



Report to the
MASSACHUSETTS BAYS PROGRAM

**EVALUATION OF CHEMICAL CONTAMINANT EFFECTS
IN THE MASSACHUSETTS BAYS**

Prepared by

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Woods Hole Oceanographic Institution
Woods Hole, MA 02543

JULY, 1995

MBP-95-05

Printed and Distributed by the Massachusetts Bays Program, a cooperative venture of
Massachusetts Coastal Zone Management Office and the U.S. Environmental Protection Agency.
Funded under U.S. Environmental Protection Agency Cooperative Agreement CEOO1534-01.

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MASSACHUSETTS BAYS PROGRAM

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FOREWORD

The roots of the Massachusetts Bays Program extend back to 1982, when the City of Quincy filed suit against the Metropolitan District Commission and the Boston Water and Sewer Commission over the chronic pollution of Boston Harbor, Quincy Bay, and adjacent waters. Outdated and poorly maintained sewage treatment plants on Deer Island and Nut Island were being overwhelmed daily by sewage from the forty-three communities in the Metropolitan Boston area. Untreated and partially treated sewage were spilling into Boston Harbor.

Litigation over the pollution of Boston Harbor culminated in 1985 when the United States Attorney filed suit on behalf of the Environmental Protection Agency against the Commonwealth of Massachusetts for violations of the Federal Clean Water Act. The settlement of this suit resulted, in 1988, in the creation of the Massachusetts Water Resources Authority, the agency currently overseeing a multi-billion dollar project to repair and upgrade Metropolitan Boston's sewage treatment system. In addition, the settlement resulted in the establishment of the Massachusetts Environmental Trust - an environmental philanthropy dedicated to improving the Commonwealth's coastal and marine resources. Two million dollars in settlement proceeds are administered by the Trust to support projects dedicated to the restoration and protection of Boston Harbor and Massachusetts Bay.

The Trust provided \$1.6 million to establish the Massachusetts Bays Program, a collaborative effort of public officials, civic organizations, business leaders, and environmental groups to work towards improved coastal water quality. The funding was used to support both a program of public education and a scientific research program focusing on the sources, fate, transport and effects of contaminants in the Massachusetts and Cape Cod Bays ecosystem. To maximize the efficiency of limited research funding, the sponsored research program was developed in coordination with research funded by the MWRA, the United States Geological Survey, and the Massachusetts Institute of Technology Sea Grant Program.

In April, 1990, following a formal process of nomination, the Massachusetts Bays Program became part of the National Estuary Program. The additional funding provided as part of this joint program of the Environmental Protection Agency and the Commonwealth of Massachusetts is being used to continue a coordinated program of research in the Massachusetts Bays ecosystem, as well as supporting the development of a Comprehensive Conservation and Management Plan for the coastal and marine resources of Massachusetts and Cape Cod Bays. The study described in this report explores the degree of biological impact associated with varying sediment concentrations of polynuclear aromatic hydrocarbons (PAHs) in coastal salt marsh ecosystems.

The information in this document has been subject to Massachusetts Bays Program peer and administrative review and has been accepted for publication as a Massachusetts Bays Program document. The contents of this document do not necessarily reflect the views and policies of the Management Conference.

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SUMMARY

Mummichogs (*Fundulus heteroclitus*), soft shell clams (*Mya arenaria*) and blue mussels (*Mytilus edulis*) were collected from some or all of 10 sites in Boston Harbor and Massachusetts and Cape Cod Bays in the Fall of 1992 and the Spring of 1993. Histopathological examination revealed a suite of changes previously known to be associated with chemical contamination in animals from the more contaminated sites. In particular, liver tumors were evident in 14% of the adult *F. heteroclitus* from the Island End River, a tributary of the Mystic River in Boston Harbor. Additionally a number of pathologies previously associated with chemical exposure were also seen in the two bivalve species at a number of contaminated sites. Induction of cytochrome P4501A was also seen in *F. heteroclitus* from the more contaminated sites, P4501A induction is a biochemical change associated with exposure to dioxin and other planar halogenated and aromatic hydrocarbons. These findings suggest that there are measurable biochemical and pathological changes in intertidal fish and shellfish from the more contaminated parts of the Massachusetts Bays system, and ongoing region-wide monitoring of these changes should be maintained to assess and encourage efforts to reduce toxic chemical pollution at source, and to improve the quality of wastewater treatment in urban areas, such as Greater Boston.

ACKNOWLEDGMENTS

We would like to thank the following people for help in this study: Matthew Liebman, Diane Gould, Marianne Farrington, Jack Paar, Brenda Jensen, Bruce Woodin, Holly and Peter Fitzgerald, George Heufelder and Judy McDowell. Chemical analyses were conducted by the US EPA Region 1 Environmental Services Division, Lexington MA. Facilities and dockage for sample preparation were provided by the New England Aquarium. Funding was provided by the Massachusetts Bays Program.

INTRODUCTION

The overall objective of this research was to evaluate the degree of biological impact associated with chemical contaminants in coastal and particularly salt marsh systems of the Massachusetts Bays, by examining multiple species, stations and endpoints, using sensitive biochemical and less sensitive pathological measures of exposure and effect. The studies were carried out over two years in 1992 and 1993.

Many studies have shown that in various parts of the Massachusetts Bays, pollutants from known point sources are linked to localized and substantive effects upon the biota. These effects include biochemical, genetic and pathological changes identified in fish and/or shellfish residing in areas impacted by the contaminants from those sources. The greatest effort in such studies has involved the Boston Harbor system, and the links between contamination and effects primarily associated with the Deer Island sewage outfall. Deer Island Flats show heavy contamination by polynuclear aromatic hydrocarbons (PAH) and chlorinated hydrocarbons (CHC) [1, 2]. Winter flounder from this same region show strongly induced mixed function oxidase (cytochrome P450 1A) [3], liver neoplasms and other cellular abnormalities [4-7], DNA and hemoglobin adducts of PAH [8, 9], and activating mutations in critical oncogenes [10]. The weight of evidence clearly indicates that chemicals in the effluent are causing these effects. Studies elsewhere in Boston Harbor and in Quincy Bay have suggested similar linkages between known effluents and effects on fish and shellfish [11].

In addition to such studies relating effects to point sources, there are ongoing efforts revealing the presence and prevalence of some of these same conditions in regions of Mass Bays (and other Massachusetts waters) that do not have major inputs from known point sources of contaminants. Thus, concentrations of PAH and CHC, elevated cytochrome P4501A content, and cellular abnormalities have been described in winter flounder from various sites [3, 6, 12, 13]. These studies are suggesting a widespread effect of contaminants that either emanate from those bays and harbors where there are known point sources, or that originate from other sources, both point and non-point.

Biological changes that can be explicitly linked to chemical contaminants, are now commonly referred to as biomarkers [14]. The studies described here employed the biomarker approach. Biomarkers may be used to determine chemical exposure and effects in several important ways. These include determination of spatial and temporal differences in the identity and source of pollutants and their effects. The establishment of cause and effect linkages is another critical aspect of biomarker studies. In using the biomarker approach in this study we have determined 1) whether there is contamination

by selected toxicants, 2) whether the contaminants are present at biologically significant concentrations, 3) which species/populations are at risk, and 4) the severity and geographic extent of these effects. The ability to sensitively and specifically detect exposure and response of organisms in a particular environment is essential in assessing the impact of chemical inputs to clean areas, or the recovery of contaminated areas following regulatory or remedial action.

The focus of this research was on salt marsh/tidal creek habitats, and mummichogs and bivalves in particular. This habitat was selected because it is the buffer zone between land and sea, it is an important nursery area for many organisms including commercially important fish species, and its high organic matter content makes it a sink for lipophilic chemical contaminants. The study therefore focusses on a habitat that is critical to the living resources in the Massachusetts Bays. Furthermore, it appears that there is inadequate critical attention presently being given these habitats in the Bays.

