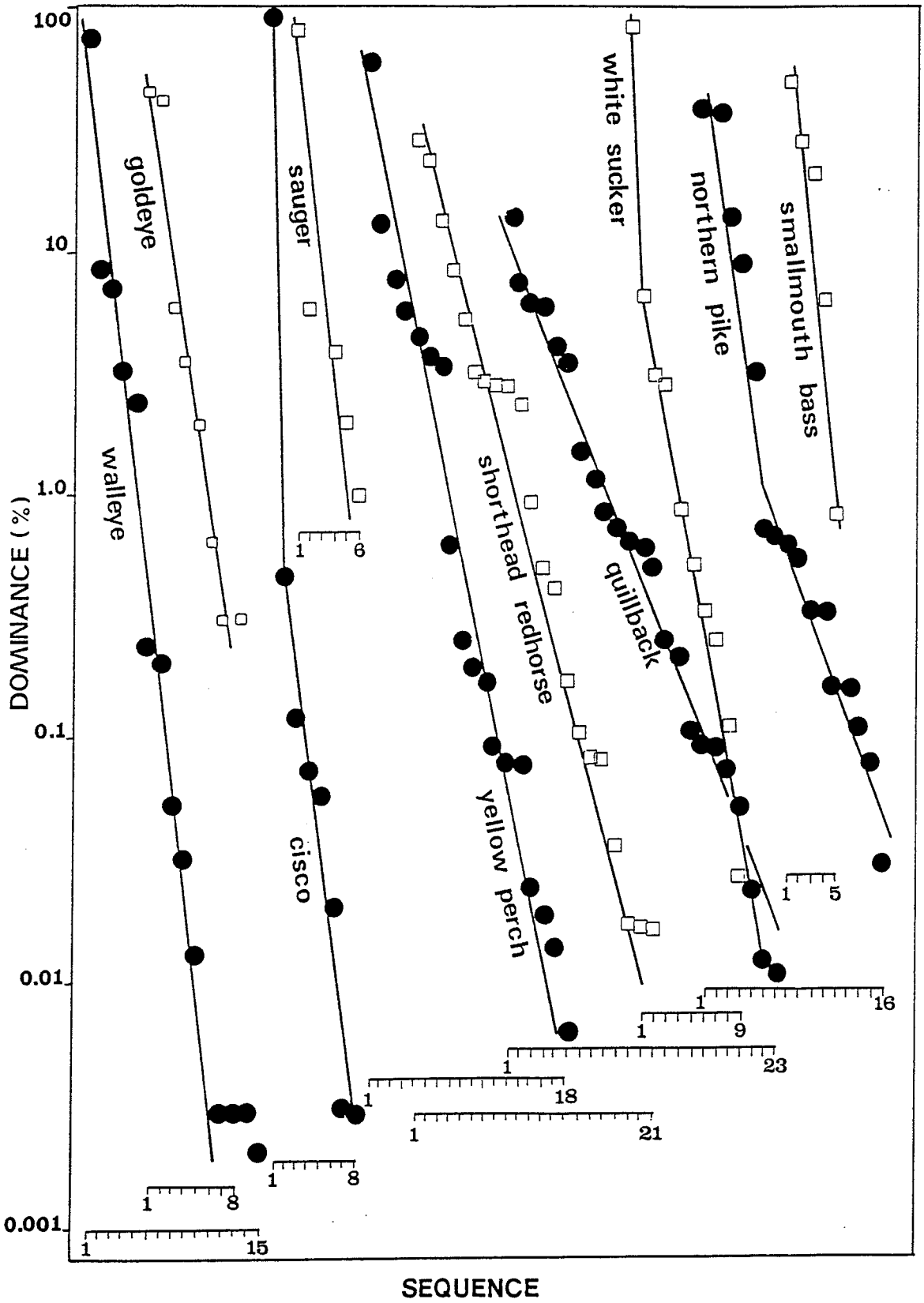
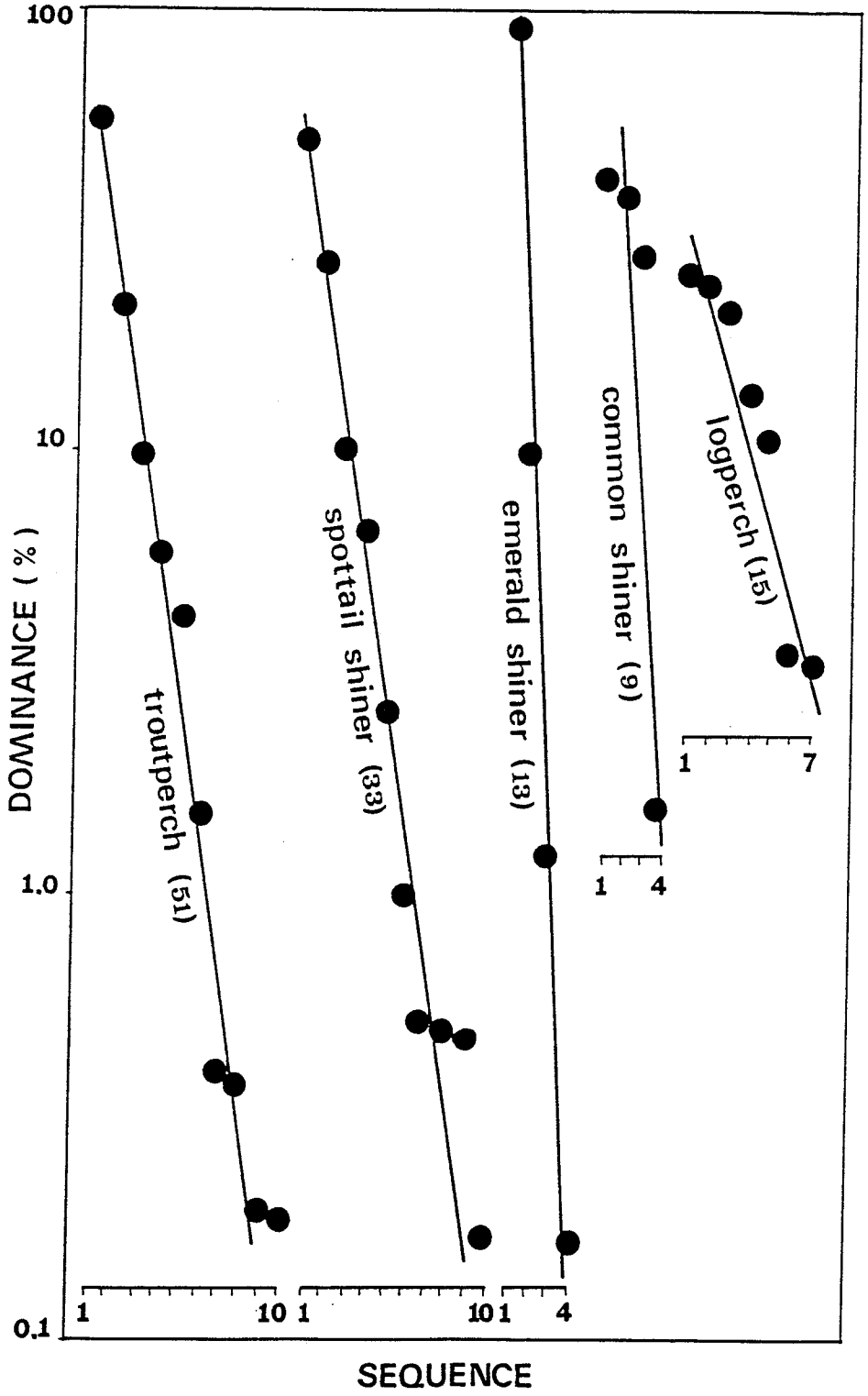

100
10
1.0
0.1
0.01
0.001
DOMINANCE (%)
walleye
goldeye
sauger
cisco
shorthead redhorse
yellow perch
quillback
white sucker
northern pike
smallmouth bass
1
8
15
1
6
1
8
1
18
21
1
9
23
1
16
1
5
SEQUENCE

Figure 9. Dominance-diversity curves for parasite metacommunities from 5 species of forage-fishes from Dauphin Lake. Sample sizes (number of fish) are indicated. Along the abscissa, parasites are arranged in a sequence from the most dominant species (nearest the origin) to the least dominant species (furthest from the origin). The species sequences (scale bars) are shown separately for each host. Regression lines were fitted by eye.


100
10
1.0
0.1
DOMINANCE (%)
troutperch (51)
spottail shiner (33)
emerald shiner (13)
common shiner (9)
logperch (15)
1
10
1
10
1
4
1
4
1
7
SEQUENCE

DISCUSSION

Parasite survey

Comparisons between the results of my survey and those of the survey by Stewart-Hay (1951) must be made cautiously, since the latter were based on a limited number (N=121) of fish collected in summer, when parasite numbers are low. For instance, although Stewart-Hay (1951) reported only 16 parasite species from Dauphin Lake, I found 51 species. Nevertheless, comparison reveals that since 1951 dramatic changes have occurred in the parasite community in Dauphin Lake. First, although in the past 'metacercariae' were abundant in forage fishes (Stewart-Hay, 1951); metacercariae are now more numerous and found in more host species. Second, although plerocercoids of L. intestinalis ('Schistocephalus' of Stewart-Hay) were presumably present, Stewart-Hay (1951) found these in low numbers and only in spottail shiners (Notropis hudsonius); today, the number of L. intestinalis has increased and plerocercoids are found in numerous host species. Third, Stewart-Hay (1951) reported finding only a few Triaenophorus nodulosus (T. stizostedionis of Stewart-Hay) in walleye; yet today T. nodulosus is found in high numbers in northern pike, cisco, yellow perch, and troutperch. Fourth, there are now at least four new species of fish in Dauphin Lake: sauger, freshwater drum, silver redhorse, and smallmouth bass.

The increased numbers and wider host distributions for L.

intestinalis and metacercariae of various digenea are no doubt a reflection of the increased use of Dauphin Lake by piscivorous birds. Stewart-Hay (1951) reported few birds in his biological survey although they are numerous on Dauphin Lake today. It is more difficult, given the limited historical data on icthyoparasites in Dauphin Lake, to account for the dramatic increase in numbers of Triaenophorus nodulosus since 1951. However, this study and others (Cunningham, 1935; Butler, 1949; Babaluk et al., 1984), have documented major shifts in the species composition of the fish community in Dauphin Lake, particularly the steady decline in the number of walleye. Perhaps this has affected predator-prey interactions in such a way that allowed T. nodulosus to become established and more widespread than in the past. The continued absence of mammalian piscivores in the area is reflected in the complete absence of mammal-transmitted parasites in the fish parasite community of Dauphin Lake. This component of the community, at least, has remained unchanged since 1951.

Several of the parasites reported here are known pathogens of fish. Briefly: Myxosoma sp. was found in a number of species and infections of the gills or muscle tissue by Myxosporidea can cause extensive damage to the host (Dogiel et al., 1961); metacercariae can cause mortalities of young fish (Lemly and Esch, 1984); and plerocercoids of T. nodulosus and L. intestinalis can severely damage the liver (Mathey, 1963; Lawler, 1969) and

cause gonadal recrudescence (Wyatt and Kennedy, 1988), respectively, in infected fishes. Furthermore, Pomphorhynchus bulbocolli is considered a serious fish pathogen (Bullock, 1963) and mortalities among fish have been attributed to Contracaecum spp. (Dechtiar, 1972), Argulus spp. (Allum and Hugghins, 1959), and L. cyprinacea (Dechtiar, 1972).

Parasite numbers

Seasonal fluctuation in numbers has been reported for a wide variety of fish parasite communities in North America (Anderson, 1976; Cloutman, 1975). These cyclical changes are usually attributed to seasonality of final host feeding behaviour and temperature-dependent mortality of parasites within the definitive host (Kennedy, 1969; Anderson, 1974). Although the specific factors controlling the amplitude and length of these cycles is unknown, evidence suggests that water temperature seems to be the dominant physical parameter determining the seasonality of parasites in many fish parasite systems (Kennedy, 1969, 1971, 1972; Anderson,1976). Especially in north-temperate lakes, seasonal patterns of feeding by fish are determined by the seasonal availability of food items, which is influenced by temperature for most aquatic invertebrates (Ward and Whipple, 1966). In Dauphin Lake: cisco feed almost exclusively on zooplankton whose numbers are determined largely by water temperature (Ward and Whipple, 1966);

catostomids feed on a variety of invertebrates and a number of these are present throughout the year; yellow perch feed on both vertebrates and invertebrates but switch to a predominantly invertebrate diet in the winter. Since aquatic invertebrates play an important role as intermediate hosts for a variety of parasites (Table 1), seasonal changes in the numbers and types of invertebrates consumed by fishes will result in concomitant changes in parasite infracommunities. As expected based on the composition of their diets: cisco show the greatest amplitude of seasonal changes in parasite numbers; seasonal fluctuations are damped out for catostomids; and yellow perch have oscillations intermediate in magnitude.

Increasing worm burden as fish age has been demonstrated for many parasites (Amin, 1974; Cloutman, 1975), and has been attributed to increased feeding (and therefore parasite recruitment) by larger fishes. Similarly, it appears that the increased numbers of parasites in female versus male yellow perch is due to the increased size and age of female perch. The relationship between age and sex of yellow perch and infracommunity size will be discussed in more detail in a later section (Chapter 4-1).

Community structure

Each of the fish species examined had a characteristic assemblage of parasites. Regardless of the fish or parasite species fish harboured about two parasite species and 4-130

individuals. Similar results were reported by Kennedy et al. (1986) for various freshwater fishes; fish harboured ~0.5-1.8 parasite species and ~2-140 individual parasites per host. Kennedy et al. (1986) identified omnivory and host movements relative to their prey (vagility) as two factors essential to the production of diverse helminth communities. In Dauphin Lake, catostomids are generalist omnivores and their ingestion of a wider range of invertebrates no doubt contributes to their higher species richness. Unfortunately, since little information is available regarding fish movements in Dauphin Lake, the extent to which increased host movement might affect parasite community diversity is not easily determined. However; northern pike, walleye, and catostomids captured, tagged, and released from pound nets in the southern half of the lake, were captured throughout the lake by commercial fisherman; and in order of abundance northern pike, white suckers, quillback, shorthead redhorses, walleye, yellow perch, goldeyes, and sauger migrate upstream in the spring to spawn (Harbricht and Franzin, 1988). Although these fish harbour many parasite species, I have no direct evidence that increased vagility is linked directly to increased complexity of the parasite community. In fact, since most spawning fish do not feed (Scott and Crossman, 1973), it is likely that few parasite species are recruited during the spawning migration.

Leong and Holmes (1981) suggested that the species richness of a parasite community within a host should

depend, in part, on the number of related host species present. The low species richness of cisco, the only member of the Salmonidae (Coregoninae) in Dauphin Lake, supports this claim and the hypothesis of Wisniewski (1958): that the parasite community within an ecosystem is characterized by parasites of the numerically dominant hosts. However, other results do not fit the hypothesis. First, although parasites of the numerically dominant cisco, yellow perch, and northern pike dominate the system, and exchange of parasites between host species was greatest between closely related hosts or predators and their prey, cisco harbours only one parasite (R. acus) common to other host species in the lake. Second, although the exact number of quillback is not known I am certain that the quillback population is smaller than that of most other fish populations in Dauphin Lake, yet N. carpiodi ranks seventh in importance in the component community. This suggests that large numbers of hosts are not essential for host-specific parasites to comprise a substantial portion of the component community. Furthermore, the low numbers of bird-transmitted parasites and the absence of mammal-transmitted parasites in Dauphin Lake contradicts the suggestions made by Wisniewski (1958) and Esch (1971): that eutrophic systems should be characterized by large numbers of these parasites. Interactions between mammals and fish and between birds and fish may be restricted due to the physical constraints imposed by Dauphin Lake. The limited shoreline relative to the surface

area of Dauphin Lake might dilute the impact of any terrestrial-aquatic interactions; whereas the choppy nature of the lake's surface forces most piscivorous birds to forage mainly in the few protected bays.

The shape of the dominance-diversity curve approaches that for a log-normal distribution of species abundance, indicating that the fish parasite component community is composed of a few very important species, many species with intermediate importance, and a few rare species. A similar tri-modal arrangement of parasites was reported by Bush and Holmes (1986) for communities of helminths from lesser scaup ducks. This community structure fits the hypotheses of Caswell (1978) and Hanski (1982); that communities are composed of a small set of predictable dominant species (P. wickliffi, B. cuspidatus, and R. acus) and a larger set of subordinate species (intermediate and rare species). When parasite metacommunities are analyzed separately a completely different pattern emerges. Dominance-diversity curves for individual metacommunities fit the geometric series (May, 1975), indicating the presence of one or two dominant species (Whittaker, 1975). This is best shown by metacommunities from cisco, northern pike, and quillback where P. wickliffi, T. nodulosus and P. pinguis, and N. carpiodi (respectively) account for 54 to 88% of all parasites found.

Dominance-diversity curves are difficult to interpret (Whittaker, 1975) since often several reasonable arguments

can be made to explain a unique pattern of organization. Recognizing this weakness, two interpretations are particularly appealing to my results. First, Whittaker (1975) proposed that the log-normal distribution (Preston, 1948) will occur if the relative importances of species populations are determined by a number of independent variables with different effects on each species. Whittaker (1975) also proposed that the geometric series might reflect the outcome of scramble competition among subsets of individuals dependent on similar resources. These are reasonable interpretations of the dominance-diversity curves for the component community and for metacommunities, respectively: the component community is composed of many metacommunities where the importance of each parasite population within an individual metacommunity might be determined by different factors; and it appears that, for N. carpiodi at least, interactions within a host species can be more important than interactions between host species.

The objective here was not to explain the mechanics that might lead to the observed arrangements for parasite communities. Instead, I attempted to show that the differences in structure seen for fish parasite communities observed from different hierarchical levels within these communities are real, and that these differences can lead to conflicting interpretations if parasite communities are considered from only a single perspective. For instance, if the fish-parasite assemblage in Dauphin Lake is

representative of fish-parasite assemblages elsewhere, and if my interpretations are correct, then an analysis restricted to the component community would suggest that fish parasites *should* be studied using an ecosystem approach, as Wisniewski (1958) and others (Noble et al., 1963; Chubb, 1970; Esch, 1971; Cloutman, 1975) have suggested. In contrast, analysis *of the same* parasite assemblage from the perspective of the metacommunity would support the ideas of Halvorsen (1971) and Wooten (1973); that analyses of individual hosts and their parasites is adequate!

The relationship between the types of parasites and fishes and parasite community structure is complex. In Dauphin Lake, parasites show varying degrees of host-specificity and differences in the number of hosts and trophic levels utilized by parasites. For example, N. carpiodi uses an unknown invertebrate intermediate host and infects only quillback; whereas R. acus uses a variety of invertebrates (Smith, 1984) and infects numerous species of fishes. Furthermore, although most populations of fish in Dauphin Lake, including quillback and northern pike, are overdispersed during the spring spawning migration (Harbricht and Franzin, 1988), field collections suggest that quillback at least, are schooling fish and maintain a clumped distribution year round. These differences are characteristic of Dauphin Lake and of other systems, and since they influence the structure of parasite infracommunities differently, generalizations regarding

component communities are difficult if not impossible to make.

To my knowledge this is the first time anyone has attempted to illustrate the differences in structure between parasite metacommunities and the component community they comprise. Furthermore, this study represents the first application of dominance-diversity curves for the analysis of a parasite community. Since interpretations based on *a priori* assumptions concerning the organization of fish parasite communities can be misleading, I suggest that parasitologists should consider both large- and small-scale community structure before interpreting any analyses on fish parasite communities.

CHAPTER 2: THE ECTOPARASITE COMMUNITY OF DAUPHIN LAKE FISHES.

INTRODUCTION

There is some information on the population biology and transmission dynamics of ectoparasites of terrestrial hosts (Werman, 1983; Glicken and Schwab, 1980), but few populations of fish ectoparasites have been studied. As mentioned earlier (Chapter 1) ectoparasites are rarely included in investigations concerned with fish-parasites. Although ectoparasites of fish have been described (Hayunga, 1984; Amin, 1981; Meyer, 1946) and enumerated (Amin, 1981), and their effects on particular hosts has been investigated (Pottinger, Pickering and Blackstock, 1984; Harrison and Hadley, 1982; Allum and Hugghins, 1959), there is a paucity of community oriented information on ectoparasites of fish. Little is known regarding the rates of infestation of fishes by ectoparasites, and how ectoparasites are transmitted to the fish host. This is not surprising as it is difficult to accurately estimate ectoparasite numbers since most ectoparasites detach from dead or dying hosts. Nevertheless, sampling procedures that minimize host stress are available and these can be used to obtain improved estimates of ectoparasite numbers.

The results presented in this chapter are from a study initiated to (1) determine the types and numbers of ectoparasites infecting fishes in Dauphin Lake, (2) compare estimates of ectoparasite numbers obtained from samples of fish collected with different sampling gear, and (3)

determine the rate at which ectoparasite reinfestation occurs when ectoparasite-free fish are returned to their natural habitats.

MATERIALS AND METHODS

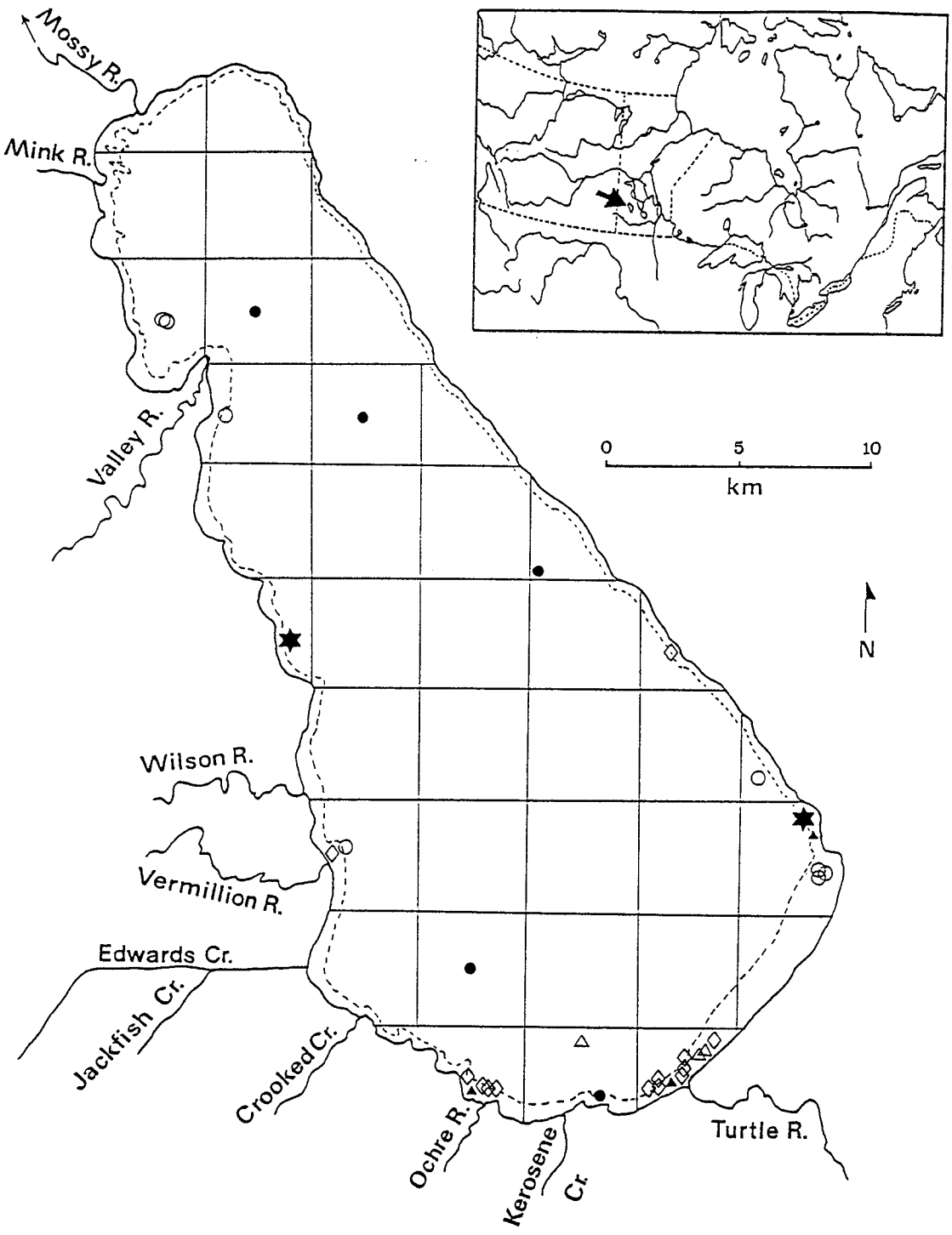
A detailed description of Dauphin Lake and a full account of the sampling procedures has already been given (Chapter 1), so only an abbreviated form of the latter and additional pertinent information will be presented here.

Study site and sampling procedures

Dauphin Lake (Fig. 10) has a surface area of approximately 700 km^2, a maximum depth of 3.5 m, and a total volume of approximately 1.645 X 10^9 m^3. The Mossy River is the only outlet stream. The lake can be separated into two distinct habitat-types: a shallow southern basin with weedy shorelines that receives most of the inflow to the lake; and a deeper northern basin with steep, rocky, sparsely vegetated shorelines. The lake is subject to large fluctuations in depth. Very little angling takes place and most of this is confined to the mouths of the rivers. Commercial harvests are limited to the winter season.

Fish were collected in 1985, 1986, and 1987 using gill nets, pound nets, and fyke nets (Everhart et al, 1976). From 1985 to 1987, gill net samples were collected during the spring, summer, and fall and a subsample of the commercially harvested fish was collected each winter. In total 14,737 fish collected with gill nets were processed and inspected for tumors and lesions. Detailed inspection of 2,865 of these provided estimates of ectoparasites.

Figure 10. Map of Dauphin Lake showing sites where pound nets (✱), fyke nets (▲), and gill nets (large open squares) were operated. Approximate locations of gill netted fish harboring ectoparasites are indicated. Symbols indicate fish infected with P. montifera (●), M. moorei (◇), neascus-type metacercariae (blackspot;○), or viral lymphocystis (tumors;△). Thin dashed line indicates approximate boundary of the 1.5 m depth contour.


Mossy R.
Mink R.
Valley R.
Wilson R.
Vermillion R.
Edwards Cr.
Jackfish Cr.
Crooked Cr.
Ochre R.
Kerosene Cr.
Turtle R.
0
5
10
km
N

Each fall fish were trapped alive using pound nets installed at two inshore stations on the lake (Fig. 10). A total of 3,869 fish was captured, examined, and ectoparasites were removed. Three-thousand four-hundred and seventy-eight (90%) of these were weighed, measured, marked with individually numbered jaw or spaghetti tags (Everhart et al., 1976), and released. Small (age-0) fish and minnows (N=2,153) were collected using fyke nets in 1986 and 1987 and these were also examined for ectoparasites.

Ectoparasites were collected, preserved, and stained according to the protocols outlined previously (Chapter 1). Leeches were identified with the aid of keys by Sawyer (1972) and Davies (1971), Branchiura were identified using Kabata (1988), and Caligidea were identified using Hoffman (1970). Viral lymphocystis, characterized by wart-like growths on the skin of diseased fish (Ribelin and Migaki, 1975), were recorded and are referred to as 'tumors' throughout the text. Throughout this chapter, wherever the term 'ectoparasite' is used I include tumors in the analyses. Although Ergasilus spp. infect many fishes in Dauphin Lake (Appendix III), no attempt was made to quantify the levels of infection of these ectoparasites on live fishes.

Statistical analysis

Mean intensity, prevalence, and abundance of ectoparasites was calculated according to Margolis et al.

(1982). Pearson's correlation coefficients (r) were used to test for correlations between the numbers of parasites and the species of hosts, and among numbers of parasites and the following variables: fork length (FL), recapture frequency, time since initial capture, year of tagging, and fish type (benthic versus limnetic). The proportions of each fish species caught in gill nets (22 September- 2 October sets only) and pound nets (7 August - 12 September) were calculated and ranked. Spearman's rank coefficient of correlation (r_r) was used to test for correlations in ranks between years and sites for pound nets, and between gill nets and pound nets. Rank correlation coefficients were also calculated for the proportions of each fish species captured by pound nets and prevalence and intensity of ectoparasites. Differences in parasite numbers between benthic versus limnetic fishes were tested using Students' t-tests. The spatial distribution of infected fish in Dauphin Lake was determined and this information used to adjust estimates of ectoparasite numbers obtained from the gill net samples. Estimates of prevalence made based on pound net samples were compared to adjusted estimates from gill net samples. A probability of $P<0.05$ was considered significant in all parametric tests, unless otherwise stated.

RESULTS

Three species of leeches (Myzobdella moorei, Placobdella montifera , and Cystobranchus verilli), two species of parasitic crustaceans (Argulus appendiculosus and Lernaea cyprinacea), neascus-type metacercariae (blackspot) and tumors (lymphocystis) were found. Eleven of 20 fish species sampled were infected with one or more ectoparasite species, and 4 of these species had tumors (Table 3). Myzobdella moorei and A. appendiculosus infected the largest number (7) of host species while C. verilli was found on shorthead redhorses (Moxostoma macrolepidotum) only (Table 3). When all fish were combined P. montifera was most prevalent (1.73% for the pound net sample), and M. moorei had the highest intensity and range of infection (2.91 ± 8.91 and 1-52, respectively) (Table 3). Walleye (Stizostedion vitreum) had the highest prevalence of tumors (2.67%) (Table 3).

A single yellow perch (Perca flavescens) infected with 52 M. moorei was emaciated, and had extensive damage to the fins. Similarly, an age-0 white sucker (Catostomus commersoni) infected with a single L. cyprinacea was emaciated. However, gross pathology associated with infections by ectoparasites was usually limited to severe but localized lesions at sites of attachment, and no mortality was observed that could be attributed directly to infection.

Table 3. Ectoparasites of Dauphin Lake fishes.

fish host	gear[a]	N[b]	M. moorei	P. montifera	C. verilli	A. appendiculosus
Stizostedion vitreum	A	681/1596	1.33±0.58 (0.44) [1-2][c]	-	-	-
	B	449/449	1 (1.11) [1]	1 (0.22) [1]	-	1 (0.45) [1]
	C	4	-	-	-	-
Esox lucius	A	673/2592	-	1 (0.15) [1]	-	-
	B	1841/1680	-	1.19±0.40 (1.95) [1-2]	-	(0.11) [1]
	C	17	-	-	-	-
Perca flavescens	A	437/3540	2.67±2.89 (0.69) [1-6]	-	-	-
	B	127/124	3.41±9.75 (21.4) [1-52]	1.50±1.0 (3.15) [1-3]	-	-
	C	41	1 (2.40) [1]	-	-	-
Catastomus commersoni	A	362/2016	-	1 (0.83) [1]	-	-
	B	543/543	1 (0.18) [1]	1.05±0.23 (3.49) [1-2]	-	1.13±0.35 (1-5) [1-2]
	C	292	-	-	-	1 (1.37) [1]
Moxostoma macrolepidotum	A	192/844	-	-	-	-
	B	603/603	1 (0.17) [1]	1 (0.99) [1]	1 (0.17) [1]	-
	C	308	-	-	-	-
Carpiodes cyprinus	A	27/27	-	-	-	-
	B	268/43	-	-	-	1 (0.37) [1]
	C	0	-	-	-	-
Coregonus artedii	A	461/4017	-	-	-	-
	B	3/3	-	-	-	-
	C	8	-	-	-	1 (12.5) [1]
Notropis atherinoides	D	994	-	-	-	1 (0.7) [1]
N. hudsonius	D	205	2 (0.49) [2]	-	-	-
Percopsis omiscomaycus	D	193	1 (5.70) [1]	-	-	1 (4.15) [1]
Percina caprodes	D	51	1 (9.80) [1]	-	-	-
pooled[d]	A	2865/14737	2±2 (0.21)[1-6]	1 (0.14) [1]	-	-
	B	3869/3478	2.9±8.9 (0.88)[1-52]	1.11±0.37 (1.73)[1-3]	1(0.03)[1]	1.08±0.28 (0.34)[1-2]
	C	670	1 (0.15) [1]	-	-	1 (0.29) [1]
	D	1483	1 (1.08) [1]	-	-	1 (1.01) [1]

[a] sampling gear: gill nets, A; pound nets, B; fyke nets (0^+ fish), C; fyke nets (minnows), D.
[b] number of fish examined: for gill nets, number examined for ectoparasites/number examined for ectoparasites and tumors: for pound nets= number examined/number examined and tagged.
[c] number of parasites: mean±standard deviation, (prevalence), [range].
[d] the following fish species were uninfected but are included in the pooled estimates: Hiodon alosoides (N=110), Aplodinotus grunniens (2), S. canadense (9), M. anisurum (8), Cyprinus carpio (3), N. cornutus (21), Pimephales promelas (14), Etheostoma nigrum (4), Rhinycthyes cataractae (1).

There was no difference in the relative proportions of fish caught by pound nets at different stations (r_r=1.0) (Fig. 10) or fished in different years (r_r=0.93). Similarly, the relative proportions of fish caught by fall gill netting were highly correlated between years (r_r=0.78). Samples collected by pound netting were pooled between years and stations and compared to the pooled fall gill netting samples. The correlation between proportions of fish caught with these two sampling gears was positive (r_r=0.53). However, higher proportions of cisco (Coregonus artedii) and lower proportions of quillback (Carpiodes cyprinus) were caught by gill nets versus pound nets (34% versus 1% for cisco, and 1% versus 22% for quillback, respectively). The length frequency distributions for fish species caught by gill nets in the fall were compared to those for fish caught by pound nets; these were nearly identical with coincident modal frequencies (data not included). In general pound nets underestimated the proportions of small (FL<300 mm) fish but these comprised ≤6% of the entire gill net sample.

Proportions of fish caught in the pooled pound net sample were compared to the numbers of ectoparasites and tumors they harbored. Prevalence for all ectoparasites and for tumors was negatively correlated with the number of fish caught (Spearman's r). Although the prevalence of P. montifera was only slightly correlated with the numbers of fish caught (r_r=-0.1), there was a strong positive correlation (r_r=0.75) between intensity and numbers of fish.

Based on their feeding habits (Scott and Crossman, 1973; Appendix I) white suckers (C. commersoni), quillback (Carpiodes cyprinus), freshwater drum (Aplodinotus grunniens), shorthead redhorses (Moxostoma macrolepidotum), silver redhorses (M. anisurum), and carp (Cyprinus carpio) were designated 'benthic' types, while those feeding primarily in the water column (northern pike, Esox lucius; yellow perch, Perca flavescens; walleye, Stizostedion vitreum; and sauger, S. canadensis) were designated 'limnetic' types. Cisco and goldeye (Hiodon alosoides) feed near the surface but were not categorized as they were rarely caught by pound nets and only a single cisco harbored ectoparasites. Benthic and limnetic fishes were compared to test for differences in their ectoparasite burdens. Cystobranchus verilli was eliminated as only a single specimen was found (Table 3). The prevalence of A. appendiculosus and blackspot was significantly higher in benthic fishes (0.63% and 1.55%, respectively) than limnetic fishes (0.16% and 0.04%, respectively); M. moorei and tumors were more prevalent in limnetic fishes (1.32% and 0.78%, respectively) than benthic fishes (0.14% and 0.07%, respectively); and the prevalence of P. montifera did not differ significantly between fish types (Z-tests for difference in proportions) (Table 4).

Table 4. Comparison between intensity (I; mean±standard deviation) and prevalence (P in %) of infection by ectoparasites for benthic versus limnetic fish caught in pound nets. Results of Z-tests for difference between prevalence are given (ns = not significant).

	fish type (no. examined)			
	benthic (N=1418)		limnetic (N=2426)	
	I[a]	P	I	P
Myzobdella moorei	1	0.14	2.97±9.48	1.32 P<0.05
Placobdella montifera	1.04±0.20	1.76	1.12±0.40	1.69 ns
Cystobranchus verilli	1	0.07	-	- P<0.05
Argulus appendiculosus	1.11±0.33	0.63	1	0.16 P<0.05
neascus-type metacercaria	-	1.55	-	0.04 P<0.05
viral lymphocystis	-	0.07	-	0.79 P<0.05

[a]number of parasites per infected fish.

Pairwise correlations were calculated for the variables fork length, recapture frequency, time since last capture, year of first capture, fish type (benthic versus limnetic), numbers of each ectoparasite species, and presence or absence of tumors. For the pooled pound net samples (all fish species combined) significant negative correlations existed between fork length and the numbers of M.moorei (r=-0.049, P=0.0038) and blackspot (r=-0.386, P=0.023), and between fish types and the numbers of A. appendiculosus (r=-0.049, P=0.001; with heavier infections on benthic fish) and blackspot (r=-0.108, P=0.0001; with heavier infections on benthic fish). The increased numbers of M. moorei on smaller fishes can be attributed mostly to the high numbers of these leeches on yellow perch, the smallest species caught (FL = 265.4±33.8 mm, N=124). Smaller walleye had lower numbers of this ectoparasite (r=-0.095, P=0.043). Increased prevalence of blackspot on shorter, benthic fishes can be attributed to white suckers which had a slightly increased prevalence of infection as fork-length decreased (r=-0.080, P=0.0634). The numbers of P. montifera increased with increasing capture frequency for walleye (r= 0.104, P= 0.017), and a similar though less significant pattern was evident for northern pike (r= 0.035, P= 0.08) and shorthead redhorses (r= 0.065, P= 0.096). Capture frequency was correlated with fork-length for all three species (r= 0.079, P= 0.096 for walleye; r= 0.132, P= 0.0001 for northern pike; and r= 0.155, P= 0.0001 for shorthead redhorses). The only significant correlation

observed for tumors was with fork-length of walleye (r= 0.184, P= 0.0001).

In general, ectoparasites were found predominantly on inshore fishes (Fig.10). All but one of 29 infected fish (including those with tumors) were gill netted in areas that intersected the shoreline. Infected fish were caught at a depth of 1.70±0.56 m (range= 1.0-3.2 m), but all major deviations toward deeper water were due to *P. montifera* or blackspot infected fish (Fig.10). When these were removed the mean depth at capture for infected fish dropped to 1.54±0.40 m (range= 1.0-2.0 m). It was estimated that the edge of the lake to a depth of 1.5 m (Fig.10), comprised approximately 14% of the total surface area, and approximately 3% of the total water volume of Dauphin Lake.

Samples of age-0 fish and minnows (Table 3) revealed patterns similar to those found for larger fishes. *A. appendiculosus* was found only on species caught in shallow water (age-0 cisco and emerald shiners, *Notropis atherinoides*) or species that rest on or near the substrate (troutperch, *Percopsis omiscomaycus*).

The proportion of tagged fish recaptured during the study period was; walleye 17%, northern pike 11%, yellow perch 3%, white suckers 9%, shorthead redhorses 11%, quillback 2%, other species 0%. Overall, 12% (N=391) of the tagged fishes were recaptured but only two recaptured fish had recruited new ectoparasites; one shorthead redhorse and one northern pike were captured 376 and 384 days after initial release,

respectively. Both had recruited a single P. montifera and both were caught in the area of initial release even though tagged fish were caught throughout the lake as early as the first winter after tagging.

Estimates of ectoparasite prevalence based on samples collected using pound nets were 4 to 12 times higher than similar estimates based on the gill net samples (Table 5). Although approximately half of all nets (46-56%) and all fish caught annually by gill nets (47-59%) were from shallow areas adjacent to the shoreline (Fig. 10), only 16% of fishes collected by gill nets in the fall (N=7,168) were captured in water ≤1.5 m deep. Since a large proportion (84%) of the fishes caught in fall were from deeper water (>1.5 m), and since these were rarely infected, estimates of prevalence for the fall gill net samples were re-calculated after excluding deep water harvests. Estimates of prevalence increased 1.5 to 5 times and approached those for the pound nets (Table 5). There was no significant difference between estimates of tumor prevalence (Table 5) calculated using pound net samples (prevalence= 0.54%) and adjusted gill net samples (prevalence= 0.77%) (Z-test).

Table 5. Estimates for prevalence (%) based on samples collected using pound nets, gill nets, and gill nets after excluding fishes caught in water > 1.5 m deep (gill nets*).

	ectoparasite			
	M. moorei	P. montifera	neascus	lymphocystis
pound nets	0.88	1.73	0.62	0.54
gill nets[a]	0.06	0.06	0.03	0.13
gill nets*,[b]	0.26	0.09	0.09	0.77

[a]fall samples only; N=7,108 fish.

[b]fall samples only; N=1,168 fish.