Biomarker endpoints

Cytochrome P4501A: Mixed function oxidase (MFO) activity is the generic name given to the type of reaction carried out by a family of enzymes known as cytochrome P450. It has been known for over 20 years that the rate of some MFO reactions could be induced (increased) by exposure of animals or animal cells to selected kinds of chemicals. Thus, aromatic hydrocarbons, and chlorinated aromatic hydrocarbons (PCBs, dioxins) induce aryl hydrocarbon hydroxylase (AHH) activities by causing synthesis of an increased amount of a specific enzyme. Induction of MFO activities in liver of fish was suggested in the mid-1970s as an index or marker of exposure to petroleum. Numerous studies have confirmed that use of induction of MFO. In the mid-1980s we provided the first evidence that antibodies to the particular MFO enzyme (the specific P450 induced by hydrocarbons, called P4501A) could be used to measure the degree of induction in fish liver, related to exposure in the environment. The induction of P4501A is one of the few molecular responses to pollutants for which we have a good understanding of the mechanism (transcriptional activation), chemical specificity (planar toxic aromatic compounds), biological specificity (vertebrates), sensitivity (dose-response relationships), influence of environmental variables and comparison with other molecular changes [15].

Importantly, our experimental studies with different pure PCBs and dibenzofurans show that P4501A is induced primarily, and possibly only, by those compounds that are the most toxic chemicals in each group [16, 17]. P4501A induction may be the single

most sensitive response to these chemicals [18]. This strongly supports the idea that P4501A content can be used as a biomarker for toxic chemical exposure. P4501A induction can provide measurements of acute or chronic exposure to hydrocarbons. Many of the inducing compounds bioaccumulate in fish and so even low levels over time may induce significant amounts of P4501A.

Several recent studies, with different fish species and in different parts of the world, have revealed close correlations between the levels of induced cytochrome P4501A and levels of PCBs or PAH either in the organisms or in their immediate environment. Studies in the flounder *Platichthys flesus* from Langsundsfjord, Norway [19], in starry flounder (*Platichthys stellatus*) from San Francisco Bay (Stegeman *et al.*, unpublished), and in rattail (*Coryphaenoides armatus*) from the deep ocean [20] have all shown close correlation between the levels of induction of P4501A in hepatic microsomes and the levels of total PCB residues. Results in lake trout larvae have also correlated MFO induction with PCB content [21]. Other fish studies have shown that levels of hepatic microsomal cytochrome P4501A also correlate with sediment contamination by PAH [22]. In Europe, there has been strong correlation between induction and proximity to kraft mill outfalls [23]. The growing number of such studies provides a consistent picture, confirming the idea that the levels of a specific cytochrome P450 isozyme can be used as a biomarker for levels of contaminants in the environment and/or in the organisms themselves. Measurement of cytochrome P4501A content can provide explicit evidence of exposure of fish to inducing compounds [24]. In this study we use two biochemical markers for cytochrome P4501A function. Immunoblot detection of the amount of cytochrome P4501A protein using the monoclonal antibody, anti-P4501A 1-12-3, and a spectrophotometric assay for the rate of catalysis of a model substrate, ethoxyresorufin-O-deethylase (EROD).

Immunohistochemical Detection: We have also demonstrated that the same antibody can be used to measure induction in tissues that are examined for histopathology. Upon exposure to inducers, P4501A is induced in various cells and organs in a fish's body. Biochemical evaluation of homogenized tissues provides accurate measurement of P4501A induction but low values can result if preparations from induced cells become diluted by unavoidable inclusion of non-inducible cell types during tissue preparation. This may be common in some organs which have few inducible cells or in embryos. In contrast, immunohistochemical staining of fixed fish tissues processed in paraffin provides cell specific information highlighting induction levels in individual cells, thus eliminating the dilution effect and allowing detection of even low induction in various organs as well as providing the ability to examine various organs with only one

test. Such examination can indicate the target cell types and the extent of change throughout the body. Surveys of archived paraffin embedded tissues can also be accomplished. We have pioneered this approach to detecting induction as a biomarker, and have employed it in analysis of experimental and environmental samples of fish [25, 26].

Histopathology: Histopathology is a definitive marker of biological effect, which can range from moderate to severe parasitic infestations or cellular abnormalities culminating in frank neoplasms. Such changes rarely can be linked to specific chemicals but usually can indicate the combined effects of the suite of chemicals found in the environment, which can include immunosuppressive agents, cell toxins, tumor initiators and tumor promoters.

Pathologies in Fish: The role of environmental chemicals as causative agent in the appearance of numerous diseases in fish is clear. Not only are there abundant data linking the prevalence of hepatic neoplasms and associated (putative) preneoplastic conditions to organic chemical pollutants [27], but in some cases there could be increased or decreased prevalence of parasitism in fish from polluted sites.

The hepatic neoplasms detected in *F. heteroclitus* have been described as hepatocellular carcinoma and hemangiosarcoma [28]. These have been closely associated with a high concentration of PAH, suggesting similar etiology to that suggested for hepatic tumors in winter flounder from Boston Harbor.

Pathologies in Molluscs: Environmental contaminants as a cause of pathology in molluscs is "...becoming increasingly well established." [29]. Pathology associated with molluscs from polluted waters are inflammation of the gills, and focal epithelial proliferation of the gills [11], regression and atrophy of digestive tubules, increased occurrence of ceroid, depression of gametogenic activity, and mantle recession [29]. Impaired immunocompetence with increased prevalence of infectious etiologies [30] such as rickettsia [11] are important findings.

Additionally, and importantly, *M. arenaria* from the northeast coast of the United States are significantly affected by a disease variably titled sarcoma, hematopoietic neoplasia, and leukemia [31]. Although pollution is not causal in this disease [32], the prevalence of the disease within a population of clams may be increased by pollution of the water in which the clams are found [33-35].

Sampling Design

Site Selection

We sampled from a total of nine intertidal sites and one pier around the Mass Bays as listed in Table 1, and illustrated in Figure 1 and in charts in the appendix. The specific sites listed were based on parameters discussed during the planning of the project, tempered with the availability of target species encountered in the field.

Sediment and clam samples for chemical analysis were collected from each site visited in 1992 in conjunction with EPA personnel from Lexington MA. Results are appended in Table A1. These data confirm that there were two reference sites, Scorton Creek and Wellfleet, both in Cape Cod Bay, and a suite of more chemically compromised sites which were, in order of increasing contamination, Weymouth Fore River, Chelsea River, Saugus River, Neponset River, Fort Point Channel and Island End River, the last of which is a tributary off the Mystic River. This last site is the prior location of a creosote manufacturing plant, close to defunct coal gasification plants, and adjacent to a major fossil fuel transportation site, which has had a recent history of recurrent oil spills. Any degree of sediment resuspension at this site results in an immediate surface slick of hydrocarbons - it appears to be extremely chemically compromised. In 1993 we also sampled two additional sites, at the Amelia Earhart Dam on the Mystic River, and Pier 2 at the Charlestown Navy Yard. Chemical analyses are not available for these sites, although the background tPAH for Pier 2 is around 250 ppm, with at least 20x that level at the creosote spill site within the Pier 2 area (pers. comm. Michael J. Wade, Wade Research, Inc.).

Sample Collection

Fish and shellfish were collected in September/October 1992, and May 1993. Fish and clams were held in water from the collecting sites and returned to laboratories at the New England Aquarium or Woods Hole Oceanographic Institution for sampling. The water was aerated during transportation using a portable 12 volt air pump. Fish were caught by using a 12 m. beach seine with extra foot-rope weights, and a 1.5 m. wooden pole at each end. The seine was dragged perpendicular to the beach, in 0 to 1.3 m. of water for about 50 m., before the outer end was moved to the beach and then both ends dragged up the beach until the catch was stranded in the center of the net. Sets were repeated until the target sample size had been obtained, where possible. Clams were collected by digging with a clam rake. Mussels were taken by manual removal. *M. edulis* were collected from 3 sites only in 1993. All animals collected were assigned a

unique sample identification number within our multi-year laboratory archive accession system (Tables 2, 13 and 18).

Sediments were collected in November 1992. Stations were visited at low tide, and sediment taken from areas near the low tide mark where fish and clams had been collected. In the case of Neponset River the fish and clam sites were divided by a culvert under a road, so separate samples were taken at each site. Samples were taken with a stainless steel polnar grab or teflon scoop from the top 3 cm. Sediment was homogenized in a polyethylene tray. All grabs, scoops and trays were cleaned with methylene chloride and methanol before each sampling. Sediment was placed in I-chem jars for PAH and moisture content analysis and whirlpak bags for total organic carbon (TOC) and percent silt-clay analyses. All samples were iced in the field and stored at -20° C, and thawed before analysis.

4. Sample Preparation and Analytical Methods

Gross pathology and Histology (Fish and Molluscs)

F. heteroclitus: Fish were killed by cervical section, examined for grossly visible external and internal abnormalities, measured for total length, observed for gender, and selected organs (liver, kidney, heart, gill, gut) were dissected and placed in 10% neutral buffered formalin or in Bouins fixative within cassettes. Samples were embedded in paraffin according to standard methods [36], and examined for histopathological change. Severity of fin erosion, and grossly visible liver lesions were scored on a scale of 0-4, and 0-2 respectively. Each histopathological lesion was scored on a scale from 0 (absent) to 4 (severe). Data from this and the studies described below are summarized in the results section and reported in full in the appendix.

M. arenaria: After collection, right valves were numbered using an indelible marker, measured, and a sample of hemolymph removed using a 25 gauge needle with a 1 ml tuberculin syringe. The left valve was removed, measured for total length, and the clam fixed in situ in 1% glutaraldehyde, 4% formalin (1G/4F) in seawater (modified from [37]). Both valves and soft tissues were examined grossly for abnormalities. Three sections of each clam were processed in paraffin. Hematoxylin and eosin stained sections included the following organs for examination: digestive diverticula, intestine, stomach, foot, gonad, heart, kidney, siphon, mantle, and gill. Lesions were described and their occurrence and severity scored on a scale from 0 (absent) to 4 (severe).

Biochemical Analysis of Cytochrome P4501A

Samples for P4501A analysis were examined from Stations 1 to 8. Five individuals were analyzed from each station. Individual sample masses were inadequate from Stations 4, 6 and 7, for which samples of 2-4 animals were pooled as necessary to obtain adequate sample masses for analysis.

Two comparable methods were used to quantitate the actual amount of P4501A protein in the liver:

1. Enhanced chemiluminescence. P4501A in hepatic microsomes was determined by immunoblotting using modifications of our previously described protocol [38]. Microsomal protein (45 μg per sample) and a range of scup P4501A standards were electrophoretically resolved on a 12% polyacrylamide gel and transferred to a Schleicher & Schuell (S & S) Rad Free™ nylon membrane. This membrane was subsequently incubated with mouse monoclonal antibody 1-12-3p6 against scup P4501A at a concentration of 10 $\mu\text{g}/\text{ml}$ in S & S blocker/TRIS buffered saline (TBS). The secondary antibody used was 1/900 diluted S & S alkaline phosphatase conjugated goat antimouse Ig in blocker/TBS. X-ray film was exposed for various times to blots incubated with S & S Enhanced Chemiluminescence (ECL) lumiphos™ substrate sheets according to the manufacturer's protocol. After standard film development, images were obtained from the autoradiograms with a Kodak Digital Camera DCS 200 driven by a Macintosh Quadra 840AV computer. Densitometric analysis of individual bands was performed using the NIH Image software program, and the amounts of P4501A in samples were estimated by comparison with the signal produced by known amounts of scup P4501A.

2. Colorimetric development. P4501A in hepatic microsomes was determined by immunoblotting using modifications of our previously described protocol [38]. Microsomal protein (45 μg per sample) and a range of scup P4501A standards were electrophoretically resolved on a 12% polyacrylamide gel and transferred to a Schleicher & Schuell (S & S) 0.2 μ nitrocellulose membrane. This membrane was subsequently incubated with mouse monoclonal antibody 1-12-3p6 against scup P4501A at a concentration of 100 $\mu\text{g}/\text{ml}$ in 5% nonfat dry milk/TRIS buffered saline (TBS). The secondary antibody used was 1/200 diluted BioRad alkaline phosphatase conjugated goat antimouse Ig in 5% milk/TBS. Color was developed using nitro blue tetrazolium and 5-

bromo-4-chloro-3-indoylphosphate. Densitometric analysis of individual bands was performed using the Scanalytics Mscan™ flat bed scanner and analytical software, and the amounts of P4501A in samples were estimated by comparison with the signal produced by known amounts of scup P4501A.

Numbers obtained for each of these methods are relative rather than absolute, as the per pmol response of the antibody is only established for scup P450. Absolute data cannot be given in the absence of purified *F. heteroclitus* P4501A protein as a standard. However, relative comparisons between animals of the same species reflect true differences. To allow comparison between the two methods we normalized all data to a percentage of maximal response.

7-Ethoxyresorufin-O-deethylase (EROD)

The EROD assay is a spectrophotometric assay for the catalytic activity of cytochrome P4501A. This assay complements the data on protein amount described above, by determining the specific activity of the enzyme present. 7-Ethoxyresorufin-O-deethylase (EROD) was determined using the Cytofluor™ fluorescent plate reader using a kinetic modification of the stopped assay procedures of Kennedy et al [39, 40] Duplicate reactions of 200 µl final volume in 50mM TRIS, 0.1 M NaCl (pH 7.8) containing 1.5 to 20 µg of microsomal protein and 2 µM 7-ethoxyresorufin were initiated by the addition of NADPH to a 1 mM final concentration in a Costar™ 48 well plate. A calibrated sample of known activity was included in each plate. Continuous production of resorufin product was determined in the Cytofluor™ fluorescent plate reader at 22° C over 6 min., and the slope of the linear portion of the plot of resorufin produced vs. time was compared to known amounts of resorufin measured under identical conditions.

Immunohistochemical Analysis of P4501A

Standard 5 µm sections were deparaffinated and hydrated in 1% bovine serum albumin/ phosphate buffered saline (BSA/PBS). During the hydration process, sections were incubated in 0.5% H₂O₂ in methanol for 45 minutes to block endogenous peroxidase [41]. Hydrated sections were immunochemically stained using a streptavidin-peroxidase stain with monoclonal antibody 1-12-3 [42] as the primary antibody, as described below. Previous immunofluorescent studies have demonstrated the specificity of MAbs 1-12-3 for P4501A in tissue sections by immunoadsorption [43].

After hydration in 1% BSA/PBS slides were mounted in a Shandon Coverplate staining system. Sections were incubated in normal goat serum (NGS) for 20 minutes to block any possible nonspecific attachment of the secondary antibody (goat antimouse IgG)[41]. Sections were washed once for 5 minutes and then incubated in 1/15,000 dilution (1.7 µg protein/ml) of MAb 1-12-3 in 1% BSA/PBS. 150µl of diluted antibody were added at the start and at 30 minutes through a 1 hour incubation. Incubation in primary antibody was followed by washing with 1% BSA/ PBS. This wash procedure followed all antibody incubations. Next, sections were incubated in a 1/200 dilution of biotin labeled goat-antimouse IgG for 20 minutes, washed and then incubated in a 1/1000 dilution of peroxidase labeled streptavidin for 20 minutes. After another wash, sections were incubated for 30 minutes in 3-amino-9-ethylcarbazole (AEC) in acetate buffer to develop a red color. Sections were rinsed and then counterstained with Mayers hematoxylin, and mounted in glycerol [44]. Two types of controls were used: (1) Sections of liver from a fish (scup; *Stenotomus chrysops*) with high and one with low concentrations of P4501A (as determined by EROD activity and immunoblotting) were included in every stained group as controls for the staining method. (2) Matching serial sections of selected tissues were stained using a nonspecific IgG (Purified mouse myeloma protein, UPC-10, IgG_{2a}, Organon Teknika, West Chester, PA) at 1.5 µg protein/ml of 1% BSA/PBS [41].

Specific staining by MAb 1-12-3 were evaluated by light microscopic examination of the stained sections. Cell types that stain and their associated occurrence and staining intensity were recorded for each tissue section examined. At least two immunochemically stained sections were examined from each of four or more fish in each sample. Comparative staining results for the group of fish analyzed from each sample is described in relationship to fish from all samples, and reported as negative, mild, mild/moderate, moderate, strong or very strong. Quantitative comparisons were made between tissue types at various sites by using the product of scaled values for intensity and occurrence.