DISCUSSION

Cystobranchus verilli and Myzobdella moorei are true fish leeches (F. Piscicolidae) that move about on the substrate in an 'inchworm' fashion when not attached to a host (Sawyer, 1972). These types of leeches are 'opportunistic' in the sense that they rely heavily on the activities of the fish hosts for transmission. It is not surprising that Piscicolids were found only on fish caught in shallow water since the rate of fish-ectoparasite contact is probably maximized there due to; a higher surface area to volume ratio, less turbid water, and congregations of fish (Scott & Crossman, 1973) that increases their density. Argulus appendiculosis is another oportunistic ectoparasite that 'sits and waits' (Poulin and Fitzgerald, 1988) for a host and, like the Piscicolids, was found only on fish caught in shallow water. Furthermore, large numbers of free-swimming Argulus sp. were found while seining near the mouth of the Turtle River. Placobdella montifera (F. Glossiphoniidae) on the other hand, has been reported from a variety of invertebrate and vertebrate hosts; members of this genus are known to be active swimmers (Sawyer, 1972) and these were the first metazoan parasites known to exhibit negative thermotaxis (Pettigrew and Fried, 1973). Placobdella montifera might prefer deeper, cooler water but it is possible that these leeches also use their swimming ability to find areas where hosts are congregated or to actively

search for hosts. Negative thermotaxis and an 'active' type of transmission could explain the presence of P. montifera on fishes collected from deep water where piscicolids and Argulus sp. were never found. If active transmission is more successful than opportunistic transmission, then the increased rate of recruitment, albeit quite low, for P. montifera is not surprising.

The differences in distribution for piscicolids and glossiphoniids are real. They are not due to differential movements of fish since fish species that served as hosts for P. montifera were captured throughout the lake. They are not the result of increased ability of P. montifera to remain attached to captured fish since P. montifera was not found on fish caught in gill nets set near shore.

There is no doubt that the use of pound nets and mark-recapture techniques provided an accurate picture of ectoparasite community structure and recruitment in Dauphin Lake, since the gear was not size selective for fish. However, I caution that even with these methods the numbers of some fish species (eg. cisco) may be underestimated while others (eg. quillback) are overestimated. Samples of fish collected with gill nets can give good estimates of ectoparasite intensity, provided that mean intensity for ectoparasites approaches unity. Since the latter is true for Dauphin Lake, the loss of ectoparasites on capture was an 'all-or-nothing' phenomenon and, although these reduced the estimate for prevalence, they did not affect estimates of

mean intensity. Estimates of prevalence from gill net samples were improved by taking into account the distribution of infected fish but were still affected by ectoparasite loss. This is best shown by the estimate for prevalence of tumors based on pound net samples and gill net samples. Since tumors are not lost on capture, the prevalence estimated from gill net samples agreed with that based on pound net samples, after making allowance for deep water harvests.

Based on the evidence presented here several generalizations can be made regarding the fish-ectoparasite assemblage of Dauphin Lake. First, ectoparasite numbers are determined primarily by the physical characteristics, habits, relative numbers, and distribution of each fish host present in the community. Second, differences in the distribution of ectoparasites exist and these can be attributed to the predominant method (opportunistic versus active) used for transmission. Third, the recruitment of ectoparasites is extremely slow but appears to be more rapid for actively transmitted types. Large scale heterogeneity in the spatial distribution of ectoparasites has important implications for the design of sampling protocols aimed at estimating ectoparasite numbers. The periphery of Dauphin Lake (from the shoreline to a depth of 1.5 m) comprises only 14% of the surface area and 3% of the total volume of the lake, and only 16% of all gill netted fish were captured there. Nevertheless, the evidence indicates that almost all

ectoparasite transmission from the environment to the fish hosts occurs in this narrow zone. Finally, although live-trapping is the preferred sampling procedure for estimating ectoparasite numbers, samples of fish collected with gill nets can give accurate estimates of ectoparasite intensity provided that the levels of infestation are low, but tends to underestimate prevalence. The latter can be improved by taking into account the distribution of infected hosts.

CHAPTER 3: *NEOECHINORHYNCHUS CARPIODI* IN QUILLBACK, *CARPIODES CYPRINUS*: EVIDENCE FOR REGULATION IN A POPULATION OF HOST-SPECIFIC PARASITES.

INTRODUCTION

To this point I have focussed on the organization of the fish-parasite community in Dauphin Lake. The evidence presented so far (Chapters 1,2) suggests that stochastic mechanisms play a large role in determining the final structure of the component community, and that similar mechanisms influence the organization of metacommunities. Density-dependent and density-independent mechanisms will ultimately affect community structure, but regulation is really a population phenomenon; density-dependent mechanisms operating on populations impart stability, while density-independent mechanisms are associated with unstable populations.

Bradley (1974) and Kennedy (1977) identified immune or other responses by the host as regulatory mechanisms that could operate on parasite populations. Since the magnitude of these responses usually depends on parasite numbers, these mechanisms are density-dependent. However, since density-dependent mechanisms may be inoperative at low population densities, these mechanisms may be difficult to identify in small populations and the existence of density-dependent mechanisms by itself does not imply that these are operating to regulate the population. Regulating factors are therefore best observed in high density infections. In addition, it is best to look at infracommunities with low diversity to minimize any confounding effects due to

simultaneous occupation of a host by competing parasite species. Fortunately, there exists in Dauphin Lake one host-parasite system that fills these criteria. In Dauphin Lake the acanthocephalan Neoechinorhynchus carpiodi infects only quillback, Carpiodes cyprinus. The parasite is extremely site-specific, occurs in almost all quillback and infrapopulations can be very large. In addition, infection of fish elicits the formation of large nodules at the sites of attachment and the production of specific serum antibodies.

A few authors (Halvorsen and Williams, 1968; Williams and Halvorsen, 1971; Simmons and Laurie, 1972; Smith, 1973) have provided evidence that infrapopulations of parasites in fish are regulated, but no real indication of the mechanisms responsible has been observed. In the next two chapters I discuss in detail the pathology and host immune responses, respectively, associated with N. carpiodi in quillback, and whether these responses might be operating to regulate the numbers of this parasite.

CHAPTER 3-1: INTESTINAL PATHOLOGY AND SITE SPECIFICITY OF THE ACANTHOCEPHALAN *NEOECHINORHYNCHUS CARPIODI* DECHTIAR, 1968 IN QUILLBACK, *CARPIODES CYPRINUS* (LESUEUR).

INTRODUCTION

The distribution of parasites within the fish intestine is variable and most do not markedly alter the intestine at the point of attachment. There are, however, examples of the induction of severe pathology by parasites (Prakash and Adams, 1960; Chaicharn and Bullock, 1967; Mackiewicz et al., 1972; McDonough and Gleason, 1981; Shostak, 1986) in which a granuloma or lesion is produced for the purpose of walling off the effect of the insult or causing a sloughing of the worms.

The generation of lesions or large nodules in response to intestinal parasites is common in the Catostomidae, but it is not clear what effects these nodules have on a particular species of parasite. Examination of fishes collected from Dauphin Lake revealed extensive nodules containing the acanthocephalan Neoechinorhynchus carpiodi Dechtiar, 1968, in the intestine of quillback (Carpiodes cyprinus). Stewart-Hay (1951) reported 'an unknown species of Neoechinorhynchus' from quillback he collected from Dauphin Lake, but failed to recognize that they represented a new species. This parasite and the nodules they produce in quillback were briefly described 17 years later by Dechtiar (1968). Little is known about the pathology induced by this parasite. The appearance and persistence of nodules in the gut of quillback throughout the year suggests a permanency not usually associated with intestinal lesions of fish.

While there are numerous references in the literature to the pathogenicity of infection by adult parasites in fish, most of these have dealt only with the histopathology of infection. Few of these have considered the implications of parasite-induced pathology as it relates to the survival of parasites or to infrapopulation regulation. The objectives of this chapter were to determine the distribution of N. carpiodi in the intestine of quillback and to investigate the relationship between pathology (size, structure, and number of nodules) and the size of the N. carpiodi infrapopulation.

MATERIALS AND METHODS

Quillback were collected from May to September and in December, 1985. Quillback of a narrow size range (395-510 mm fork length) were sampled in order to minimize any effects due to size and age of hosts. Fish were killed by a blow to the head and their fork length, round weight, sex, and maturity was noted. The gastrointestinal tracts (from the oesophageal-intestinal junction to the anus) of all fish were removed, and within 5 min of death of the fish these were divided into 10 equal sections using the biased-grid technique (Brambell, 1965). Each section of the intestine (section 1 was nearest the oesophagus) was slit longitudinally, placed in water, and examined for the presence of helminths. The location and number of any nodules was recorded. For histological study, pieces of intestine (about 25 cm^2) with nodules were pinned flat on cardboard, fixed (24 h) in Bouin's fixative (Humason, 1979), washed repeatedly, and stored in 70% ethanol. The intestinal contents were examined later for the presence of additional parasites.

All nodules found were ranked according to their size (largest to smallest) and position (anteriormost to posteriormost), and the number and position of each nodule cluster was recorded. Ninety-two nodules containing N. carpiodi were collected from quillback. A cluster was a group of nodules situated within a localized region of the

gut. Two of 73 quillback had a pair of clusters. Each nodule was measured to determine maximum width (MW), length (L), and depth (D), and the normal gut thickness (T) was measured 2 cm from the edge of the nearest nodule. The external volume of each nodule was determined using the formula for one-half of an ellipsoid, $1/12\pi LW^2$, where $W=[MW+(D-T)]/2$. The internal volume of each nodule was estimated by injection of known volumes of water into Bouin's-fixed nodules.

All N carpiodi were removed from each nodule and total numbers recorded. All parasites found were fixed, stored, and stained according to the methods outlined previously (Chapter 1). Acanthocephalans were classed as males, non-gravid females (presence of ovaries or ovarian balls but no shelled acanthors), gravid females (shelled acanthors present), and immature worms (the sex of these was noted). The sex of approximately 10% of the worms could not be determined due to improper clearing; the maturity of these was estimated based on worm size.

Gross pathology was described from fresh specimens. Some nodules with worms *in situ* were fixed in Bouin's fixative, embedded in paraffin, and serially sectioned at 5-10μ. Some of these sections were stained with Gill's haematoxylin and eosin (H&E) for general histology, some with picro-Sirius red F3BA for collagen (Puchtler et al., 1973), and others with fast-green for mucoid secretions. Identification of cell types and intestinal tissue layers was based on the

terminology and descriptions of Weisel (1962), Chaicharn and Bullock (1967), and Roberts (1978).

Throughout the text of this chapter the term 'intensity' is used as in Margolis et al. (1982). For consistency with previous chapters, the term 'density-dependent' is used rather than 'intensity-dependent' (Margolis et al., 1982).

Statistical analysis

A 3 x 3 test of independence using χ^2 (Sokal and Rohlf, 1969) was used to analyze the relationship between nodule type and position. Students' t-tests were used to analyze the distribution of N. carpiodi among nodules and along the intestines of quillback. Regression equations for the relationships between external nodule volume and worm numbers, size of clusters and worm intensity, and distribution of worms and external nodule volume were derived using simple linear regression. Data on nodule volume and worm numbers were log-transformed prior to analysis. Nodules were placed in three categories on the basis of pathology type, and stepwise discriminant analysis (Bennet and Bowers, 1976) was used to choose independent variables that best predicted the category of pathology assigned to a nodule. A probability of $P<0.05$ was considered significant in all tests.

RESULTS

The intensity and distribution of N. carpiodi infections in quillback were not affected by host sex, age, size, or the season of capture. There were no seasonal differences in the distribution of proportions of male and female worms, and gravid and non-gravid worms (t-tests).

Distribution along the intestine.

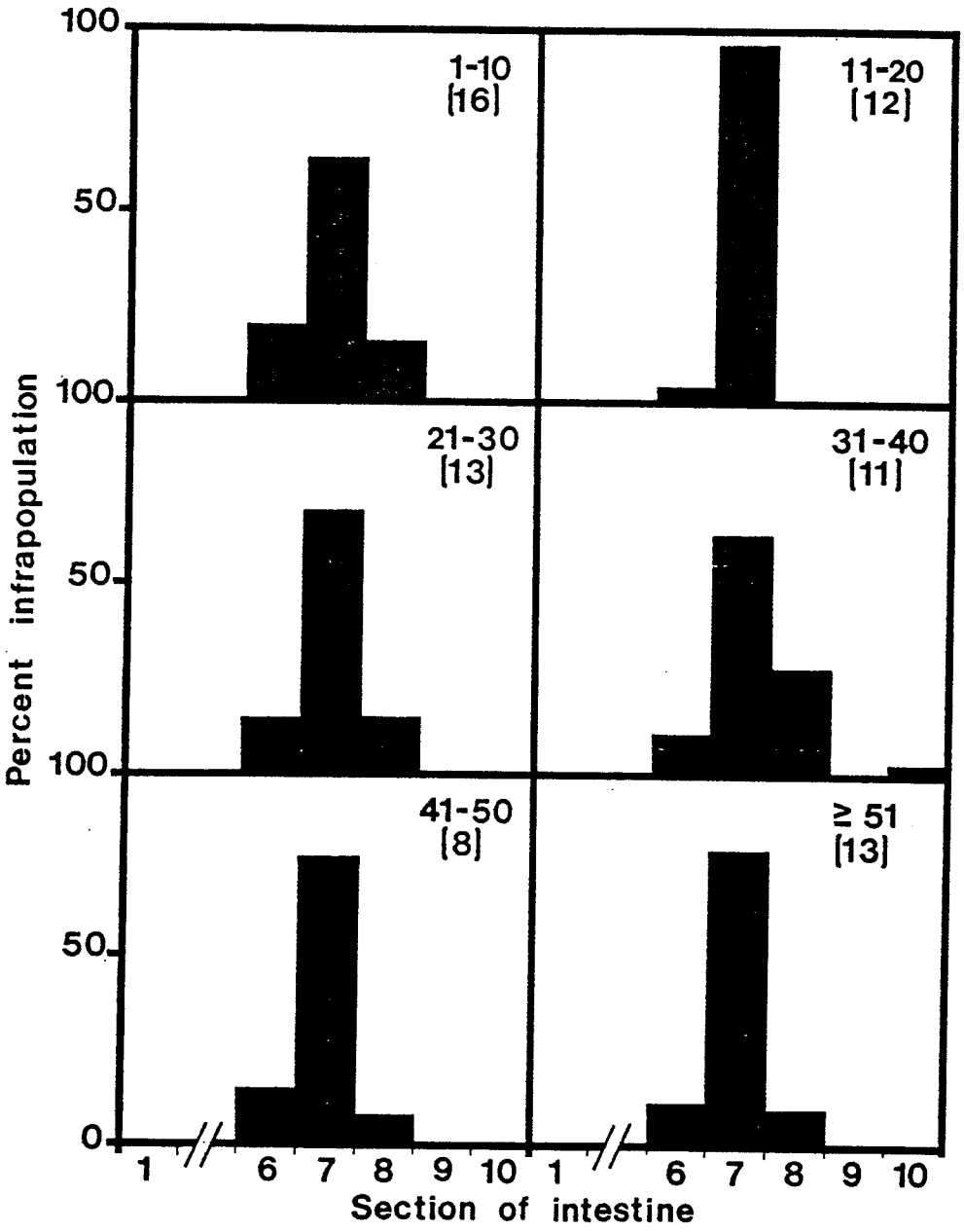
Over 98% of the N. carpiodi recovered from 73 infected quillback were attached in sections 6-8 of the intestine, with section 7 being the most frequently parasitized region regardless of intensity (Fig. 11). The distribution of worms by sex, and gravid and non-gravid females as a proportion of the total number of worms is summarized in Table 6. Female N. carpiodi outnumbered males in all regions where they occurred except section 7, where the number of males did not differ significantly from the number of females (t-test). Although section 7 harbored the fewest females, the proportion of gravid females was significantly higher than the proportion of non-gravid females in this region ($P<0.05$, t-test). Furthermore, section 7 was the most frequently occupied region (Fig. 11). The number of males did not differ among sections of intestine, nor did the number of females (ANOVAs).

Table 6. Distribution of worms by sex and reproductive state of females (percent infrapopulation; mean±standard deviation) among sections and nodules. The results of Student's *t*-tests are given.

	sex			reproductive state		
	male	female	*t*-test	gravid	non-gravid	*t*-test
section of intestine						
6	31.7±23.9 (8)[b]	68.3±23.9 (8)	$P<0.005$	36.9±30.7 (7)	63.1±30.7 (7)	ns[a]
7	52.1±24.7 (31)	47.9±24.7 (31)	ns	57.6±32.6 (27)	42.4±32.6 (27)	$P<0.05$
8	37.8±6.3 (2)	62.2±6.3 (2)	$P<0.05$	29.6±24.2 (2)	70.4±24.2 (2)	ns
nodule volume[c]						
0-0.079	45.2±39 (13)	54.8±39 (13)	ns	82.3±30.3 (7)	17.7±30.3 (7)	$P<0.005$
0.08-0.16	42.1±26.1 (7)	57.9±26.1 (7)	ns	53.7±20.4 (5)	46.3±20.4 (5)	ns
0.16-0.24	47±12 (9)	53±12 (9)	ns	52±19.6 (9)	48±19.6 (9)	ns
≥0.24	38.9±14.8 (10)	61.1±14.8 (11)	ns	42.1±18.9 (8)	57.9±18.9 (8)	ns

[a] ns; not significant.
[b] N; sample size.
[c] cm^3.

Figure 11. The distribution of N. carpiodi along the small intestine of quillback. Numbers in upper right corners indicate range of intensities (number of fish examined).


100
50
100
50
100
50
0
Percent infrapopulation
1-10
[16]
11-20
[12]
21-30
[13]
31-40
[11]
41-50
[8]
≥ 51
[13]
1
6
7
8
9
10
1
6
7
8
9
10
Section of intestine

These results suggested that worm distribution was probably related to the size and number of nodules and associated pathology at the sites of parasite attachment. Therefore, a description of this pathology is given prior to an assesment of the distribution of worms among nodules.

Gross pathology

The pathology associated with N. carpiodi was apparent as an extreme localized swelling (Fig. 12) when viewed from the serosal surface. Occasionally, a large polypoid outgrowth projected from the swelling into the body cavity, and often a small (2-5 mm) whitish pustule was centered on this outgrowth. When the gut was opened in the area of swelling, several nodules grouped together were observed on the lumenal side of the intestine (Figs. 13, 14). Nodules varying in number (1-8 nodules per cluster) and size (23.5 x 15.2 x 7 mm maximum) harbored 2-106 N.carpiodi (Fig. 13), and were firm to the touch and creamy to deep pink in color. The appearance of the lumenal surface varied with the number of N. carpiodi attached. Sites with few worms were characterized by small ellipsoid nodules. In sites where worms were more densely attached, nodules were larger and more pendulous (Figs. 13, 14), with one or more narrow channels through which the acanthocephalan trunks protruded. Villi over the lumenal surface of the nodules appeared intact on gross examination. No nodules or other signs of pathology were observed along the intestine when N. carpiodi

was absent even though other parasite species were sometimes present.

Histopathology

The gut of quillback has 5 layers. The mucosa (Fig. 15) consists of columnar epithelial cells, goblet cells, and small numbers of rodlet cells and granulocytes. The lamina propria is thick and granulocytes are present between the epithelial cells of the mucosa and within the lamina propria. A thick (acellular) stratum compactum is present (Fig. 15). The muscularis (Fig. 15) has an inner circular muscle layer ensheathed in a thin outer longitudinal layer. A unicellular serosa completely surrounds the intestine. All five layers are observed adjacent to the sites of N. carpiodi attachment (within 0.5 cm of the outer margin of a nodule) but within the nodule or at the point of attachment of the acanthocephalans some layers are indistinct or absent (Fig. 16). The stratum compactum acts as a barrier that prevents the worms from breaching the intestine (Fig. 15).

The pathology observed was arbitrarily classified into three categories related to the number of worms, the depth of penetration of proboscides, and the size of nodules (Table 7). Type I pathology (Fig. 18) was associated with small nodules (< 0.3 cm^3) harboring few N. carpiodi and where the proboscides of the worms were loosely attached.

Table 7. Relationship between volume of nodules and pathology classifications for clusters of 1-4 nodules.

	nodule volume[a] (cm^3)			
N[b]	1	2	3	4
4 (5)	0.234±0.168[c] 0.104-0.497[d] (II-III)[e]	0.095±0.07 0.044-0.218 (II-III)	0.028±0.044 0.001-0.105 (I-II)	0.012±0.009 0.001-0.024 (I)
3 (5)	0.139±0.06 0.091-0.224 (I-III)	0.032±0.032 0.003-0.073 (I-II)	0.027±0.025 0.006-0.058 (I)	
2 (13)	0.227±0.182 0.006-0.67 (II-III)	0.14±0.133 0.035-0.49 (I-II)		
1 (22)	0.434±0.635 0.024±2.33 (I-III)			

[a] 1, largest nodule in cluster; 4, smallest nodule in cluster.

[b] number of nodules per cluster, (sample size)

[c,d,e] mean±standard deviation, range, (pathology class).

Type II pathology was associated with medium-sized nodules (0.3-0.6 cm^3) which protruded into the lumen of the intestine. These had larger numbers of worms and the proboscides had penetrated to the stratum compactum, but not through it (Fig. 15). An epithelium-lined channel was present in most of these nodules (Fig. 17), the mucosal layers being disrupted in the vicinity of proboscis attachment only (Fig. 19). Hyperplasia was evident in the lamina propria (Fig. 15, 19), accompanied by an increase in vascularization. The lumenal surface of nodules showing Type II pathology had an intact epithelium but goblet cells (Fig. 15) were more numerous than in the normal epithelium. Occasionally host tissue showed cellular compression (Fig. 15) where the trunk of N. carpiodi contacted it, and the number of goblet cells was low (Fig.16), and more granulocytes were present in the epithelium. Collagen deposition was restricted to regions immediately adjacent to the proboscides and necks of the worms.

Type III pathology (Figs. 16, 19) occurred when the point of attachment of gravid females was below the stratum compactum and was characterized by large nodules (ca. >0.6 cm^3). Collagen deposits occurred in the lamina propria and throughout the muscularis (Figs. 16, 19). Well-defined collagenous pads formed around the proboscides (Fig. 16) and hyperplasia was extensive (Figs. 16-19). Large vascularized outgrowths (Fig. 12) were associated with the serosal surface of Type III nodules, and large blood vessels leading

to the serosa were sometimes present (Fig. 16). In general, Type III pathology was associated with large numbers of densely packed worms (Figs. 13, 19) where the host tissue was disrupted in the region of attachment, but was intact as the worms reached the channel lined with epithelial cells (Fig. 17). Other characteristics were like those described for Type II pathology.

Distribution among nodules

The number of nodules increased linearly with intensity ($\underline{r}^2$=0.31, $\underline{P}>\underline{F}$=0.0001), and external nodule volume increased with worm numbers ($\underline{r}^2$=0.68, $\underline{P}>\underline{F}$=0.001). Quillback (N=71) with a single cluster of nodules had significantly fewer nodules (26.4±26.4 worms per fish) than those (N=2) with two clusters (126±14.1; $\underline{P}$<0.001, $\underline{t}$-test). When two clusters were present the anteriormost cluster occupied a significantly larger total volume ($\underline{P}$<0.05, $\underline{t}$-test). Large nodules were generally situated anterior to small nodules but this pattern varied. Seventy-five percent of all nodules found in the most anterior position of a cluster were Type III, 56% of the nodules immediately posterior to these were Type II, and 61% of the most posterior nodules were Type I (Fig. 20). The smallest nodules had proportionately more gravid females (Table 6), but females in Type I nodules were always smaller than females in Type III nodules.

Figures 12-19. N. carpiodi in the intestine of quillback. **12.** External view of infected intestine. Note the protrusion (P) and expansion of the intestine compared to the normal gut (NG). Bar=5 mm. **13.** N. carpiodi *in situ*. A cluster of 3 nodules (N) is visible. Note the largest nodule containing most of the worms (W). Bar=5 mm. **14.** Lumenal view of nodules (worms removed). Bar=11.5 mm. **15.** Cross-section showing Type II pathology. The worm lies deep within the lamina propria (LP) but the proboscis (PR) has not penetrated the stratum compactum (SC). Goblet cells (GC) are numerous in the epithelium (E), and the LP is expanded (stars) around the worm. H&E. Bar=0.15 mm. **16.** Type III nodule. All tissue layers are disrupted or destroyed. Note the large collagenous pads (CP), breakdown of the SC (black arrows), and blood vessels (BV; white arrow) at the sites of PR attachment. Picro-Sirius red. Bar=0.6 mm. **17.** Cross section through a channel (C) containing worms. Note the absence of goblet cells and the lack of an interface layer between worms and host tissue. There is some damage (arrows) but the gut is largely intact. H&E. Bar=0.18 mm. **18.** Cross-section of Type I nodule. The proboscis is attached in the LC. Cellular compression (c) is restricted to cells adjacent to the proboscis. H&E. Bar=0.06 mm. **19.** Cross-section of Type II nodule. Collagen deposition (dark material) and tissue damage is extensive. Picro-Sirius red. Bar=0.8 mm. CM, circular muscle; LM, longitudinal muscle; S, serosa.

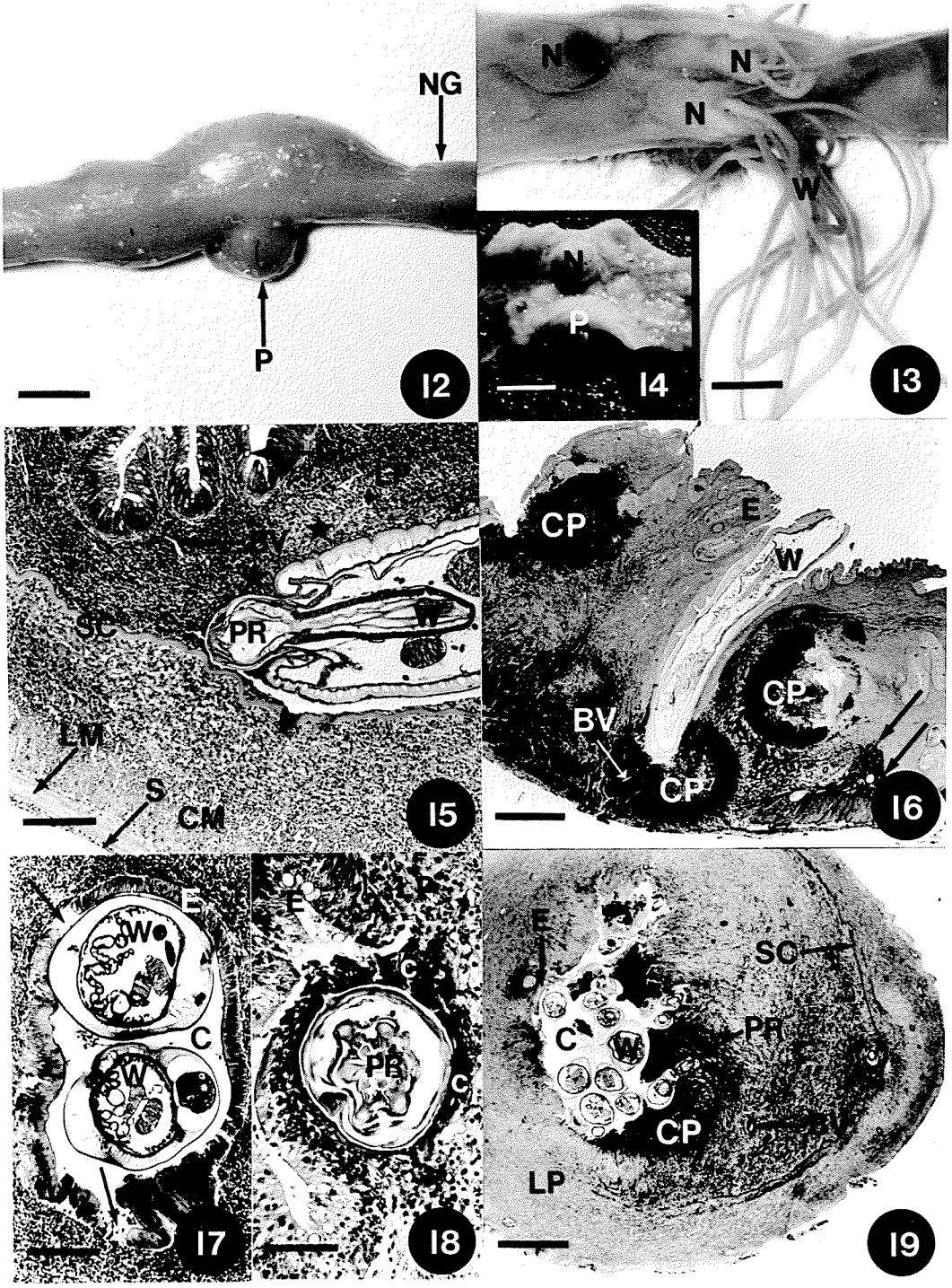
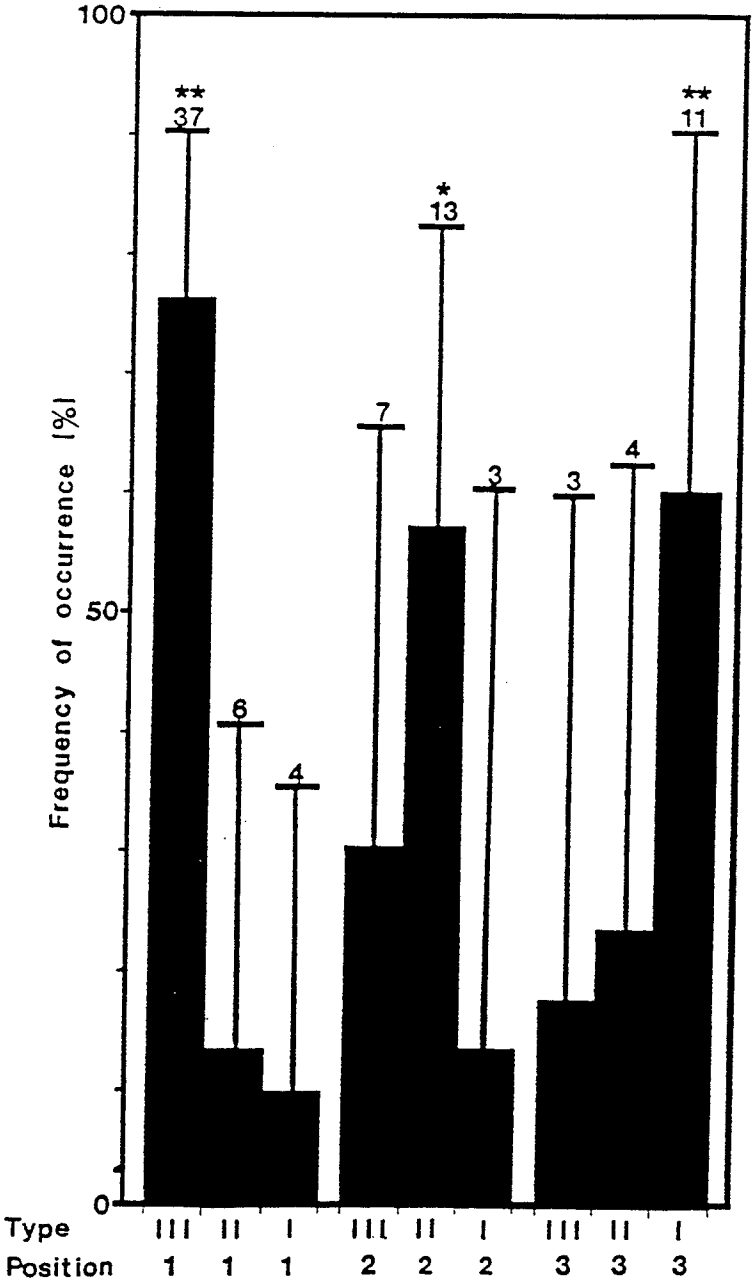

NG
P
12
N
N
N
W
13
N
P
14
E
SC
PR
W
LM
S
CM
15
CP
E
W
CP
BV
CP
16
E
W
C
W
17
E
C
PR
C
18
E
SC
C
W
PR
CP
LP
19

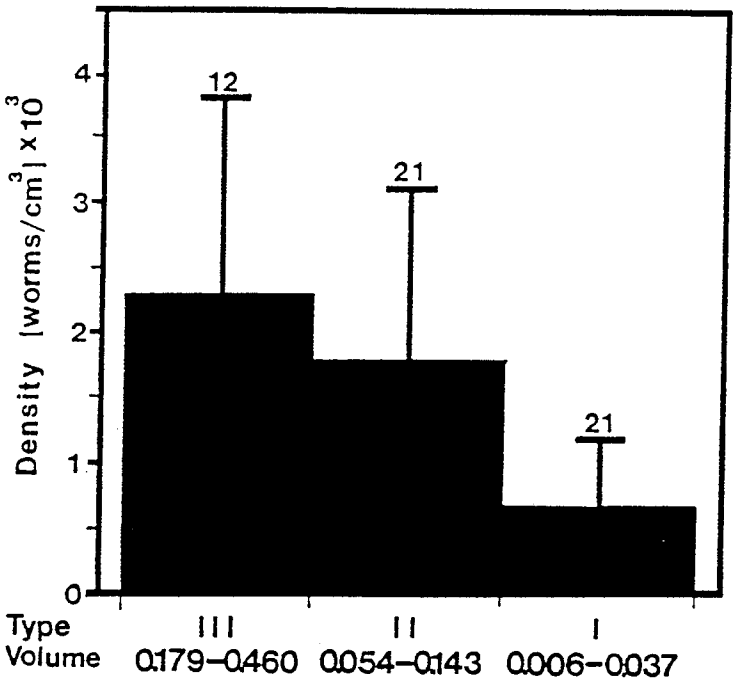
Figure 20. Distribution of nodule types within clusters. Position 1 is most anterior; 3 is most posterior. Columns and bars represent mean frequencies and upper 95% confidence limits, respectively. Results of Students' *t*-tests for mean frequencies are indicated as: *, $P<0.05$; **, $P<0.005$. Sample sizes are also given.


100
50
0
Frequency of occurrence [%]
**
37
6
4
7
*
13
3
3
4
**
11
Type
III II I III II I III II I
Position
1 1 1 2 2 2 3 3 3

Sixty-six percent of the worms in Type III nodules were immature; significantly greater than the proportions in Type II or Type I nodules (51.2% and 0%, respectively: $P<0.005$, t-test). Type III nodules occurring singly had a higher proportion of immature worms (69.7±20.8%) than Type III nodules occurring within a cluster (51.6±16.1%) and regardless of cluster size, Type III nodules maintained a higher proportion of immatures than any other nodule. The proportion of immatures was higher in more anteriorly situated nodules. This is best shown by Type II nodules which had a significantly higher proportion of immatures (75±35.4%) at position 1 (most anterior) than at position 2 (43.3±35.7%: $P<0.005$, t-test).

The relationship between external nodule volume and worm density is presented in Figure 21. The classification of nodules was based on external volume (corresponding to the 95% confidence intervals for Type I, II, and III nodules), and a stepwise discriminant analysis based on nine variables (number of females per nodule, gravid females per nodule, mature males per nodule, worms per nodule, worms per fish, number of nodules, position of nodules, section of intestine, and normal gut thickness) was performed. The stepwise discriminant procedure chose the number of gravid females per nodule and normal gut thickness as the best variables for discriminating among nodule types. The discriminant function correctly classified 87% of the nodules based on knowledge of these two variables only.

Figure 21. Relationship between worm density and type of nodule. Volume ranges given are 95% confidence limits for external volume (cm^3). Vertical lines and bars indicate means and standard deviations, respectively. Sample sizes are indicated above each bar.


Density [worms/cm^3] x10^3
4
3
2
1
0
12
21
21
Type
Volume
III
0.179–0.460
II
0.054–0.143
I
0.006–0.037

Nodule volume was more strongly correlated to the number of gravid females present ($\underline{r}^2$=0.55) than to normal gut thickness ($\underline{r}^2$=0.02).

DISCUSSION

Parasite-induced pathology

The distribution of N. carpiodi in the intestine of quillback is more restricted than that reported from other studies on acanthocephalans in fish (Hine and Kennedy, 1974; McDonough and Gleason, 1981). The pronounced pathology evident in all infections is an important aspect of N. carpiodi distribution within quillback. This pathology differs somewhat from that described for other host-parasite systems. Although Dechtiar (1968) noted inflammation of surrounding tissues and a prominent connective tissue nodule around the penetrating proboscides of N. carpiodi, he made no references to differences in the degree of pathology.

Pathology corresponding to the Type I classification has been described for Leptorhynchus thecatus and N. cylindratus in bass, Micropterus salmoides (Venard and Warfel, 1953; Esch and Huffines, 1973, respectively), Acanthocephalus jacksonii in trout, Salmonidae (Bullock, 1963), A. dirus in darters, Etheostoma caeruleum (McDonough and Gleason, 1981), and N. cristatus and N. prolixoides in white suckers, Catostomis commersoni (Chaicharn and Bullock, 1967). The Type II response is similar to the response of the carp (Cyprinus carpio) intestine (Mackiewicz et al., 1972) to the presence of the caryophyllid cestode Biacetabulum biloculoides but differs in that nodules induced by B. biloculoides are smaller, harboring only 1-6 worms, and

debris and necrotic tissue surround the scoleces. The Type III classification is similar to pathology described by Prakash and Adams (1960) for Echinorhynchus lageniformis in flounder, Platicthys stellatus, where female worms penetrated the stratum compactum and induced the formation of a nodular outgrowth on the lumenal surface of the intestine. Octospinifer macilentus and Pomphorhynchus spp. elicit similar responses (Chaicharn and Bullock, 1967; McDonough and Gleason, 1981).

There are three unusual features associated with N. carpiodi infections. First, there is no mucoid layer between the parasite and the host tissues. A mucoid layer has been reported by Bullock (1963) and also by Mackiewicz et al. (1972), who suggest that it may serve as a nutrient source for the worms. Although such an interface was absent, numerous goblet cells were observed on the lumenal surface and in the epithelium of the nodules where it was not in direct contact with the worms. Second, the formation of nodules in the absence of deep proboscis penetration is atypical as Chaicharn and Bullock (1967) reported that O. macilentus did not induce nodule formation unless their proboscides penetrated the stratum compactum. Third, the epithelium of the host was intact even when pathology was most severe, except immediately adjacent to the proboscides. Attachment by other acanthocephalans is often associated with total destruction of the epithelium (Bullock, 1963).

Based on the relationship between pathology and the characteristics of the worms attached, it is likely that the following sequence of events describes the dynamics of nodule formation. Recent infections characteristically have few worms attached primarily in the epithelium, but occasionally in the lamina propria. Few gravid females (0-5) are present and the pathology fits the description of a Type I nodule. As these attached worms mature, the number of gravid females increases and penetration is deeper into the lamina propria. Additional worms are recruited and Type II pathology occurs. With the passage of time, the number of gravid worms increases to 8-14, and their proboscides penetrate to and then through the stratum compactum, resulting in Type III pathology. Concurrently, collagenous pads are laid down around the deepest proboscides and a polypoid outgrowth forms beneath the serosa, probably due to the extensive vascularization that occurs. These nodules appear to be long-lived because no nodules without worms, and no scars were noted. Nor was there any evidence of seasonal changes in the number of nodules or worms. Furthermore, the extensive vascularization, lack of damage to the intestinal lumenal surface or to the channels of the nodules, and necrosis limited to the region adjacent to the sites of proboscides attachment suggests that these nodules are a relatively permanent part of the quillback intestine.

Pathology versus infrapopulation structure.

Since these nodules have some degree of permanency, albeit induced by the presence of the parasites, and since there appears to be some localization of the N. carpiodi infrapopulation within a preferred region of the intestine, perhaps the size and numbers of nodules affect the carrying capacity of the intestinal environment. It is likely that the quality of this microhabitat affects the biotic potential of the parasite, as measured by the number of gravid females. I attempted to answer these questions by determining (*i*) if the position of the largest nodule is always most anterior and therefore would be expected to acquire more worms, (*ii*) if the position of the largest nodule affects the proportion of mature and immature worms, (*iii*) if the presence of more nodules increased the biotic potential, particularly the number of gravid female worms, and (iv) whether position and cluster size affected recruitment of worms by Type III nodules.

The most anterior nodules were usually Type III and acquired more worms. However, the largest proportion of worms in these large nodules was not simply the result of a more anterior position in the gut because, regardless of their position, Type III nodules had the highest proportion of immatures and harbored more gravid females and mature males. Furthermore, the number of nodules and their size was directly correlated with higher numbers of total worms, including gravid females. If carrying capacity is increased

by increasing the number of nodules, why is recruitment greatest into the largest nodule? Nodule position no doubt plays a role but the nature of the nodules as a unique microhabitat appears to be more important. Perhaps recently acquired cystacanths of N. carpiodi actively select existing Type III nodules because they provide the best attachment sites, have a better or more plentiful nutrient source, or they are attracted to a species-aggregation or sex pheromone. Whatever the cues for increased recruitment into Type III nodules, it is certainly not related entirely to an anterior position, nor is it correlated with distances between Type III nodules and Types II and I.

These observations are interesting in that the parasite-induced pathology, in the form of a well-vascularized nodule, creates a long-lasting and stable microhabitat. This host-parasite association may minimize leakage of body fluids from the host and the possibility of secondary bacterial infections because of the limited area of the lesions exposed. Furthermore, the most severe pathology (Type III) appears to be the best microhabitat for the parasite, as it attracts proportionally more recruits and harbors more gravid females regardless of its position relative to other nodules in the gut. Whether or not worm densities are high enough for parasite-induced pathology or host immunity to exert a regulating effect on infrapopulations of N. carpiodi in quillback, will be dealt with further in the next chapter.

CHAPTER 3-2: INTESTINAL LEAKAGE AND PRECIPITATING ANTIBODIES IN THE SERUM OF QUILLBACK, *CARPIODES CYPRINUS* (LESUEUR), INFECTED WITH *NEOECHINORHYNCHUS CARPIODI* DECHTIAR, 1968 (ACANTHOCEPHALA:NEOECHINORHYNCHIDAE)

INTRODUCTION

The pathology associated with infections of the acanthocephalan Neoechinorhynchus carpiodi in quillback, Carpiodes cyprinus has already been described (Chapter 3-1). Briefly, N. carpiodi elicits nodule formation at the sites of proboscis attachment and nodule numbers, size, and pathology are related to the number of worms and to the depth of proboscis penetration. When gravid female N. carpiodi penetrate beyond the stratum compactum, the resulting breakdown of the mucosal barrier and extensive vascularization may cause the intestine to be more leaky in the region of the nodules, thereby enhancing fluid contact between parasite and host and the possibility of developing an immune response by the host. The persistence of N. carpiodi and the nodules further suggests that there might be prolonged stimulation of the quillback immune system.

Little information is available on the immune response of fish to intestinal metazoan parasites (Harris, 1972; Kennedy and Walker, 1969; McArthur, 1978). Precipitating antibodies to intestinal infections of Pomphorhynchus laevis and Telogaster opisthorchis were reported by Harris (1972) and McArthur (1978), but neither of these studies considered the possibility of false positives due to C-reactive protein (CRP) or alpha-migrating factor which have been identified in some fish sera (Baldo and Fletcher, 1973; Alexander, 1980). These proteins are non-specific and form

precipitation bands similar to specific antigen-antibody precipitin bands observed in Ouchterlony double-diffusion plates, and therefore must be considered if precipitin reactions in gels are to be interpreted accurately. The objectives of this study were to determine if (*i*) the pathology associated with nodules and the acanthocephalan N. carpiodi in the quillback intestine enhanced the movement of fluids into the gut lumen, and (*ii*) quillback produced precipitating antibodies to N. carpiodi, and (*iii*) how host immunity might affect parasite numbers.

MATERIALS AND METHODS

Measuring gut leakiness

Quillback were collected with live traps during August and September, 1986, and in June, 1987. Length, weight, sex, and maturity of fish were noted at their time of death.

Plasma protein concentration was measured using Evans blue (EB) as an indicator. Evans blue (2.0 ml, 1% solution, autoclaved) in phosphate buffered saline (PBS) was injected into the heart of unanesthetized fish with an 18 ga. needle. Quillback were kept in a 27 m^3 enclosure at water temperatures of 12-16°C and sacrificed 12, 15, 17, or 23 h post-injection. Fish were killed by a blow to the head. The gastrointestinal-tract (from the oesophageal-intestinal junction to the anus) was removed and placed on a biased-grid wax board (Brambell, 1965) and the entire outer surface of the gut wiped clean with a damp sponge to avoid contamination of the lumenal contents with blood. Intestines were divided into three sections; a pre-nodular section including all the intestine anterior to the sites of N. carpiodi attachment (Fig. 12), a central nodular section where all the worms and nodules were found, and a post-nodular section posterior to the nodular section. Each section was flushed with 4-8 ml of PBS and then gently squeezed to remove any lumenal contents. All helminths recovered from the intestines were enumerated, identified, and recorded.

The lumenal contents of each section were homogenized using a 10 ml ground glass tissue homogenizer and the homogenate adjusted to 12 ml with PBS. A 5 ml aliquot of the diluted homogenate was mixed with an equal volume of 10% trichloroacetic acid (TCA) to test if EB was bound to protein and not present as unbound dye. Formation of a blue precipitate indicated the absence of unbound dye (Nawa, 1979). Serum (0.075 ml) was also tested with TCA to check that EB had been successfully injected. Quillback which were improperly injected had sera that did not produce a blue precipitate and these were not used in the analyses.

Four ml of acetone (-10°C) was added to an equal volume of diluted homogenate to extract protein-bound EB and to remove turbidity (Allen, 1951). The solution was mixed and then centrifuged for 8 min at 7000 rpm in a Model CL clinical centrifuge (International Equipment Co., Needham HTS., Mass.). The optical density (OD) of the resulting supernatant was immediately measured at 620 nm using a Bausch & Lomb Spectronic 20 spectrophotometer, and compared to a PBS-acetone blank.

Blood samples (2-6 ml/fish) collected from the ventral aorta by cardiac puncture were left approximately 30 min at room temperature and then placed at 4°C and allowed to coagulate for at least 4 h. These samples were then centrifuged (2 min at 7000 rpm) and the serum collected. Serum samples (0.15 ml) were diluted 100 times with PBS before the OD was measured.

A standard curve for EB concentrations was prepared by mixing 4 ml of EB-PBS solution (serial dilutions of 10 to 4 x 10^{-5} g/ml EB) with 4 ml acetone and absorbance measured at 620 nm (Bradford, 1976). The OD recorded for each sample was transformed into corresponding measures of EB concentration (CEB) for lumenal (CEBL) or serum (CEBS) contents using the standard curve. Since the CEB values were determined from samples previously diluted with PBS and acetone, an estimate of undiluted CEB (uCEB) was made where; uCEBL = CEBL x 2, and uCEBS = CEBS x 200.

Six uninjected fish were controls and these were used to determine the levels of background absorbance at 620 nm from the lumenal contents. A correction for background absorbance was determined using the equation CEBLc = uCEBL - B; where CEBLc is the concentration of EB after adjusting for the dilution factor and background absorbance, and B is the average uCEBL value attributable to background absorbance. A further calculation was used since pre-nodular, nodular, and post-nodular sections of the gut were not always of equal length. All CEBLc values were divided by the length (cm) of the section and recorded as CEBLc/cm of intestine to permit comparison between sections of gut in the same fish and among sections from different fish.

The concentration of EB in the intestine (LI) was determined using LI = ((CEBLc/uCEBS)/cm); where LI is the ratio of the concentration of EB in lumen (after corrections) versus serum contents.

After the lumenal contents had been collected the open ends of each gut section were clamped to prevent loss of worms. These sections were kept at 4°C and examined within 24 h of host death by immersing in water and slitting longitudinally. Location and number of all N. carpiodi and/or nodules were recorded, and sex and maturity of all worms noted. Worms were removed, washed twice in PBS, and stored at -80°C. Each nodule was measured for maximum width (MW), maximum length (L), and maximum depth (D), and normal gut thickness (T) was measured 2 cm from the edge of the nearest nodule. The volume of each nodule was determined as previously discussed in Chapter 3-1 using the formula for one half of an ellipsoid.

Testing for antibody

Antigens were prepared from immature and mature male and gravid and non-gravid female N. carpiodi that had been stored at -80°C. Worms were thawed and immediately homogenized in 5 ml of PBS until microscopic examination of the suspension revealed no large particles. Homogenates were centrifuged at 17,000 rpm for 30 min at 4°C in a Beckman J2-21 centrifuge, and the supernatants pipetted into dialysis tubing (12,000-14,000 m.w. exclusion/ Spectropore, Spectrum Medical Industries Inc., Los Angeles, U. S. A.) and dialysed for 48 h against PBS (4 changes of 1 l each). Solutions containing purified antigens were collected after dialysis and centrifuged as above. Aliquots (0.5 ml) of purified

antigen were stored at -80°C. Protein determinations were made on each aliquot using the micro-method of Lowry et al. (1951).

Blood samples were collected from 61 quillback and undiluted serum was isolated and stored in 1.5 ml microcentrifuge tubes at -80°C until needed. Five uninfected quillback showing no signs of previous infection (no nodules) served as controls. Serum was tested for precipitating antibody against antigens of N. carpiodi using a modification of the Ouchterlony double-immunodiffusion technique (Ouchterlony and Nilsson, 1973). Samples were tested in gels (1% agarose: 3% polyethylene glycol-6000) made up in 0.1 M tris(hydroxymethyl)aminoethane (TRIS) buffer at pH 8.3. After diffusion the gels were rinsed (30 min) in 5% sodium citrate, washed (12 h) in 2% saline and distilled water (1 h), air dried, and stained with 1% Amido Schwartz 10B (Keleti and Lederer, 1974).

To ensure that precipitin bands were due to the presence of specific antibody and not due to CRP or alpha migrating factors 0.1 M disodium ethylenediaminetetracetic acid (EDTA) was incorporated into the gel buffer to block any non-specific precipitation reactions (Ellis, 1985). Serial dilutions of fish sera and worm antigens were tested for possible relationships between antibody titre and number of worms per fish. Antibody titre was taken as the reciprocal of the highest antigen dilution giving visible precipitation with undiluted antibody.

Statistical analysis

Regression equations were derived using simple linear and polynomial regression. To reduce variance, concentration values were *log* transformed prior to analysis. Each section of intestine was placed into one of two categories based on *log* LI values and stepwise discriminant analysis (Bennet and Bowers, 1976) was applied to choose independent variables that best predicted the assignment of intestinal sections to each category. Quillback were put into one of two classes based on the number of N. carpiodi they harbored and a standardized measure of skewness based upon the third moment around the mean (G_1) (Bennet and Bowers, 1976) was calculated to compare the frequency distributions of titres for positive sera from fish with low or high numbers of worms. A probability of $P < 0.05$ was considered significant in all tests.

RESULTS

Concentration of EB in the intestine

Sixty-one quillback were treated with EB and 60 of these were infected with N. carpiodi. The extent of normal intestinal leakiness was determined from the remaining fish whose intestine showed no evidence of previous infection by N. carpiodi. Injection of EB into the uninfected quillback showed that little protein-bound EB passed from the body to the lumen of the intestine. The concentration of EB in the normal quillback intestine (*log* LI=$^{-}$4.25) was outside the 99% confidence limits for infected quillback pre-nodular (*log* LI=$^{-}$2.24 to $^{-}$2.99), nodular (*log* LI=$^{-}$0.23 to $^{-}$1.25), and post-nodular (*log* LI=$^{-}$2.19 to $^{-}$2.94) regions. Eight of the quillback treated with EB harbored Lissorchis gullaris (3.3±2.6 worms per infected fish), 11 had Rowardleus pennensis (10.9±14.4), and 3 had Monobothrium hunteri (2.3±1.2). Pathology was not observed in quillback without N. carpiodi. Neoechinorhynchus carpiodi were not recovered when the intestines were flushed with PBS and were firmly attached and alive when nodules were examined 24 h after collection.

There was no significant difference in the concentration of EB (LI) in the three regions of intestine at 12, 15, 17, and 23 h post-injection although the amount of EB in the nodular sections was highest (*log* LI = 1.11±2.36) at 15 h post-injection. The concentration of EB in the lumen of the

intestine was greatest in the section containing the nodules and in slightly higher concentrations in the post-nodular region than in the pre-nodular region (Fig. 22).

Sections of intestine were classified as having high (*log* LI>$^{-}$1.62) or low (*log* LI<$^{-}$1.61) concentrations of EB and this was compared to the total volume of nodules. A stepwise discriminant analysis based on 5 variables (time post-injection, length, site of worm attachment, number of worms per fish, and number of nodules per fish) was done. The discriminant procedure chose number of nodules as the best variable for discriminating between intestinal sections. Eighty percent of the intestinal sections could be correctly classified based on the number of nodules present and the number of nodules was correlated ($\underline{r}^2$=0.64) with the number of gravid female worms.

Serum tests

The protein concentrations of the undiluted antigens were: gravid females, 5.8 mg/ml; mature male, 4.2 mg/ml.

Precipitin bands (Fig. 23) were observed in 33 of 61 (54%) sera tested (titre=8.12±12.36), and the distribution of positive sera was skewed towards high titres (G_1=3.199). Fourty-two fish had low numbers of N. carpiodi (0-40 worms per fish) and only 40% of these tested positive. Nineteen fish had high numbers of worms (>41 worms per fish) and 84%

Figure 22. Relationship between the concentration of protein-bound Evans blue (Log LI) in the lumen and the total volume of nodules (cm) in the intestine for N. carpiodi-infected quillback. Regression equations for concentration of bound EB versus volume are given and plotted separately for pre-nodular (PRN,●), nodular (N,▲), and post-nodular (PN,■) regions. Dashed line indicates the level of protein-bound EB in the lumen of an uninfected, nodule-free quillback.

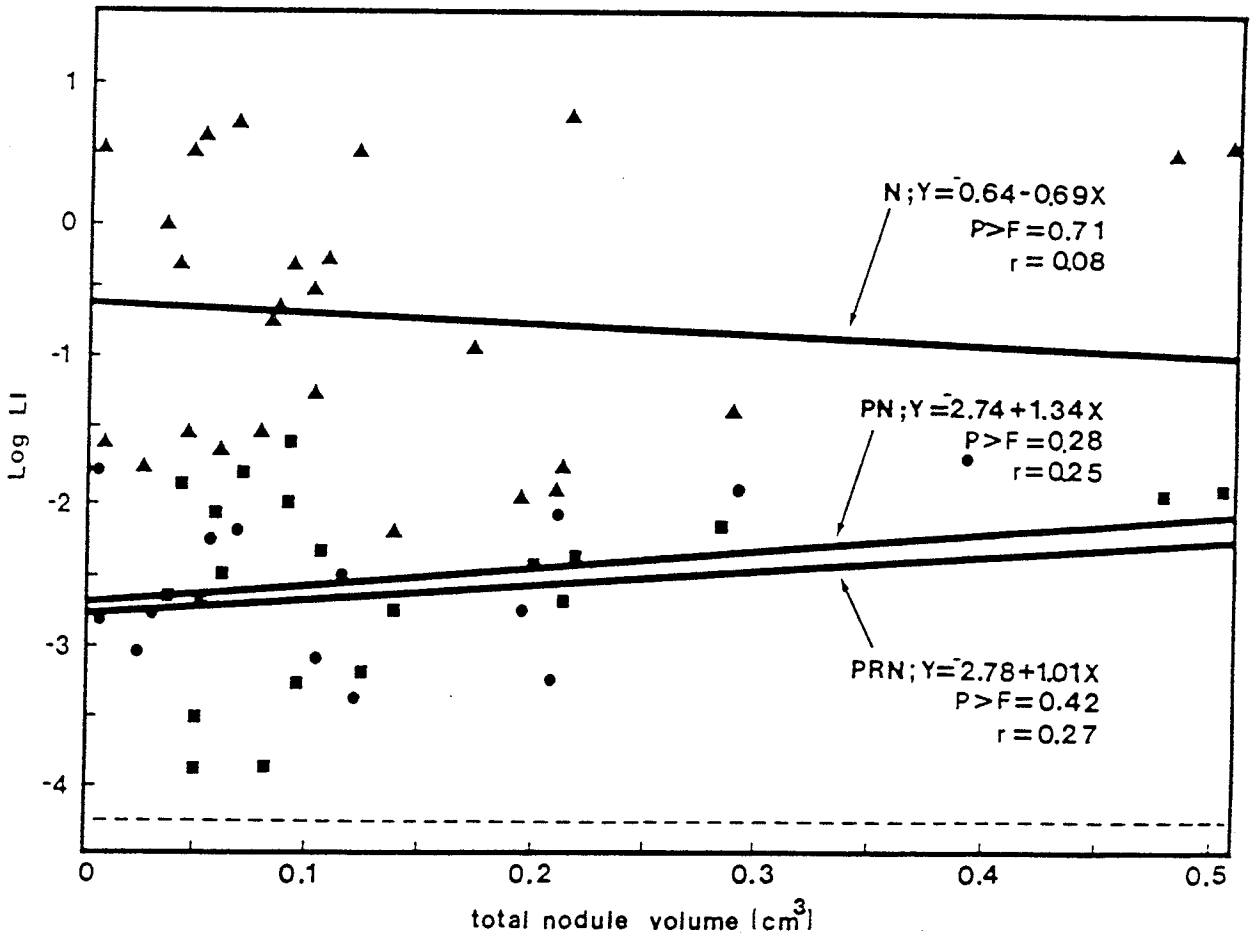
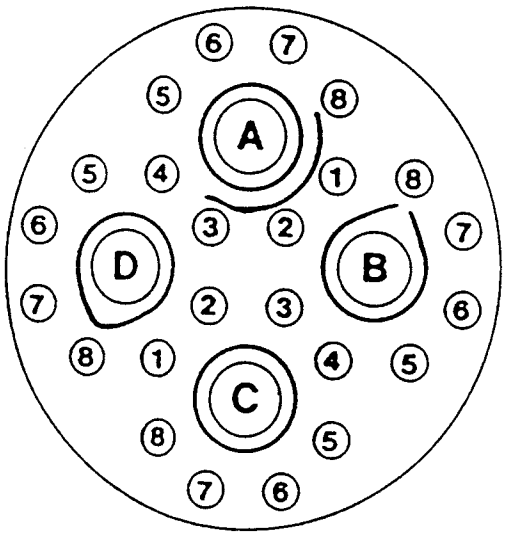

N; Y = 0.64 - 0.69X
P>F = 0.71
r = 0.08
PN; Y = 2.74 + 1.34X
P>F = 0.28
r = 0.25
PRN; Y = 2.78 + 1.01X
P>F = 0.42
r = 0.27
Log LI
1
0
-1
-2
-3
-4
0
0.1
0.2
0.3
0.4
0.5
total nodule volume (cm3)

Figure 23. Precipitin bands resulting from double-immunodiffusion of quillback serum against N. carpiodi antigens. **A-D.** Large wells containing quillback serum (A, undiluted serum; B, 50% dilution; C, 25% dilution; D, 12.5% dilution). **1-4.** Small wells containing gravid female N. carpiodi antigens (1, undiluted antigen; 2, 50% dilution; 3, 25% dilution; 4, 12.5% dilution). **5-8.** Wells containing mature male antigens (5, undiluted antigen; 6, 50% dilution; 7, 25% dilution; 8, 12.5% dilution).


A
B
C
D
1
2
3
4
5
6
7
8

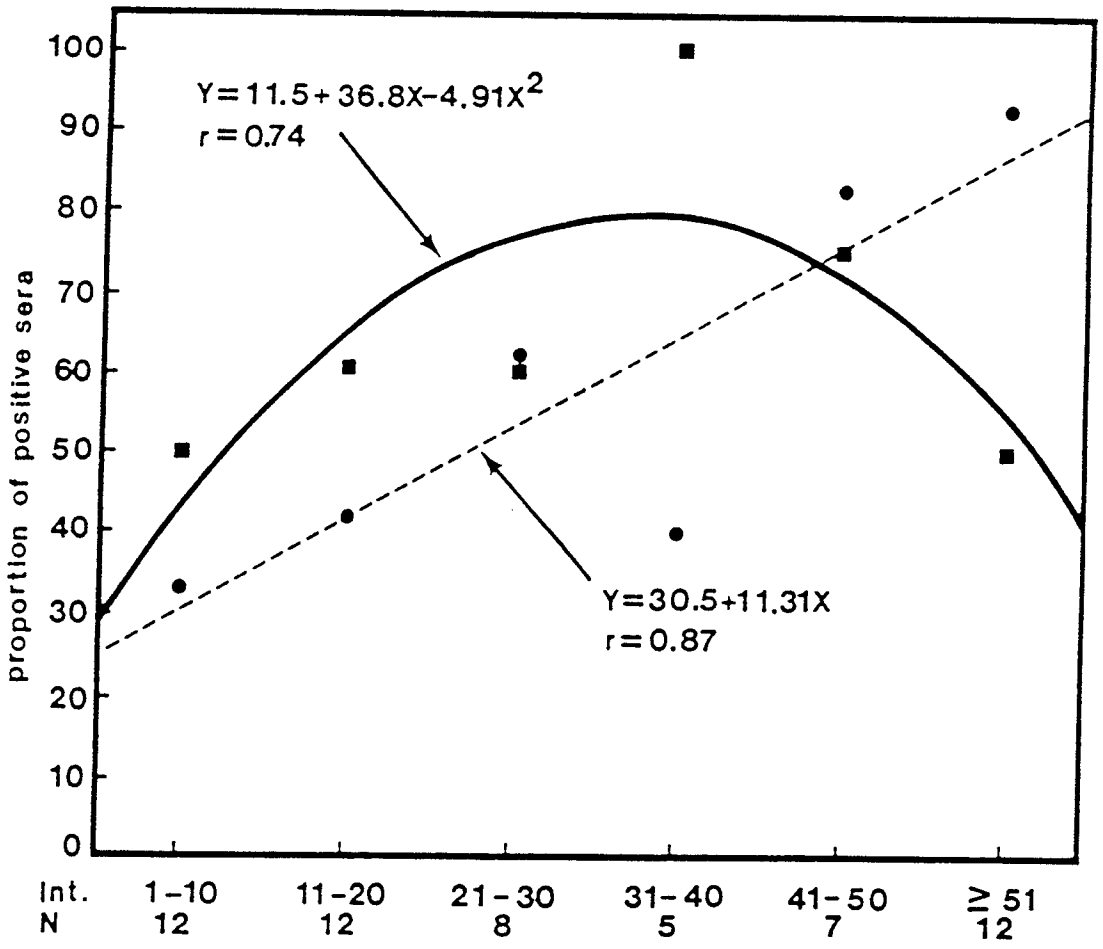
were positive for N. carpiodi antibodies. The titre for fish with low numbers of worms (7.88±9.78) was not significantly different from that for fish with high numbers of worms (8.38±15.31) (t-test), but the distribution of positive sera was more skewed for fish with high numbers of worms (G_1=2.9-59) than for fish with fewer worms (G_1=1.689).

Four of five sera from fish with no worms but showing evidence of previous infection (nodules) tested positive (titre=3.0±1.15). Sera from 4 fish known to be uninfected and with no nodules or other signs of previous infection by N. carpiodi all tested negative. One sample of serum from an uninfected quillback with no nodules gave a weak positive response (titre=2).

The proportion of positive sera increased linearly with intensity of infection (r^2=0.75). However,the proportion of positive sera with high (>4) titres reached a maximum for fish with 21 to 40 worms, and decreased for fish with more worms (Fig. 24).

Seven of 21 sera tested in a separate experiment gave false positives (titre=3.14±2.27) in gels not containing EDTA; the remaining sera (N=14) gave responses in EDTAtreated gels that were lower than or equal to the responses in untreated gels. The ability of EDTA-treatment to reduce non-specific precipitation was confirmed when sera from different quillback were diffused against each other. Precipitation reactions occurred between one pair of sera but these were absent in EDTA-treated gels.

Figure 24. Relationship between the number of N. carpiodi per quillback (Int.) and (*i*) the proportion of positive sera (●) and (*ii*) the proportion of positive sera with a titre ≥4 (■). Regression equations for positive sera (----) and for positive sera with a titre ≥4 (——) are given. N, number of fish.


Y=11.5+36.8X-4.91X2
r=0.74
Y=30.5+11.31X
r=0.87
proportion of positive sera
100
90
80
70
60
50
40
30
20
10
0
Int.
N
1-10
12
11-20
12
21-30
8
31-40
5
41-50
7
≥ 51
12

DISCUSSION

Gut leakiness and host immunity

This study has clearly shown that the intestine of quillback infected with the acanthocephalan N. carpiodi is leaky to large proteins from the blood. This leakiness, expressed through the complexing of EB with serum proteins, is most pronounced in the region of nodules induced by N. carpiodi. The EB measured in the intestine of quillback is due primarily to leakage into the lumen, where breakdown of the mucosal-tissue barrier occurred, since uninfected quillback showed extremely low levels of EB in the absence of any such pathology. The concentration of EB in the lumen of the gut of uninfected quillback was much lower than that recorded from the anterior and posterior sections of infected quillback intestines, and this difference was most pronounced where nodules were present. Increasing concentrations of EB in the intestine of quillback was correlated with increasing numbers of worms and nodules and further supports the idea that gut leakiness in quillback is largely due to the presence of N. carpiodi and associated pathology.

The leaky nature of the intestine in the region of the nodules indicates that large molecules including antigens, could cross the mucosal-tissue barrier. Indirect evidence for this is shown through an immune response in the form of precipitating antibodies to N. carpiodi antigens. Furthermore, there is no doubt that this response involves

specific antibody-antigen reactions since I ruled out the effects of non-specific reactions due to CRP and alpha migrating factor. Whether host tissue needs to be damaged to allow parasite antigens access to the humoral system (Smyth, 1969; Harris, 1972) or whether such antigens can be transported through the intact epithelium (Rombout et al., 1985) is not known. Rees (1967) suggested that immune responses to intestinal fish parasites will develop only if the host mucosa is invaded. Harris (1972) found specific anti-worm antibody in the intestinal mucosa of chubs infected with the acanthocephalan Pomphorhynchus laevis and concluded that non-specific leakage of precipitins from the blood to the mucous might be due to associated tissue damage. In the present study there was a strong immune response to antigens derived from male and female acanthocephalans and it is likely, in natural infections of quillback, that N. carpiodi-antigens reach the blood stream via these parasite induced lesions.

Evidence for regulation

Whatever the mechanism of antigen presentation, a continued but low level stimulation of the quillback immune system seems likely since the intensity and distribution of N. carpiodi infections was not affected by host sex, size, or the season of capture (Chapter 3-1). It appears that in quillback infected with N. carpiodi the antibody response is persistent and due to a continuous stimulation of the host

immune system. The average quillback in Dauphin Lake harbors about 34 N. carpiodi (Appendix III-G); coinciding with the range of infrapopulation sizes (31-40 worms) in quillback showing the maximum proportion of strong antibody responses (Fig. 24). At lower intensities (≤30 worms) few quillback produce antibodies and those that do respond only weakly. Furthermore, fish with the highest intensity infections had lower titres and this suggests the possibility of immunosupression. At higher intensities (≥41 worms) more quillback respond, but fewer are able to mount a strong immune response.

This host-parasite system, because of the persistent humoral and cellular responses manifested in nodule formation, and the possibility of immunosupression, suggests that the numbers of N. carpiodi in quillback might be regulated by host responses. However, since vascularization appears to play an important role by bringing blood fluids next to the only break in the mucosal-tissue barrier, the persistence of the nodules and the limited area and leaky nature of the lesions might also ensure that a limited but steady supply of nutrients is available to the parasites.

CHAPTER 4: *RAPHIDASCARIS ACUS* IN YELLOW PERCH, *PERCA FLAVESCENS*, AND NORTHERN PIKE, *ESOX LUCIUS*; REGULATION OF NON HOST-SPECIFIC PARASITES.

INTRODUCTION

Thus far, I have considered regulation in populations of fish-parasites with simple life cycles and well developed host specificity (i.e. Neoechinorhynchus carpiodi in quillback). Parasite populations, however, can be influenced by regulatory mechanisms operating at any stage in the life-cycle or within any host (Kennedy, 1977). In addition, most fish-parasites exhibit little or no host-specificity and have complex life cycles involving a variety of hosts. For the majority of fish parasite populations, it is therefore likely that these increasingly complex interactions between hosts and between hosts and their parasites play a vital role in determining whether or not regulation occurs.

Density-dependence in parasite size, known as the 'crowding effect' (Read, 1951), has been reported for various cestodes (Roberts, 1961; Hesselberg and Andreassen, 1975). Furthermore, fecundity of several nematode and cestode parasites has been reported to be related to parasite burden (Krupp, 1961; Michel, 1974; Jones and Tan, 1971; Mello, 1974; Croll et al., 1982). Since for most animals fecundity is a size-related trait (Blueweiss et al., 1978), perhaps the sub-lethal consequences of parasite density on size can be measured by translating their effects into influences on parasite fecundity, a biological parameter which directly determines transmission rates of parasites and their long term evolution.

The nematode Raphidascaris acus has a complex life-cycle involving a variety of hosts; briefly, this parasite uses aquatic invertebrates as paratenic hosts, fish as intermediate hosts, and piscivorous fish as the definitive host (Smith, 1984). In North America, R. acus utilizes yellow perch, (Perca flavescens) and northern pike (Esox lucius) as the main intermediate and definitive host, respectively (Margolis and Arthur, 1979). In Dauphin Lake: R. acus ranks second in overall importance (Fig. 7), it is the dominant parasite species in yellow perch (Appendix III-M, Fig. 8), and in northern pike ranks third in importance (Appendix III-C, Fig. 8). Furthermore, pike and perch are abundant in the lake and share many parasite species (Table 2) due to heavy predation of perch by pike (Appendix 1).

In the remaining sections of this thesis, the R. acus-yellow perch-northern pike system in Dauphin Lake is examined for evidence of regulation. Larvae of R. acus are found predominantly in the liver of infected yellow perch where they elicit a wide range of pathological responses by the host (Poole and Dick, 1984). Since liver function is closely related to growth, high burdens of invasive larvae should directly affect growth of perch. Furthermore, since life-time fecundity for this parasite can be measured directly, the effects of stochastic and deterministic forces on parasite fecundity can be measured. In Chapter 4-1, I provide evidence that yellow perch are subject to parasite-induced mortality due to infection by larvae of R. acus. In

chapter 4-2, I show that density-independence is nevertheless the dominant force determining the structure of the adult population in northern pike. Finally in Chapter 4-3, I evaluate by computer simulation the influence of stochastic mechanisms on the maintenance of long-term stability in populations of R. acus, and show that for unregulated (unstable) parasite populations increased genetic variation and variation in fecundity may act to minimize the dangers of extinction.

CHAPTER 4-1: PARASITE-INDUCED MORTALITY OF YELLOW PERCH DUE TO INFECTION BY *RAPHIDASCARIS ACUS*.

INTRODUCTION

The main proximate causes of mortality in fish are thought to be predation, parasitism, reproductive demands, and 'senescence', but quantifying mortality rates and determining the underlying causes of mortality is difficult. Most fisheries studies consider proximate causes of mortality as part of natural mortality, and usually deal with two causes of death: natural mortality and mortality due to fishing. Parasites have been implicated in mortalities of fish, and the importance of parasite-induced mortality in the regulation of fish parasite populations has been discussed (Anderson, 1978; Anderson and May, 1978; May and Anderson, 1978), but the impact of parasite-induced mortality is usually not considered by fisheries biologists. A few parasitologists have suggested that parasite-induced mortality of fish occurs (Klein et al., 1969; Henricson, 1977; Lester, 1977; Anderson and Gordon, 1982; Gordon and Rau, 1982; Kennedy, 1984; Lemly and Esch, 1984), but convincing evidence is elusive. Evidence is usually graphical; peaked age-intensity curves concomitant with a decrease in mean abundance of parasites, and a slope of less than two for a log-log graph of variance versus mean intensity of infection (Anderson and Gordon, 1982).

The nematode Raphidascaris acus is found in fish throughout Eurasia and North America (Hoffman, 1970; Margolis and Arthur, 1979). The complex life-cycle of

R. acus was summarized by Smith (1984); briefly, this parasite uses aquatic invertebrates as paratenic hosts, fish as intermediate hosts, and piscivorous fish as the definitive host. Although some information is available on the pathology of infection by this parasite (Poole and Dick, 1984; Smith, 1986) and its transmission to the definitive host (Smith, 1986), it is not known whether R. acus causes significant parasite-induced mortality in natural populations of fish. The only report of mortality of fish due to infection by R. acus is for bream, Abramis brama, in the USSR (Osmanov, 1953 *in* Petrushevski and Shulman, 1961).

In North America yellow perch, Perca flavescens, is one of the main intermediate hosts for R. acus (Margolis and Arthur, 1979). Few studies have dealt with the effects of parasitism on yellow perch (Lawler, 1969; Lester, 1977; Poole and Dick, 1984) so the impact of parasitism on populations of yellow perch remains unclear. The objectives of this chapter were: *i*), to estimate the natural mortality rate of yellow perch in Dauphin Lake; *ii*), to determine the proportion of total mortality that can be attributed to predation by northern pike, Esox lucius; *iii*), to determine the levels of infection by R. acus in yellow perch, and the effects of increasing density of R. acus on growth and mortality of perch; and *iv*), to discuss the significance of infection by R. acus on the life-history of yellow perch.

MATERIALS AND METHODS

A complete description of Dauphin lake has already been given (Chapter 1) and will not be repeated here. Some characteristics of the lake which are more pertinent to the present discussion are: *i*), Dauphin Lake has a diverse fish fauna (23 species); *ii*), very little angling takes place and most of this is directed towards walleye (Stizostedion vitreum vitreum) and northern pike (Esox lucius); and *iii*), a small fishery, supported mainly by harvests of walleye and northern pike, is operated each winter. Furthermore, yellow perch are rare in the annual commercial harvest.

Collection and examination of yellow perch

The total sample of fish used in this study was collected as part of the biological survey conducted by the Canadian Department of Fisheries and Oceans (DFO). A complete description of the sampling gear used and a full account of the gill netting programme have already been given (Chapter 1, *sampling procedures*). The total sample included 3,619 yellow perch, comprising about 25% of the entire DFO harvest. All yellow perch were inspected for external parasites and the presence of lymphocystis was recorded. The fork length (FL; mm) and round weight (W; g) was measured, and opercular bones were collected for age determinations (LeCren, 1947). The sex and maturity of each fish was determined according to the criteria of Treasurer and Holliday (1981) and Craig

(1987). To provide estimates of internal parasites a complete necropsy was performed on a random subsample of perch (N=292). The weight of the liver (LW; g) was measured for 71 perch from a second, independent subsample comprised of fish collected in spring, summer and fall of 1986.

Necropsy procedures, specimen collection, and specimen preparation were as outlined previously (Chapter 1; *Parasite enumeration and identification*). The number and types of parasites found, their sites of infection and the stomach contents were recorded for each perch. Furthermore, the entire liver of each perch was examined microscopically with the aid of a trichinoscope. Some encysted or encapsulated parasites were removed with fine forceps for later identification.

Enumeration of Raphidascaris acus

Since most R. acus found in yellow perch were larvae (three of 9,948 R. acus found were adults), and since 95% of these were found in the liver (Appendix III-M) the remaining text deals only with R. acus larvae in the liver of perch.

Infection of yellow perch by R. acus is characterized by the presence of second, third, and fourth-stage larvae (L_2, L_3, and L_4 larvae, respectively) (Smith 1984, 1986). Fourth-stage larvae are infective to northern pike (Esox lucius), in which they moult and develop into adults (Smith 1984, 1986). Pathology associated with the development of worms from L_2 to L_4 in the liver of yellow perch was described by Poole and Dick (1984). Briefly: the most recent infections

are characterized by free larvae (L_2, L_3 and infective L_4 larvae) in the major hepatic blood vessels; in older infections encapsulated L_4 larvae are found throughout the parenchyma; and in the final stages of host response, larvae are walled off to form collagenous nodules. Aside from slight hemorrhaging there are no lesions associated with free larvae (L_f); encapsulated larvae (L_e) appear normal but are surrounded by a cellular wall of host origin composed of fibroblasts, granulocytes, macrophages, and hepatocytes; nodules (L_n) are discrete accumulations of collagen surrounding degenerating larvae, but with time these become opaque and lose their integrity, and larvae are destroyed (Poole and Dick, 1984). Free larvae, capsules, and discrete nodules are easily identified and counted; but although scattered deposits of collagen from older nodules that have lost their integrity remain visible even after worms are destroyed, it is difficult to determine the number of older nodules by counting. Since nodule formation contributes to the accumulation of non-functional parenchyma in the liver as yellow perch age, counts of the number of nodules must include an estimate of the number of older nodules.

Initial analyses showed that the number of free larvae and well-defined nodules was age-invariant in yellow perch; whereas the number of encapsulated larvae accumulated with age. That the number of free larvae did not increase with age was not unexpected, as new immigrants are quickly walled-off (Smith, 1984) and contribute to the accumulation

of capsules. If the rate of formation of nodules is balanced by the rate of degeneration and loss of nodules, then the total number of nodules should accumulate at the same rate as capsules; if the turnover rate for nodules is faster, they should accumulate more slowly; and if turnover is slower, they should accumulate more rapidly.

The total number of nodules (L_n) was estimated based on the regression of the number of discrete nodules (L_{n*}) versus age of yellow perch (L_{n*}= ¯1.61+1.17 x age, r^2=0.094, $\underline{P}$=0.0001). The accumulation of nodules was assumed to be additive and to occur on an annual basis, as suggested by the reports of Poole and Dick (1984) and Smith (1984, 1986). Since the intercept of the regression of L_{n*} versus age was not significantly different from zero ($\underline{t}$-test), the total number of nodules was estimated using the formula: $L_n = L_{n*}+[1.17 \times (age-1)]$. For age-0 yellow perch, $L_n=L_{n*}$ since degenerating nodules will not appear until age-1. Parasite density (D; number of $\underline{R}$. $\underline{acus}$ larvae/g of liver) was estimated for all perch. Weights of livers (LW) were not recorded for the initial subsample of yellow perch. These were estimated based on the regression of LW versus W for the second subsample ($\underline{r}^2$=0.769) using the formula: LW = (0.01068 x W)-0.05821. The density of free larvae (D_f), encapsulated larvae (D_e), nodules (D_n), and all categories combined (L_t) was calculated.

Total mortality and mortality due to predation

Annual natural survival rates (S) and natural mortality rates (A=1-S) of yellow perch were estimated using Chapman and Robson's minimum variance unbiased estimator (Chapman and Robson, 1960; Ricker, 1975). According to Chapman and Robson the best estimate of S from age census data is: $S = T/(N + T - 1)$ with sampling variance estimated as: $S \times (S-[T - 1/ N + T - 2])$, where $T = N_1 + 2N_2 + 3N_3 + \ldots$ and $N = N_0 + N_1 + N_2 + \ldots$

To determine the validity of estimates of S and A based on the subsample, these were compared to estimates obtained using the total sample of yellow perch (N=3,619) collected by the DFO. Chapman and Robson's method requires that ages of fish are known, and since ages were not available for all of the latter these were estimated. Since growth of yellow perch in Dauphin Lake is rapid, ages were estimated based on the regression equation for FL versus age from our subsample using the formula: age=*antilog*(FL/[1.03+291.3])-1 (r_2 =0.81), rather than the age-length key method of Ricker (1975).

To determine the proportion of total natural mortality attributable to predation by northern pike, catch curves (Ricker, 1975) based on age-frequencies for our subsample were adjusted for age-specific predation by northern pike, the top predator and main definitive host for R. acus in Dauphin Lake. The magnitude of age-specific predation by northern pike was estimated based on the frequency of

occurrence for length-classes of yellow perch recovered from northern pike stomachs (N=356), compared to the frequencies of these length classes of yellow perch in the total sample.

Parasite-induced mortality

The condition of perch was measured using Fulton's condition factor, $FK=[W/(FL \times 10)^3] \times 100$ (Ricker, 1975), and the relationship between density of larvae (D) and FK was examined using Pearson's correlation coefficient (r) and simple linear regression. Since few age-0 yellow perch were collected and growth of males and females was non-asymptotic, logistic growth models were discarded in favour of a simpler exponential model. Preliminary comparisons of exponential growth curves based on FL with those based on W revealed only minor differences in slope, and since r^2 increased for the latter all further analyses were based on growth in weight. To check for interaction between density of R. acus and growth of yellow perch, perch were separated into two groups based on the density of larvae they harboured ('Heavy' infections with: total density, $D_t>50$ worms/g; density of free larvae, $D_f>10$ worms/g; density of encapsulated larvae, $D_e>20$ worms/g; or density of nodules, $D_n>10$ worms/g. 'Light' infections with: $D_t\leq50$, $D_f\leq10$, $D_e\leq20$, or $D_n\leq10$ worms/g), and growth curves for perch with heavy and light infections were compared. Frequency-distributions for density of larvae and age and weight of fish aided the interpretation of growth curves.

Since fishing mortality of yellow perch is negligible, A and S could be estimated, and the contribution of predation mortality was removed; the remaining mortality must be due to parasitism, reproductive demands, and senescence. Plots of mean intensity and variance-to-mean ratios for intensity of R. acus versus age of perch, and a log-log plot of variance versus mean for age-related intensity, were inspected for trends indicative of parasite-induced mortality (Anderson and Gordon, 1982; Kennedy, 1984).

Statistical analysis

Differences in intensity were tested between years using ANOVA, and between male and female and inshore and offshore yellow perch using Students' t-tests. Changes in the proportion of each category of larvae comprising the R.acus population were tested across age-classes of perch using ANOVA. A probability of $P<0.05$ was considered significant. Mean intensity, prevalence, abundance, and density were calculated according to Margolis et al. (1982); mean intensity refers to mean intensity of all categories of larvae combined (L_t), including nodules and scars, unless specifically stated otherwise. All analyses were performed using SAS. Pertinent data on the Eurasian perch (Perca fluviatilus) are discussed since Eurasian perch and yellow perch are equivalent biologically (Thorpe, 1977).

RESULTS

Since the loss of yellow perch in Dauphin Lake due to fishing is negligable, any estimate of total mortality includes the contributions made by proximate causes of mortality. Before discussing these, I consider the accuracy of my estimates of total natural mortality of yellow perch.

Natural mortality

Based on the subsample of fish whose ages could be determined, annual survival rates (S) were calculated for male and female yellow perch, and for combined sexes using Chapman and Robson's (1960) minimum-variance unbiased estimator (Table 8). Mortality (A) is the complement of survival (A=1-S) (Ricker, 1975). Since the subsample of yellow perch was collected over a three-year period, substantial differences in A and S between years could invalidate my estimates. To test the validity of my estimates these were compared to A and S calculated from the total samples collected in 1985 (N=1,153), 1986 (N=1,134), and 1987 (N=1,332). Ages were not available for the latter so these were estimated based on the regression equation for FL versus age for yellow perch from the subsample (FL = 1.0255+291.279 x *log*[age+1], r^2=0.8083, P=0.0001) using the equation: age= *antilog*(FL/[1.03+291.3])-1. Since my estimates for annual mortality in 1985, 1986, and 1987 (58%, 58%, and 61%, respectively), were comparable,

Table 8. Annual survival rate (S) and mortality rate (A) for yellow perch from Dauphin Lake. S and A were estimated using the minimum-variance unbiased estimator of Chapman and Robson (1960) with age-2 yellow perch as the first fully recruited age-class. Values of S and A for perch reported by other authors are listed for comparison.