Hemocyte Evaluation (clams)

Hemocyte evaluation (immunoperoxidase staining) for neoplastic cells was done with at least 20 clams from each site. Hemolymph was sampled within two days of collection from the same animals that were examined for histopathology. Methods were as described previously [45] with some modifications. Briefly, 0.1 ml of hemolymph containing hemocytes are removed from the clams through the posterior adductor blood

sinus with a 1 ml syringe containing 0.9 ml of filtered sea water. This hemolymph mixture was placed on a poly-L-lysine coated coverslip in a multiwell plate and hemocytes allowed to settle onto the coverslip for 30 minutes. Fluid was removed from each chamber and cells fixed for 5 minutes in 1G/4F. Wells were then rinsed in phosphate buffered saline 4 times. Hemocytes were stained in an indirect immunoperoxidase staining method using, as the primary monoclonal antibody, MAB 1E10 (developed by Carol Reinisch) which is specific for neoplastic cells that occur in the leukemic disease. A second peroxidase tagged antimouse antibody was then applied. Finally the peroxidase complexes were developed with AEC to produce a red color on the leukemic cells. Using these stained cells, a percentage of neoplastic to normal cells was determined and staged according to previously published methods [45].

Sediment Analyses

Total organic carbon analysis was performed by IEA, Inc., Monroe CT. This was analyzed according to a 7/27/88 modification of EPA 9060 from SW-46, by Lloyd Kahn, US EPA Edison NJ. The method involved acidification to remove inorganic carbon, combustion, separation of gases in a UV-persulfate reaction chamber and measurement of carbon in an infrared detector. Detection limit = 100 mg/kg (0.01%). Silt clay content was determined by wet-sieving using ASTM D422 by Geotesting Express, Acton, MA.

PAH measurements were made by US EPA Region 1, Environmental Services Division, Lexington, MA. Extractions were conducted using EPA Region 1 'PAH procedure in sediment and soil samples' standard protocol (PAHsell1.sop - Ultrasonic extraction - method 3550b). Clean-up procedures were silica gel clean-up method 3630b. Extracts were analyzed on GC/MS in Selected Ion Monitoring mode similar to EPA method 680 using different characteristic ions. Practical quantitation limits (= detection limits) were 2 ppb ($\mu\text{g}/\text{kg}$) for all PAHs except for dibenzo(a,h)anthracene, which was at 20 ppb.

Sediment PAH data are used for comparisons with a variety of measures in the results. They are expressed as total PAH for each station. Each station value is a mean of the replicate samples from each station. Where samples were analyzed in duplicate, the first reported value only was used in each duplicate case. Non-detects were assumed to be zero. Of three replicate samples at Scorton Creek two were non-detects, and one had trace levels of a few compounds. This station mean was also taken to be zero.

Statistical Analysis

Histology

Due to the nature of the data collected in this survey of histopathological conditions of organisms from the Massachusetts Bay area, all analyses were conducted using multivariate statistical techniques [46]. Multivariate techniques are applicable when the data collected consists of several independent and dependent variables, all of which are correlated with each other to some degree. The presence of multiple partially correlated independent and dependent variables made univariate and bivariate statistical analyses inappropriate.

The analysis of this data was further constrained because the pathological conditions were evaluated using a system of discrete variables where each condition was ranked using an ordinal rating system.

The objective of the analyses was to investigate the ability of the ranked pathologies to allow the sites to be grouped according to some commonality, in this case level of environmental perturbation. In order to achieve this objective the data were subjected to discriminant function analysis. Discriminant function analysis, a multivariate technique mathematically equivalent to multivariate analysis of variance, permits the analyst to predict group membership on the basis of a set of predictor variables, in this case the histopathological rankings. The groupings were defined in terms of the relative contaminant levels at each site (i.e. no, low, or high levels of contamination). The predictor variables for both fish and bivalves are listed in Tables 7 and 16 respectively.

The end product of a discriminant analysis are subsets of predictor variables combined to maximize the differences between groups. The first combination, termed discriminant function 1, may account for a large percentage of the variability observed between sites. But it may require a second or third combination of the predictor variables, not accounted for in discriminant function 1, to allow the analyst the ability to significantly predict membership in the defined groups. The inverse of this analysis is that one may not be able to significantly differentiate group membership with the data given.

Results of discriminant analysis may be interpreted in two different manners. The first interpretation of the analysis consists of the analyst's ability to predict group membership based on a set of predictor variables. In the present study this allows us to predict whether the organism can be assigned to having been retrieved from an area with

a predictable level of contamination based on histopathological analysis of the organism. The second interpretation resulting from discriminant function analysis is to ascertain which predictor variables contribute the most to predicting, from a statistical viewpoint, which group the data set belongs in.

In the present study, for fish 236 observations per variable were analyzed and for bivalves a total of 178 observations per variable were made for *M. arenaria* and 60 observations per variable were made for *M. edulis*. Missing data, due to tissue missing from the histological preparation, were randomly distributed throughout the data sets and these missing values were replaced by group means to facilitate the analysis. Discriminant function analysis can be highly influenced by the presence of outliers. In the present study the data were tested for both univariate and multivariate outliers using residuals analysis, with the resultant removal of three fish and one clam from the dataset for the discriminant analyses.

The overall conditions of each analysis are listed in Table 8. In each of these analyses an F- test was performed, based on Wilk's lambda, to ascertain whether group membership could be reliably predicted. In all cases the significance of the discriminant functions to predict group membership was $p < 0.01$ (Table 8). We can reliably predict the contaminant range from where the organism was retrieved within our study sites based on the set of histopathological observations made on that organism.

To demonstrate these results graphically, we have plotted the centroids of the first 2 discriminant functions for each species. The centroid represents the mean of the discriminant function group following reduction of the data matrix to a single dimension.

Biochemistry

Variation for P4501A and EROD was analyzed for between-station differences by ANOVA. These data were also compared with levels of sediment contamination by simple logistic regression. For analyses that failed to detect PAH, a value of 0.0 $\mu\text{g/g}$ was arbitrarily assigned for the purposes of logistic regression.

5. RESULTS

Sediment Analysis (Table A1)

Silt, clay, moisture and organic carbon content varied substantially but without any marked parallel with PAH content. In order of increasing PAH burden per gm. sediment the stations fell into three major groups: 1) Low: Wellfleet, Scorton, 2) Medium: Weymouth Fore River, Neponset River, Chelsea River, Saugus River, and 3) High: Fort Point Channel, Island End River. Sediment data is unavailable for the Earhart Dam site. For the statistical analysis of clam pathology, the absence of clams from the Island End River lead us to regard the Saugus and Chelsea sites in addition to the Fort Point Channel as "high" for that analysis.

Fundulus heteroclitus

Abundance

Mummichogs were caught from all of the stations listed in Table 1 except station 10. Date of collection, sample sizes, sample ID numbers and mean total length are given in Table 2. *F. heteroclitus* were often hard to find in the Boston Harbor system. The Island End River site appeared to be on the border of being azootic. There appeared to be greater life in 1993 than in 1992. Common terns were observed feeding there in that year (Holly Fitzgerald, pers. comm.). Likewise mummichogs were hard to find from the Neponset River: attempts to find fish failed at Fox Point, the gas tanks, and further up the river, before we found fish at the site described. The Fort Point Channel was hard to sample, given the extensive bulkheading, and the available fish were small. The Saugus River failed to yield fish at 4 different sites downstream of the described site. In contrast, mummichogs were abundant at both of the reference sites on Cape Cod.

Histopathology (Tables 3 to 6)

Neoplasms

The most dramatic histological change evident in the mummichogs was in adults from the Island End River, the most contaminated site, in 1993. At that site 14% of the fish had liver neoplasms, and 40% had associated foci of tinctorially altered hepatocytes. The absence of neoplasms in the fish from the Island End River in 1992 is not a significant finding, as the animals sampled in 1992 were subadult on the basis of their

total length, as shown in Table 2, and one would not necessarily expect to see such lesions in fish of this age. Neoplasms and altered hepatocyte foci were absent from fish from all the other less contaminated sites.