	S[a]	A	A (authority)
total sample			
1985	0.4236±0.4233	0.5764	
1986	0.4176±0.4174	0.5824	
1987	0.3691±0.3688	0.6309	
pooled	0.3868±0.3923	0.6132	
subsample			
male	0.2973±0.2778	0.7027	0.39-0.73 (M^{c}Cormak, 1965)
female	0.4288±0.4277	0.5712	0.64-0.71 (M^{c}Cormak, 1965)
pooled	0.4204±0.4940	0.5796	0.15-0.69 (various authors)[b]

[a] mean±variance.

[b] Craig and Kipling, 1983 (A= 0.15-0.67); Goedde and Coble, 1981 (A= 0.68-0.69); LeCren, 1987 (A=0.42).

and since my estimate for 1985-1987 combined (61%) is similar to that obtained from my subsamples (58%) and to those reported by others (Table 8), I am confident that my estimates of S and M are representative of the true rates of natural mortality and survival for yellow perch in Dauphin Lake.

Mortality due to predation

Since northern pike is the top predator in Dauphin Lake and serves as the main definitive host for R. acus, I considered whether or not feeding by northern pike on yellow perch is size-selective and to what extent this might contribute to the increased mortality of yellow perch.

Ten percent of the northern pike stomachs examined (N=356) contained yellow perch, and all of these were less than 250 mm FL. Comparison of the length-frequency distribution for perch $\leq$250 mm FL in the total sample (159.1$\pm$523.9 mm, N=200) with that for perch recovered from pike stomachs (92$\pm$58.1 mm, N=40), confirmed that yellow perch selected by pike had significantly lower FL than yellow perch that were available in the lake (P=0.0001, t-test). Furthermore, ages estimated from FL indicated that about 75% of the yellow perch eaten by pike were $\leq$2 years old.

The ratio between the proportion of yellow perch in age-classes recovered from northern pike stomachs (P) and the proportion in each age-class in the total sample (A) (Table 9) gives an estimate of mortality due to predation by

Table 9. Data used to estimates the losses of yellow perch to predation by northern pike. Fork lengths are based on a subsample of yellow perch with known ages. Fork length was used to estimate age of yellow perch ≤250 mm in the total sample. The proportions of each age-class of yellow perch in the total sample (A) was calculated and compared to the proportions recovered from northern pike stomachs (P).

		fork length (mm)		proportion (%)			
age	n	$\bar{X} \pm$ S.D.	LL - UL[a]	A[b]	P[c]	P/A	%[d]
0	1	60	-	10.5	52.5	5	100
1	24	110.1±25.5	99.9-120.3	6.5	15	2.31	67
2	32	139.3±27.1	124.2-148.7	39.5	5	0.127	88
3	65	158.9±25.7	152.7-165.2	18.5	0	0	88
4	25	190.0±37.5	173.4-204.7	7	5	0.714	68
5	20	238.6±29.9	225.5-251.7	15.5	2.5	0.161	85

[a] Lower (LL) and upper (UL) 95% confidence limits.
[b] Proportion of yellow perch. Based on the length frequency distribution for all yellow perch ≤ 250 mm FL in the total sample (n = 200). As yellow perch with fork length outside the 95% confidence limits were excluded, A does not add up to 100%.
[c] Proportion of yellow perch predated. Based on 40 yellow perch recovered from northern pike stomachs.
[d] Proportion of female yellow perch.

northern pike. For this comparison age of yellow perch recovered from northern pike was estimated based on FL. These estimates are valid since for perch within the size-range preyed upon by pike, the 95% confidence limits for mean FL of successive age-classes do not overlap (Table 9).

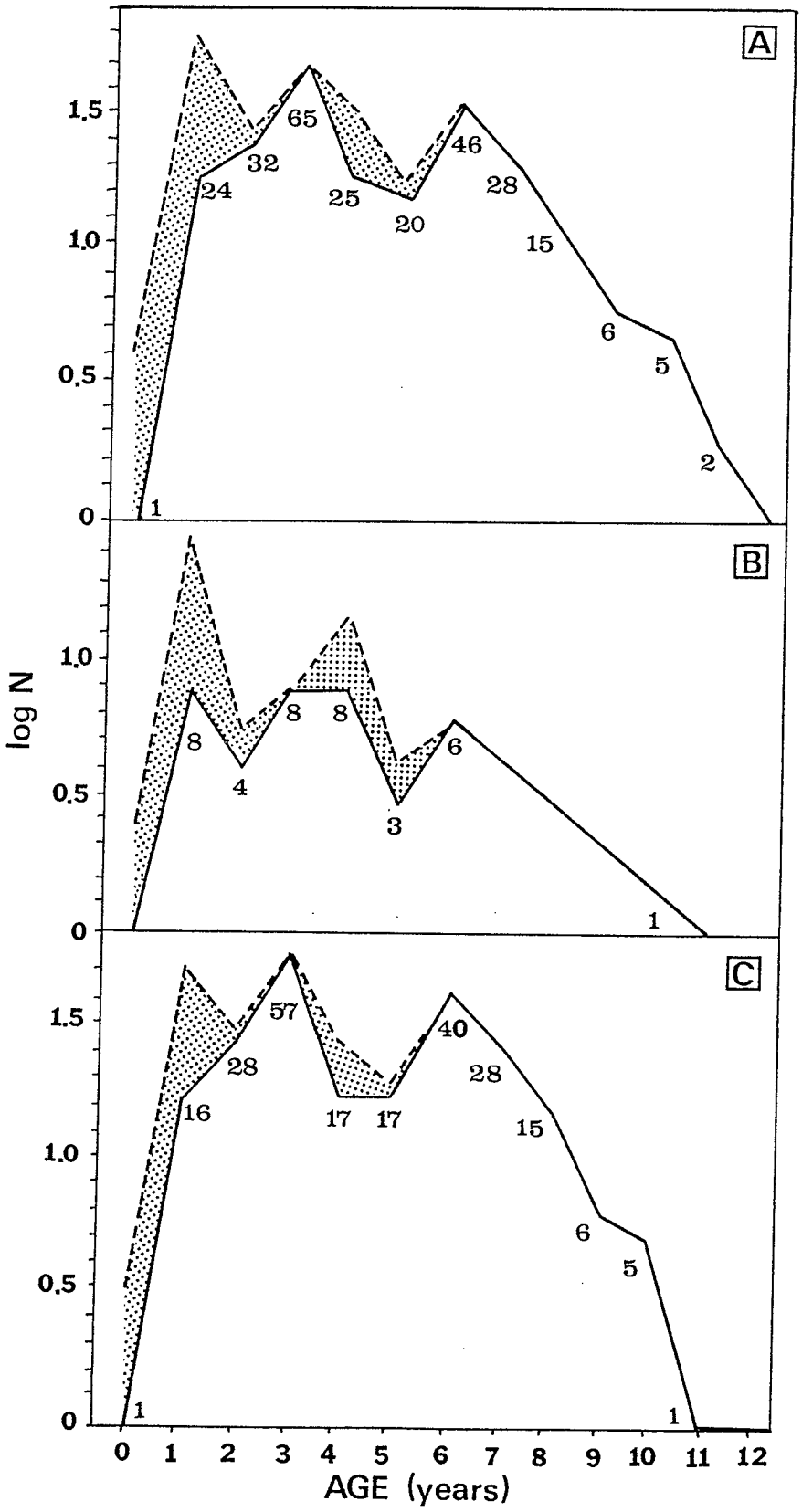
To estimate the magnitude of predation by northern pike on different age-classes of perch, catch curves (*log*N versus age) based on age-frequencies (F_N) for the subsample were plotted and compared to catch-curves with age-frequencies adjusted to account for losses due to predation (F_{N*}). The difference between normal and adjusted catch-curves reflects the magnitude of predation by pike for each susceptible age-class of yellow perch. For the combined subsample, the adjusted number of fish was calculated using:

$F_{N*}=(F_N \times P/A)+F_N$. For males and females, the proportions of each sex at large in the total sample (Table 9) were considered in calculations of F_{N*}; the adjusted number of males (F_{NM*}) was calculated using:

$F_{NM*}=F_{NM}+[(F_{N*}-F_N) \times (1-p)]$, and the adjusted number of females (F_{NF*}) was calculated using:

$F_{NF*}=F_{NF}+[(F_{N*}-F_N) \times p]$, where p is the proportion of females in the total sample (Table 9). About 24% of the total mortality of yellow perch was due to predation by northern pike (Fig. 25A). Predation by pike accounted for 45% of all mortality of male perch (Fig. 25B), and for females (Fig. 25C) 19%. For both males and females, mortality due to predation was highest for age-0 and age-1 yellow perch

Figure 25. Catch curves for yellow perch before (-----) and after (- - -) adjusting for losses to predation by northern pike. A . Males and females combined. B . Males only. C . Females only. Shaded region represents the magnitude of mortality of yellow perch due to predation. Note logarithmic scale.


A
1.5
1.0
0.5
0
1
24
32
65
25
20
46
28
15
6
5
2
B
1.0
0.5
0
8
4
8
8
3
6
1
C
1.5
1.0
0.5
0
1
16
28
57
17
17
40
28
15
6
5
1
0 1 2 3 4 5 6 7 8 9 10 11 12
AGE (years)
log N

(85% and 70%, respectively), decreased to zero at age-3, and increased again by age-4 (42%) (Fig. 25; note logarithmic scale).

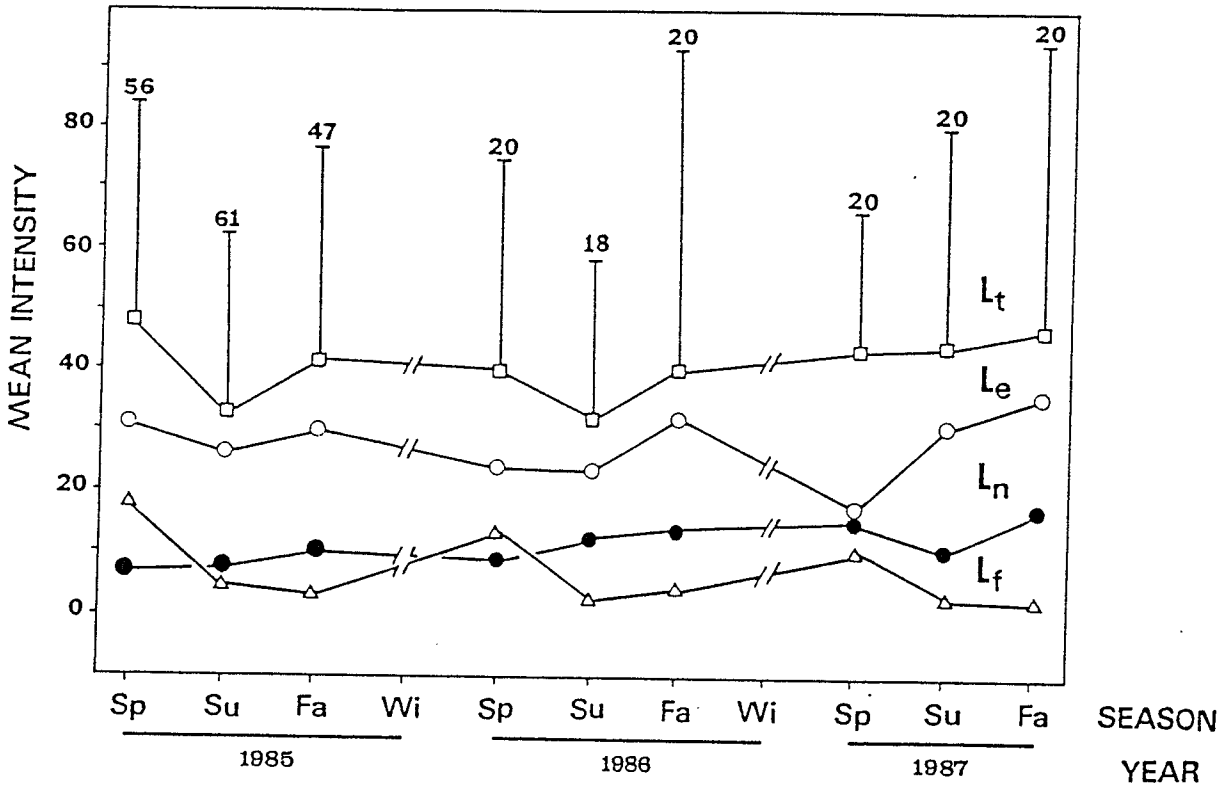
Predation alone does not account for all of the mortality seen for yellow perch in Dauphin Lake. The contribution of parasites, senesence, and reproductive demands to total mortality are discusssed below. In the next section I provide evidence that parasite-induced mortality due to infection by R. acus comprises a large portion of the mortality not explained by predation.

Parasite intensity

Eighteen species of parasites and viral lymphocystis were found in yellow perch (Appendix III-M). R. acus was the most abundant species present; larvae of R. acus had the highest overall prevalence (over 95%) and the highest intensity (35.77 $\pm$ 32.69 larvae/infected yellow perch) of all the helminths found (Appendix III-M). Since larval R. acus were found almost exclusively (three of 9,948 worms were adults) and predominantly in the liver (about 95% of all larvae), and since most yellow perch were infected and mean intensity was high, all further investigation focussed on the interaction between larvae of R. acus and yellow perch.

Mean intensity of larvae did not vary between years (ANOVA). A weak seasonal pattern of infection characterized by reduced intensity of infection in summer was evident (Fig. 26). The number of free larvae (L_f) was highest in

Figure 26. Seasonal changes in mean intensity of R. acus in yellow perch. Symbols represent mean intensity of free larvae (△; L_f), encapsulated larvae (○; L_e), nodules (●;L_n), and all categories combined (□;L_t). Vertical bars and numbers represent one standard deviation and sample size (number of yellow perch examined), respectively.


MEAN INTENSITY
80
60
40
20
0
56
61
47
20
18
20
20
20
L_t
L_e
L_n
L_f
Sp
Su
Fa
Wi
Sp
Su
Fa
Wi
Sp
Su
Fa
SEASON
1985
1986
1987
YEAR

spring and declined steadily towards fall, concomitant with an increase in the number of encapsulated larvae (L_e); but the total number of nodules (L_n) was uniform among seasons (Fig. 26). Yellow perch caught inshore (water depth $\leq$1 m) and those caught offshore (>1 m) had similar intensities of infection (*t*-test); and females harboured more larvae (37.54 $\pm$ 33.87, N=231) than males (25.82 $\pm$ 23.73, N=44) (P=0.0068, *t*-test).

Overall, mean intensity of larvae (L_t) increased with age of yellow perch (Fig. 27). The number of free larvae (L_f) and well-defined nodules (L_{n*}) in yellow perch is limited by capsule formation and degeneration of nodules, respectively, and is age-invariant (Fig. 27). The number of encapsulated larvae (L_e) increased with age as worms became walled-off and capsules accumulated (Fig. 27). After adjusting for the number of degenerating nodules, total number of nodules (L_n) increased with age. I am confident that my counts of capsules are accurate and since all capsules eventually form nodules, the displacement between regression lines for encapsulated larvae and nodules is an estimate of the residence-time of larvae in capsules. When advanced along the abscissa (Fig. 27), the curve for encapsulated larvae agrees well with the curve for nodules, indicating that the residence-time for larvae in capsules is approximately two years, and that capsule formation and nodule formation occur at similar rates. Furthermore, a decreasing proportion of free larvae in older yellow perch (Fig. 28) could only occur

Figure 27. Age-specific mean intensity of R. acus in yellow perch. Exponential curves for mean intensity of free larvae (L_f), encapsulated larvae (L_e), nodules (L_n), and all categories combined (L_t) are indicated. Mean intensity of discrete nodules (dotted line; L_{n*}) is shown for comparison. The regression line for encapsulated larvae, advanced two years along the abscissa, is indicated (dashed line). The total number of nodules (L_n) was estimated based on the regression of the number of discrete nodules (L_{n*}) versus age of yellow perch (L_{n*}= ¯1.61+1.17 x age, r^2=0.094, P=0.0001). The accumulation of nodules was assumed to be additive and to occur on an annual basis. Regression equations and r^2 are given.

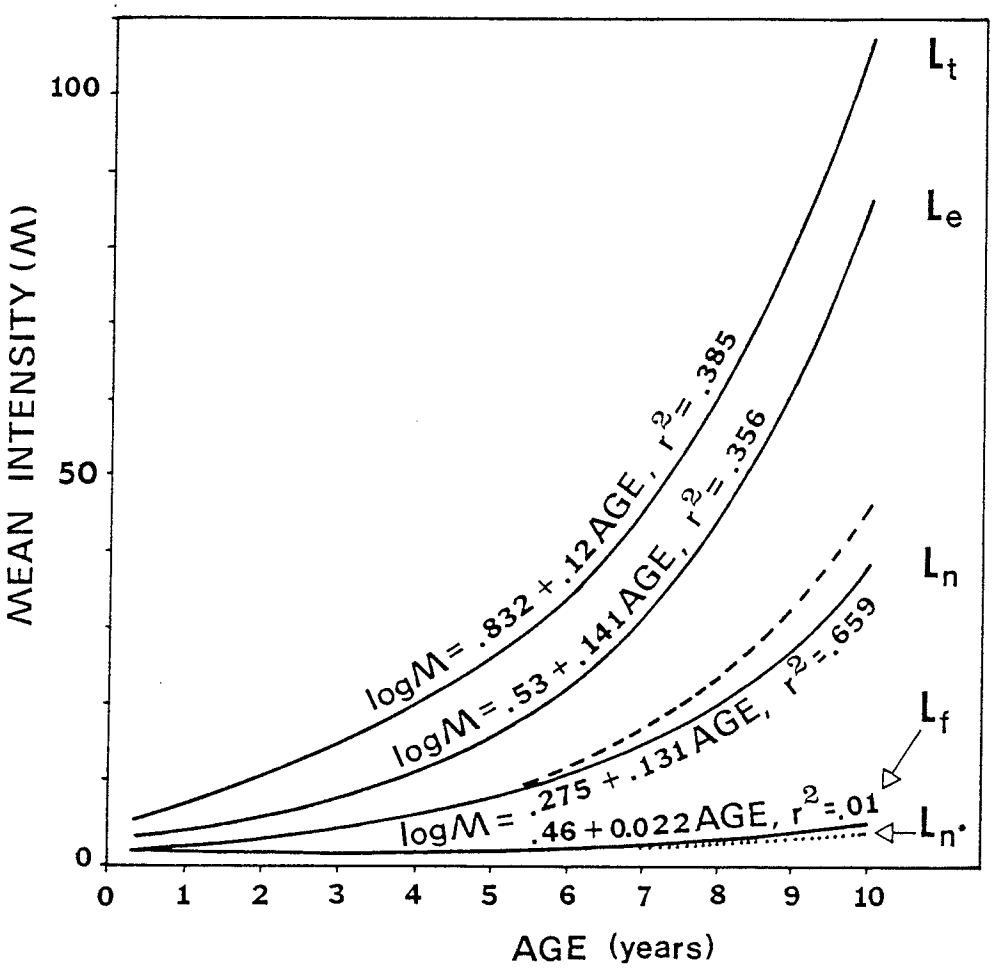
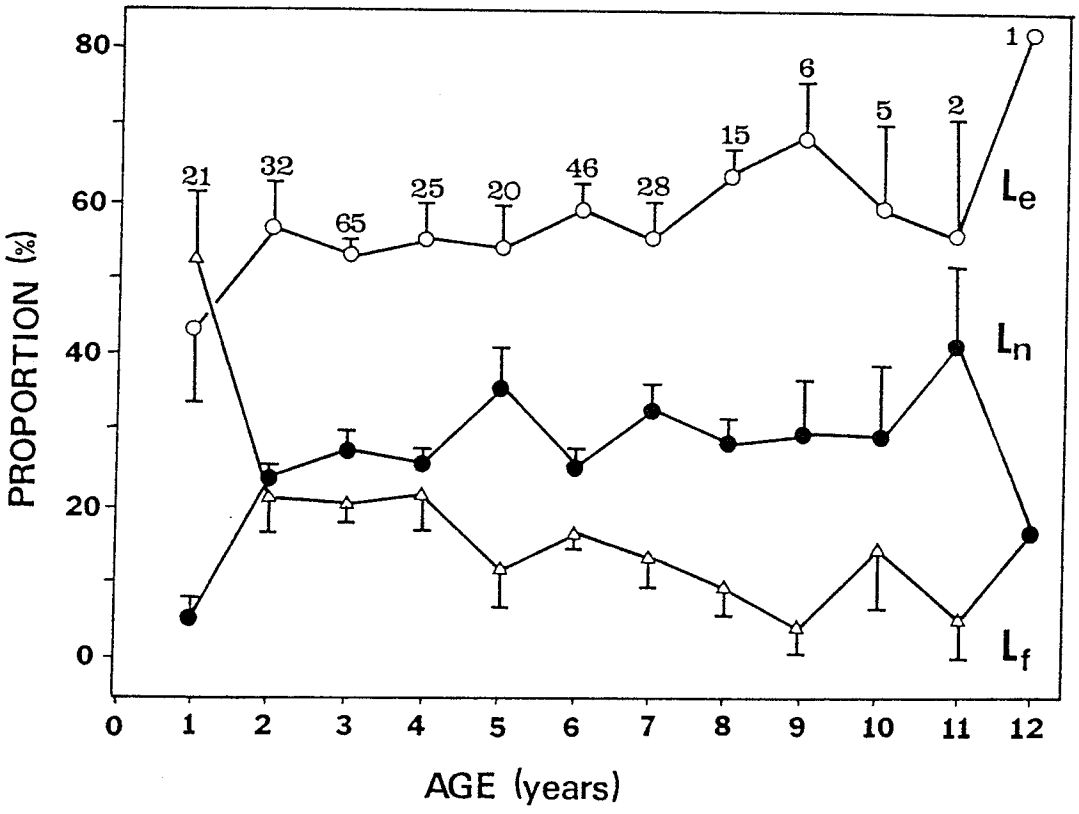

MEAN INTENSITY (M)
100
50
0
0
1
2
3
4
5
6
7
8
9
10
AGE (years)
L_t
L_e
L_n
L_f
$L_{n'}$
$\log M = .832 + .12 AGE,\ r^2 = .385$
$\log M = .53 + .141 AGE,\ r^2 = .356$
$\log M = .275 + .131 AGE,\ r^2 = .659$
$\log M = .46 + 0.022 AGE,\ r^2 = .01$

Figure 28. Changes in the proportions of free larvae (△;L_f), encapsulated larvae (○; L_e), and nodules (●;L_n), in populations of R. acus from the liver of yellow perch. Vertical bars and numbers represent one standard error and sample size (number of yellow perch examined), respectively.


80
60
40
20
0
PROPORTION (%)
0 1 2 3 4 5 6 7 8 9 10 11 12
AGE (years)
21
32
65
25
20
46
28
15
6
5
2
1
Le
Ln
Lf

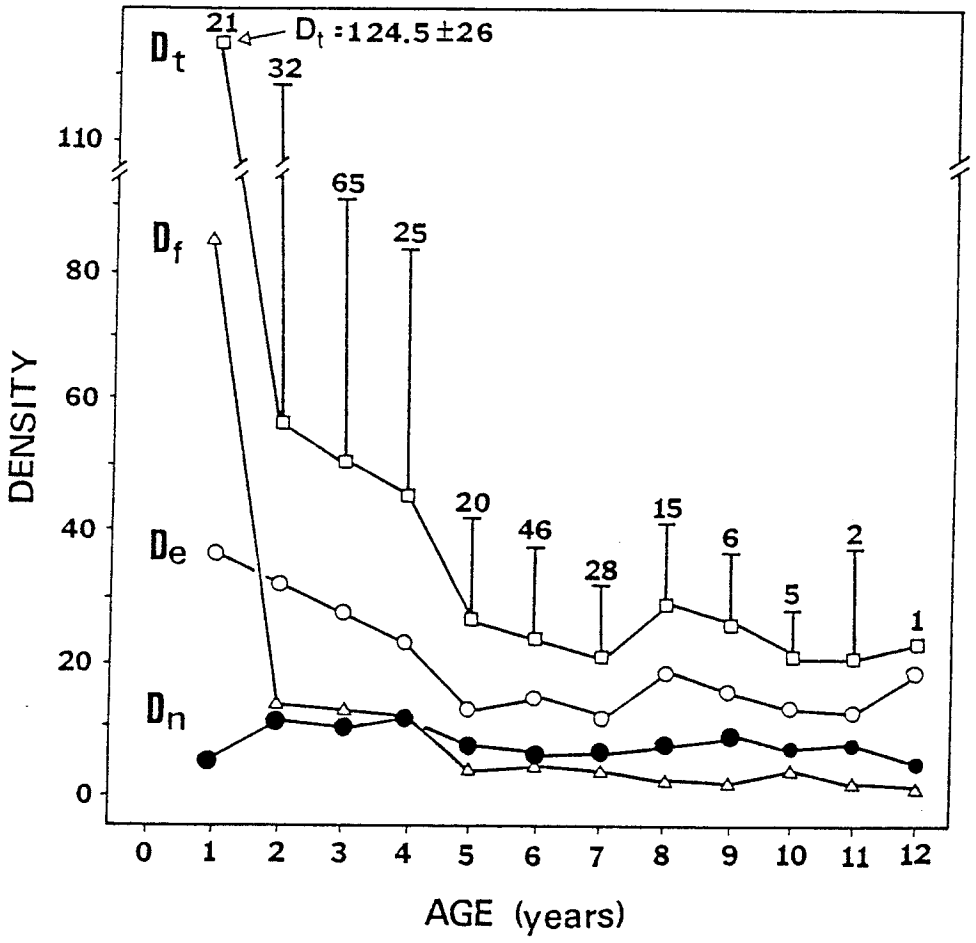
if the rate of immigration was constant or decreased with age. Analysis of stomach contents of perch suggests that recruitment of R. acus is age-invariant. Similarly, increasing proportions of nodules in older yellow perch is not due to decreased immigration and must be due to a more rapid host reponse in older fish. The proportions of free and encapsulated larvae and nodules were similar between sexes and were not correlated with water temperature (ANOVAS).

Parasite density and growth of yellow perch

Density of R. acus (D_t, larvae/g of liver) was highest in young perch and decreased with age (Fig. 29). Densities of free and encapsulated larvae (D_f and D_e, respectively) also decreased with age of perch, but densities of nodules (D_n) remained uniform (Fig. 29). Decreased larval density with age occurred for both male and female yellow perch, and for both sexes the greatest reduction in larval density coincided with the average age at maturity (2.33 $\pm$ 1.16 and 3.2 $\pm$ 0.45 years for males [N=16] and females [N=73], respectively). Maximum larval density occurred one year later for females (D_t=58 $\pm$ 40 at age-2) compared to males (D_t=277 $\pm$ 427 at age-1).

Since infections of yellow perch by R. acus are characterized by high densities of larvae in the liver, an organ whose function is closely tied to growth, the relationship between larval density and growth of yellow

Figure 29. Changes in the density (D; larvae/g liver) of free larvae (△; D_f), encapsulated larvae (○; D_e), nodules (●; D_n), and all categories combined (□; D_t) in the liver of yellow perch. A . Males plus females (N=266). B . Females only (N=229). C . Males only (N=37). Vertical bars and numbers represent one standard deviation and sample size (number of yellow perch examined), respectively. Note breaks in vertical axis.


D_t
D_f
D_e
D_n
D_t = 124.5 ± 26
21
32
65
25
20
46
28
15
6
5
2
1
110
80
60
40
20
0
DENSITY
0 1 2 3 4 5 6 7 8 9 10 11 12
AGE (years)

perch was investigated. In general, growth of male and female perch (Fig. 30) was similar until maturation, when sexually dimorphic growth begins. Mature females grew faster and reached a larger ultimate size than mature males (Fig. 30). Initial analyses indicated a significant correlation ($\underline{r}$=-0.245, $\underline{P}$=0.0001) between condition factor (FK) and larval density for females, but not for males. Since the size of the gonads will influence W and therefore affect FK (Ricker, 1975), the data was re-analyzed. Comparison of mean FK for yellow perch with different states of maturity revealed that the relationship between larval density and FK was influenced by higher FK for mature perch (FK=1.41 $\pm$ 0.13 and FK=1.58 $\pm$ 0.46 for males [N=24] and females [N=46], respectively). When mature yellow perch were considered in isolation, increased density of larvae had no affect on FK for mature females ($\underline{r}$=0.118, $\underline{P}$=0.321) but mature males with high density infections had reduced FK ($\underline{r}$=0.733, $\underline{P}$=0.0008).

Perch were categorized as harbouring 'heavy' infections (total density, D_t>50 worms/g; density of free larvae, D_f>10 worms/g; density of encapsulated larvae, D_e>20 worms/g; and density of nodules, D_n>10 worms/g) or 'light' infections ($D_t \leq 50$, $D_f \leq 10$, $D_e \leq 10$, and $D_n \leq 20$ worms/g, respectively) based on the frequency distributions for larval density, and growth curves for perch with heavy versus light infections were compared. In general, the slope of growth curves for perch with heavy infections was lower than the slope of growth curves for perch with light infections (Fig. 31).

Figure 30. Absolute growth curves for male (●) and female (○) yellow perch from Dauphin Lake. Open and closed circles represent average fork length; vertical lines represent one standard deviation; and numbers indicate sample size (number of fish). Ages at maturity (mean ± standard deviation) are indicated (horizontal blocks).

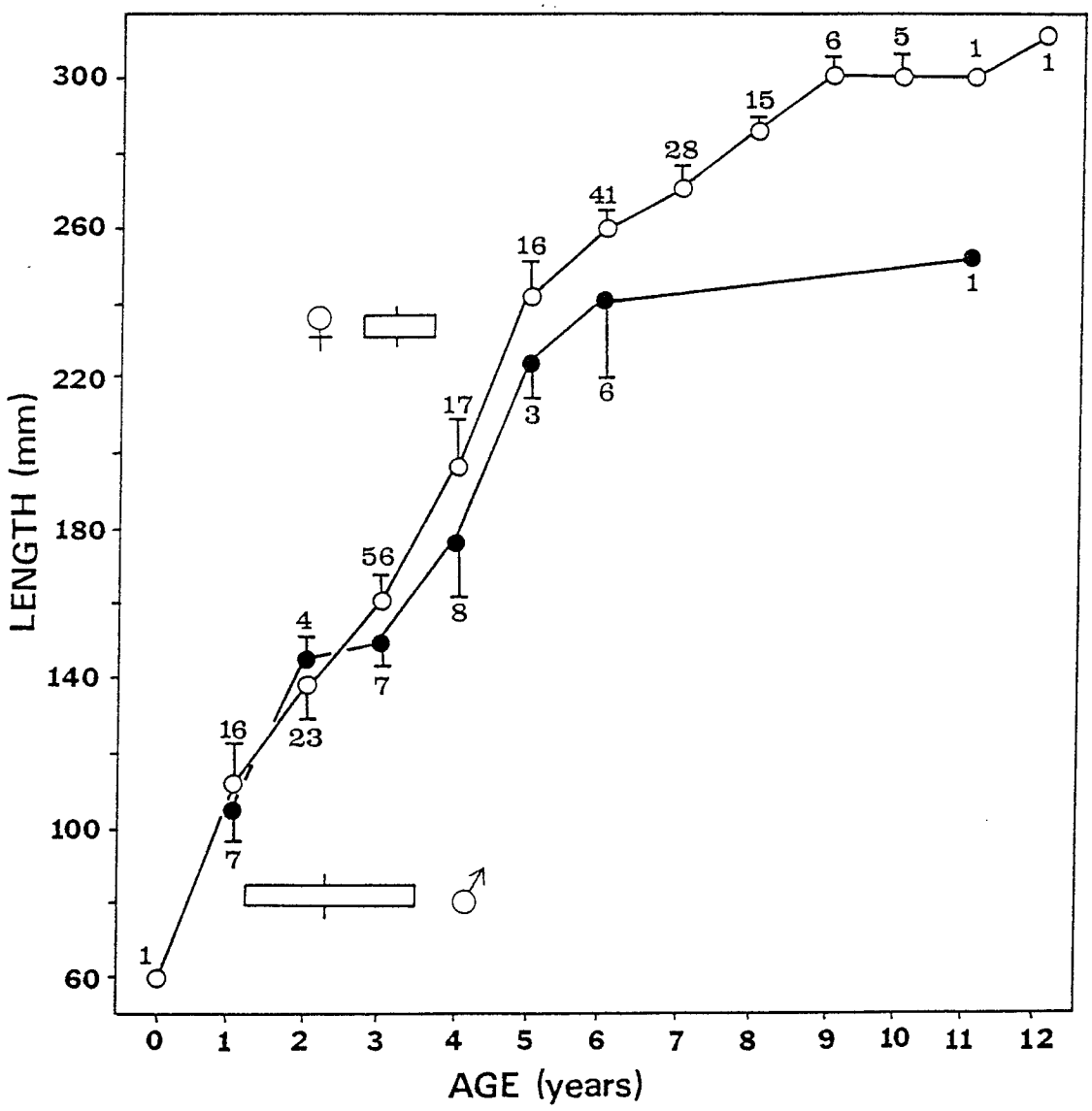
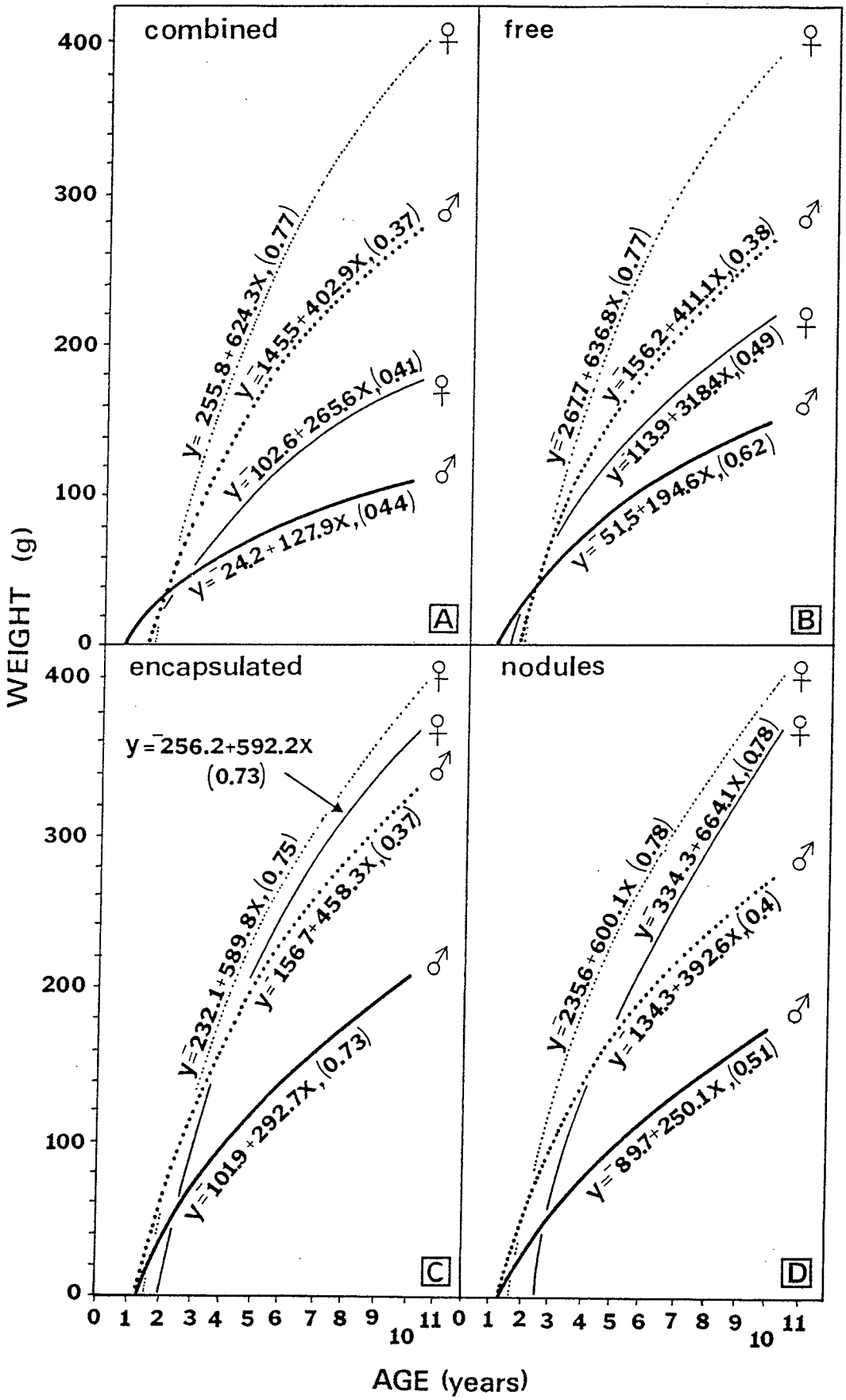

LENGTH (mm)
300
260
220
180
140
100
60
0
1
2
3
4
5
6
7
8
9
10
11
12
AGE (years)
♀
♂
1
16
7
4
23
56
7
17
8
16
3
41
6
28
15
6
5
1
1
1

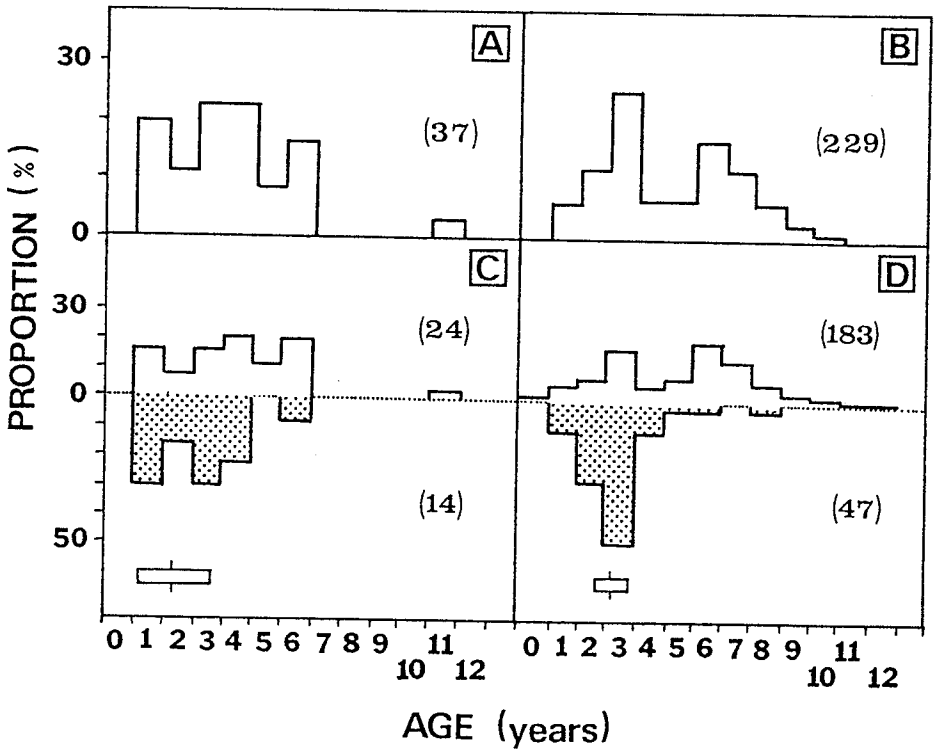
Figure 31. Growth curves for male (thick lines) and female (thin lines) yellow perch with heavy (solid lines) or light (dashed lines) infection by R. acus. All densities (D) are in larvae/g of liver. A . Growth curves for yellow perch with $D_t>50$ and $D_t\leq 50$. B . Growth curves for yellow perch with $D_f>10$ and $D_f\leq 10$. C . Growth curves for yellow perch with $D_e>20$ and $D_e\leq 20$. D . Growth curves for yellow perch with $D_n>10$ and $D_n\leq 10$. Regression equations and r^2 for the model $Y=a+b\log X$ are indicated. $P<0.0001$ for each regression.


combined
free
encapsulated
nodules
A
B
C
D
WEIGHT (g)
AGE (years)
0
100
200
300
400
0 1 2 3 4 5 6 7 8 9 10 11
y = 255.8+624.3x, (0.77)
y = 1455+402.9x, (0.37)
y = 102.6+265.6x, (041)
y = 24.2+127.9x, (044)
y = 2617+636.8x, (0.77)
y = 156.2+4111x, (0.38)
y = 1139+3184x, (0.49)
y = 515+194.6x, (0.62)
y = 256.2+592.2x
(0.73)
y = 232.1+589.8x, (0.75)
y = 1567+458.3x, (0.37)
y = 1019+292.7x, (0.73)
y = 2356+600.1x, (0.78)
y = 334.3+6641x, (0.78)
y = 1343+3926x, (0.4)
y = 89.7+250.1x, (0.51)

Assuming homogenous age-related mortality this indicates reduced growth rates for heavily infected fish. However, the growth curves for heavily infected males shifted towards the ordinate axis (Fig. 31), suggesting that young, heavily infected males were over-represented by larger bodied fish. Unfortunately, this could not be tested statistically since only one heavily infected immature male was collected. Reduced growth was most apparent with high densities of free worms (Fig. 31B); but was also apparent for males with high densities of capsules and nodules (Fig. 31 C, D).

Frequency distributions for density of infection and age and weight of yellow perch supported the suggestion that the observed shifts in the growth curves might be due to increased losses of young, heavily infected yellow perch and reduced growth of older, heavily infected yellow perch. Larval densities $\geq$125 worms/g were found in 43% of age-1 male perch (N=7), but in none of the older males (N=30); similarly, infections with densities $\geq$ 100 worms/g were found in 11% of females less than 5 years old (N=13), but in none of the older females examined (N=129). Age-frequency distributions for yellow perch with heavy and light infections (Fig. 32) revealed that the decrease in numbers of young, heavily infected females coincides with the onset of maturation. Age-distributions for lightly infected females (Fig. 32D.) were strongly bimodal, but for heavily infected females the distributions had a single mode favouring immature fish; either increased numbers of heavily

Figure 32. Age-distributions for male (left) and female (right) yellow perch with heavy (D_t>50 larvae/g; shaded columns) and light ($D_t \leq 50$ larvae/g; open columns) infections of R. acus. The distribution for all males (A) and all females (B) combined, for males with heavy and light infections (C), and for females with heavy and light infections (D) are presented separately. For each sex the age at maturity (mean ± standard deviation) is indicated by a horizontal bar. Number of fish examined (N) is given.


A
B
C
D
(37)
(229)
(24)
(183)
(14)
(47)
30
0
30
0
50
PROPORTION (%)
0 1 2 3 4 5 6 7 8 9 10 11 12
0 1 2 3 4 5 6 7 8 9 10 11 12
AGE (years)

infected females died at age-3, or fewer of these matured. Age-distributions for male perch (Fig. 32. A,C) were not bimodal and no increase in the proportion of immature males occurred for heavily infected males. Changes in weight-frequency distributions with age of yellow perch provided additional evidence for differential mortality of male and female yellow perch. Briefly, the distribution for weight of female yellow perch revealed two modal classes; a group of small fish ($W \leq 80$ g) less than 4 years old and a second group of heavier fish ($W > 80$g) 2 years old or older. In males bimodality was only apparent at age-4. For both sexes, the smaller modal group did not appear after age-4. Immature females from the first modal group had greater larval densities ($D_t = 58.4 \pm 36.9$) than larger, mature females from the second modal group ($D_t = 21.2 \pm 10.6$; $\underline{P} = 0.0001$), and immature males had higher densities of nodules ($D_n = 12.6 \pm 15.3$) than their mature counterparts ($D_n = 6.1 \pm 2.7$; $\underline{P} = 0.0458$) ($\underline{t}$-tests).

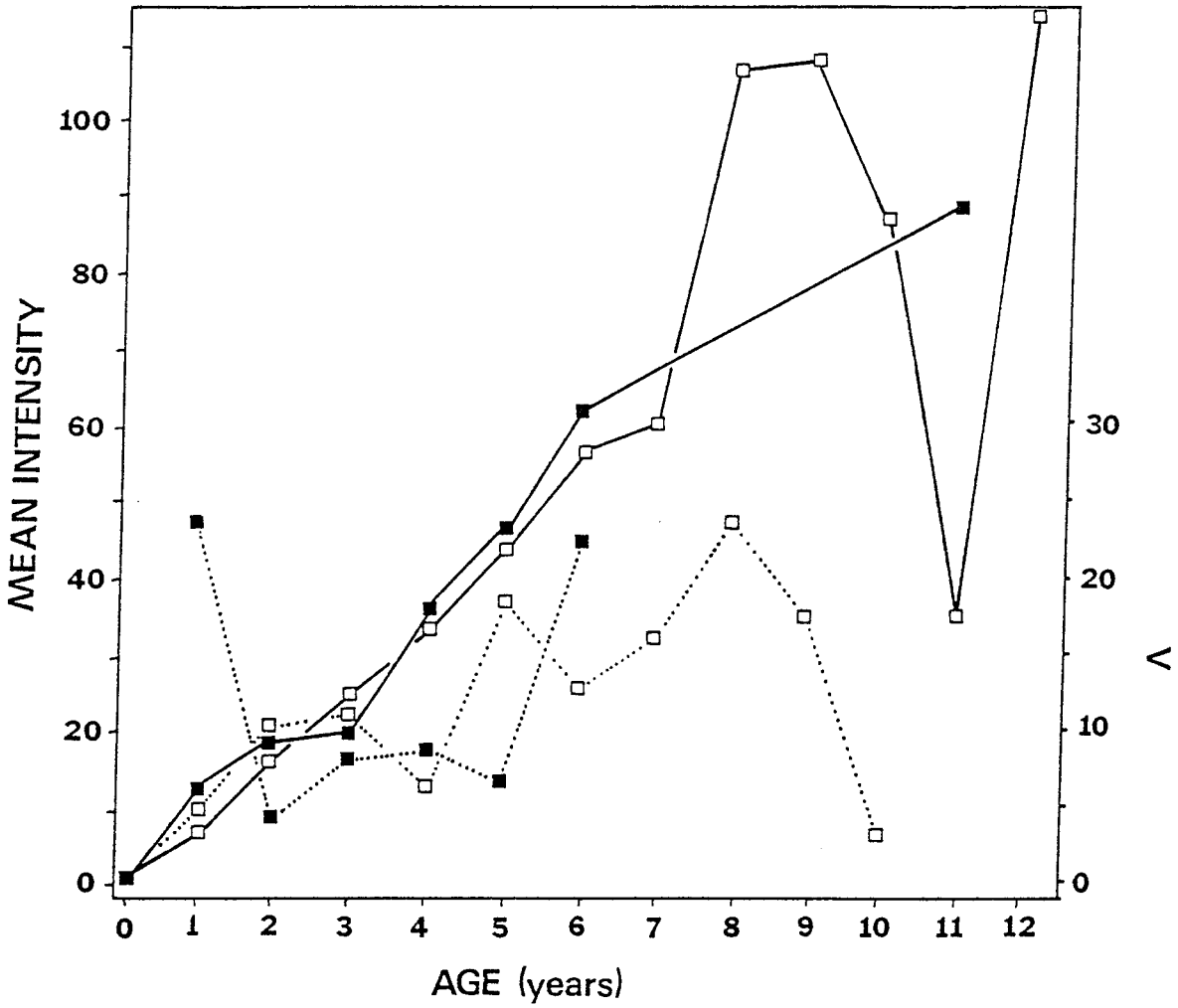
Perhaps the high proportion of immature females seen with high density infections (Fig. 32) is due to delayed initial maturation of perch. Although immature, virgin females with high D_t were smaller (36.8 ± 17.9 g, N=46) than their lightly infected counterparts (53.9 ± 36.3 g, N=48) ($\underline{P} = 0.0001$, $\underline{t}$-test), the average age of maturing virgin females with high densities of recently-formed nodules or scars (2.9 ± 0.7 years, N=40) was significantly greater than those with low densities of recently-formed nodules or scars

(2.39 ± 1.05 years, n=54) (P=0.0001, *t*-test). Furthermore, 94% of females with high larval densities were immature compared to only 25% of females with low density infections. Finally, female yellow perch from the second modal group (weight ≥80g) which I assume have survived early infection, were in better condition (FK=1.52 ± 0.349, n=139) than those from the first modal group (<80 g) (FK=1.35 ± 0.12, n=92) (P=0.001, t-test).

Parasite-induced mortality

Since the perch population in Dauphin Lake exhibits no significant fishing mortality, estimates of A represent total natural mortality. However, even after correcting for predation by pike some age-classes (especially age-2) are under-represented (Fig. 25), indicating that other proximate causes of mortality are operating. I proposed earlier that significant parasite-induced mortality occurs in yellow perch concomitant with maturation. I now provide additional evidence, albeit indirect, that infection by R. acus causes parasite-induced mortality in yellow perch. First, mean intensity of R. acus peaks at age-9 and then declines (Fig. 33); second, the variance-to-mean ratio for intensity of R. acus decreases for older age-classes (Fig. 33); and third, the slope of the regression of *log* mean intensity (*log*M) versus *log* variance (*log* V) (*log*V=0.839+[1.152 x *log*M], r^2=0.71, P=0.0011) is less than two. These traits are

Figure 33. Plots of mean intensity versus age (———) and variance-to-mean ratio (V) versus age (·····) for male (■) and female (□) yellow perch infected with R. acus.


MEAN INTENSITY
100
80
60
40
20
0
0
1
2
3
4
5
6
7
8
9
10
11
12
AGE (years)
30
20
10
0
V

considered by parasitologists to indicate parasite-induced mortality (Anderson and Gordon, 1982; Kennedy, 1984).

DISCUSSION

Size-selective predation which I attribute to northern pike has been demonstrated for other piscivorous fishes feeding on yellow perch, with smaller members of yellow perch cohorts suffering increased mortality (Nielson 1980; Post and Prankevicus 1987). Post and Prankevicus (1987) were the first to suggest that the intensity of size-selective mortality by piscivores may be controlled by the next trophic level, and that density-dependent factors may be more important than predation in determining year-class strengths in yellow perch. Although these suggestions apply to the quantity and quality of prey available to yellow perch, they relate equally well to the interaction between R. acus and yellow perch. If yellow perch with heavy infections by R. acus have reduced growth, they remain longer in the size-ranges susceptible to predation by northern pike. From the standpoint of the yellow perch, larger size should reduce the risk of capture by northern pike (Nielson 1980); and from the standpoint of the parasite, infecting perch 1-year old or older (FL$\leq$120 mm) ensures a higher rate of transmission to the definitive host, since the former harbour the highest densities of larvae and are selectively preyed upon by northern pike.

There is evidence that yellow perch must reach a critical size (80-100 mm FL and 5-10 g W) before sexually dimorphic growth will begin (Malison et al. 1985, 1986). This size

range corresponds to age-0 and age-1 yellow perch from Dauphin Lake. Furthermore, attaining a minimum body size is important in determining the initial onset of vittelogenesis and spermatogenesis in yellow perch (Malison et al. 1986). Vitellogenin synthesis is one of several energy demanding processes that occur in the liver; in yellow perch, vittellogenesis begins in early fall and continues until early spring when fish spawn (Treasurer and Holliday 1981). Immature females grow rapidly from April to May but mature females delay somatic growth until May or June due to the metabolic demands imposed by egg production (Craig 1987).

The accumulation of non-functional tissue due to infection by R. acus decreases the ability of the liver to store and process glycogen. This, combined with decreased feeding by perch in the fall (Craig 1987) and the increased energy demands of vitellinogenesis, could lead to substantial mortalities of heavily infected female yellow perch due to exhaustion of stored energy supplies. This is not unrealistic since increased mortality in female yellow perch prior to spawning due to insufficient energy storage has been reported (Newsome and Leduc 1975). Furthermore, Craig (1987) estimated that a 260 mm FL female undergoing vitellogenesis consumes 87% of the energy stored by the soma in one year. By comparison, a 225 mm FL male consumes only 10% during spermatogenesis (Craig 1987).

Based on published accounts of the life-history of yellow perch and on the evidence I have presented for parasite-

induced mortality, I suggest the following scenario for R. acus infections in yellow perch. Recruitment of R. acus likely begins within the first year of life, probably when yellow perch are about two months old, concomitant with the onset of exogenous feeding and increased use of aquatic invertebrates as food items (Craig 1987). Initially, the density of R. acus larvae is low and predation by northern pike appears to be the most important cause of mortality in both sexes of yellow perch. The intensity of larvae increases as fish grow, and males and females begin to diverge as the critical size for maturation is approached. As density of larvae is highest prior to this stage the effects on yellow perch metabolism are most pronounced. Since males are usually smaller than females they have increased weight-specific metabolic demands and increased mortality. Even though they expend less energy on spermatogenesis, small males with heavy infections succumb and larger, surviving males grow slower and are in poorer condition. Although females are generally larger than males they expend much more energy on vitellogenesis. Small females that initiate vitellogenesis eventually succumb as energy reserves are used up, while females that are initially larger or those that delay maturation to allow for increased somatic growth, survive the added stress of infection. These also have reduced growth.

Increased numbers of females versus males in populations of yellow perch has been reported by many authors (Weller

1938; Jobes 1952; Bregazzi and Kennedy 1982; Craig 1987), but no satisfactory explanation for this phenomenon has been presented. Differential mortality of the sexes has been proposed to explain the shifting sex ratio of yellow perch with age (Weller 1938; Jobes 1952; Craig 1987), but the proximate causes of this sex-related mortality were not considered. The contrasting responses of male and female yellow perch to infection by R. acus could account for the increasing proportion of females as yellow perch age. Furthermore, my interpretation could account for the presence of bimodality for the weight-distribution of female yellow perch.

I presented graphical evidence that infections by R. acus causes parasite-induced mortality in yellow perch, but these graphical methods need clarification. Anderson and Gordon (1982) cautioned that: one, peaked age-intensity curves can be generated by age-related differences in infection rates and if the lifespan of the parasite is greater than that of the host and mortality is greater than recruitment in older hosts; two, decreased variance-to-mean ratios (V/M) in older fish may be due to decreased variance of infection, acquired immunity, or spatial limitations in older fish; three, the mean and V/M could decrease due to independent but correlated causes of host mortality such as increased susceptibility to predation due to infection; and four, different genotypes of yellow perch may exhibit different growth rates or varying degrees of susceptibility. However,

the data shows that recruitment of worms, as indicated by the number of free larvae, is constant. Furthermore, I accounted for all deaths of parasites by counting recently-formed nodules and estimating the number of scars. There is no evidence for increased variance of recruitment in older fish and the very high densities of infection found in male yellow perch suggests that space is not a limiting factor. Finally, although larger yellow perch with many worms are available prey items, northern pike are size-selective for smaller fish with fewer worms. The confounding effects of genetic variance on growth and susceptibility to disease in yellow perch can not be ruled out, but Heath and Roff (1987) have shown that differences in growth between stunted and non-stunted populations of yellow perch are principally an effect of environmental variation, not genetic differentiation.

Previous authors suggest for R. acus in yellow perch, that encapsulation of L_4 larvae is temperature-dependant (Smith 1984, 1986), and there is no correlation between increasing numbers of larvae and condition of yellow perch (Poole and Dick 1984; Smith 1984). By contrast, my data clearly shows that the proportion of encapsulated larvae and nodules is not correlated with temperature, and heavily infected yellow perch are in poorer condition and heavily infected yellow perch exhibit reduced growth. Furthermore, although temperature (Henderson 1985), predation by Diacyclops sp. (Hartig and Jude 1984) and by fishes (Lyons and Magnuson

1987), and cannibalism (Tarby 1974) have been proposed to explain the large variation in growth and mortality observed in yellow perch; I have shown that a large component of this variation can be attributed to parasite-induced mortality resulting from the metabolic stresses imposed by the combined actions of: *i*), infections by R. acus; and *ii*), the metabolic demands of reproduction.

It appears that projections of growth for yellow perch requires that additive effects of parasite-induced mortality and the differential effects of parasites on male and female yellow perch be considered. Perhaps as we learn more about the direct effects of tissue invading parasites on fish growth and mortality, predictive growth models for natural populations of fish will include the effects of parasitism as a variable, and fisheries biologists will consider the presence of invasive parasites in vitally important organs as a major factor contributing to reduced growth and mortality in natural populations of fish.

CHAPTER 4-2: DIFFERENCES IN NUMBERS AND INEQUALITIES IN MASS AND FECUNDITY DURING THE EGG PRODUCING PERIOD FOR RAPHIDASCARIS ACUS.

INTRODUCTION

It is generally accepted that for most animals increased adult body size is correlated to increased fecundity and that mass is an accurate index of fecundity (Blueweiss et al., 1978). A correlation between size and fecundity exists in some plant populations but the distribution of sizes is skewed (Wilson and Levin, 1986). Botanists have applied a variety of statistical methods to analyze this phenomena, but Weiner and Solbrig (1984) argued that measures of inequality such as the Lorenz curve (LC) and Gini Coefficient (GC) are more appropriate than standardized measures of skewness when addressing questions concerning plant population structure. Briefly, a Lorenz curve is a bivariate plot of the cumulative proportion of a ranked variable (such as mass) against the cumulative proportion of individuals, while a Gini coefficient is the arithmetic mean difference for the ranked variable between all pairs of individuals. The Gini coefficient is zero when all individuals are equal (maximum equality), and one when all individuals but one have values of zero for the variable in question (maximum inequality). These measures have been used to analyze the relationship between size and fecundity (Scheiner, 1987) and the consequences of inequalities in fecundity (Wilson and Levin, 1986) for plant populations.

Most parasite populations also exist as size hierarchies of individuals and it is generally assumed for parasites

that mass is correlated to fecundity. Dobson (1986), using body size and the number of ovarian balls as indices of fecundity for the cestode Hymenolepis diminuta and the acanthocephalan Moniliformis moniliformis, respectively, was the first to apply LCs and GCs to the analysis of parasite populations. Shostak and Dick (1987) recognized the shortcomings of using an index to estimate fecundity and compared inequalities in mass with (absolute) lifetime fecundity for the cestode Triaenophorus crassus. Shostak and Dick (1987) found that inequalities in mass for T. crassus were correlated with, but always lower than, corresponding inequalities in fecundity. However, it was not clear to what extent these observations were affected by changing rates of recruitment, growth of parasites or other stochastic and deterministic factors since these could not be assessed due to synchronous egg-production by T. crassus (Shostak and Dick, 1987).

The nematode Raphidascaris acus has an annual cycle in northern pike, Esox lucius. Since this parasite has an asynchronous life-cycle and lifetime fecundity can be measured directly, samples taken at different times allow us to follow changes in inequalities in the parasite population. Furthermore, the effects of recruitment, growth, and stochastic and deterministic factors on parasite inequalities can be measured. The objectives of this paper were to determine: *i*), the extent of variation in numbers, maturation, fecundity, and mass in R. acus; *ii*), whether

inequality in size is expressed as inequality in fecundity; *iii*), to what extent stochastic and deterministic factors affect parasite size and fecundity; and *iv*), the usefulness of the Gini coefficient and the Lorenz curve in understanding the relationship between size and fecundity for R. acus.

MATERIALS AND METHODS

Pike were collected with trap nets set in 1.5 m of water in the Turtle River, an inlet stream to Dauphin Lake (Fig.1) and with gill-nets at approximately bi-weekly intervals (Table 10) in 1987. Water temperature at 1 m depth (10-22°C) was recorded at sampling time. Pike were removed within 0.5 h of capture and killed by a blow to the head; fork length, round weight, sex and maturity were noted and an ageing structure was taken from each fish. The gastro-intestinal tract of each fish was removed and placed on ice. Samples were transported to the laboratory and examined within 1 h of collection.

The intestine and stomach were slit open longitudinally, their contents recorded, and the spleen was weighed. The mucosa was scraped into a shallow tray and the intestinal contents diluted and stirred in water and examined for helminths. Individual Raphidascaris acus were collected and their sex and maturity noted where possible. Worms were classified as adult males, adult females (no eggs in the uterus), gravid females (eggs present) and immatures (sex unknown) based on the descriptions given by Smith (1984). The open ends of the intestine of 40 pike were ligatured immediately after the viscera was removed and the intestinal contents was examined to determine if R. acus shed eggs while in the host. Adult male, adult female and immature worms were temporarily stored in petri dishes containing

lake water and gravid worms were placed individually into glass vials containing lake water (8°C). Vials containing gravid females were loosely stoppered and refrigerated (8°C) for 24 h to allow for egg release.

The maximum length (to the nearest mm) and width (to the nearest 10^{-3} mm) of each worm was measured and each worm was blotted dry on a paper towel and weighed (to the nearest 10^{-4} g). After 24 h gravid females were removed from storage and weighed and their uteri examined for residual eggs. The number of eggs released in 24 h by each worm was estimated by calculating the average number of eggs in five 10 µl samples and adjusting for dilution. The stage of development of eggs was noted.

The frequency distribution of R. acus individuals among pike for each sample period and for the combined data and the frequency distribution of fecundity for gravid worms were fitted to negative binomial frequency distributions. To measure size and fecundity hierarchies Gini coefficients (adjusted for sample size) were employed and graphically portrayed using Lorenz curves (Dobson, 1986; Weiner and Solbrig, 1984). Gini coefficients were calculated for infrapopulations, metapopulations at each sample time and for the pooled metapopulation of gravid female R. acus in pike. Gini coefficients determined for metapopulations using fecundity are called G'_{Fm} and those using mass are called G'_{Mm}. The corresponding values averaged over several infrapopulations are G'_{Fi} and G'_{Mi}, respectively.

Negative binomial distributions were fitted to the data using APL and all other analyses were performed using SAS. Gravid female worms were separated into two groups based on fecundity and stepwise discriminant analysis (Bennet and Bowers, 1976) was used to choose an independant variable that best predicted the group membership of each worm.

Data are presented as $\bar{X} \pm$ S.E. unless otherwise indicated. Prevalence, mean intensity and infrapopulation are used as in Margolis et al. (1982). The term metapopulation (Riggs et al., 1987) refers to all infrapopulations sampled from a given host species within an ecosystem. Fecundity is the total number of eggs released by an individual, whereas an index of fecundity is any measurement assumed to be positively correlated with fecundity when the exact relationship is not known.

RESULTS

Worm numbers

Three thousand one hundred and sixteen R. acus were collected from 232 pike (15.9 ± 18.5 worms per fish, prevalence= 85%). The frequency distribution for R. acus pooled across all pike and sample periods fit a negative binomial distribution (k=0.436, p=4.227; P=0.022). As the season progressed intensity and prevalence declined (Table 10) and the frequency distribution became less skewed (Fig. 34). The number of immature worms increased through April (Fig. 35A) but was significantly reduced (P<0.025, t-test) in mid-May (3.7 ± 4.3, N=15). Numbers increased significantly (P<0.125, t-test) by late May (7.5 ± 11.0, N=21), but declined steadily therafter. Generally, immature worms accounted for an increasing proportion of the metapopulation as the season progressed. The pattern for adult males (Fig. 35B) and adult females (Fig. 35C) was similar with maximum intensity and prevalence occurring in spring followed by steadily decreasing numbers towards autumn. Gravid worms (Fig. 35D) had peak intensity and prevalence in mid-May, declining numbers in June, and lower levels throughout the summer and autumn; 41% of all gravid worms were found in May.

Table 10. Changes in intensity, prevalence and range of infection for Raphidascaris acus in pike from Dauphin Lake.

Sample period	Number of fish	Intensity $\bar{X} \pm$ S.D.	Prevalence (%)	Range
April 14	43	23.9 ± 21.9	98	1 - 104
April 29	39	23.7 ± 21.5	92	1 - 85
May 13	20	22.6 ± 18.3	100	3 - 72
May 31	33	13.5 ± 17.6	97	1 - 63
June 25	26	8.3 ± 7.2	92	1 - 31
July 20	31	4.8 ± 5.7	87	1 - 27
August 19	20	4.3 ± 3.4	60	1 - 11
September 25	20	2.0 ± 2.0	20	1 - 5
Pooled	232	15.9 ± 18.5	85	1 - 104

Figure 34. Changes in the frequency distribution for Raphidascaris acus in pike from Dauphin Lake. Maximum likelihood estimates for the parameters k and p of the negative binomial distribution and values of P are given where variance > mean. LCL, lower class limit.

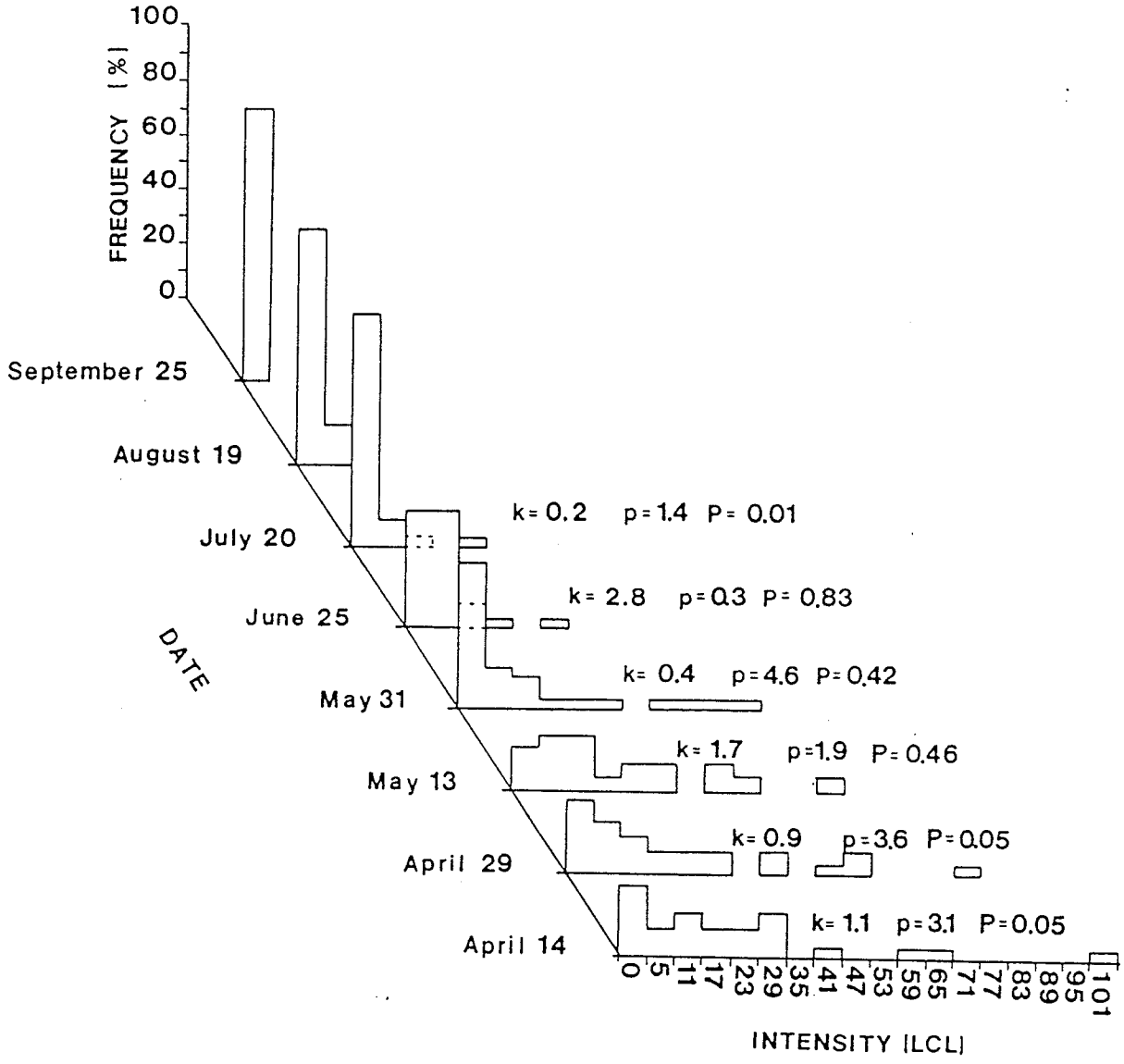
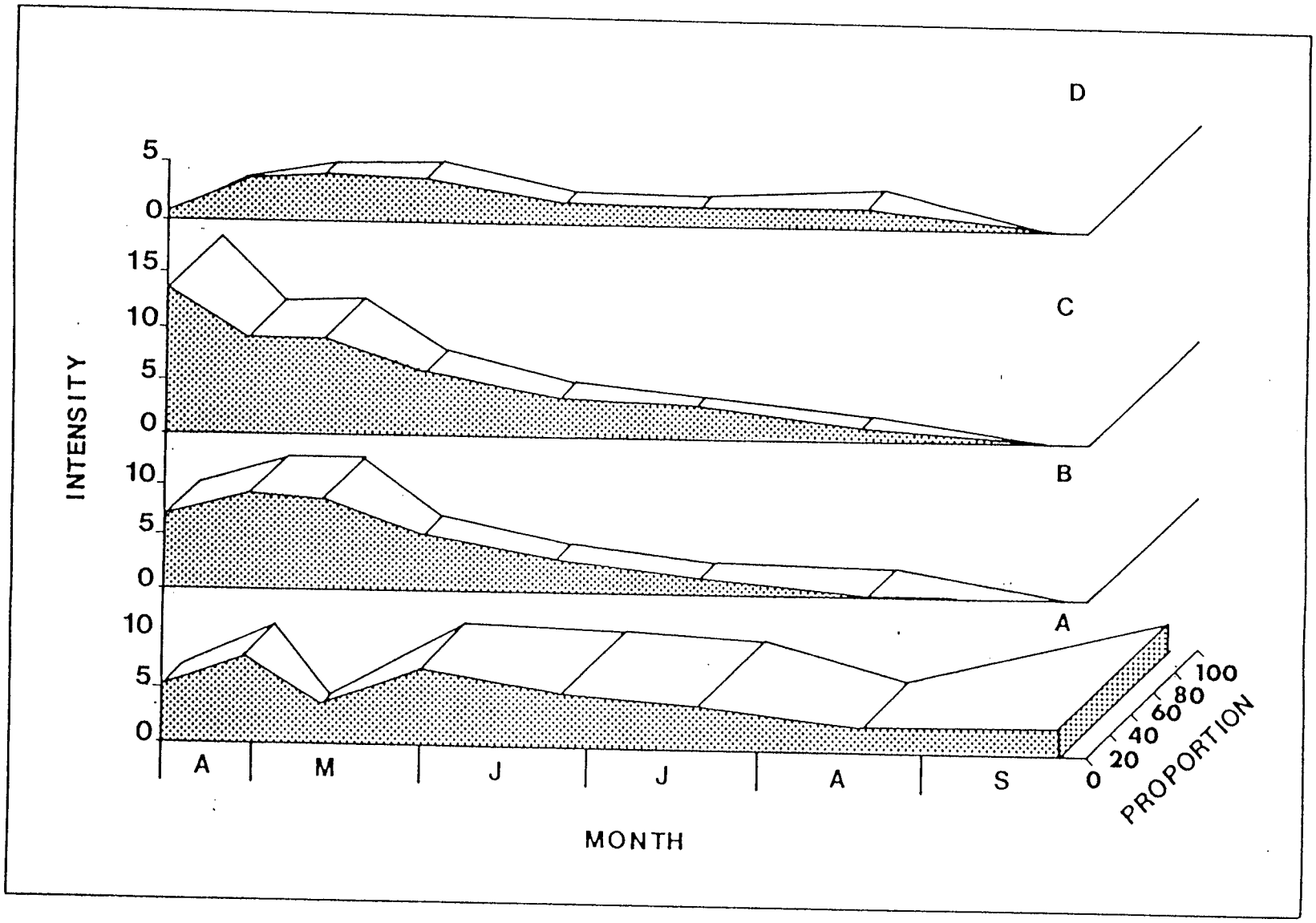

100
80
60
40
20
0
FREQUENCY [%]
September 25
August 19
July 20
June 25
May 31
May 13
April 29
April 14
DATE
k= 0.2 p= 1.4 P= 0.01
k= 2.8 p= 0.3 P= 0.83
k= 0.4 p= 4.6 P= 0.42
k= 1.7 p=1.9 P= 0.46
k= 0.9 p=3.6 P=0.05
k= 1.1 p=3.1 P=0.05
0 5 11 17 23 29 35 41 47 53 59 65 71 77 83 89 95 101
INTENSITY [LCL]

Figure 35. Changes in intensity and proportion of the metapopulation (%) for Raphidascaris acus in pike from Dauphin Lake. A. Immature worms. B. Adult male worms. C. Adult female worms. D. Gravid female worms.


D
C
B
A
INTENSITY
5
0
15
10
5
0
10
5
0
10
5
0
A
M
J
J
A
S
0
20
40
60
80
100
PROPORTION
MONTH

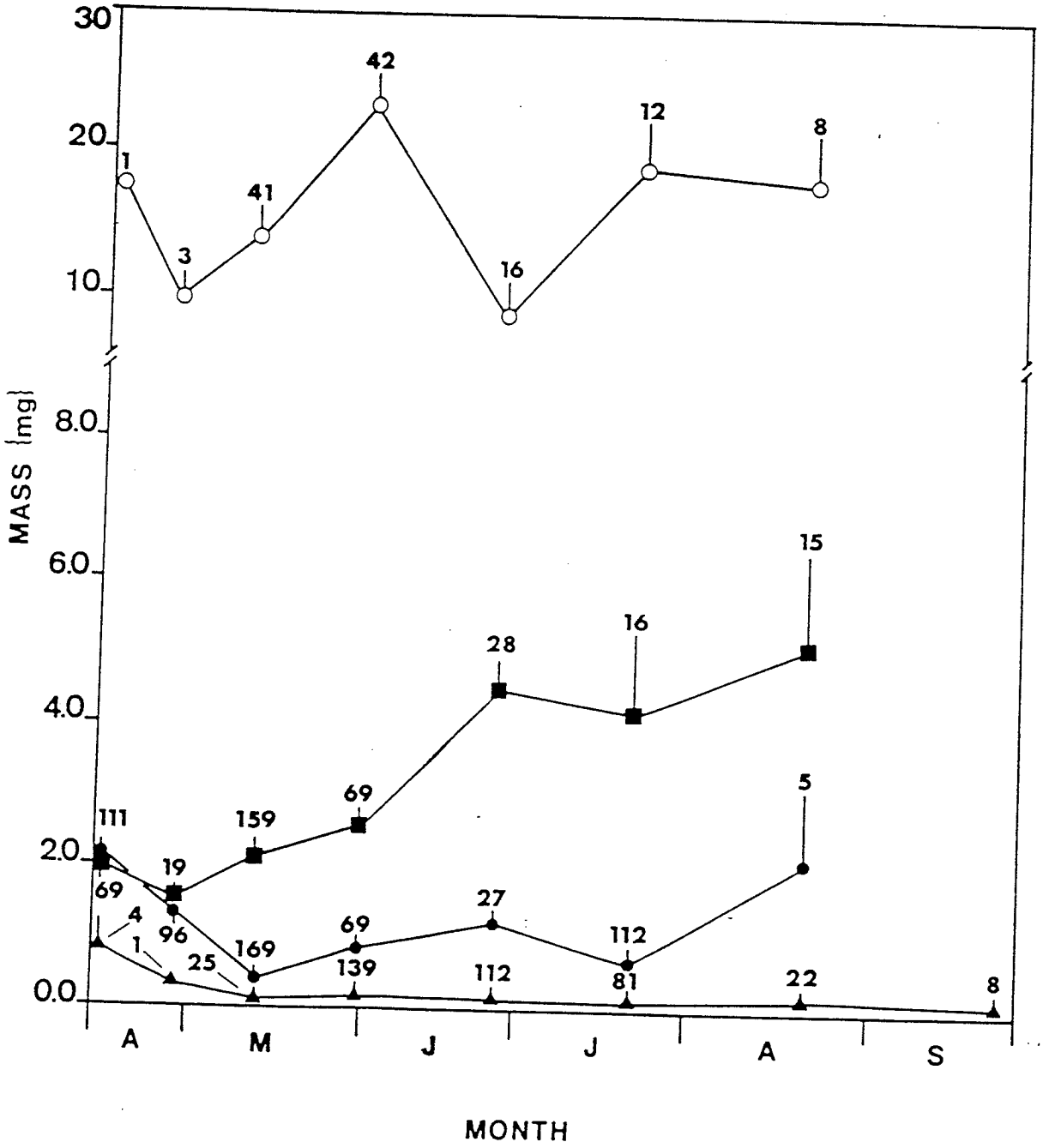
Worm mass

The mass per worm was 0.14 ± 0.49 mg (N=392, range= 0.1 - 9.6 mg) for immature worms, 2.39 ± 2.93 mg (N=450, range= 0.1 - 20.8 mg) for adult male worms, 1.10 ± 1.63 mg (N=479, range= 0.1 - 14.9 mg) for adult female worms and 18.91 ± 12.27 mg (N=127, range= 0.7 - 61.2 mg) for gravid female worms.

There were significant differences in the mass of adult males (F=8.36, P=0.0001), adult females (F=15.98, P= 0.0001), and gravid females (F=5.13, P=0.0001) between sample periods but no differences in the mass of immature worms (ANOVAs) (Fig. 36). To test for density-dependant effects on worm mass, worms were split into two groups based on intensity of infection. Infrapopulations with ≥40 worms (more than twice the population mean) accounted for approximately 10% of all the worms collected, and these were compared to the remaining worms (90%) from lighter infections. The mass of adult male and adult female worms varied with infection intensity (F= 4.18 and F=5.05, respectively. $P < 0.05$) but there were no significant difference in the mass of immature and gravid worms in heavy versus light infections (ANOVAs). Spleen weight was correlated with intensity of infection (r= 0.36, P= 0.0003) but not with worm mass (Pearson correlation coefficients).

Changes in water temperature experienced for a given sample period were followed by corresponding changes in the mass of adult male (r=0.23, P=0.0001) and gravid female

Figure 36. Changes in mass for immature (▲), adult male (■), adult female (●), and gravid female (○) Raphidascaris acus from pike in Dauphin Lake. Points and bars represent $\bar{X} \pm$ S.E., respectively. Sample sizes are given. Note changing scale.


30
20
10
8.0
6.0
4.0
2.0
0.0
MASS (mg)
A
M
J
J
A
S
MONTH
1
3
41
42
16
12
8
111
69
4
1
25
19
96
159
169
69
139
69
112
28
16
15
27
112
81
5
22
8

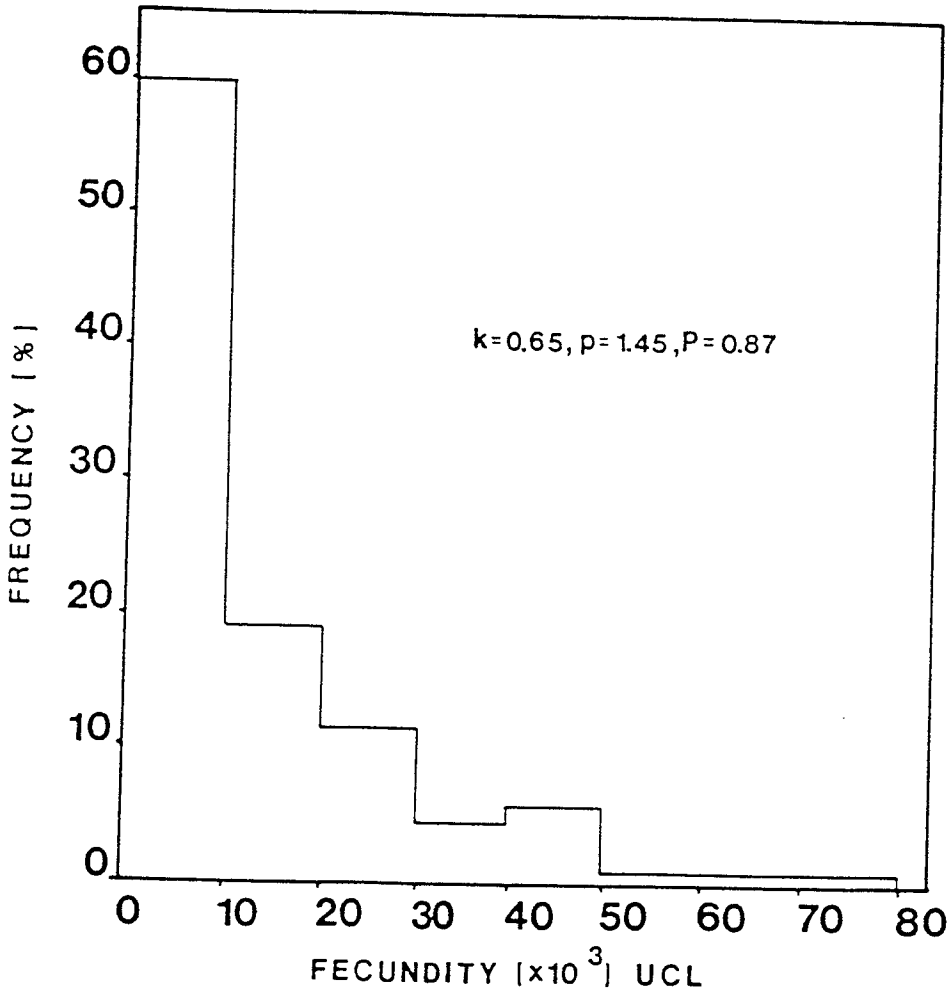
($\underline{r}$=0.32, $\underline{P}$=0.0001) worms during the subsequent sample period. In particular, periods of rapid increase in water temperature (11 to 16°C from 14 April to 13 May, and 13 to 21°C from 31 May to 25 June, respectively) were followed by periods when growth rate of R. acus was most rapid (0.426 mg/day from 29 April to 31 May and 0.407 mg/day from 25 June to 20 July). With these rates of growth larval worms would attain their maximum weight (18.91 ± 12.27 mg for gravid females) 15 to 78 days after ingestion by a pike.