Other Lesions

In the liver, macrophage aggregations were evident in adults from the Island End River, coccidia were only evident from the Wellfleet reference site and hepatic necrosis was evident in adults from the Island End River, Weymouth and Wellfleet. Hepatic clear cells were only evident from fish from the Fort Point Channel as isolated nests of 1-3 cells that were distinct from the typical foci of clear cells described in other species of fish from other chemically contaminated sites. Hepatic megalocytosis was only seen in fish from Scorton Creek. There was no other changes evident in these livers to suggest as to why this change, which has in the past been associated with chemical exposure, was observed. Cardiac nematodes were seen in one fish each from Wellfleet, Earhart Dam, and Neponset River. Gill trematodes were found in fish from Neponset, Saugus, Wellfleet, Earhart Dam and the Island End River, but were only prevalent from Neponset. Gill hyperplasia was present at all sites to varying degrees. Splenic macrophages were observed in fish from Weymouth, Neponset, Saugus, Scorton Creek, and the Island End River.

Discriminant Analysis of Fish Histopathology

Site groups were condensed into groupings defined by the sediment contaminant loads at each site as shown in Table A1. The contaminant loadings were classified as either non-detectable (Stn. #'s 5 and 6), low (Stn. #'s 1 and 9), or high (Stn. #'s 2,4, 7 and 8) and the fish pathological evaluations were used as predictor variables to separate these groups. Two discriminant functions were generated which defined the groups. These are plotted in Figure 2. DF1 was defined liver coccidian parasites (Lv2) and gill trematodes (G11). DF2 included the other 5 pathological predictors with liver macrophage aggregates (Lv1) and hepatic necrosis (Lv3) having the highest correlations within DF2.

It is important to emphasize that these discriminant function analyses were not biased by any presumption as to potential causation by chemical exposure. Furthermore visual examination of the data in Tables 3 and 4 and knowledge of published data would strongly suggest that only hepatic neoplasms and altered cell foci are positively correlated with chemical exposure. As in all matters we must maintain a sense of the differences between statistical and biological significance.

Cytochrome P4501A

Biochemical data on *F. heteroclitus* are listed in Tables 9 and 10. These data were generated using two techniques as described in the methods. The comparability of these techniques is shown in Figure 3, where normalized data from each method is compared for samples that were analyzed with both techniques. We took this relationship to demonstrate that the P4501A protein values were method independent, and to allow pooling of the data from each method to give a comparison between all sites. Data were segregated by site and season of collection and by gender, for the spring collection. It is important to account for inherent physiological differences in addition to chemically induced changes. Specifically, we know that spawning female *F. heteroclitus* have a markedly reduced P4501A expression even in the face of chemical exposure. This reduction results from the liver of spawning females being primarily occupied with production of an egg constituent, vitellogenin. This effect is not seen in *F. heteroclitus* caught in the Fall. Thus genders were not separated for Fall collected samples. Tables 9 and 10 respectively show Fall and Spring values of P4501A protein levels, and P4501A enzymatic activity, as shown by ethoxyresorufin-o-deethylase (EROD) activity. Figure 4 shows anti-P4501A immunoblots from a selection of these animals. In Table 9 there is a general trend of increasing P4501A protein content and EROD activity with increasing chemical exposure (Figure 5). In Table 10 the Spring 1993 data shows a marked induction of P4501A protein and EROD activity in the males in fish from the Island End River, as compared to Wellfleet. In contrast females from this Spring sample set showed lower P4501A protein, and EROD activity from both sites, although there was still a relative increase in EROD of females from the Island End River as compared to Wellfleet. Individual data from all Fall animals are plotted in Figure 5 as a simple regression against sediment contamination levels normalized to percent carbon. Data from this regression analysis and others are summarized in Table 11.

Immunohistochemical data is presented as a mean of tissue P4501A protein expression for all animals regardless of gender or season in Table 12. Major trends were not evident in this data set, although in general increased expression was seen in the liver and gill epithelium from more contaminated sites.

Bivalves

Abundance

Clams were collected in both years. One of the more dramatic observations we made was the extensive presence of empty softshell clam valves in the mudflats of the lower half of the Island End river, with a total absence of live animals, even after extensive digging.

Extensive histopathologic evaluations of soft shell clams were done in 1992 in order to determine what pathologies might be correlated with polluted stations. In 1993, (based on the results from 1992) soft shell clams and blue mussels were examined for an abbreviated list of pathologies that were identified as potentially resulting from pollution.

***Mya arenaria* - Histopathology**

Clams collected are summarized in Table 13, and the frequencies of pathologies in clams are shown in Tables 14 and 15 for 1992 and 1993 respectively. Published pathologies as well as previously undescribed diseases/conditions were identified in *M. arenaria* in 1992. Leukemia, gill hyperplasia (papilloma), and parasitic metacercaria were present in connective tissues of various portions of the animals' bodies. Also seen were siphon, gill and mantle inflammation and brown cell accumulation in the digestive gland and kidney. Brown cells are probably lipofuscin-filled inactive hemocytes also called ceroid bodies [29, 47, 48]. Chlamydial infection of the digestive gland, pericardial/brown exudate in the kidney, gill parasites and green gland (pericardial gland) changes were also seen. All of the above have previously been reported in *M. arenaria*. New pathologies/conditions identified in this study are 3 types of parasitic infections of the kidney, kidney hyperplasia, gonadal inflammation, and probable gill carcinomas. Gill carcinomas were identified in animals from one location .

Discriminant analysis statistics for *M. arenaria* are listed in Table 8. The contaminant loadings were classified as either low (Stn #'s 5 and 6), moderate (Stn #'s 1,2 and 9), or high (Stn #'s 3,4, 8). The pathological variables that are utilized for each of the DF's are listed in Table 16. As can be seen in Figure 6, the groups are widely separated when plotted according to the group centroids for DF1 and DF2.

***Mya arenaria* - Hemocyte Evaluation**

Immunohistochemical evaluation of hemocyte samples revealed the presence of varying levels of hemopoietic neoplasia in clams from 4 sites (Table 17). The most severely impacted site was the Fort Point Channel, with 25% of the sample affected. Low prevalences were also seen at Neponset, Chelsea and Wellfleet, but not Scorton, Saugus, or Weymouth. No apparent correlation with chemical exposure was detected.

***Mytilus edulis* - Histopathology**

The blue mussel *M. edulis* was collected at selected sites during 1993 (Table 18) and subjected to a similar protocol of pathological evaluation as for the soft shell clam. Pathologies observed (Table 19) included trematode infections of the gonads with associated inflammation, possible coccidial infections of the gonad, gill parasites, green gland (pericardial gland) abnormalities, leukemia (sarcoma) and brown cells in the digestive gland. All of the above have been previously identified in *M. edulis* or related bivalves. Trematode infections were severe in station 8 and 10.

The data were analyzed using discriminant function analysis for parameters listed in Table 16 with the groupings based on the location of the site. The groups could be discriminated based on 2 discriminant functions calculated from the 13 pathological predictor variables. A plot of the site separation based on the 2 discriminant functions can be found in Figure 7. DF1 was calculated from the variables G11, Kd2, Kd1, and Kd5 while DF2 was calculated from the remainder of the variables.

6. DISCUSSION

Fish Abundance

From our field observations, the abundance of fish from the areas sampled appeared to be inversely related to the degree of chemical contamination. This suggests that the chemical-associated pathological and biochemical changes evident in this study may well have resulted in population level recruitment effects to some degree. The methods utilized in this study were not designed to make quantitative estimates of population abundance, but our qualitative observations suggest this supposition. Another major

factor in the low availability of this species in the Boston stations may be substantial habitat degradation. In particular the extensive bulkheading of the intertidal zone, with loss of marsh grass and other salt marsh organisms presumably restricts the alongshore colonization, and recolonization following die-offs, of suitable habitat by mummichogs. It should be noted that only subadult fish were found in 1992 at the Island End River site, whereas at one time point in 1993 a good collection of adults was made. This must imply significant alongshore movements of this species with time. It is unclear whether these adults were the parents of the subadult fish caught the previous year.

Fish Histology

The most important observations on fish histology in this study are that of chemical-associated neoplasms and altered cellular foci in the adult fish from the Island End River. These lesions demonstrate a severe longstanding carcinogenic exposure of fish at this site. The other lesions and parasitic infections are interesting, but given our current knowledge they cannot be associated with differing levels of chemical exposure. We report them here to record the data so that should better understanding of their significance be available in the future, the data can be easily reappraised.