Worm fecundity

Worms isolated from the intestine of pike were covered by a thick layer of mucous and gravid worms released eggs into water only after removal of this mucous layer. Most eggs were shed in the four to eight cell stage and only rarely were incompletely developed eggs found. None of the gravid worms showed signs of uterine prolapse during egg collection and eggs were never found in the intestines of pike harboring gravid worms.

A total of 122 of 127 gravid female worms released eggs and the average fecundity of these was 13,763±14,225 eggs per worm (range= 60-71,520). Egg release relative to body size (Appendix IV) was 699±664 eggs/mg (N=112, range= 1-4,696). Overall the distribution of fecundity was skewed and fit a negative binomial distribution (Fig. 37). Gravid worms found in May (20.4 ± 12.8 mg, 15,213 ± 15,414 eggs per worm. N=85) and those found between July and August (19.4±10.5 mg,

Figure 37. Frequency distribution for fecundity of individual Raphidascaris acus from pike in Dauphin Lake. UCL, upper class limit.


60
50
40
30
20
10
0
FREQUENCY [%]
k=0.65, p=1.45, P=0.87
0
10
20
30
40
50
60
70
80
FECUNDITY [x10 3] UCL

13,918 ± 13,018 eggs per worm. N= 20) were larger and more fecund than those present in June (10.7 ± 8.4 mg, 6,912 ± 4,637 eggs per worm. N= 16) (*t*-tests, $P < 0.05$) and the variability in fecundity was lowest for worms collected in June (*F*-tests, $P< 0.05$). The number of eggs per worm was not related to intensity of infection or spleen weight (ANOVAs), but all other measured variables (width, length and mass of worms, water temperature and fish sex and length) were significantly correlated with fecundity (Pearson correlation coefficients, $P< 0.05$). Mass had the highest correlation with fecundity ($r= 0.534$, $P= 0.001$) and explained 50 ± 38% (range= 7-95%) of the variation in fecundity within any monthly sample, and 56 ± 33% (range= 0-99%) at the infrapopulation level. When the data were pooled mass explained only 28% of the total variation in fecundity (Appendix IV).

Gravid worms could be separated into two groups; a large group with low fecundity (≤ 10,000 eggs per worm) and a smaller group with high fecundity (> 10,000 eggs per worm) (Fig. 37). A stepwise discriminant analysis based on worm mass, intensity of infection, pike fork length and time of capture identified worm mass as the best variable for discriminating among low and high fecundity worms. Discriminant Function Analysis correctly classified 74% of the gravid worms based on knowledge of mass only.

Inequalities in mass and fecundity

Gini coefficients (Table 11) calculated separately for each infrapopulation in 45 pike harboring gravid female worms showed that average inequalities in fecundity (G'_{Fi}= 0.73 ± 0.32, range= 0.04 - 1.0) were consistently larger than those for mass (G'_{Mi}= 0.60 ± 0.41, range= 0.05 - 1.0) (t-test, $P < 0.05$). The metapopulation collected on June 25 was the only one where G'_{Fm} was lower than G'_{Mm} (Table 11) and this was also the only time when the coefficient of variation for mass was greater than that for fecundity (Table 11) and aggregation was reduced (Fig. 34). Lorenz curves (Fig. 38) showed that these inequalities arose from a small number of highly fecund or heavy worms. Overall, the most fecund 10% of the R. acus metapopulation produced about 33% of all the eggs, while the heaviest 10% of the worms provided about 25% of all the biomass (Fig. 38H).

Table 11. Changes in the Gini coefficient (G') for fecundity (G'_F) and mass (G'_M) of gravid female Raphidascaris acus in pike from Dauphin Lake.

Sample[a] period	G'_{Fm}	G'_{Fi}[b] [C.V.][c]	G'_{Mm}	G'_{Mi} [C.V.]	
April 14	1.00	1.00 ± 0.00 (1)	1.00	1.00 ± 0.00 (1)	NS[d]
April 29	0.77	0.77 ± 0.00 (1) [127.7]	0.21	0.21 ± 0.00 (1) [32.6]	NS
May 13	0.51	0.51 ± 0.33 (11) [109.3]	0.25	0.39 ± 0.33 (10) [51.3]	NS
May 31	0.50	0.72 ± 0.30 (11) [89.7]	0.30	0.55 ± 0.44 (11) [59.0]	NS
June 25	0.39	0.77 ± 0.34 (7) [67.1]	0.52	0.73 ± 0.39 (8) [79.3]	NS
July 20	0.51	0.82 ± 0.30 (9) [101.1]	0.31	0.70 ± 0.45 (9) [57.6]	NS
August 19	0.47	0.83 ± 0.37 (5) [88.6]	0.26	0.78 ± 0.44 (4) [51.1]	NS
Pooled	0.56	0.73 ± 0.32 (45)	0.41	0.60 ± 0.41 (44)	P <.05

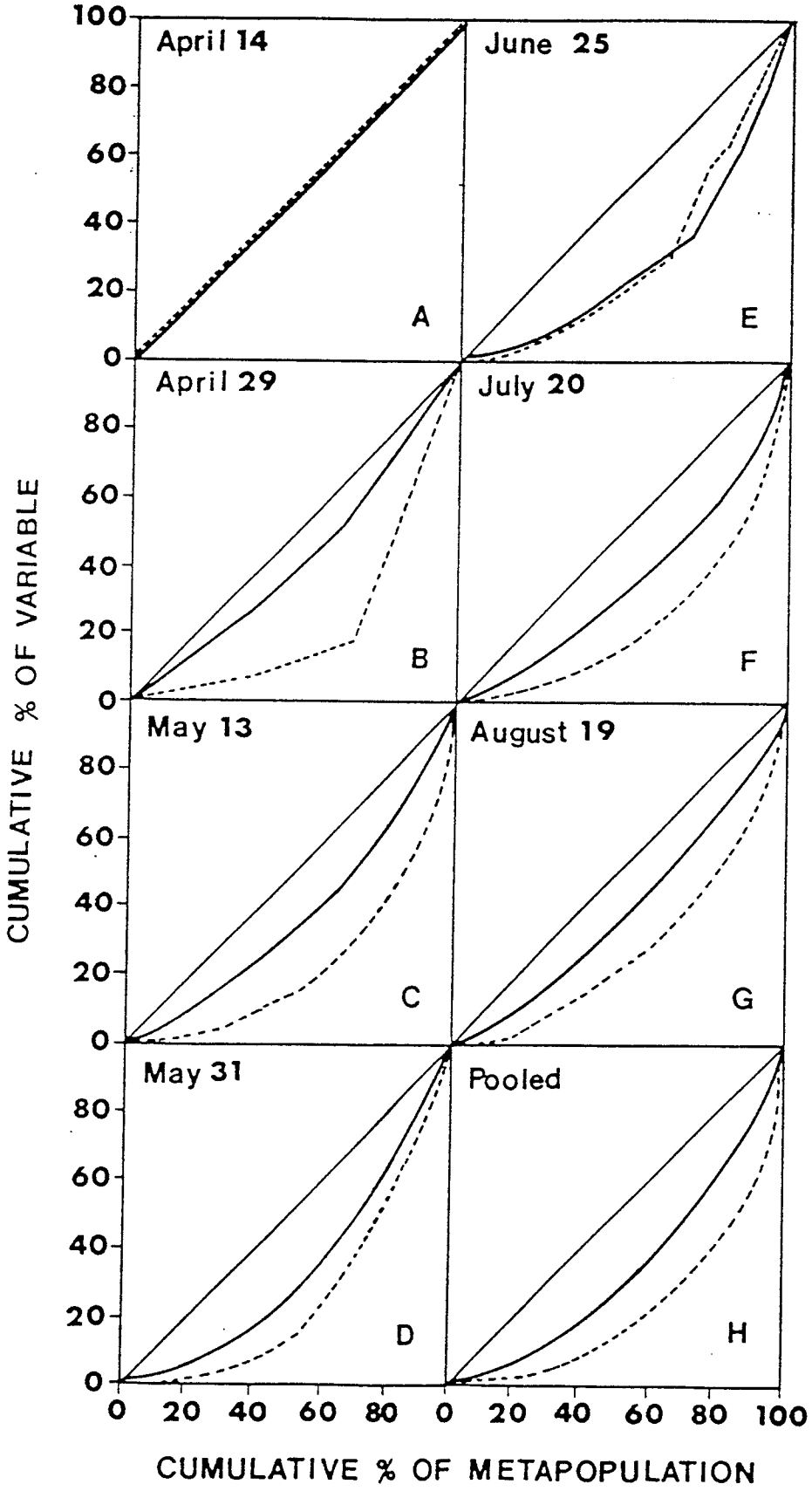
[a] no gravid worms found on September 25.

[b] $\overline{X}$ ± S.D. (n).

[c] C.V.,coefficient of variation.

[d] NS, not significant.

Figure 38. Lorenz curves for fecundity (-----) and mass (———) of gravid female Raphidascaris acus metapopulations at different dates (A-G) and for the pooled data (H). Gini coefficients for fecundity and mass are given in Table 11. Thin diagonal lines represent perfect equality.


April 14
A
June 25
E
April 29
B
July 20
F
May 13
C
August 19
G
May 31
D
Pooled
H
0
20
40
60
80
100
CUMULATIVE % OF VARIABLE
CUMULATIVE % OF METAPOPULATION

DISCUSSION

Prior to discussing the ecological consequences of morphological variability for R. acus as it relates to its reproductive potential, I consider (*i*) the biology of this parasite in Dauphin Lake, (*ii*) the effects of various biotic and abiotic factors on variation within infrapopulations and metapopulations of R. acus and (*iii*) the relationship between mass and fecundity as measured using the Lorenz curve and the Gini coefficient.

Changes in numbers

The number of immature worms in northern pike decreased significantly in May, suggesting that recruitment of R. acus in Dauphin Lake occurs predominantly in early spring and again in late summer. The spring peak in intensity of immature worms is likely the result of an accumulation of worms in pike over winter, when recruitment rates are low and the feeding period is prolonged. The second prolonged phase of recruitment is due to increased and intense feeding by post-spawning pike on yellow perch (Perca flavescens), as the number of yellow perch consumed by pike in Dauphin Lake is generally greatest in early spring and early autumn (Appendix I). These two annual sub-populations of immature R. acus in pike account for the number of gravid females since numbers of the latter peak two to eight weeks after corresponding increases in the numbers of immature worms.

Intensity decreased rapidly after May primarily due to loss of mature worms, since recruitment of new worms continued. Declining numbers of immature worms after May were due mainly to reduced recruitment but was also a consequence of more rapid growth rates for worms at higher temperatures. Smith (1984) states that R. acus develops to maturity in pike (experimental infections at 12-14°C) in about one month and from my data I estimate that R. acus in Dauphin Lake would attain maximum size in 15 to 78 days. These results indicate that R. acus continues to grow after reaching maturity. Therefore, adult and gravid worms collected in early April must represent the immature worms recruited by pike over winter and early spring, while those found in August represent immature worms recruited in June and July. Furthermore, adult and gravid worms found in late June and early July when the two sub-populations overlap represent primarily the large fecund worms of the spring peak and the smaller less fecund worms from the fall, respectively.

Most models describing size-fecundity relationships (Smith and Fretwell, 1974) assume that the energy available for reproduction is limited and that fecundity is therefore density-dependent. This is an unlikely situation for R. acus since the intestine of pike appears to have a surplus of nutrients for parasites (Shostak, 1986), and since these worms continue to grow after maturation with little concomitant increase in fecundity. Consequently, one reason

for the weak correlations between fecundity and mass observed for R. acus may be that growth of worms in pike is not restricted by the energy demands of egg production. My calculations showing that R. acus develops very quickly suggests that the turnover rate for this parasite is rapid, and this can further reduce recruitment of worms (Smith, 1986). Finally, the changing physiological condition of the hosts as the season progresses may be involved in decreasing the intensity of R. acus in pike during the summer. The relationship between spleen weight and parasite intensity in pike is no surprise, as this organ is involved in the immune responses of fish and antibody production by fish is related to temperature (Finn, 1970).

Inequalities in mass and fecundity

I found that mass is not a reliable index of fecundity for R. acus and in most cases inequalities in fecundity measured using the Lorenz curve and Gini coefficient were greater than corresponding inequalities in mass. Similar results have been reported for Triaenophorus crassus in pike (Shostak and Dick, 1987) but My results indicated, for R. acus in Dauphin Lake, that similarities between inequalities in mass and fecundity measured using the Lorenz curve and Gini Coefficient were affected by the coefficient of variation for these variables. Weiner and Solbrig (1984) stated that the Gini coefficient is the preferred summary statistic as it scales the skew to account for the mean, but

also cautioned that highly skewed frequency distributions would not be of interest if the coefficient of variation of sizes was very low. Similarily, Dobson (1986) pointed out that although the Gini coefficient and the Lorenz curve are both realistic indices of inequality for comparing populations with different means they should always be used together, since Lorenz curves for populations with many large individuals and many small individuals can have similar Gini coefficients. I have shown that one must be cautious when interpreting these indices as similarities between inequalities based on mass and fecundity may be due to extreme variation in one or both of the variables being considered and not due to some presumed biological dependence between egg production and body size. In addition, when working with long-lived organisms, measurements made on individuals in a single sampling period may not adequately characterize those individuals. This is especially true with traits such as fecundity that can be size or age dependant.

The value of the Gini Coefficient and Lorenz curve may be more limited than previously thought, especially when the biology and nature of variability of the parasite population is not fully understood. Even when the effects of growth, recruitment and other biotic and abiotic factors were considered I was still unable to show a strong statistical correlation between fecundity and mass for the entire egg-producing population of R. acus. The weak statistical

correlation between mass and fecundity for R. acus means that an index of fecundity may have little predictive value for estimating the fitness of a parasite. If further studies confirm, for other parasite systems, that allometric correlations do not accurately represent biological relationships among life-history parameters, then interpretations based on statistical inference and limited biological data will add little to our understanding of parasite populations.

CHAPTER 4-3: THE ROLES OF ENVIRONMENTAL HETEROGENEITY AND SKEWED FECNDITY DISTRIBUTIONS IN THE MAINTENANCE OF GENETIC VARIATION IN *RAPHIDASCARIS ACUS*

INTRODUCTION

It is generally accepted that biological evolution depends on changes in fitness due to genetic differences in survival, mating success, and fecundity. There is evidence that fecundity is heritable (Neel and Schull, 1972) and that fecundity is correlated with heterozygosity (Hamrick et al., 1979), and it is known that the fecundity distribution is skewed for many biological populations (Wilson and Levin, 1986; Neel and Schull, 1972). In particular, fecundity distributions for various parasites (Shostak and Dick, 1987; Dobson, 1986; Chapter 4-3) fit the negative binomial distribution. Nevertheless, most genetic theory is based on the assumption that fecundity fits a normal approximation to the Poisson distribution (Wilson and Levin, 1986) and that fecundity differs little among genotypes (Falconer, 1981). Since a highly skewed fecundity distribution violates many of the assumptions made in standard selection theory (Falconer, 1981), alternative approaches must be taken. Wilson and Levin (1986) used computer simulations to show that, with skewed fecundity distributions, plants respond to selection faster and have increased variation compared to Poisson based theory.

The parasitic nematode Raphidascaris acus from pike (Esox lucius) has high mean fecundity and variance for fecundity, and populations of R. acus maintain a fecundity distribution that fits the negative binomial distribution (Chapter 4-2).

R. acus matures in pike. Adult males and females copulate and gravid females shed eggs which are ingested by a variety of invertebrate and vertebrate intermediate hosts (Smith, 1986), mainly yellow perch (Perca flavescens). Eggs kept at 8-10°C remain viable for more than a year and it is thought that larvae can survive in perch for up to two years (Smith, 1986).

In this chapter I show, using computer simulations based on empirical data, how stochastic processes can create a situation that retards the evolutionary process for parasites. By maintaining increased genetic variation, populations of R. acus can persist for longer periods of time than expected for unregulated populations of parasites.

MATERIALS AND METHODS

I simulated 20 infrapopulations of R. acus and examined the influence of various random effects on the fecundity distribution for successive generations of progeny. All simulations were based on a one locus-two allele (F and f) model. The fecundity of a parasite, and hence it's position in the dominance hierarchy (Wilson and Levin, 1986), was assumed to be determined by both genotype and environment. Since natural selection depends on additive genetic variance (Falconer, 1981) the genotype (G) was divided into additive (G_A) and dominance (G_D) components so that $G=G_A+G_D$ for all individuals. Values of G_A were fixed at 0, 6, and 12 for homozygous recessive (ff), heterozygous (Ff and fF), and homozygous dominant (FF) genotypes, respectively. Individuals bearing the FF genotype are most fecund. A dominance effect ($G_D=2$) was added when calculating G for heterozygotes.

In this model the environment was subject to random fluctuations from several sources and these fluctuations were assumed to be long term in relation to the length of one parasite generation. An environmental effect (E) on parasite fecundity (plasticity) was represented by a random number (range=1.0-10.0) drawn from a uniform distribution, where 0.0 and 10.0 represented the worst and best environments, respectively. Finally, the fecundity (F') of each of the progeny was calculated using the equation

$\underline{F}' = (\underline{G}_A + \underline{G}_D + \underline{E}) \times \underline{M}$, where $\underline{M}$ is a fixed variable ($\underline{M}$=100) used only to scale the results. At the start of each simulation: the distribution of alleles fit a Hardy-Weinberg distribution, the proportion of dominant alleles ($\underline{p}^D$) was equal to 0.5, and 50% of the adult worms were female.

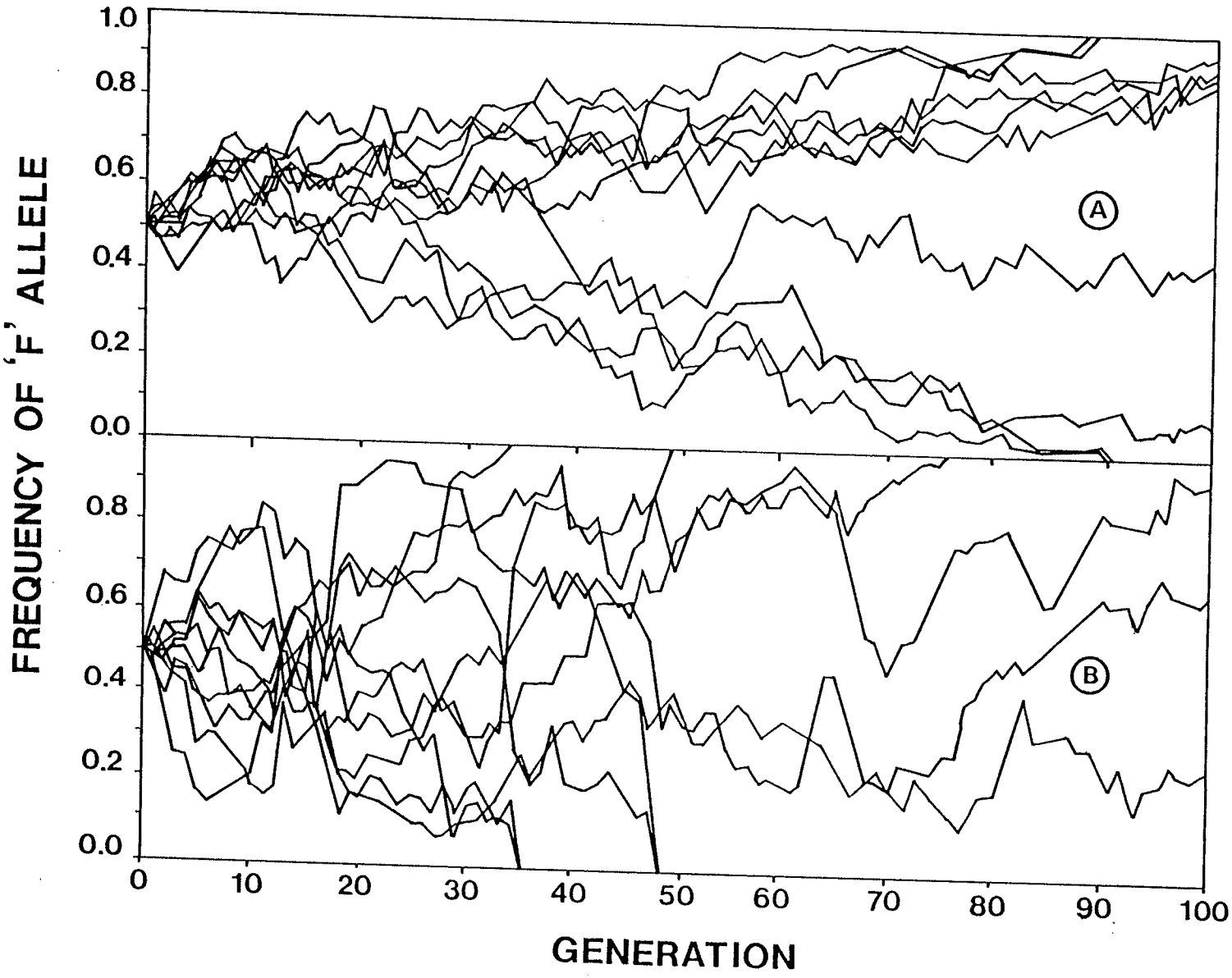
Changes in $\underline{p}^D$ and the mean, coefficient of variation, and skewness of the fecundity distributions for ten control infrapopulations (200 adults per generation, no mortality of adults, 100% survival of larvae, random $\underline{E}$) were followed for 100 generations. These were compared to values for ten chaotic infrapopulations influenced by a combination of simultaneous random effects; in addition to $\underline{E}$ the number of adults, mortality of adults, and survival of larvae (0-200, 0-50%, and 0-50%, respectively) were allowed to vary randomly and independently each generation.

For these comparisons I was less concerned with the shape of the resulting fecundity distributions than whether or not these were positively skewed, since the selective process is not qualitatively changed by the degree of skewness of fecundity distributions (Wilson and Levin, 1986). Sample skewness was measured using the formula:
$n/(n-1) \times (n-2)\sum(X_i-\bar{X})^3/S^3$ where n=number of $\underline{R}$. $\underline{acus}$, X_i and $\bar{X}$ are the fecundity of worm i and mean fecundity of the population, respectively, and S is the standard deviation. All simulations were based on programmes written in SAS (Appendix V).

RESULTS

Control infrapopulations showed the expected (Falconer, 1981) dispersion of gene frequencies and associated drift (Fig. 39A). By generation 100, four infrapopulations were fixed or lost and only six infrapopulations were still segregating (Fig. 39A). Infrapopulations experiencing chaotic conditions responded faster but more erratically to selection, fluctuations in gene frequencies due to drift were more pronounced, and only three lines were still segregating by generation 100 (Fig. 39B). Selection for the dominant allele (F) lead to increased mean fecundity (Fig. 40A) but decreased the coefficient of variation (CV) for fecundity (Fig. 40B). Conversely, selection against F resulted in decreased mean (Fig. 40A) and increased CV (Fig. 40B) for both control and chaotic infrapopulations. Generally, chaotic infrapopulations responded much faster (Fig. 40A,B). Positively skewed fecundity distributions occurred rarely when F was fixed (Fig. 41B) but frequently when F was being lost from the gene pool (Fig. 41A). In the latter case, skewed fecundity distributions were maintained for up to 90 generations.

Figure 39. Changes in the frequency of the dominant allele (F) for fecundity in 10 populations of R. acus experiencing (A) random environmental variation (control populations) and (B) where the environment, number (0-200) and mortality (0-50%) of adults, and survival of larvae (0-50%) varied simultaneously (chaotic populations). In all simulations the maximum number of adult worms was set to 200 (50% females), a single locus with two alleles (F and f) was used, and no selective advantage was assumed.


FREQUENCY OF 'F' ALLELE
1.0
0.8
0.6
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0.2
0.0
A
B
0
10
20
30
40
50
60
70
80
90
100
GENERATION

Figure 40. Changes in mean fecundity (A) and coefficient of variation (B) for two control infrapopulations (thin lines) and for two chaotic infrapopulations (thick lines) of R. acus. The fate of the dominant allele is indicated.

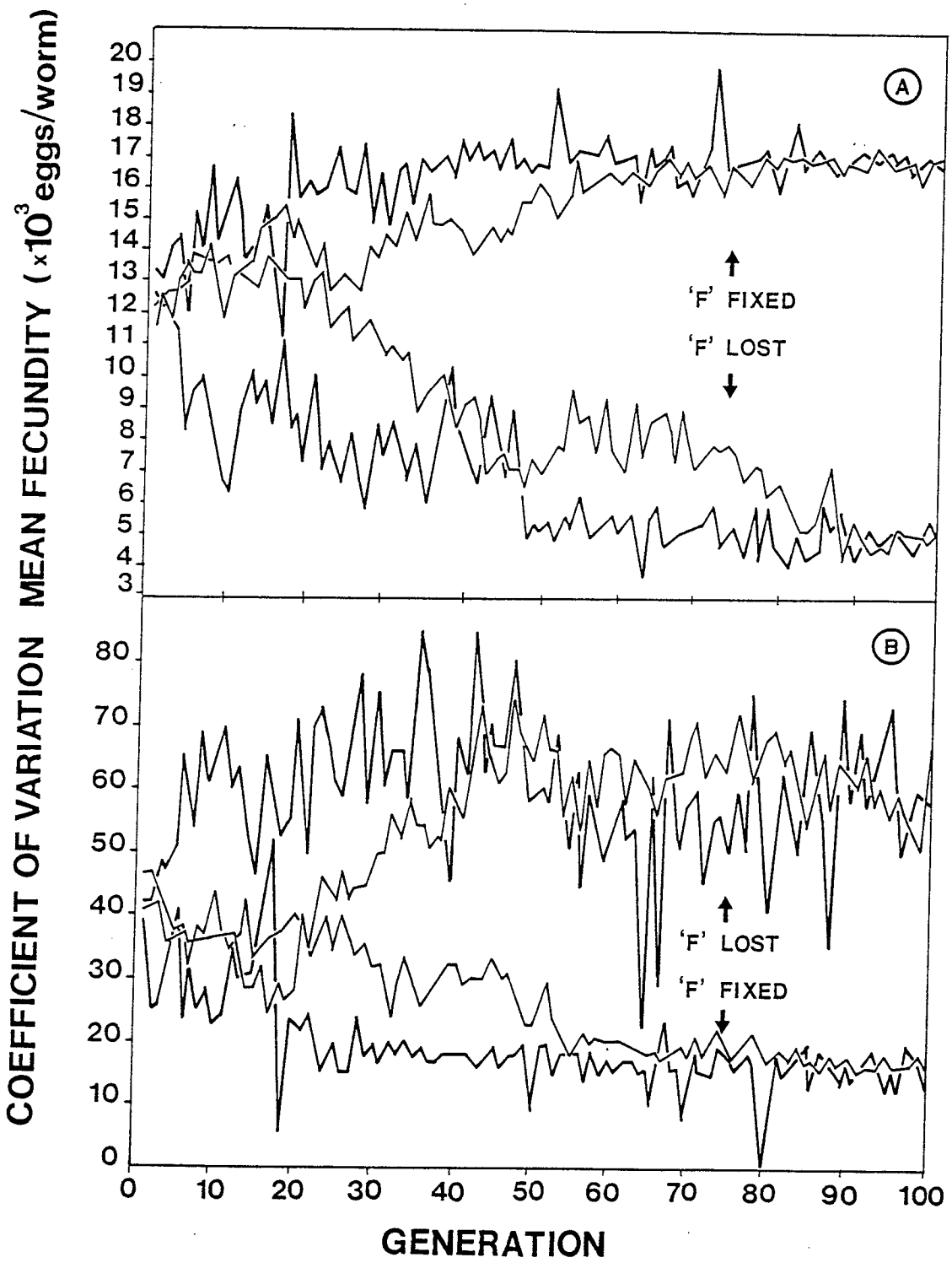
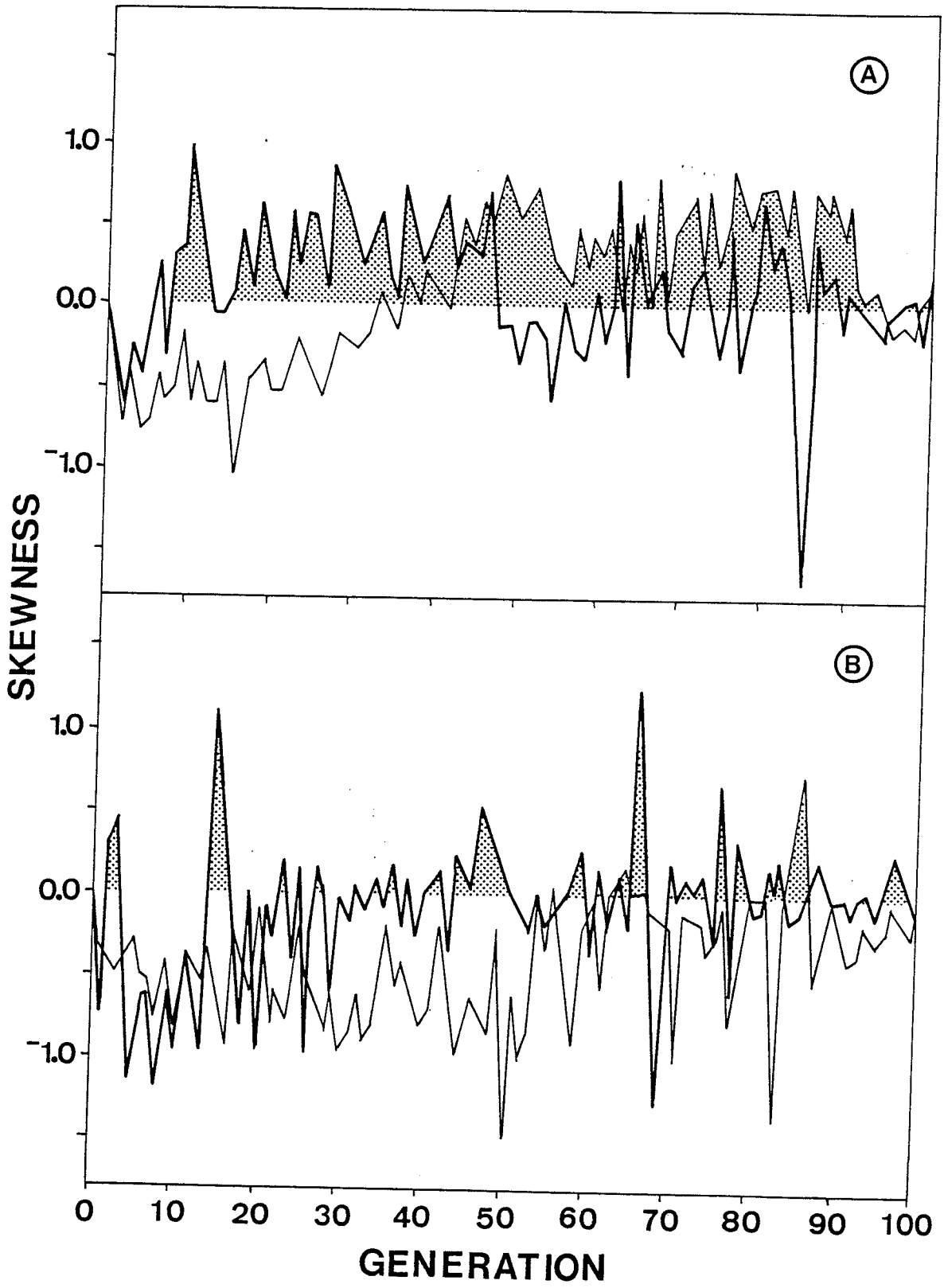

COEFFICIENT OF VARIATION MEAN FECUNDITY (x10³ eggs/worm)
A
'F' FIXED
'F' LOST
B
'F' LOST
'F' FIXED
GENERATION
0
10
20
30
40
50
60
70
80
90
100

Figure 41. Changes in skewness for the fecundity distribution for the control (thin lines) and chaotic (thick lines) infrapopulations of R. acus shown in Figure 2. Periods when the fecundity distribution was positively skewed are indicated by shading. A; F lost. B; F fixed.


A
B
1.0
0.0
-1.0
SKEWNESS
0
10
20
30
40
50
60
70
80
90
100
GENERATION

DISCUSSION

It is generally accepted that size of most organisms is correlated with fecundity and survival (Calder, 1984) and that selection operating on differences in sizes between individuals leads to rapid genetic change (Wilson and Levin, 1986). However, since there is only a weak correlation between size and fecundity for R. acus (Chapter 4-2) it is unlikely that an evolutionary strategy based on differences in worm size is operating on this species. I have shown, using a one locus diploid model with heritable fecundity, that prolonged periods of skewness of the fecundity distribution (Fig. 41 A) can be maintained for R. acus if selection is against the dominant allele and if several independant and unpredictable environmental effects are operating (Fig. 40). I caution, however, that this interpretation is based on the assumption that fecundity is heritable and that consequently, variation in fecundity is a measure of genetic variation for a population. Before discussing the mechanisms that might be responsible for the maintenance of genetic variation for R. acus, I provide evidence, albeit indirect, that R. acus does have a high degree of genetic variation.

Genetic variation is usually estimated based on average heterozygosity (H) for electrophoretically detectable allozymes (Powell, 1975), and these have been analyzed for a wide range of non-parasitic invertebrates. Nevo (1978) found

that in general, heterozygosity for invertebrates ($\underline{H}$=0.122) is higher than that for vertebrates or plants. Similarly, Hamrick et al. (1979) reported that genetic variance for invertebrates is roughly equivalent to that for plants ($\underline{H}$=0.156). Few estimates of the heterozygosity of parasitic invertebrates exist, but it can be estimated. Hamrick (1979) found a significant correlation ($\underline{r}$=0.66, $\underline{P}$<0.001) between the natural log of lifetime fecundity ($ln\underline{F}'$) and $\underline{H}$ for 101 animal species including invertebrates (Powell, 1975). Lifetime fecundity for R. acus ($\underline{F}'$=13 763± 14 225 eggs per female) is known (Chapter 4-2) and based on the least squares regression of $\underline{F}'$ versus $\underline{H}$ ($\underline{H}$= -0.0588+0.0287$ln\underline{F}'$), I estimate $\underline{H}$=0.21 for R acus. The only available comparison (Bullini et al, 1986) is $\underline{H}$=0.04±0.01 and $\underline{H}$=0.15±0.02 for ascaridoid nematodes with one homeothermic host and one or more homeothermic and poikiliothermic hosts, respectively. The higher value for mean heterozygosity in R. acus is not surprising, given that R. acus has a wider variety of hosts and all of these are poikiliotherms (Smith, 1986).

How does variable fecundity and a chaotic environment lead to increased genetic variance, and how can these simulations be interpreted with respect to the natural system? First, 'white noise' variation in the environment has been reported to prevent complete fixation and loss of alleles (Gillespie, 1973), and lead to quasi-fixation and quasi-loss (Kimura, 1954) instead. Using a diploid model incorporating white noise variation, Gillespie (1973) showed

that both alleles can be maintained even when the fitness of the heterozygotes is less than or equal to that of the homozygotes, providing that the variation in fitness of the heterozygotes is reduced. In such a system overall fitness decreases (Gillespie, 1973). Similarly, I have shown for R. acus that decreased mean fecundity is accompanied by increased variation. Second, with increased mean fecundity more recombinant progeny can be produced and this increases the probability of infecting new hosts since these represent highly variable environments. Increased recombination by itself, could maintain high genetic variation even if establishment of new individuals is a chance event (Hamrick et al., 1979). Third, it is possible that increased levels of variation at the suprapopulation level can be attributed to different infrapopulations experiencing different selection pressures. Karlin and McGregor (*in* Nevo, 1978) made a similar proposal, stating that stable polymorphism in a total system comprised of several sub-systems, can be maintained as a result of one or more 'main effects' in each system if the sub-systems are coupled. Dispersion among infrapopulations of R. acus might serve to maintain rare alleles in the suprapopulation, since both eggs and larvae are mixed in paratenic and intermediate hosts prior to each adult generation. Mixing of larval generations in yellow perch ensures that each generation of adult worms contains representatives from several generations in the past. Any larvae surviving to maturity would maintain a record of past

selection events, and the continued 'injection' of genotypes from these natural gene banks retards the decay of genetic variation. Finally, if there is selection for certain genotypes in the intermediate hosts and selection for others in the definitive host, a form of balancing selection is possible (Clegg and Allard, 1973).

What advantage is imparted to R. acus by maintaining high genetic variation? Populations with little or no genetic variation would respond instantaneously to changes in the environment, a phenomenon known as 'tracking' (Ginzburg, 1981). Any genetic variation imparts a population with some inertia and this invokes a time delay in response (Ginzburg, 1981). A high degree of inertia might be more adaptive for R. acus since this parasite lives in an extremely unpredictable and erratic environment. Increased genetic variation and inertia would reduce any subsequent fluctuations in populations (Braumann, 1981). Classical selection is still at work, but at a reduced rate.

These interpretations support the ideas of Bradley (1974), Kennedy (1976), and Keymer (1982) that the magnitude of reproductive rates of parasites compensates for the low efficiency of transmission links in helminth life-cycles. I propose that chaos resulting from the combined effects of several random environmental and genetic processes acting in concert, serves to limit or prevent the effects of selection on R. acus and maintains a high degree of genetic variation in this species. High genetic variation increases the

probability of transmission since a greater variety of hosts can be accomodated, and this decreases the likelihood of extinction by ensuring that at least a few larvae survive to maturity each generation. Furthermore, genetic variation is ensured since some individuals with high fecundity mature each generation, even though this is a rare event. To my knowledge this is the first time that a mechanism for the production and maintenance of skewed fecundity distributions in parasite populations has been proposed. Perhaps similar mechanisms might explain the persistence of unregulated parasite populations in natural systems.

CHAPTER 5: GENERAL DISCUSSION

Many of my observations are similar to those reported for other fish-parasite systems, i.e.:

i) Significant long-term changes occur in the numbers and types of parasites concomitant with changes in the composition of the host community.

ii) Short-term variation in parasite numbers is related to the physical characteristics of the fish hosts, and their habitats, relative numbers, and distribution.

iii) Diet, age, and sex of hosts determines, to a large extent, the types and numbers of parasites harboured by fish.

iv) Stochastic factors play an important role in determining the structure of the parasite community.

These observations have already been discussed and will not be dealt with further. On the other hand, some of the observations conflict with accepted theory, have been interpreted in new ways, or have important implications for parasitology and fisheries biology. These warrant further discussion and each will be dealt with separately.

Community organization

If the organization of the component community in Dauphin Lake is representative of fish-parasite communities in general, then studies on fish-parasites should not be based

entirely on an ecosystem approach as Wisniewski (1958) and others (Noble et al., 1963; Chubb, 1970; Esch, 1971; Cloutman, 1975) suggested, nor should they be based entirely on analyses of individual hosts and their parasites as Halvorsen (1971) and Wooten (1973) suggested. Not only does fish-parasite community structure differ from one hierarchical level to the next, but more importantly, analyses at different levels within this hierarchy leads to conflicting interpretations. In the future, investigators should consider both large- and small-scale structure of fish-parasite communities, before basing entire research programmes on *a priori* judgements concerning the organization of fish-parasite systems.

Parasite-induced pathology

My observations of Neoechinorhynchus carpiodi in quillback suggest that parasite-induced pathology is not only beneficial to the parasite, but may also benefit the host. Parasite-induced pathology, in the form of a well-vascularized nodule, creates a long-lasting and stable microhabitat for N. carpiodi; whereas the limited area of the lesions exposed minimizes the possibility of secondary bacterial infections and host death. Furthermore, this pathology is density-dependent and appears to be the best microhabitat for the parasite. If this type of interaction occurs in other host-parasite systems where parasites alter their attachment sites, then perhaps the role of parasite-

induced pathology in the regulation of fish parasites should be reconsidered.

Parasite-induced mortality

Many hypotheses have been proposed to explain the large variation in growth and mortality observed in species of freshwater fish. I have suggested, for yellow perch in Dauphin Lake, that a large component of this variation can be attributed to density-dependent, parasite-induced mortality. This mortality results from metabolic stress imposed by the combined actions of heavy infections by Raphidascaris acus, and the energy demands of reproduction. It is apparent from the results for parasite-induced mortality in yellow perch, that parasites located in vitally important organs, like the liver, are a major contributor to mortality in natural populations of fish. Furthermore, my analyses reveals that any relationship between mortality of fish and parasite-induced pathology is difficult to explain unless one considers parasite *density*, rather than parasite *intensity*.

Fecundity versus mass

Although density-dependent factors are important for R. acus in yellow perch, stochastic factors provide the dominant force in the definitive host. Furthermore, even when the effects of growth, recruitment and other biotic and abiotic factors were considered I was unable to show a

strong correlation between fecundity and mass for the egg-producing population of R. acus. This suggests that an index of fecundity may have little predictive value for estimating the fitness of a parasite.