Neoplasms

Liver tumors and associated foci of altered hepatocytes were seen in adult mummichogs from the Island End River. These two lesions have been closely associated with chemical exposure in other wild populations of fish [27, 49], including *F. heteroclitus* [28]. Similar lesions have been induced experimentally in a variety of aquarium fish species following exposure to a number of hepatocarcinogens, including polynuclear aromatic hydrocarbons [50-53] and to extracts of contaminated sediments [54]. The absence of chemical-associated lesions in mummichogs from the Island End River in 1992 is unlikely to reflect major chemical changes at that site between the two years. It is almost certainly a result of the much smaller size of the fish sampled in 1992 (32 mm mean length), as compared to 1993 (76 mm total length) - see Table 2. It is known that altered liver cell foci and liver tumors apparently take a substantially longer time to develop in wild species than in aquarium-bred species such as medaka and trout. The time frame is not known for mummichogs, but in winter flounder from Boston Harbor we know that the youngest tumor-bearing animals were 5 years old [55], i.e. about 50% of their life expectancy. Therefore, we would expect the tumor-bearing

mummichogs to be at least 2 years old. The fish sampled in 1992 were probably spawned in 1991 and hence below the likely tumor-bearing age. Further sampling and age analysis of this population is warranted.

Macrophage Aggregations

Macrophage aggregates were only evident in fish from the Island End River in 1993. The reason for the absence of macrophages from the Island End River in 1992 compared with 1993 is that the 1992 animals were subadults, whereas the 1993 animals were adults. Age, along with chemical exposure, parasitism, and nutrition are known factors to affect macrophage aggregation [56]. The 1992 animals were therefore presumably too young to have acquired notable aggregations of macrophages. The absence of macrophage aggregations from other contaminated sites in the Boston area may reflect the short lifespan of this species in contrast to other species in Boston that have shown severe macrophage aggregation at similar levels of contamination [4].

Hepatic Coccidiosis

In this study hepatic coccidiosis was only found at one of the two reference sites, Wellfleet. Increased coccidiosis at cleaner sites was also found by Vogelbein (pers. comm.) in a study of mummichogs in the Chesapeake Bay. In the discriminant analysis hepatic coccidiosis proved to be a useful parameter for site discrimination, however it is important to stress that there is no obvious known causal relationship between chemical exposure and parasite burden. Such a relationship has at times been suggested [57], but will always depend on the host, parasite and vector in question. There is certainly no consensus at this time on a general relationship between chemical exposure and parasitism. Significant parameters depend on the relative toxicities of chemical(s) to the host, parasite, and vector. The chemicals might be immunosuppressive to the host, allowing greater parasitism at contaminated sites, or the chemicals might be either parasiticidal or toxic to vector species, allowing more parasitism at cleaner sites. For these reasons one cannot expect field studies such as described here to demonstrate a predictable net effect of chemical exposure on host-parasite relationships. There are various anecdotal reports of both increased and decreased parasitism with chemical exposure. We have such unpublished data in our studies, and probably the net result depends on the specific interactions of the parameters described above. There is a need

for experimental studies of these relationships to evaluate the cause and effect relationships between parasitism and chemical exposure

Hepatic Necrosis

This lesion was predominantly found at the Island End River Site in adult fish in 1993. It was also seen in a few fish from Wellfleet in 1992, probably associated with coccidian infection. Necrotic cell death reflects cells dying for non-physiological reasons, from any of a great number of causes and again cannot be used to imply a cause and effect relationship with chemical exposure.

Hepatic Clear Cells

In fish from the Fort Point Channel we observed isolated nests of clear cells. These cells differed significantly from larger foci of clear cells often described in fish and rodents exposed to chemicals in the field and laboratory [58]. The significance of our observation is unclear.

Hepatic Megalocytosis

In fish from Scorton Creek we observed a significant prevalence of hepatocytes with substantially enlarged nuclei. In the past this lesion has been associated with chemical exposure in some marine fish species [58]. In this study no such relationship appears to be present and again the significance of the observation is unclear.

Gill Hyperplasia

This lesion was observed at a variety of prevalences in this study. The respiratory epithelium of fish proliferates following a wide range of noxious stimuli, including altered pH of the water, anoxia, parasitism and chemical exposure. One particular problem in interpreting this data is that gill exoparasites will induce gill hyperplasia, and the diagnostic methods used in this study would not have detected many of the unicellular parasites commonly found on fish gills. Therefore the observations of gill epithelial hyperplasia cannot be explained in this dataset.

Spleen Macrophage Aggregations

This lesion was only seen with any abundance in adult fish from the Island End River (55%). Other studies have linked this lesion to chemical exposure (G. Gardner pers. comm.), although the same caveats expressed above for this lesion in the liver also apply here.

Other Parasitic Infections

Tables 3 and 4 also list a series of other parasites in a variety of tissues. The same comments above regarding hepatic coccidiosis also apply in these cases.

Discriminant analysis

From the statistical analysis we have also demonstrated the utility of a number of specific lesion types in predicting levels of contaminant exposure at particular sites, although the primary predictors were parasitic lesions, which are determinant variables whose association with chemical exposure is unclear and probably different for different hosts, parasites and sites..

Fish Biochemistry

Cytochrome P4501A protein levels have been shown to increase with exposure to dioxin and other aromatic compounds that mimic dioxin in their ability to bind the aromatic hydrocarbon receptor and induce cytochrome P4501A. These compounds include halogenated biphenyls, dibenzofurans and polynuclear aromatic hydrocarbons. Increasing levels of these compounds increase the amount of P4501A protein usually with concomitant increases in enzymatic activity, measured here by metabolism of a substrate ethoxyresorufin-o-deethylase (EROD). The data presented in Tables 9 and 10 clearly show statistically significant increased P4501A and EROD activity at the contaminated sites in comparison with the two reference sites. Specifically fish from the Island End River, Fort Point Channel, Saugus River, and Weymouth Fore River showed statistically elevated P4501A protein content as compared with the Wellfleet and Scorton Creek sites. As described in the results section there are physiological factors that affect P4501A expression involving oogenesis in the spring caught females. The reduced expression in females caught in the spring (Table 10) is evidence of this effect.

The variability in the P4501A and EROD data could result from two primary types of variable:

1. The only available data on chemical contamination is for polynuclear aromatic hydrocarbons in the sediment. Other significant compounds that also affect cytochrome P4501A protein expression, and EROD activity include dioxins, dibenzofurans and coplanar polychlorinated biphenyls (PCB's) in the sediment and in the fish themselves. A fuller understanding of this data would be possible if analyses for all of the above compounds were available. PCB analysis should be congener specific as there is a major difference in the effects of different congeners on cytochrome P4501A. Specifically the coplanar congeners are the primary inducers of cytochrome P4501A protein. Those same congeners also induce EROD activity up to a certain level, above which they then inhibit EROD activity. Such confounding relationships may underlie the differences between the cytochrome P4501A protein expression and EROD activity shown in this data set.
2. It is also possible that P4501A is also induced by certain natural plant products similar in structure to the manmade chemicals described above. This possibility arises from the assumption that the cytochrome P450 systems in vertebrates evolved to metabolize natural endogenous and exogenous compounds. There are numerous studies showing the metabolism of a diversity of natural substrates in vertebrates. This issue has recently been discussed in relation to fishes [59]. If such effects are operating in these fish they appear to at most have reduced the closeness of fit, without destroying obvious trends.

The data expressed here for P4501A protein content are all relative, as we do not have purified P4501A protein from mummichog to use as an internal immunoblot standard. However, availability of absolute numbers would not alter the interpretation of the data significantly.