Patterns of dispersion

Patterns of parasite dispersion within host populations have been the focus of much attention by parasitologists (Anderson and Gordon, 1982; Taylor et al., 1979; Pennycuick, 1971). Broadly speaking, these can be divided into three categories, described empirically by three different probability distributions; under-dispersed (regular or homogeneous) populations that fit the positive binomial distribution, random patterns described by the Poisson distribution, and over-dispersed (contagious, aggregated, or heterogeneous) patterns that fit the negative binomial distribution. Most often, the majority of hosts tend to harbour few parasites while a few hosts harbour the major proportion of the parasite population; the over-dispersed pattern of the negative binomial (Anderson and Gordon, 1982). It has been shown that heterogeneity in host behaviour and aggregated spatial distributions of infective stages can generate over-dispersion (Keymer and Anderson, 1979), and that in the few heavily-infected hosts density-dependent processes exert a regulatory influence via suppression of parasite or host fecundity or survival. Furthermore, it is widely accepted that over-dispersed

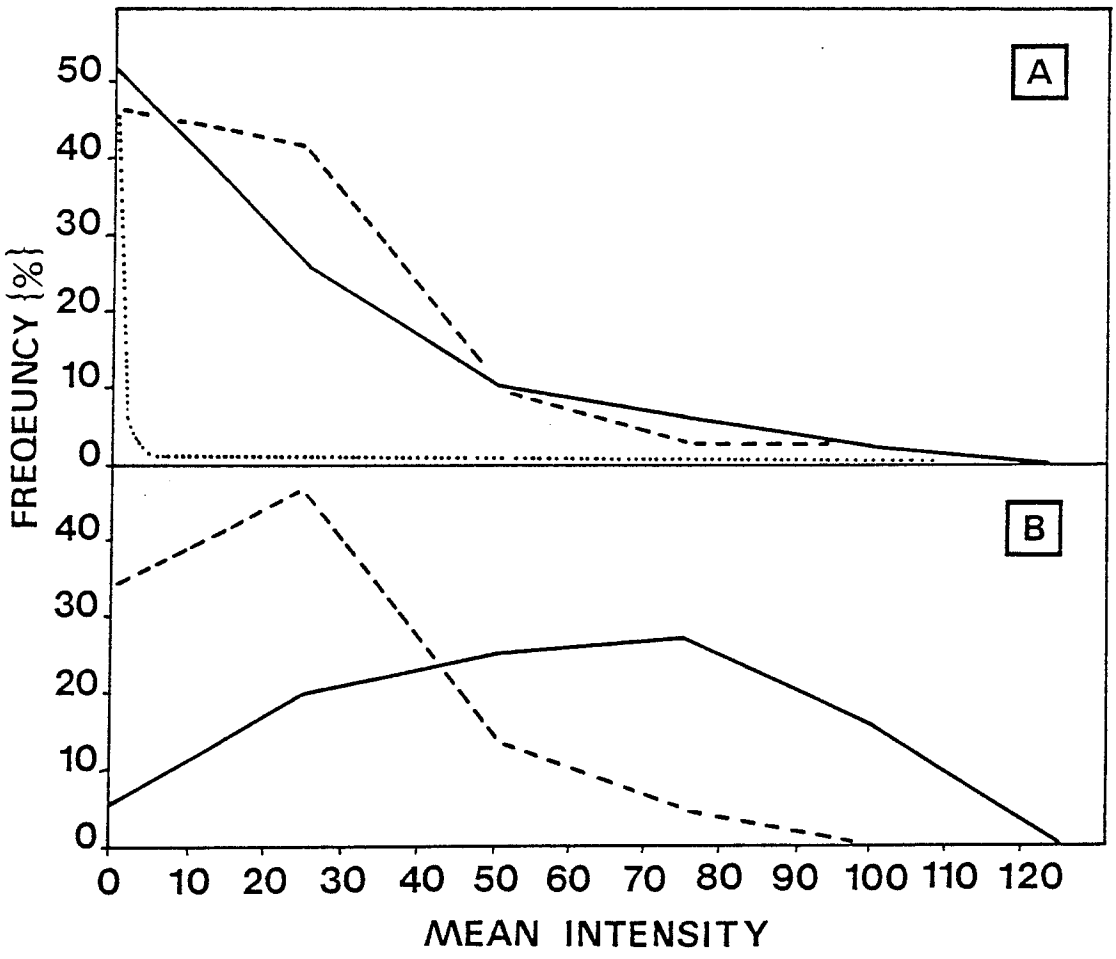
patterns of parasite numbers/host observed in nature, act to enhance the density-dependent regulation of both host and parasite populations (Anderson and Gordon, 1982).

Analyses of parasite populations in Dauphin Lake revealed some density-dependent mechanisms. Parasite-induced pathology in quillback due to infection by N. carpiodi, and reduced growth and parasite-induced mortality in yellow perch due to R. acus, are density-dependent. Increased pathology in quillback increases the number of N. carpiodi the host can accomodate. Clearly, this does not provide the negative-feedback required for regulation. On the other hand, whether or not reduced growth and parasite-induced mortality in yellow perch exert any regulatory control on populations of R. acus is not so easy to resolve. One could argue that the R. acus population is regulated, based solely on the evidence for density-dependent mortality in yellow perch infected with R. acus, but mechanisms exist that may be preventing density-dependent mortality from exerting any significant influence. First, any parasite-induced mortalities of older perch ($\geq$4 years) will exert no control, since older perch contribute little to transmission of R. acus to pike. Second, reduced transmission requires significant losses of young perch, since each of these harbours relatively few larvae. Third, reduced transmission of larvae due to significant losses of young perch, might be compensated for by alternative sources of food and therefore infection for pike (especially troutperch and cisco).

Fourth, reduced growth due to infection with R. acus means perch remain in the size-range susceptible to predation by pike for longer periods of time, further compensating for mortality. Finally, over-dispersion of R. acus is dependent on the age and size of the host. For example, the degree of overdispersion for R. acus in perch susceptible to predation by pike was greater compared to non-susceptible perch that contribute little to parasite transmission (Fig. 42). This demonstrates that extrinsic factors (eg. size-selective predation) can have different consequences for transmitted versus non-transmitted components of a parasite population.

Classical theory suggests that in the absence of density-dependent forces, parasite-populations will be unregulated, unstable, and in constant danger of extinction (Kennedy, 1985). The evolutionary implications of regulation versus non-regulation are different. Since regulated populations of parasites are thought to persist for long periods of time (Kennedy, 1985), they will exert strong selection pressures on their hosts and vice versa. On the other hand, unregulated parasite populations are characterized by frequent extinctions and so are not able to exert any selection pressure on their hosts. All the parasite populations analyzed in this study were over-dispersed, and based on historical information and simulations it appears that catastrophic extinctions of parasite species are rare. If regulation by density-

Figure 42. Patterns of dispersion for fish-parasites from Dauphin Lake. **A.** Frequency-distribution for Placobdella montifera (dotted line), Neoechinorhynchus carpiodi (dashed line), and Raphidascaris acus (solid line) infecting all types of fishes from Dauphin Lake. **B.** Frequency distributions for R. acus larvae from yellow perch susceptible to predation by northern pike (≤ 250 mm; dashed line), and for larvae in non-susceptible perch (>250 mm; solid line). The distribution for P. montifera is an approximation only, since infected fish rarely harboured more than a single leech. All other distributions are based on empirical data.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MEAN INTENSITY

dependence is not occurring, how can these parasite populations persist? Perhaps unregulated populations of parasites are able to persist in nature because they possess a repertoire of life-history adaptations to counteract the numerous stochastic processes acting on them. These processes are common to all fish-parasites, but their relative importance for a particular parasite and its host(s) varies.

Table 12 summarizes the life history traits and their implications for three parasite populations, which I think represent the range of parasite types infecting fish; the ectoparasite (Placobdella montifera), the host-specific endoparasite (Neoechinorhynchus carpiodi), and the generalist endoparasite (Raphidascaris acus). Placobdella montifera; reduced host-specificity, and active transmission related to stratification of the host species along the shoreline, maximizes the probability of transmission and leads to a highly aggregated distribution among the fish hosts. High genetic variation is ensured by sexual reproduction, and the increased mobility, dispersal, and longevity of adults ensures gene flow between populations. Neoechinorhynchus carpiodi; over-dispersion is reduced (Fig. 42) compared to P. montifera due to the use of an intermediate host and well-developed host-specificity. Furthermore, the complex relationship between quillback and N. carpiodi minimizes host mortality due to infection, while maximizing the carrying capacity of the host for the

Table 12. Summary of ecological factors that can influence the structure of populations of *Placobdella montifera*, *Neoechinorhynchus carpiodi*, and *Raphidascaris acus* and their predicted effects.

ecological factor	*P. montifera*	*N. carpiodi*	*R. acus*
degree of parasitism	facultative: leaves host to breed*	obligate:	obligate:
reproduction	dioecious, sexual: high genetic variation within populations	dioecious, sexual: high genetic variation within populations	dioecious, sexual: high genetic variation within populations
dispersal of eggs	deposits eggs:* clumped distribution reduces variability	via hosts faeces: dispersal increases variability	released into water: dispersal increases variability
invertebrate hosts	none: no dispersal from site of deposition	one (?): directed dispersal to definitive host	many: directed dispersal to intermediate host and mixing of larvae
vertebrate intermediate hosts:	none: limits dispersal	none: limits dispersal	various fish: directed dispersal to definitive host. High mean intensity, density, and abundance in yellow perch
paratenic hosts:	none:	none (?):	various aquatic insects: increases potential genetic variation
definitive hosts:	various fish;	*Carpiodes cyprinus*:	mainly *Esox lucius*:
host specificity:	none	well-developed	intermediate
transmission to definitive hosts:	active transmission low mean intensity, abundance	passive transmission via food-web, high mean intensity, high abundance	directed transmitted via food-web, intermediate mean intensity and abundance in northern pike pike.
distribution in vertebrate hosts:	ectoparasitic on surface.	localized in narrow region of intestine	northern pike: throughout intestine and stomach. yellow perch: primarily in liver.
geographic range:	cosmopolitan*	Mississippi, Missouri watersheds	cosmoplitan
trophic levels utilized:	one	two	many
life cycle and phenology:	long-lived*, seasonal,	long-lived (?), asynchronous	annual, seasonal, asynchronous, two sub-populations annually
fecundity:	low	intermediate-high	high ($>10^4$)
position in community:	rare	intermediate	dominant
regulatory mechanisms:	none	severe pathology, gut leakiness, antibody response	reduced growth and mortality of yellow perch. No effect on northern pike
stochastic mechanisms:	distribution and behaviour of hosts	patchy distribution of quillback (?)	age and sex of yellow perch

* Based on available literature.

parasite through increased pathology. High genetic variation is maintained through sexual reproduction, dispersal of eggs and directed dispersal of larvae (likely related to the schooling nature of the host and their clumped distribution in the lake), asynchronous reproduction, and high fecundity. Raphidascaris acus; transmission to the intermediate hosts is largely through asynchronous, broadcast dispersal of eggs in the environment. The ability of the parasite to infect a variety of fish hosts further decreases the degree of overdispersion in the population (Fig. 42). However, the longevity of larvae of R. acus in perch, the large size of the perch population in the lake, and size-selective feeding by pike on perch concentrates R. acus in the top trophic level in northern pike. High genetic variation is maintained via sexual reproduction, mixing of sub-populations, high fecundity, and by mixing of eggs and larvae in the environment and in the intermediate hosts. The relative magnitude of these processes differ for each type of parasite, but depend on some degree of spatial, temporal, or behavioural heterogeneity to ensure that genetic variation is maintained and that catastrophic extinctions are avoided.

Concluding remarks

Kennedy (1985) predicted for fish-parasite systems that: *i*), relationships between different species of fish and parasites will differ in time and space; *ii*), parasites in freshwater systems will often exist in non-equilibrium

conditions; and *iii*), because freshwater habitats are separate, discrete, and discontinuous, gene flow between systems will be restricted and result in differences in fish and parasite gene pools between habitats. Prediction *i* is true for most fish-parasite systems, but regardless of the particular adaptations employed, the ultimate goal for parasites appears to be maximizing transmission and preventing extinction. Prediction *ii* is also correct, but the existence of non-equilibrium conditions does not imply instability, since parasite populations can be stabilized by existing stochastic interactions. Kennedy found no evidence for genetic differences in species of parasites from different habitats, nor of any genetic interaction between parasites and fish, and this contradicts prediction *iii*. Based on empirical data and computer simulations, I proposed that chaos resulting from the combined effects of several random environmental and genetic processes acting in concert, can limit or prevent the effects of selection on parasites and maintain a high degree of genetic variation. Similar mechanisms could explain the absence of any genetic differences for fish parasites from different lakes.

Throughout this thesis I have made reference to the contradictory views of fish-parasite systems held by contemporary parasitologists. I am convinced that many of these contradictions can be explained by an integrated approach to the study of fish-parasite systems that takes into account theoretical, experimental, and field studies

concomitantly. Anderson (*in* Meerovitch, 1982) and Barret (1982) realized this and recognized that an approach combining both genetics and ecology is essential to our understanding of host-parasite systems. Although many of the factors that affect parasite population structure and dynamics are not completely understood, it is apparent that density-dependent factors are not as important in fish-parasite systems as they are reported to be in homeothermic systems. In fact, density-dependence may play little or no role in regulation of fish-parasite populations. Furthermore, the "ghost of competition past" may be inappropriate for fish-parasite systems as there is increasing evidence for underutilization of resources by fish-parasites.

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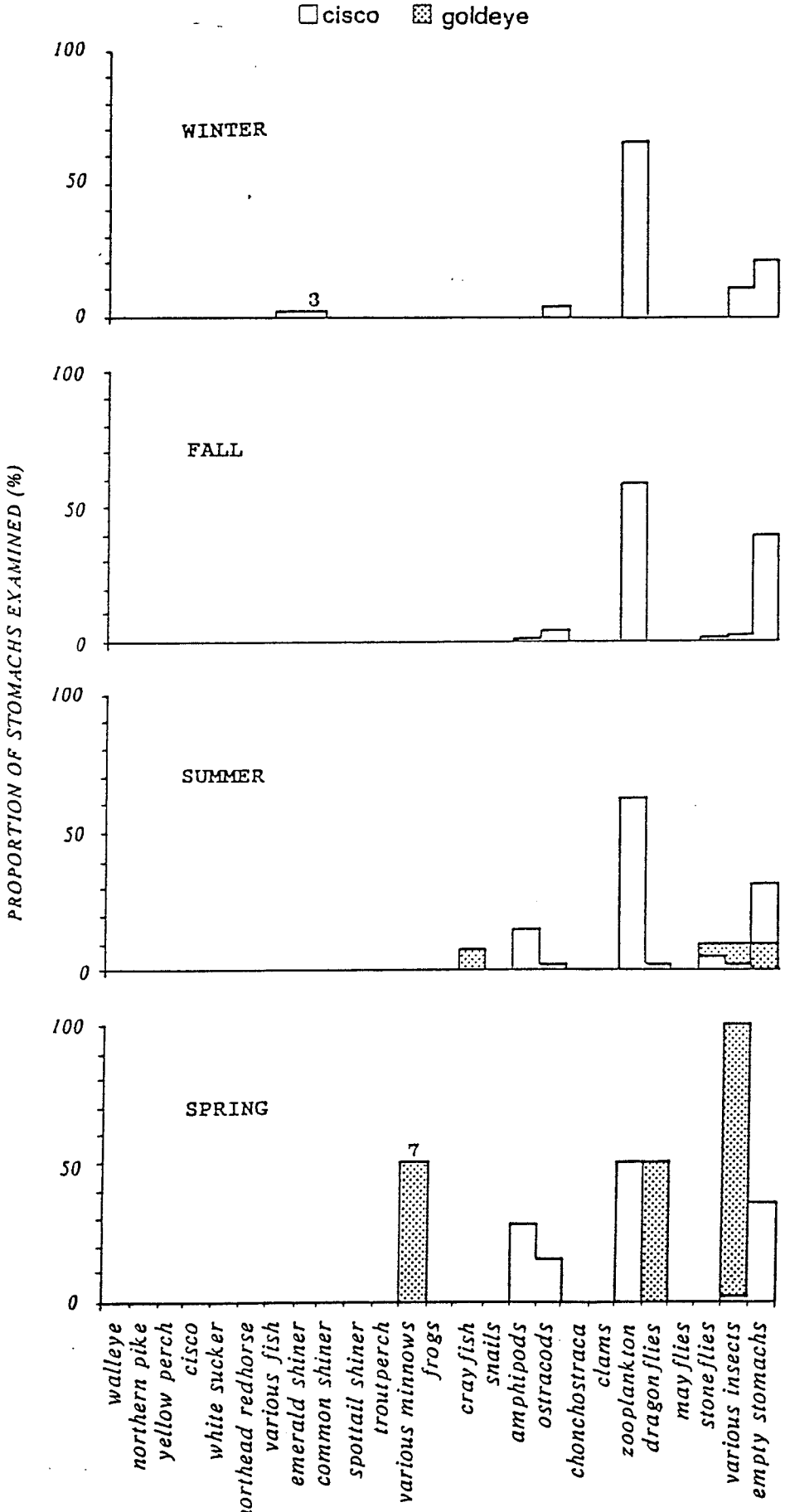
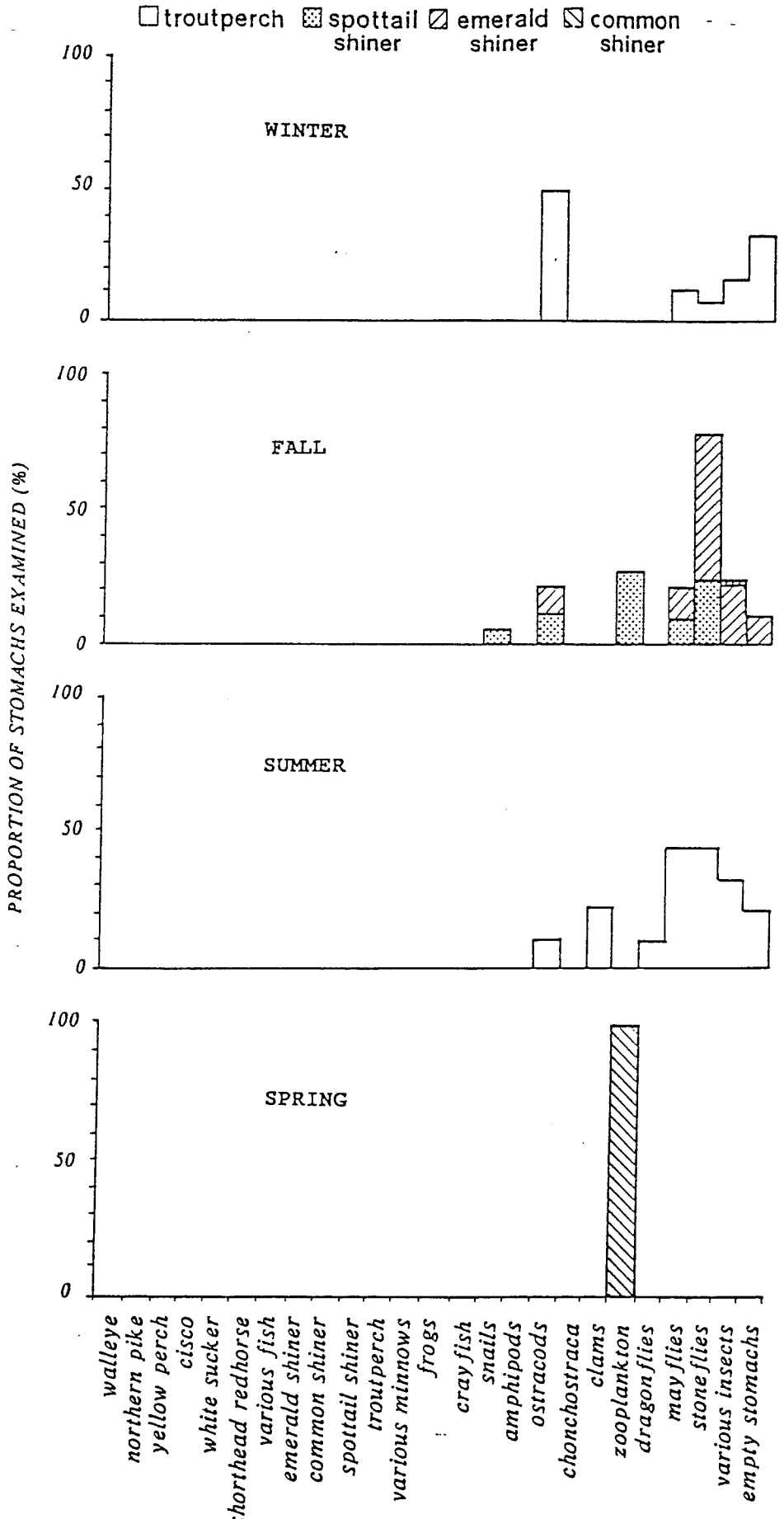
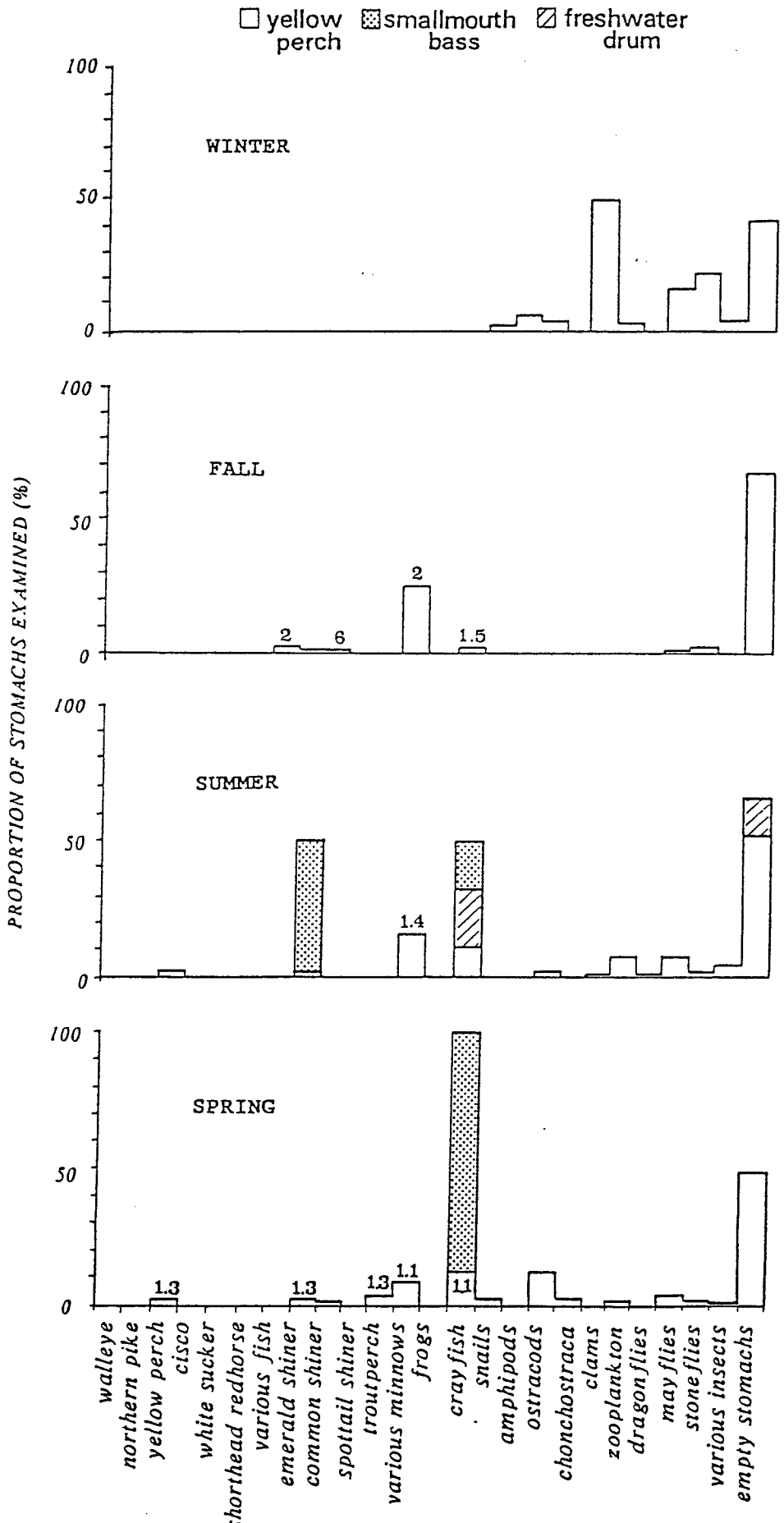
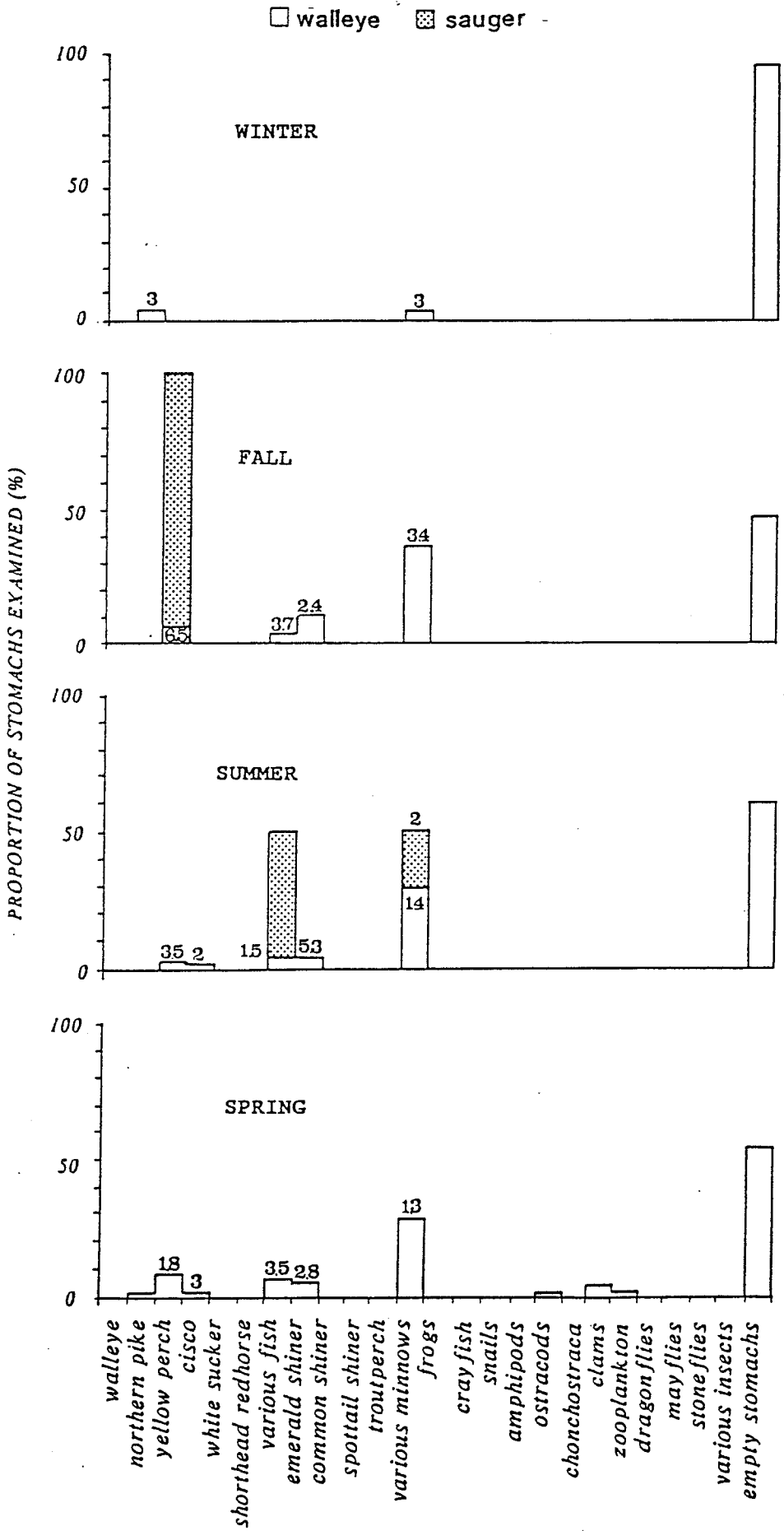
Appendix I

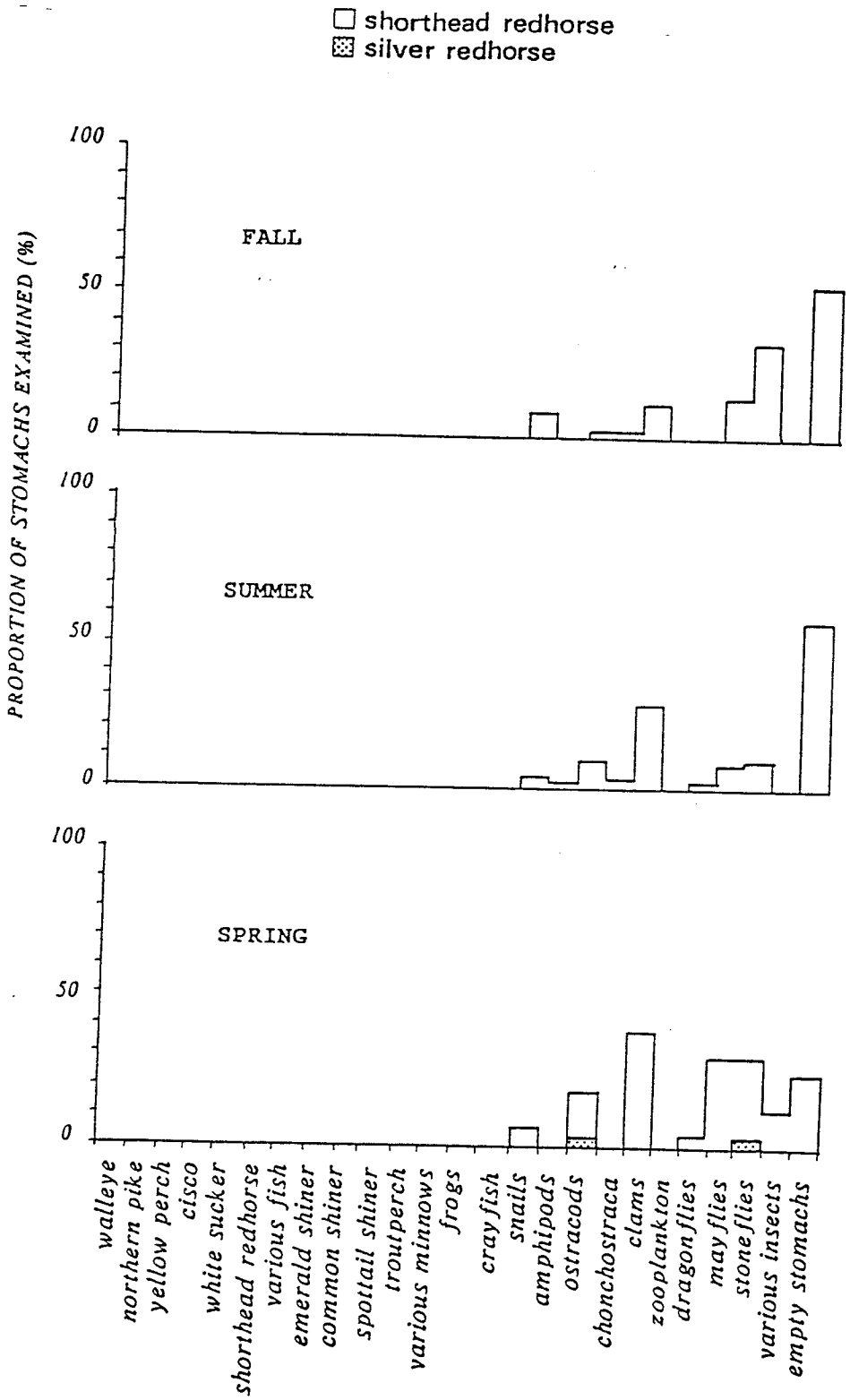
Seasonal changes in the diets of fishes from Dauphin Lake, Manitoba. For each species of fish the diet is presented as a frequency histogram for the proportion of fish stomachs examined containing particular food-items. Numbers above columns represent mean number of food-items per stomach where this mean was ≥ 1.

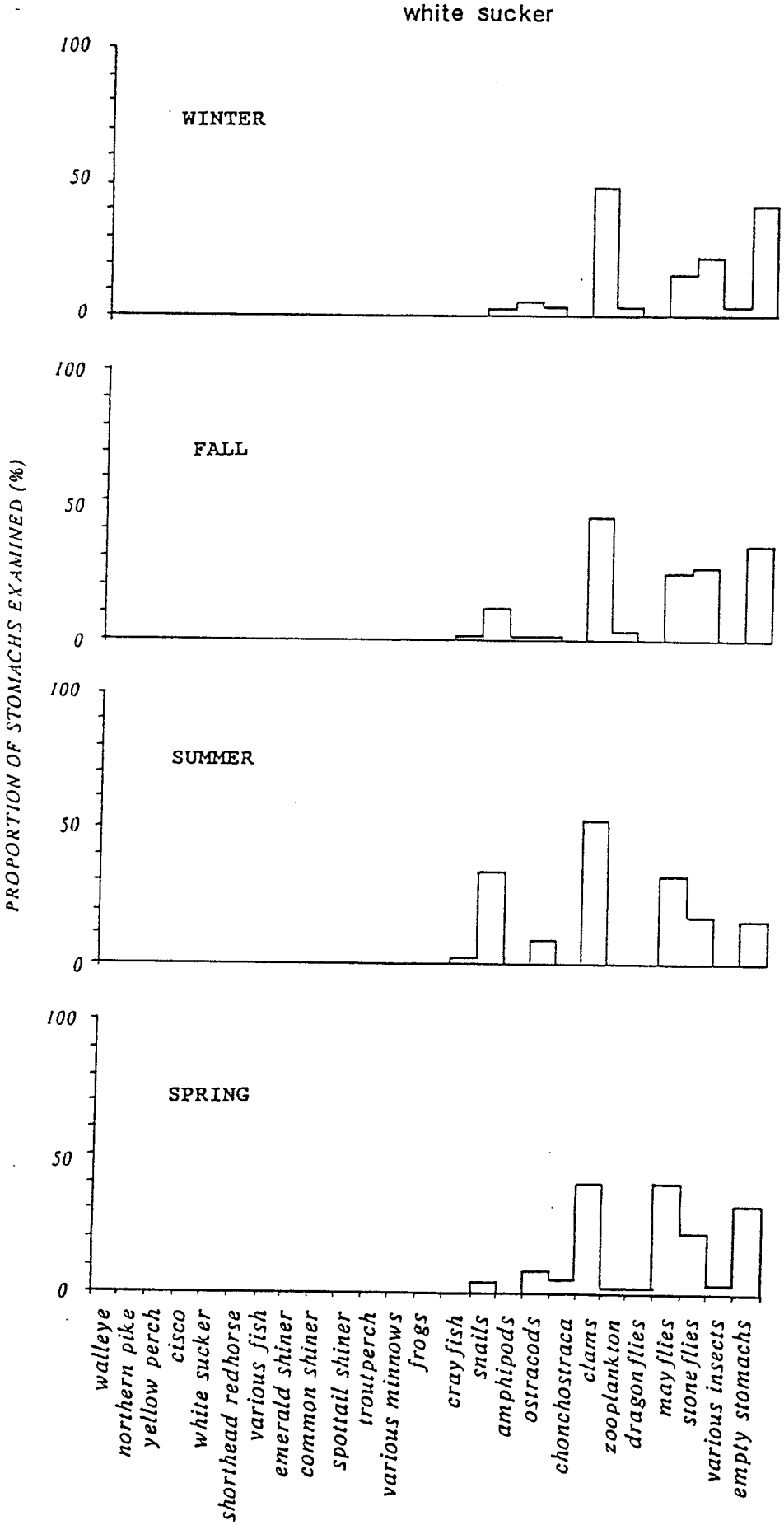

cisco
goldeye
WINTER
FALL
SUMMER
SPRING
100
50
0
3
7
PROPORTION OF STOMACHS EXAMINED (%)
walleye
northern pike
yellow perch
cisco
white sucker
shorthead redhorse
various fish
emerald shiner
common shiner
spottail shiner
trout perch
various minnows
frogs
crayfish
snails
amphipods
ostracods
chonchostraca
clams
zooplankton
dragonflies
mayflies
stoneflies
various insects
empty stomachs

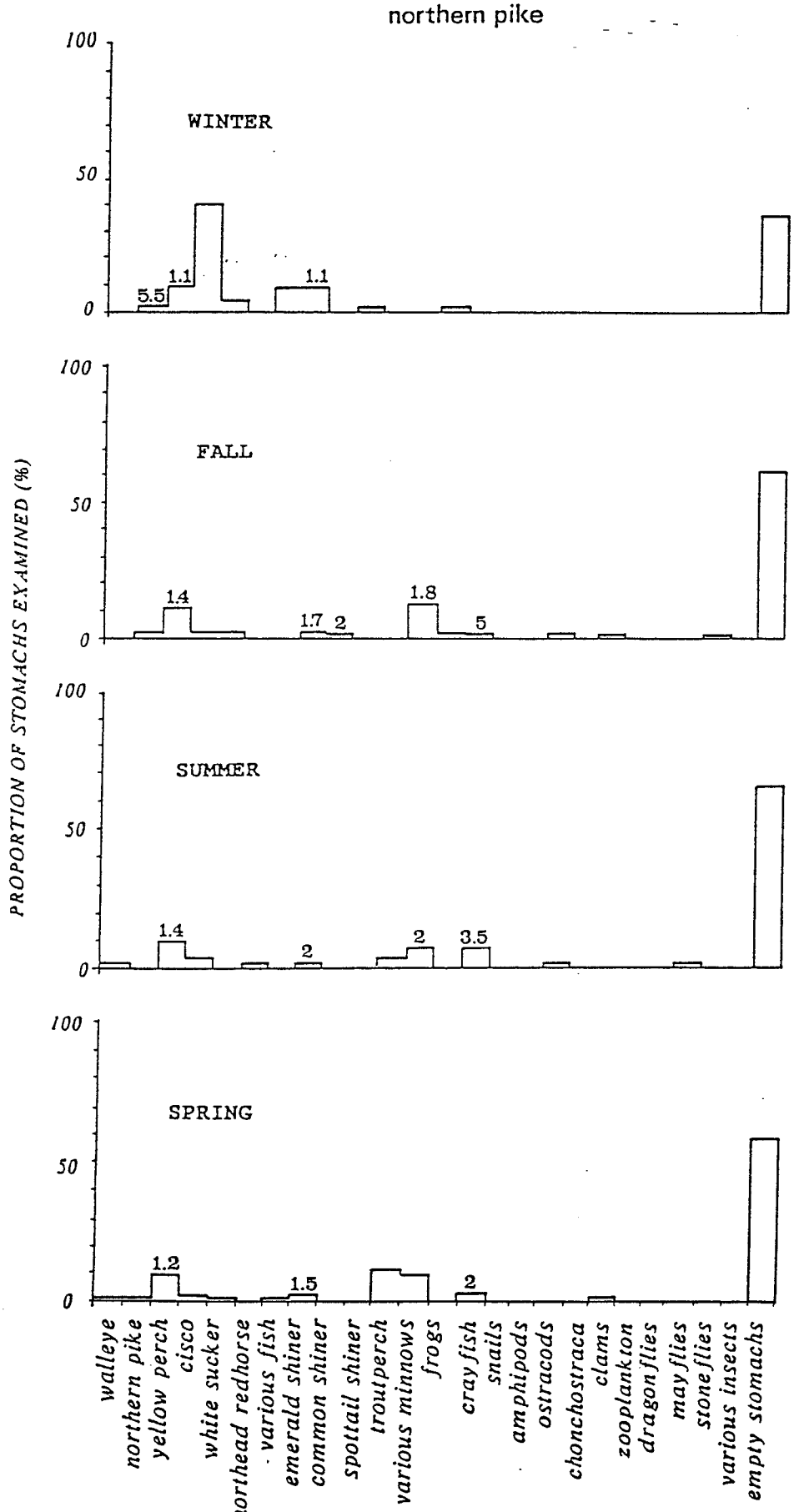

troutperch
spottail shiner
emerald shiner
common shiner
WINTER
FALL
SUMMER
SPRING
100
50
0
PROPORTION OF STOMACHS EXAMINED (%)
walleye
northern pike
yellow perch
cisco
white sucker
shorthead redhorse
various fish
emerald shiner
common shiner
spottail shiner
troutperch
various minnows
frogs
crayfish
snails
amphipods
ostracods
chonchostraca
clams
zooplankton
dragonflies
mayflies
stoneflies
various insects
empty stomachs


yellow perch
smallmouth bass
freshwater drum
PROPORTION OF STOMACHS EXAMINED (%)
WINTER
FALL
SUMMER
SPRING
100
50
0
2
6
2
1.5
1.4
1.3
1.3
1.3
1.1
1.1
walleye
northern pike
yellow perch
cisco
white sucker
shorthead redhorse
various fish
emerald shiner
common shiner
spottail shiner
trout perch
various minnows
frogs
crayfish
snails
amphipods
ostracods
chonchostraca
clams
zooplankton
dragonflies
mayflies
stoneflies
various insects
empty stomachs


walleye
sauger
PROPORTION OF STOMACHS EXAMINED (%)
WINTER
FALL
SUMMER
SPRING
100
50
0
3
3
65
37
24
34
35
2
15
53
2
14
18
3
35
28
13
walleye
northern pike
yellow perch
cisco
white sucker
shorthead redhorse
various fish
emerald shiner
common shiner
spottail shiner
trout perch
various minnows
frogs
crayfish
snails
amphipods
ostracods
chonchostraca
clams
zooplankton
dragonflies
mayflies
stoneflies
various insects
empty stomachs


shorthead redhorse
silver redhorse
PROPORTION OF STOMACHS EXAMINED (%)
100
50
0
FALL
SUMMER
SPRING
walleye
northern pike
yellow perch
cisco
white sucker
shorthead redhorse
various fish
emerald shiner
common shiner
spottail shiner
troutperch
various minnows
frogs
crayfish
snails
amphipods
ostracods
chonchostraca
clams
zooplankton
dragonflies
mayflies
stoneflies
various insects
empty stomachs


white sucker
WINTER
FALL
SUMMER
SPRING
100
50
0
PROPORTION OF STOMACHS EXAMINED (%)
walleye
northern pike
yellow perch
cisco
white sucker
shorthead redhorse
various fish
emerald shiner
common shiner
spottail shiner
trout perch
various minnows
frogs
crayfish
snails
amphipods
ostracods
chonchostraca
clams
zooplankton
dragonflies
mayflies
stoneflies
various insects
empty stomachs


northern pike
WINTER
FALL
SUMMER
SPRING
100
50
0
5.5
1.1
1.1
1.4
1.7
2
1.8
5
1.4
2
2
3.5
1.2
1.5
2
PROPORTION OF STOMACHS EXAMINED (%)
walleye
northern pike
yellow perch
cisco
white sucker
shorthead redhorse
various fish
emerald shiner
common shiner
spottail shiner
trout perch
various minnows
frogs
crayfish
snails
amphipods
ostracods
chonchostraca
clams
zooplankton
dragonflies
mayflies
stoneflies
various insects
empty stomachs

Appendix II

Mean intensity (MI), prevalence (P), range of infection (R), and literature sources used for identification of parasites from fishes from Dauphin Lake, Manitoba.