A further potential variable affecting changes in cytochrome P4501A expression with chemical exposure is that these compounds also have other systemic toxicity in addition to P4501A induction. These effects, especially at high doses will affect the ability of the organisms to respond with P4501A alteration. Thus the variable P4501A increase at the high levels of exposure may well reflect systemic toxicity associated with other known and unknown toxic effects of measured and unmeasured chemicals and heavy metals. There are many other toxic and carcinogenic compounds and elements potentially present in coastal sediments. Cases could be made for the analysis of amongst others, aromatic amines, chlorinated pesticides, and heavy metals. The decision to include such analyses on a routine basis must be made after a cost benefit analysis. In the case of this study we opted for a pair of integrative biomarkers that were relatively independent on choice of the correct chemical target analytes, and one representative

class of chemicals, PAH's. Essentially the biomarker approach uses the assumption that measurable biological effects are an economically viable and more informative substitute for specific broad spectrum chemical analysis. Choice of biomarker(s) is of course a major topic for consideration and has been discussed extensively elsewhere [14].

Comparison of Mummichog Data with other Fish Histopathology and Biochemistry from the Massachusetts Bays

Most of the available toxicologic histopathology on fish in the Mass. Bays has been on the winter flounder [4, 55, 60]. In both winter flounder and mummichog severe chemical exposure leads to liver tumors. However, the nature of the tumors and preneoplastic conditions in the two species are markedly different. In the winter flounder, tumors are preceded by stepwise development of a series of lesions described as hydropic vacuolation [5, 6, 61] and other primarily biliary changes. The prevalence of hydropic vacuolation has proven to be a very useful, repeatable assay for chemical exposure effects in winter flounder from a series of sites monitored for the Massachusetts Water Resources Authority [7, 13, 62]. Hydropic vacuolation has not been seen in mummichogs. It appears to precede the formation of tumors in winter flounder, which are primarily cholangiocellular in nature. The analogous lesion in mummichogs, rarely seen in winter flounder, are foci of altered hepatocytes, seen in this study in 50% of the adult mummichogs from the Island End River. These lesions are thought to precede the predominantly hepatocellular neoplasms in mummichogs. We suspect that mummichogs from the Fort Point Channel site might well have yielded a low but significant prevalence of these focal lesions if we had been able to sample mature adults from that site (Table 2). Likewise there may well be other heavily contaminated sites in the Mystic/ Chelsea River system with similar prevalences. A detailed survey of these two rivers looking for hepatic histopathology in adult mummichogs would be an economically viable method for highlighting other hot spots in addition to the Island End River. It appears that chemical-associated histopathology in the mummichog is primarily useful at the highly contaminated end of the environmental spectrum. Most of the discriminatory changes seen in mummichogs from the cleaner sites were parasitic in nature, and not necessarily associated with chemical exposure. Thus histology of mummichogs from cleaner sites is of less obvious value in monitoring for the effects of chemical contaminants. This is in contrast to the winter flounder, where liver histopathology has proven useful even at only fairly uncontaminated sites such as the future MWRA outfall site.

The biochemical alterations described here can also be compared to other studies. There are no published data on mummichog P4501A levels in fish from the Mass. Bays. In winter flounder P4501A induction and EROD activity was higher in fish from Boston Harbor and Plymouth as compared to fish from Nantucket and Buzzards Bay [3]. Also, winter flounder from the Narrow River in Rhode Island were substantially more induced than from Georges Bank [63]. Thus the findings in this study are comparable to those in winter flounder, and the monitoring of cytochrome P4501A content and EROD activity should be routinely included in monitoring programs in both winter flounder, as sentinels of coastal bottom sediments, and mummichogs, of estuarine intertidal sediments in sites ranging from low to high chemical contamination.

Fish Immunohistochemistry

Immunohistochemical detection of cytochrome P4501A expression in hepatocytes (Table 12) showed a certain degree of similarity with biochemical analysis of liver homogenates (Tables 9 and 10). Other tissues showed a variety of patterns, without necessarily following the trend seen in the liver. The assessment of immunohistochemically stained protein in this species is less quantitative than the densitometric assessment of immunoblots, as used for the biochemical analysis. Variables that can confound immunohistochemical analysis include masking of the antigen by fixation, variable stain development, and variation in observer interpretation. Our experience in this species proved to be less straight forward than in other species where we have often used the technique successfully. For these reasons we used the biochemical assays to a greater degree than originally planned, as described above.

To summarize the fish data we have, without incurring the substantial cost of a broad scale chemical analysis, provided evidence of significant chemical-biological effect at the contaminated sites both at the biochemical and morphological levels. These biomarker endpoints should be used both to define chemically compromised sites, and monitor attempts to remediate them.

Bivalve abundance

Soft shell clams were present at all of the intertidal sites samples, except the Island End River. The methods we employed to collect these animals were not designed to be

quantitative, but they were surprisingly abundant at the Fort Point Channel, Chelsea River and Neponset River sites, indeed their abundance throughout the Boston Harbor system, wherever a shelving intertidal soft bottom was present, was as remarkable as the relative absence of mummichogs.

Bivalve Histopathology

Previous studies have identified many types of lesions in various species of mollusks that exclusively occur in, or appear to be more commonly found in, animals from polluted waters or in animals exposed in the laboratory to polluted sediments. Bivalve lesions previously associated with pollution and found in the soft shell clams and blue mussels examined in this study (Tables 13 & 14) are: gill hyperplasia (papilloma) [64, 65], leukemia[31], kidney hyperplasia [64, 65], increased brown cells² (ceroid bodies) [29], and gonadal inflammation (unassociated with parasites) [29]. Of the other lesions seen in these animals, the association of kidney parasites and green gland (pericardial gland) hyperplasia with pollution has not been described to date.

While gill papillomas have been induced with polluted sediments in the laboratory [65] and have been associated with polluted natural environments [64], the occurrence of carcinomas of possible gill origin have not been identified before. Extensive metastasis of tumor to various other tissues of the clams was identified in all affected animals. However, only in one animal was a possible gonadal tubule identified as containing neoplastic cells. While gonadal tumors with metastasis to the gill have been identified in *M. arenaria* [66], because of the large size of the gill tumors, appearance of continuity with the gill epithelium, extensive metastatic foci of tumor within other tissues of these animals, and lack of involvement of gonadal tubules with tumor, carcinoma of primary gill origin was favored over carcinoma of gonadal origin. However, transmission electron microscopic examination of the tumor cells should confirm the cell of origin. Carcinomas of possible gill origin were identified in several soft shell clams in this study, but from only one location (Saugus). It is not known if this lesion is directly due to the effects of a specific pollutant or combination of pollutants, or to some other reason such as virus infection , or even a combination of the above

²Brown cells [47, 48] are probably non-circulating hemocytes that contain enough brown granules of various sizes to cause the cell to round up and appear granular and brown on hematoxylin and eosin staining. The term has been used in the literature to describe inflammation in bivalves. The nature of the granules is not known in most cases, but is assumed to represent "a change in host-fat metabolism" [48] and probably represents lipofuscin pigments.

afflictions. However, the occurrence of the tumor in animals from one area indicates that it is a site specific lesion. Thus it will probably not be a useful lesion for monitoring pollution. Further studies of the cause of this lesion are needed.

M. edulis showed decreased numbers of pathologies associated with polluted waters as compared with *M. arenaria*. However, the possible causative relationship of pollution with parasitic gonadal infections in *M. edulis* needs to be further investigated.

An increased occurrence of leukemia (sarcoma) in *M. arenaria* and *M. edulis* collected from polluted waters was not identified in this study. The finding of 4/20 clams from the Fort Point Channel with leukemia was not statistically significant. However, it may well be biologically significant. To evaluate this a larger sample from that site should be obtained and analyzed. It is possible that the lack of positive correlation between leukemia in bivalves and pollution resulted either from the type of pollution in the sites studied, or the lack of some other promoting or initiating factor necessary for the development of leukemia in the clams and mussels.

In this study individual lesion types (such as gonadal inflammation or kidney hyperplasia) show an increase in animals from more severely polluted sites. The more important finding however, is the cumulative increase in the frequency of certain lesion types and severities in animals from polluted waters. Such an increase in the number and severity of lesions in polluted animals may reflect a decrease in condition and cellular and humeral defense abilities [29] as well as a possible direct effect on the target cells by the pollutants [65]. The use of bivalves as biological monitors of pollution may be very appropriate and could provide information both in assessing the severity of pollution at a point in time as well as the effectiveness of remedial efforts.