Appendix IIA. Mean intensity (MI), prevalence (P), and range of infection (R) for parasites from 1,842 fish from Dauphin Lake, Manitoba.

parasite species[a] authority	stage[b]	MI[c]	R	P[d]
Anoncohaptor anomalum Mueller, 1938	A	5.37±5.92	1-25	1.03
Argulus appendiculosus Wilson, 1907	A	1.08±0.28	1-2	0.34
Azygia longa (Leidy, 1851) Manter, 1926	A	1.50±0.76	1-3	0.43
Biacetabulum infrequens Hunter, 1927	A	1	1	0.16
Biacetabulum sp.	A	3.47±3.85	1-15	1.03
Bothriocephalus cuspidatus Cooper, 1917	A	57.03±114.25	1-1656	23.62
Caecincola sp.	M	1	1	0.05
Centrovarium lobotes (MacCallum, 1895) Stafford, 1904	M,A	6.69±10.48	1-74	12.54
Contracaecum sp.	L	6.58±7.78	1-41	5.27
Crepidostomum cooperi Hopkins, 1931	A	20.11±53.32	1-300	5.80
C. illinoiense Faust, 1918	A	14.78±22.87	1-75	0.49
Creptotrema funduli Mueller, 1934	A	2.25±1.24	1-5	0.87
Cystobranchus verilli Meyer, 1940	A	1	1	0.03
Diplostomulum sp.	M	3.57±4.82	1-28	2.66
Dorylaimida Pearse, 1942	A	5.16±6.81	1-30	1.36
Ergasilus lizae Kroyer, 1863	A	3.00±2.65	1-10	0.60
E. luciopercarum Henderson, 1926	A	19.71±23.80	1-113	7.38
E. nerkae Roberts, 1963	A	7.74±9.21	1-42	1.03
E. versicolor Wilson, 1911	A	7.74±8.12	1-40	1.03
Hunterella nodulosa Mackiewicz and McCrae, 1962	A	2.00±1.41	1-3	0.11
Khawia iowensis Calentine and Ulmer, 1961	A	2	2	0.05
Lernaea cyprinacea Linnaeus, 1758	A	1	1	0.15
Ligula intestinalis (Linnaeus, 1758) Gmelin, 1790	P	1.38±0.52	1-2	0.43

Mean intensity (MI), prevalence (P), and range (R) of infection for parasites from 1,842 fish from Dauphin Lake, Manitoba (continued).

parasite species[a] authority	stage[b]	MI[c]	R	P[d]
Lissorchis crassicrurum Haderlie, 1953	A	8.78±24.01	1-163	2.66
L. gullaris Self and Campbell, 1956	A	1.10±11.46	1-46	1.25
lymphocystis	T			0.11
Monobothrium hunteri Mackiewicz, 1963	A	4.67±2.66	2-9	0.33
Myxosoma sp.	C	1.73±2.14	1-10	1.19
Myzobdella moorei (Meyer, 1940) Meyer and Moore, 1954	A	2.91±8.91	1-52	0.88
Neascus sp.	M			0.54
Neoechinorhynchus carpiodi Dechtiar, 1968	A	20.92±19.03	1-88	2.06
N. crassus Van Cleave, 1919	A	4.45±5.43	1-45	10.59
N. cristatus Lynch, 1936	A	6.03±9.06	1-43	1.57
N. distractus	A	5.45±11.59	1-66	2.99
N. tennelus (Van Cleave, 1913) Van Cleave, 1919	A	70	70	0.05
Paurorhynchus hiodontis Dickerman, 1954	A	3.00±2.83	1-5	0.11
Philometroides nodulosa (Thomas, 1929) Dailey, 1967	A	1	1	0.05
Placobdella montifera Moore, 1906	A	1.11±0.37	1-3	1.73
Pomphorhynchus bulbocolli Linkins in Van Cleave, 1919	A	14.52±36.29	1-337	10.80
Posthodiplostomum minimum Hoffman, 1958	M	28.5±31.82	6-51	0.11
Proteocephalus luciopercae Wardle, 1932	A	12.09±16.86	1-88	9.88
P. pearsei LaRue, 1919	A	6.53±13.44	1-83	4.61
P. pinguis La Rue, 1911	A	22.20±33.41	1-233	11.45
P. wickliffi Hunter and Bangham, 1932	A	122.34±186.11	1-1188	13.08
Raphidascaris acus (Bloch, 1779) Ralliet and Henry, 1915	L,A	18.04±25.93	1-216	39.69
Rhabdochona canadensis Moravec and Arai, 1971	A	8.00±10.52	1-45	1.47

Mean intensity (MI), prevalence (P), and range (R) of infection for parasites from 1,842 fish from Dauphin Lake, Manitoba (continued).

parasite species[a] authority	stage[b]	MI[c]	R	P[d]
Rowardleus pennensis Mackiewicz and Deutsch, 1976	A	9.78±7.07	1-23	0.49
Spinitectus gracilis Ward and Magath, 1917	A	2.45±2.43	1-12	2.88
Tetracotyle sp.	M	4.56±7.85	1-37	2.11
Tetraonchus monenteron (Wagener, 1857) Diesing, 1858	A	13.83±15.23	2-90	4.40
Triaenophorus nodulosus (Pallas, 1760), Rudolphi, 1819	P,A	22.89±51.11	1-452	11.18
Unionidae	G	29.98±80.85	1-358	2.48
Urocleidus adspectus Mueller, 1936	A	10.10±13.28	1-82	3.91
total	T,G,C,M,L,P,A	57.75±107.78	1-1663	91.21

[a] status according to Margolis and Arthur (1979) and Lubinsky and Loch (1979): *, new Manitoba record; **, new Canadian record.
[b] A, adults; L, larvae; P, plerocercoids; M, metacercariae; C, cysts containing trophozoites; G, glochidia; T, tumors.
[c] mean ± standard deviation.
[d] percent.

Appendix IIB. Sources used to identify parasites from 1,842 fish from Dauphin Lake, Manitoba.

parasite species	source
Anoncohaptor anomalum	Dechtiar and Dillon, 1974
Argulus appendiculosus	Beverly-Burton, 1984
Azygia longa	Kabata, 1988
Biacetabulum infrequens	Schell, 1985
Biacetabulum sp.	Amin, 1982
Bothriocephalus cuspidatus	
Caecincola sp.	Schell, 1985
Centrovarium lobotes	Schell, 1985
Contracaecum sp.	Hoffman, 1970
Crepidostomum cooperi	Schell, 1985
	Amin, 1982
C. illinoiense	Schell, 1985
	Amin, 1982
Creptotrema funduli	Schell, 1985
	Mueller, 1934
Cystobranchus verilli	Elliot and Mann, 1979
	Davies, 1971; Sawyer, 1972
Diplostomulum sp.	Schell, 1985;
Dorylaimida	Mai and Lyon, 1975
Ergasilus lizae	Roberts, 1970; Kabata, 1988
E. luciopercarum	Roberts, 1970; Kabata, 1988
E. nerkae	Roberts, 1970; Kabata, 1988
E. versicolor	Roberts, 1970; Kabata, 1988
Hunterella nodulosa	Amin, 1986
Khawia iowensis	Calentine and Ulmer, 1961
Lernaea cyprinacea	Kabata, 1988
Ligula intestinalis	
Lissorchis crassicrurum	Schell, 1985
	Christensen et al., 1982
L. gullaris	Schell, 1985;
	Self and Campbell, 1956
lymphocystis	Ribelin and Migaki, 1975
Monobothrium hunteri	Amin, 1986
	Mackiewicz, 1963
Myxosoma sp.	Hoffman, 1970
Myzobdella moorei	Davies, 1971; Sawyer, 1972
Neascus sp.	Schell, 1985
Neoechinorhynchus carpiodi	Dechtiar, 1968
N. crassus	Amin, 1985
N. cristatus	Amin, 1985
N. distractus	Amin, 1985
N. tennelus	Amin, 1985

Sources used to identify parasites from 1,842 fish from Dauphin Lake, Manitoba (continued)

parasite species	source
Paurorhynchus hiodontis	Schell, 1985; Dickerman, 1954
Philometroides nodulosa	Hoffman, 1970
Placobdella montifera	Davies, 1971; Sawyer, 1972 Elliot and Mann, 1979
Pomphorhynchus bulbocolli	Samuel et al., 1976
Posthodiplostomum minimum	Schell, 1985
Proteocephalus luciopercae	
P. pearsei	
P. pinguis	
P. wickliffi	
Raphidascaris acus	Smith, 1984
Rhabdochona canadensis	Moravec and Arai, 1971
Rowardleus pennensis	Mackiewicz and Deutsch, 1976
Spinitectus gracilis	
Tetracotyle sp.	Schell, 1985
Tetraonchus monenteron	Beverly-Burton, 1984
Triaenophorus nodulosus	
Unionidae	Hoffman, 1970
Urocleidus adspectus	Beverly-Burton, 1984

Appendix III

Host-parasite list for metacommunities of parasites from Dauphin Lake fishes. The arrangement and common names of fish hosts is according to Scott and Crossman (1973), and parasites are listed alphabetically for each host. For each host species: N (number of fish examined); $\underline{N}$ (estimated relative abundance of host population in Dauphin Lake); and S (number of parasite species found; mean $\pm$ standard deviation) is given. For each parasite species: mean intensity (MI); range (R); prevalence (P); relative abundance (A); and the developmental stages and sites of infection in the fish hosts are listed. Where available, comparative data from Stewart-Hay (1951) and Lubinsky and Loch (1979) is included (round brackets and square brackets, respectively). A single asterisk (*) after the parasite name indicates a new host record for this species.

Appendix III-A: Parasites of goldeye (Hiodon alosoides) from Dauphin Lake, Manitoba.

N=13 (0)
N=0.63
S[a]=9 [1], 2.23±0.93

parasite	stage[b]	MI[c]	R	P[d]	RA[e]	sites
Azygia longa*	A	1	1	7.69	0.31	intestine
Bothriocephalus cuspidatus	A	12.08±23.43	1-78	92.31	45.6	intestine
Crepidostomum illinoiense	A	14.78±22.87	1-75	69.23	41.8	intestine
Ligula intestinalis*	P	2	2	7.69	0.63	stomach
Lissorchis crassicrurum*	A	1	1	7.69	0.31	intestine
Paurorhynchus hiodontis	A	3±2.83	1-5	15.38	1.89	intestine, coelom
Raphidascaris acus*	A	2±1.41	1-3	15.38	3.46	intestine
	L	3.5±3.54	1-6	15.38		liver
Rhabdochona canadensis*	A	3.8±3.03	1-8	38.46	5.97	intestine, stomach
combined	L,P,A	23±28.38	2-86			

[a] Ergasilus nerkae was also found but no other data is available. Plerocercoids of L. intestinalis probably represent worms liberated from digested troutperch.
[b] A, adults; L, larvae; P, plerocercoids.
[c] mean ± standard deviation.
[d,e] percent.

Appendix III-B: Parasites of cisco (Coregonus artedii) from Dauphin Lake, Manitoba.

N=286 (11)
N=26.81
S[a]=8 (0) [21], 1.28±0.48

parasite	stage[b]	MI[c]	R	P[d]	RA[e]	sites
Argulus appendiculosus*	A	1	1	12.5	0.12	skin
Dorylaimida	A	1	1	0.35	0.003	intestine
Ergasilus luciopercarum*	A	3.67±3.2	2-10	2.1	0.074	gills
Proteocephalus wickliffi	A	122.34±186.1	1-1118	84.27	99.3	stomach, intestine
Raphidascaris acus*	L	2.25±1.72	1-10	20.98	0.454	liver, serosa swim bladder
Spinitectus gracilis	A	2.13±1.55	1-5	2.8	0.057	stomach, intestine
Tetracotyle sp.	M	1.50±1.	1-3	1.4	0.02	pericardium
Triaenophorus nodulosus*	P	1	1	0.35	0.003	liver
combined	M,L,P,A	118.19±183.79	1-1118	87.76		

[a] data for A. appendiculosus is from 8 cisco captured in fyke nets.
[b] A, adults; L, larvae; P, plerocercoids; M, metacercariae.
[c] mean ± standard deviation.
[d,e] percent.

Appendix III-C: Parasites of northern pike (Esox lucius) from Dauphin Lake, Manitoba.

parasite	N=356 (21) stage[b]	N=14.25 MI[c]	S[a]=17 (2) [24], 2.30±1.04 R	P[d]	RA[e]	sites
Argulus appendiculosus*	A	1	1	0.11	0.003	skin
Azygia longa	A	1.75±0.96	1-3	1.12	0.055	intestine
Bothriocephalus cuspidatus	A	2.25±1.5	1-4	1.12	0.07	intestine
Centrovarium lobotes	A	7.37±13.6	1-74	14.6	3.04	intestine
Contracaecum sp.*	L	1	1	0.56	0.016	spleen
Diplostomulum sp.	M	1	1	0.56	0.016	vitreous humor
lymphocystis				0.38	0.011	skin
Neascus sp.	M			0.28	0.008	skin
Placobdella montifera*	A	1.19±0.4	1-2	1.95	0.066	skin
Pomphorhynchus bulbocolli	A	1	1	0.56	0.016	rectum
Proteocephalus pinguis	A	22.4±33.51 (12.15±9.05)	1-233 (2-31)	58.71 (71.43)	37.14	stomach, intestine
Raphidascaris acus	A	7.44±8.9 (2.89±3.52)	1-54 (2-12)	64.04 (42.86)	13.45	stomach, intestine
	L	1	1	0.28		stomach, intestine
Spinitectus gracilis*	A	1.33±0.52	1-2	1.69	0.064	stomach, intestine
Tetracotyle sp.	M	2	2	0.56	0.032	pericardium
Tetraonchus monenteron	A	13.83±15.23	2-90	22.75	8.88	gills
Triaenophorus nodulosus	A	24.53±52.73	1-452	53.65	37.1	stomach, intestine
Unionidae*	G	4	4	0.28	0.032	gills
combined	G,M,L,P,A	36.87±51.83	1-453	96.07		

[a] data for A. appendiculosus , lymphocystis, and P. montifera is from 449 northern pike captured in pound nets.
[b] A, adults; L, larvae; P, plerocercoids;M, metacercariae; G, glochidia.
[c] mean ± standard deviation.
[d,e] percent.

Appendix III-D: Parasites of emerald shiners (Notropis atherinoides) from Dauphin Lake, Manitoba.

N=13 (ca. 500)
N= not estimated
S^a=4 (0) [3] 1±0

parasite	stage[b]	MI[c]	R	P[d]	RA[e]	sites
Argulus appendiculosus*	A	1	1	0.70	0.113	skin
Centrovarium lobotes*	M	8.33±14.82	1-47	69.23	93.65	musculature
Contracaecum sp.*	L	1	1	7.69	1.25	stomach
Diplostomulum sp.	M	4	4	7.69	4.99	vitreous humor
combined	M,L,A	7.27±13.48	1-47	84.61		

[a] data for A. appendiculosus is based on 994 emerald shiners captured in fyke nets.
[b] A, adults; L, larvae; M, metacercariae.
[c] mean ± standard deviation.
[d,e] percent.

Appendix III-E: Parasites of common shiners (Notropis cornutus) from Dauphin Lake, Manitoba.

N=9 (31)
N= not estimated
S[a]=4 (1), 2.33±0.5

parasite	stage[b]	MI[c]	R	P[d]	RA[e]	sites
Centrovarium lobotes*	M	4.0±3.09	1-9	66.67	36.34	musculature
Diplostomulum sp.	M	3.6±3.58	2-10	55.56	27.26	vitreous humor
Pomphorhynchus bulbocolli*	A	1	1	11.11	1.51	intestine
Spinitectus gracilis*	A	2.56±1.59	1-5	100	34.9	intestine
combined	M,A	7.33±6.08	3-22	100		

[a] the 'metacercariae' reported by Stewart-Hay (1951) probably correspond to C. lobotes.
[b] A, adults; M, metacercariae.
[c] mean ± standard deviation.
[d,e] percent.

Appendix III-F: Parasites of spottail shiners (Notropis hudsonius) from Dauphin Lake, Manitoba.

N=33 (8)
N= not estimated
S[a]=10 (1), 1.86±0.91

parasite	stage[b]	MI[c]	R	P[d]	RA[e]	sites
Bothriocephalus cuspidatus	A	1	1	3.03	0.525	intestine
Centrovarium lobotes	M	13.86±18.39	2-55	21.21	50.96	musculature
Contracaecum sp.	L	3.33±1.21	2-5	18.18	10.49	intestine
Diplostomulum sp.	M	4.55±2.54	2-10	33.33	26.29	vitreous humor
Ligula intestinalis	P	1.25±0.5 (1)	1-2 (1)	12.12 (50.0)	2.63	coelom
Myzobdella moorei*	A	2	2	0.49	0.17	skin
Pomphorhynchus bulbocolli	A	1	1	3.03	0.525	intestine
Proteocephalus pinguis	A	1	1	6.06	1.05	intestine
Raphidascaris acus*	L	2.17±1.6	1-5	18.18	6.84	serosa, intestine, liver
Triaenophorus nodulosus	P	1	1	3.03	0.525	liver
combined	L,M,P,A	9.05±13	1-62	63.64		

[a] Scistocephalus sp. plerocercoids reported by Stewart-Hay (1951) were probably L. intestinalis
[b] A, adults; P, plerocercoids; L, larvae; M, metacercariae.
[c] mean ± standard deviation.
[d,e] percent.

Appendix III-G: Parasites of quillback (Carpiodes cyprinus) from Dauphin Lake, Manitoba.

N=104 (4) $\bar{N}$=5.72
S[a]=13 (3) [6], 1.4±1.0

parasite	stage[b]	MI[c]	R	P[d]	RA[e]	sites
Anoncohaptor anomalum	A	5.6±5.9	1-25	17.3	2.81	gills
Argulus appendiculosus*	A	1	1	0.37	0.011	skin
Diplostomulum sp.*	M	4.5±2.6	2-7	3.8	0.496	vitreous humor
Ergasilus lizae*	A	3.1±2.8	1-10	9.6	0.864	gills
Lissorchis gullaris*[f]	A	9.4±11.7	1-46	23.0	6.27	intestine
		(1)	(1)	(25.0)		
Monobothrium hunteri*[g]	A	3	3	2.9	0.25	intestine, liver
		(150)	(150)	(25.0)		
Myxosoma sp.	C	2±1.8	1-12	5.8	0.337	skin, intestine
Neoechinorhynchus carpiodi	A[h]	33.6±20	1-136	88.0	85.81	intestine
		(19±15.6)	(8-30)	(50.0)		
Pomphorhynchus bulbocolli	A	1	1	0.9	0.026	intestine
Rowardleus pennensis*	A	10.8±7.4	1-23	9.6	3.0	intestine
Unionidae	G	1.33±0.58	1-2	2.9	0.11	intestine
combined	G,C,M,A	31.39±23.71	3-101	78.85		

[a] plerocercoids of L. intestinalis were occasionally seen in quillback but no other data is available on these.
[b] A, adults; M, metacercariae; C, cysts containing trophozoites; G, glochidia of an unidentified unionid mollusc.
[c] mean ± standard deviation.
[d,e] percent.
[f] 'flukes' in Stewart-Hay (1951).
[g] probably Spartoides wardi of Stewart-Hay (1951).
[h] 'Neoechinorhynchus sp.' of Stewart-Hay (1951).

Appendix III-H: Parasites of white suckers (Catostomus commersoni) from Dauphin Lake, Manitoba.

N=253 (46) $\bar{N}$=11.87 S^a=24 (3) [37], 2±0.97

parasite	stage[b]	MI[c]	R	P[d]	RA[e]	sites
Argulus appendiculosus	A	1.13±0.35 (1)	1-2 (1)	1.5 (2.17)	0.1	skin
Biacetabulum sp.	A	4.0±4.6	1-15	3.16	0.746	intestine
Contracaecum sp.	L	1.50±0.71	1-2	0.79	0.07	intestine
Diplostomulum sp.	M	4.90±5.32	1-14	3.95	1.15	vitreous humor
Ergasilus versicolor*	A	6.75±4.27	1-10	1.58	0.63	gills
Hunterella nodulosa	A	2.00±1.41	1-3	0.79	0.094	intestine
Lernaea cyprinacea[f]	A	1	1	0.3	0.02	skin
Ligula intestinalis	P	1.33±0.58	1-2	1.19	0.09	coelom
Lissorchis crassicrurum*	A	12.00±32.53	1-163	10.28	7.3	intestine
lymphocystis[g]	T			0.18	0.011	skin, musculature
Myxosoma sp.	C	5.00±3.83	2-10	1.58	0.47	gills
Myzobdella moorei*[g]	A	1	1	0.18	0.01	skin
Neascus sp.[g]	M			4.05	0.24	skin
Neoechinorhynchus crassus	A	4.15±5.65	1-45	55.33	13.16	intestine
N. cristatus	A	8.28±11.37	1-43	6.72	3.3	intestine
N. distractus*	A	7.31±13.99	1-66	14.23	6.16	intestine
Philometroides nodulosa	A	1	1	0.4	0.024	fins
Placobdella montifera*[g]	A	1.05±0.23	1-2	3.49	0.217	skin, pericardium
Pomphorhynchus bulbocolli	A	19.3±43.43	1-337	51.78	5.92	intestine
Raphidascaris acus*	L	3.38±4.07	1-12	3.16	0.63	intestine, vitreous humor
Rhabdochona canadensis*	A	9.25±12.82	1-28	1.58	0.866	intestine
Tetracotyle sp.	M	6.65±9.7	1-37	9.09	3.58	pericardium
Unionidae	G	1	1	0.79	0.05	gills
combined	T,G,C,M,P,L,A	19.01±39.85	1-405	87.75		

[a] A. stizostethi of Stewart-Hay (1951) is probably A. appendiculosus.
[b] A, adults; L, larvae; P, plerocercoids; M, metacercariae; C, cysts containing trophozoites; G, glochidia of an unidentified unionid mollusc; T, tumors.
[c] mean ± standard deviation.
[d,e] percent
[f] based on 292 white suckers captured in fyke nets.
[g] based on 543 white suckers captured in pound nets.

Appendix III-I: Parasites of silver redhorses (Moxostoma anisurum) and shorthead redhorses (M. macrolepidotum) from Dauphin Lake, Manitoba.

silver redhorse:	N=2 (0)	N=0.31	S[a]=1 [11]			
shorthead redhorse:	N=124 (9)	N=5.72	S[a]=21 (2) [1], 2.07±1.23			
parasite	stage[b]	MI[c]	R	P[d]	RA[e]	sites
Biacetabulum infrequens*	A	1	1	0.81	0.082	intestine
Biacetabulum sp.*	A	3.09±3.39	1-13	8.87	2.79	intestine
Contracaecum sp.*	L	1.2±0.45	1-2	4.03	0.493	intestine
Cystobranchus verilli[f]	A	1	1	0.17	0.017	skin
Diplostomulum sp.	M	1	1	0.34	0.035	vitreous humor
Dorylaimidae[g]	A	30	30	50		stomach, intestine
Ergasilus versicolor*	A	8±10.23	1-42	12.1	9.86	gills
Khawia iowensis*	A	2	2	0.81	0.165	intestine
Lissorchis crassicrurum*	A	5.27±5.4	1-20	17.74	9.52	intestine
Myxosoma sp.*	C	1	1	4.03	0.41	gills
Myzobdella moorei*[f]	A	1	1	0.17	0.017	skin
Neascus sp.*	M			0.17	0.017	skin
Neoechinorhynchus crassus*	A	5.22±4.76	1-20	44.35	23.57	intestine
N. cristatus*	A	2.83±2.48	1-9	9.68	2.79	intestine
N. distractus*	A	1.95±1.47	1-5	15.32	3.04	intestine
Placobdella montifera*[f]	A	1	1	0.99	0.1	skin, pericardium
Pomphorhynchus bulbocolli	A	6.16±11.04	1-67	44.35	27.82	intestine
Raphidascaris acus*	L	1	1	0.81	0.082	intestine, vitreous
Rhabdochona canadensis*	A	9.35±11.65	1-45	13.71	13.05	intestine
Tetracotyle sp.	M	1.83±2.04	1-6	4.84	0.90	pericardium
Unionidae*	G	7±3.46	4-12	3.23	2.3	gills
combined						
M. macrolepidotum	G,C,M,L,A	9.93±10.66	1-67	86.29		
M. anisurum	A	30	30	50		

[a] A. stizostethi of Stewart-Hay (1951) is probably A. appendiculosus.
[b] A, adults; L, larvae; M, metacercariae; C, cysts containing trophozoites; G, glochidia of an unidentified unionid mollusc.
[c] mean ± standard deviation.
[d,e] percent.
[f,g] based on 603 shorthead redhorses captured in pound nets.

Appendix III-J: Parasites of trout-perch (Percopsis omiscomaycus) from Dauphin Lake, Manitoba.

N=51 (19)
N=not determined
S=10 (0) [7], 2.16±2.22

parasite	stage[b]	MI[c]		RP[d]	RA[e]	sites
Argulus appendiculosus*[a]	A	1	1	4.15	0.392	skin
Centrovarium lobotes	M,A	5.71±5.18	2-22	41.18	22.2	intestine (A), vitreous humor (M)
Crepidostomum cooperi*	A	7.67±4.93	2-11	5.88	45.1	intestine
Diplostomulum sp.	M	10.67±15.01	2-28	5.88	5.93	vitreous humor
Myzobdella moorei*	A	1	1	3.92	0.37	skin
Raphidascaris acus*	L	9.03±12.54	1-62	64.7	55.18	liver
Rhabdochona canadensis*	A	1	1	1.96	0.185	skin
Spinitectus gracilis	A	3.53±3.93	1-12	29.41	9.81	intestine
Tetracotyle sp.	M	1	1	1.96	0.185	skin, musculature
Urocleidus adspectus*	A	2	2	7.84	1.48	gills
combined	M,L,A	14.13±17.59	1-90	74.51		

[a] data for A. appendiculosus is based on 193 trout-perch captured in fyke nets.
[b] A, adults; M, metacercariae; L, larvae.
[c] mean ± standard deviation.
[d,e] percent.

Appendix III-K: Parasites of smallmouth bass (Micropterus dolomieui) from Dauphin Lake, Manitoba.

N=3 (0)
N=0.31
S=5 [16], 3.33±1.15

parasite	stage[a]	MI[b]	R	P[c]	RA[d]	sites
Bothriocephalus cuspidatus	A	1	1	33.33	0.84	intestine
Contracaecum sp.	L	7.33±2.08	5-9	100	18.48	mesentary
Ergasilus luciopercarum*	A	10.67±9.87	4-22	100	26.89	gills
Pomphorhynchus bulbocolli	A	7	7	33.33	5.88	intestine
Posthodiplostomum minimum	M	28.5±31.82	2	66.67	47.89	liver, spleen
combined	M,L,A	39.67±28.22	20-72	100		

[a] A, adults; M, metacercariae; L, larvae.
[b] mean ± standard deviation.
[c,d] percent.

Appendix III-L: Parasites of longnose dace (Rhinicthyes cataractae), johnny darters (Etheostoma nigrum), and freshwater drum (Aplodinotus grunniens) from Dauphin Lake, Manitoba.

longnose dace:	N=1 (9)	N= not determined	S=0 [4]
johnny darter:	N=1 (2)	N= not determined	S=1 (0) [6]
freshwater drum;	N=3	N= 0.31	S=1

parasite	stage[a]	MI[b]	R	P[c]	RA[d]	sites
Diplostomulum sp.[e]	M	2	2	100		vitreous humor
Pomphorhynchus bulbocolli[f]	A	3	3	33.33		intestine

[a] M= metacercariae.
[b] mean.
[c,d] percent.
[e] johnny darter.
[f] freshwater drum.

Appendix III-M: Parasites of yellow perch (Perca flavescens) from Dauphin Lake, Manitoba.

N=292 (5) N=24.74 S=19 (0) [29] 2.97±1.62

parasite	stage[a]	MI[b]	R	P[c]	RA[d]	sites
Azygia longa	A	1.33±0.58	1-2	1.03	0.024	stomach
Bothriocephalus cuspidatus	A	8.42±12.82	1-85	36.99	5.5	intestine
Caecincola sp.*	M	1	1	0.34	0.006	vitreous humor
Centrovarium lobotes	A	5.78±10.45	1-46	6.16	0.63	intestine
Contracaecum sp.	L	7.49±8.35	1-41	26.71	3.54	spleen, stomach
Crepidostomum cooperi	A	20.47±54.51	1-300	34.93	12.64	intestine
Creptotrema funduli*	A	2.46±1.27	1-5	4.45	0.19	intestine, stomach
Diplostomulum sp.	M	1.18±0.4	1-2	3.77	0.079	vitreous humor
Ergasilus luciopercarum	A	3.33±4.03	2-16	4.11	0.24	gills
lymphocystis[e]	T			0.79	0.79	skin, musculature
Myzobdella moorei[e]	A	3.41±9.75	1-52	21.4	0.014	skin
Placobdella montifera*[e]	A	1.5±1	1-3	3.15	0.083	skin
Proteocephalus pearsei	A	6.53±13.44	1-83	29.11	3.36	intestine
Raphidascaris acus	A	1.5±0.71	1-2	0.68		stomach, intestine
	L	35.77±32.69	1-216	95.21	60.22	liver, rectum, heart
Spinitectus gracilis	A	1.15±0.55	1-3	4.45	0.09	stomach, intestine
Tetracotyle sp.	M	1	1	1.03	0.018	pericardium
Triaenophorus nodulosus	P	2.15±1.82	1-7	4.45	0.169	hindgut, liver
Unionidae	G	56.73±110.51	2-358	7.53	7.55	gills
Urocleidus adspectus	A	10.72±13.57	2-82	22.95	4.35	gills
combined	T,G,M,P,L,A	58.22±71.11	1-499	95.89		

[a] A, adults; L, larvae; M, metacercariae; P, plerocercoids; G, glochidia of an unidentified unionid mollusc; T, tumors.
[b] mean ± standard deviation.
[c,d] percent.
[e] based on 127 yellow perch captured in pound nets.

Appendix III-N: Parasites of logperch (Percina caprodes) from Dauphin Lake, Manitoba.

N=15 (3)
N= not determined
S= 7 (0) [11], 2.18±1.54

parasite	stage[a]	MI[b]	R	P[c]	RA[d]	sites
Centrovarium lobotes*	A	1.75±0.96	1-3	26.67	24.17	intestine
Diplostomulum sp.	M	1	1	6.67	3.45	vitreous humor
Myzobdella moorei*	A	1	1	26.67	13.81	skin
Neascus sp.	M			40	20.07	skin, musculature
Pomphorhynchus bulbocolli	A	1.16±0.41	1-2	40	24	intestine
Raphidascaris acus*	L	1	1	6.67	3.45	liver
Spinitectus gracilis*	A	1.50±0.71	1-2	13.33	10.36	stomach, intestine
combined	M,L,A	4.27±6.71	1-24	73.33		

[a] A, adults; M, metacercariae; L, larvae.
[b] mean ± standard deviation.
[c,d] percent.

Appendix III-O: Parasites of sauger (Stizostedion canadense) from Dauphin Lake, Manitoba.

N=6 (0)
N=0.31
S=6 [18],

parasite	stage[a]	MI[b]	R	P[c]	RA[d]	sites
Bothriocephalus cuspidatus	A	5	5	16.67	4.86	intestines
Centrovarium lobotes	A	3.00±1.41	2-4	33.33	5.83	intestines
Crepidostomum cooperi*	A	40	40	16.67	3.89	intestines
Creptotrema funduli*	A	2	2	16.67	1.94	intestines
Proteocephalus luciopercae	A	1	1	16.67	0.97	intestines
Raphidascaris acus	L	28.33±18.01	16-49	50	81.9	liver
combined	L,A	23.17±18.79	4-50	100		

[a] A, adults; L, larvae; M, metacercariae.
[b] mean ± standard deviation.
[c,d] percent.

Appendix III-P: Parasites of walleye (Stizostedion v. vitreum) from Dauphin Lake, Manitoba.

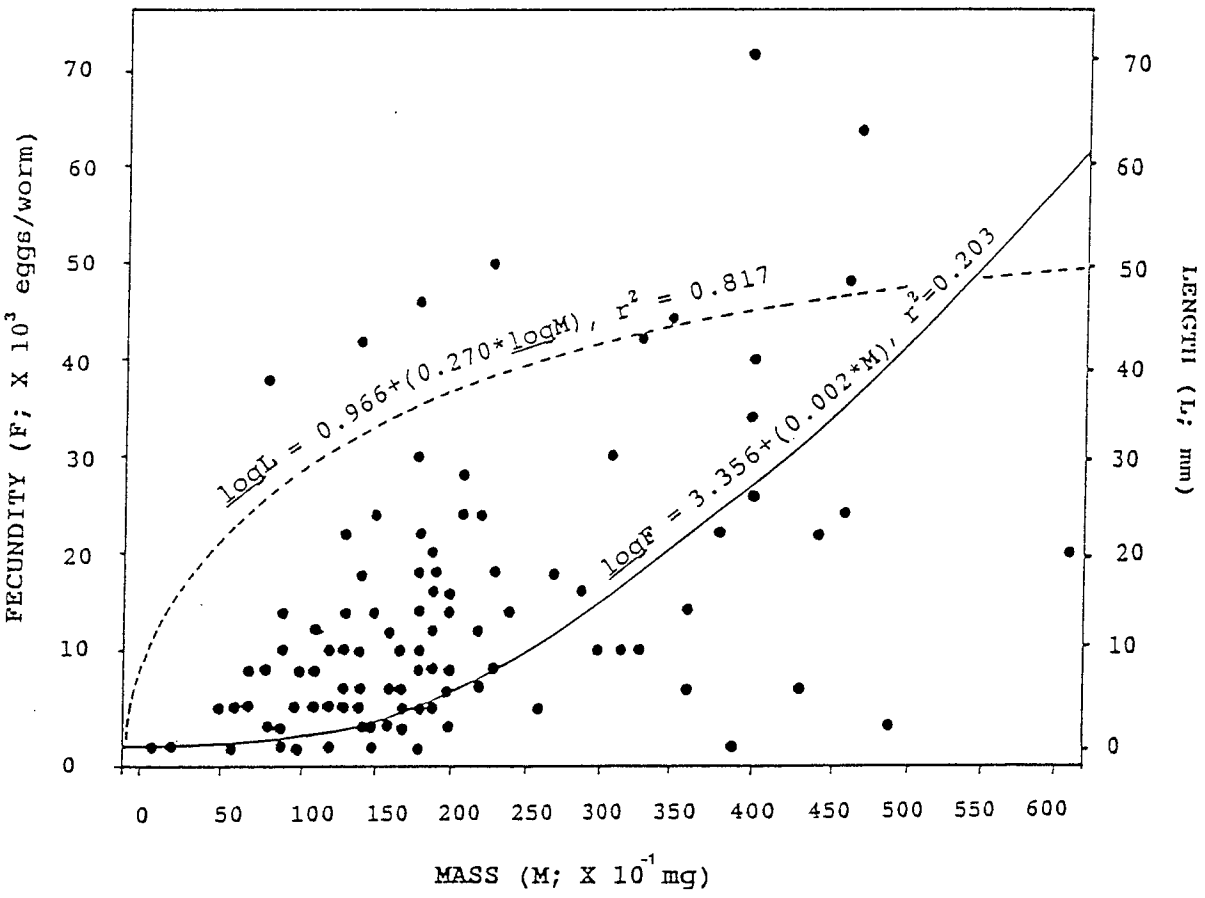
N=329 (21) N=8.82 S=16 (7) [21], 2.64±1.12

parasite	stage[a]	MI[b]	R	P[c]	RA[d]	sites
Argulus appendiculosus[e,f]	A	1	1	0.45	0.052	skin
		(1)	(11)	(4.76)		
Bothriocephalus cuspidatus	A	77.07±130.37	1-1656	93.62	78.11	intestine, stomach
Centrovarium lobotes	A	6.51±8.99	1-70	34.04	2.4	intestine
		(2.33±0.58)	(2-3)	(14.29)		
Crepidostomum cooperi	A	1	1	0.3	0.003	intestine
Creptotrema funduli*	A	1	1	0.3	0.003	intestine, stomach
Diplostomulum sp.	M	1	1	0.3	0.003	vitreous humor
Ergasilus luciopercarum	A	22.5±24.81	1-113	34.95	8.51	gills
lymphocystis[g]	T			2.67	0.031	skin, musculature
Myzobdella moorei	A	1.33±0.58	1-2	0.91	0.013	skin
Neascus sp.	M			0.3	0.3	skin, musculature
Neoechinorhynchus tennelus	A[f]	70	70	0.3	0.23	intestine
Placobdella montifera*[g]	A	1	1	0.22	0.002	skin
Proteocephalus lucioperca	A	12.15±16.88	1-88	55.01	7.24	intestine, stomach
Raphidascaris acus	A	13.83±41.95	1-147	3.64		stomach, intestine
	L	7.82±7.87	1-46	31.31	3.2	liver, rectum, eye
Unionidae	G	4.85±4.40	2-16	3.95	0.21	gills
Urocleidus adspectus	A	1	1	0.3	0.003	gills
combined	T,G,M,L,A	94.31±136.49	1-1663	97.87		

[a] A, adults; L, larvae; M, metacercariae; G, glochidia; T, tumors.
[b] mean ± standard deviation.
[c,d] percent.
[e] Argulus stizostethii of Stewart-Hay (1951).
[f] probably 'Pomphorhynchus' of Stewart-Hay (1951)
[g] based on 449 walleye captured in pound nets.

Appendix IV

Scatterplot for fecundity (F) versus mass (M) of Raphidascaris acus from northern pike. Regression lines for fecundity versus mass (———) and for worm length (L) versus mass (- - - -) are indicated. Regression equations and r^2 are given.


FECUNDITY (F; X 10³ eggs/worm)
LENGTH (L; mm)
MASS (M; X 10⁻¹ mg)
logL = 0.966+(0.270*logM), r² = 0.817
logF = 3.356+(0.002*M), r²=0.203
0
10
20
30
40
50
60
70
0
50
100
150
200
250
300
350
400
450
500
550
600

Appendix V

Program used for computer simulations in Chapter 4-3.

Abbreviations:

- g: generation
- N: number of worms
- M: percent mortlality
- N_S: number of survivors
- p_g: P(F) for each generation. For all simulations, p=0.5 for g=1.
- G: genotype code (0= ff, 1= Ff or fF, 2= FF)
- G_A: additive genetic effect
- G_D: dominance effect
- E: environmental effect
- GV: genotypic value
- SEX: 0= female, 1= male
- $\underline{F}$: fecundity
- $\overline{X}$: mean fecundity

```
FOR GENERATION = 1 TO g
      N ⟵ 1 + DU[0, 200]
      M ⟵ U[0, 0.5]
      N_S ⟵ N - (N x M)
      p_g ⟵ BIN[N_S, p_g-1] - N_S
      FOR WORM = 1 TO N_S
            G ⟵ BIN[2, p_g]
            FOR G=0: G_A ⟵ 0; G_D ⟵ 0, GENOTYPE= ff
            FOR G=1: G_A ⟵ 5; G_D ⟵ 2, GENOTYPE= Ff or fF
            FOR G=2: G_A ⟵ 10; G_D ⟵ 0, GENOTYPE= FF
            E ⟵ U[0, 10]
            GV ⟵ G + G_A + G_D + E
            SEX ⟵ BIN[1, 0.5]
            IF SEX = 0
                  F = GV x X̄
            END FOR
            OUTPUT
      END FOR
END FOR
```