7. Conclusions.

The following conclusions can be drawn from these studies:

- Extreme chemical stress at the Island End River appears to have lead to a high prevalence of liver neoplasia in mummichogs from this site. Soft shell clams were assumed to once being abundant but only shells were found at this site in 1992 and 1993.
- Sites in and around Boston Harbor showed elevated levels in fish of cytochrome P4501A protein and activity indicative of exposure to significant levels of hydrocarbons known to induce that protein.
- Soft shell clams and mummichogs exhibit a range of pathological change, some associated with chemical exposure, that allow prediction of levels of chemical contamination at each sample site.
- The Cape Cod Bay sites, Wellfleet, and Scorton Creek both exhibit low levels of contamination, and little biological change associated with chemical exposure.

8. Recommendations

- Attention should continue to be paid to the sources of chemical contamination in and around Boston Harbor. This area, especially its inner reaches, such as the Island End River are substantially more impacted than cleaner sites in Cape Cod Bay. In particular, attempts to minimize the ongoing toxic chemical inputs into the Mystic River, and the Island End River would allow gradual reduction of the severe biological problems evident in that area.
- Further interpretation of the biochemistry data is dependent on greater understanding of how complex mixtures of anthropogenic and biogenic aromatic compounds affect cytochrome P4501A in bottom-feeding fish. Research on this topic is highly necessary.
- Ongoing region wide biochemical and pathological biomarker monitoring should be continued to assess and encourage efforts to reduce toxic chemical pollution at source, and to improve the quality of wastewater treatment in urban areas, such as Greater Boston. Analysis of the changes reported here should be integral to the monitoring of environmental remediation efforts.
- Further research is necessary to define how liver tumors develop in mummichogs with time, possible causative agents of tumors in this species and *Mya arenaria*, and how the changes described here affect the growth and reproduction of these fish and shellfish.

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Table A1 - Sediment polynuclear aromatic hydrocarbon concentrations from 8 sites in the Massachusetts Bays

| Station Location | Stn. # | Stn. Samp. # | Duplicate? | Moisture (%) | Sediment description | Silt-clay (%) | TOC (mg/kg) | TOC (%) | Acenaphthene (ug/kg) | Acenaphthylene |
|------------------|--------|--------------|------------|--------------|--|---------------|-------------|---------|----------------------|----------------|
| Weymouth | 10 | 88970 | | 16 | dark gray sand, trace shells, some gravel | 5 | 7620 | 0.76 | 2.4 | nd |
| Weymouth | 11 | 88971 | | 23 | gray sand and silt | 28 | 10600 | 1.06 | 3.1 | 4.7 |
| Weymouth | 11 | 88971 | Yes | 23 | | | | | 3.3 | 4 |
| Neponset - Mya | 12 | 88972 | | 21 | gray silty sand with some shells | 8 | 5700 | 0.57 | 2.6 | 4.4 |
| Neponset - Mya | 12 | 88972 | Yes | 21 | | | | | 2.1 | 3.5 |
| Neponset - Mya | 13 | 88973 | | 23 | dark brown sand with shells | 7 | 4560 | 0.46 | 2.2 | 2.4 |
| Neponset -Fund | 14 | 88974 | | 55 | dark brown sand, silt, trace gravel & organics | 50 | 31800 | 3.18 | 150 | 58 |
| Neponset -Fund | 15 | 88975 | | 55 | dark brown organics with silt | 96 | 53200 | 5.32 | 33 | 42 |
| Neponset -Fund | 16 | 88976 | | 63 | dark brown silt with sand and organics | 98 | 47000 | 4.7 | 28 | 20 |
| Fort Point Chan. | 17 | 88977 | | 50 | black silt with sand and some organics | 54 | 89400 | 8.94 | 1600 | 250 |
| Fort Point Chan. | 18 | 88978 | | 56 | black organics with silt | 79 | 68600 | 6.86 | 490 | 190 |
| Fort Point Chan. | 19 | 88979 | | 20 | dark brown sand with silt, trace of organics | 13 | 68400 | 6.84 | 100 | 25 |
| Chelsea | 20 | 88980 | | 34 | dark gray silty sand with some gravel | 50 | 33000 | 3.3 | 26 | 29 |
| Chelsea | 21 | 88981 | | 29 | gray sandy silt with some shells | 63 | 17000 | 1.7 | 5.9 | 8.7 |
| Island End | 22 | 88982 | | 30 | gray silty clay with sand | 72 | 68400 | 6.84 | 1600 | 3700 |
| Island End | 22 | 88982 | Yes | 30 | | | | | 1700 | 2900 |
| Island End | 23 | 88983 | | 39 | gray silty clay with some gravel | 54 | 46100 | 4.61 | 3900 | 8100 |
| Saugus River | 24 | 88984 | | 20 | black sand with silt, trace shells | 10 | 10100 | 1.01 | 12 | 33 |
| Saugus River | 33 | 88993 | | 47 | black silty sand | 46 | 27000 | 2.7 | 25 | 70 |
| Wellfleet | 26 | 88986 | | 24 | gray sand | 2 | 2890 | 0.29 | nd | nd |
| Wellfleet | 27 | 88987 | | 16 | gray sand with some gravel | 2 | 2880 | 0.29 | nd | nd |
| Wellfleet | 27 | 88987 | Yes | 22 | | | | | nd | nd |
| Scorton Creek | 28 | 88988 | | 19 | light gray fine sand with trace shells | 1 | 1330 | 0.13 | nd | nd |
| Scorton Creek | 29 | 88989 | | 30 | light gray sand | 1 | 1460 | 0.13 | nd | nd |
| Scorton Creek | 30 | 88990 | | 15 | light brown sand | 10 | 2620 | 0.26 | nd | nd |

Table A1 - Sediment polynuclear aromatic hydrocarbon concentrations from 8 sites in the Massachusetts Bays

| Station Location | Anthracene | Benzo(a)anthracene | Benzo(b)fluoranthene | Benzo(k)fluoranthene | Benzo(a)pyrene | Benzo(ghi)perylene | Chrysene |
|------------------|------------|--------------------|----------------------|----------------------|----------------|--------------------|----------|
| Weymouth | 6.2 | 22 | 30 | 24 | 23 | 21 | 30 |
| Weymouth | 11 | 52 | 65 | 47 | 60 | 45 | 66 |
| Weymouth | 12 | 46 | 53 | 43 | 54 | 43 | 58 |
| Neponset - Mya | 7.2 | 39 | 55 | 44 | 56 | 55 | 54 |
| Neponset - Mya | 6.6 | 36 | 56 | 42 | 51 | 45 | 52 |
| Neponset - Mya | 5.6 | 29 | 44 | 34 | 42 | 40 | 40 |
| Neponset -Fund | 630 | 1600 | 1200 | 1400 | 1400 | 1100 | 1600 |
| Neponset -Fund | 100 | 470 | 610 | 580 | 580 | 450 | 670 |
| Neponset -Fund | 80 | 600 | 730 | 620 | 710 | 640 | 780 |
| Fort Point Chan. | 3200 | 7200 | 6800 | 6000 | 7400 | 6000 | 7500 |
| Fort Point Chan. | 1400 | 4600 | 4200 | 3900 | 4400 | 4200 | 5100 |
| Fort Point Chan. | 330 | 1000 | 1100 | 1000 | 1000 | 870 | 1400 |
| Chelsea | 130 | 560 | 510 | 540 | 570 | 400 | 600 |
| Chelsea | 49 | 110 | 120 | 110 | 120 | 93 | 130 |
| Island End | 5500 | 16000 | 20000 | 16000 | 15000 | 14000 | 22000 |
| Island End | 6400 | 20000 | 30000 | 30000 | 20000 | 2200 | 30000 |
| Island End | 15000 | 50000 | 70000 | 50000 | 60000 | 30000 | 60000 |
| Saugus River | 120 | 600 | 500 | 510 | 610 | 400 | 640 |
| Saugus River | 140 | 880 | 960 | 890 | 960 | 900 | 1100 |
| Wellfleet | nd | nd | nd | nd | nd | nd | nd |
| Wellfleet | nd | nd | nd | nd | nd | nd | nd |
| Wellfleet | nd | nd | nd | nd | nd | nd | nd |
| Scorton Creek | nd | nd | nd | nd | nd | nd | nd |
| Scorton Creek | nd | nd | nd | nd | nd | nd | nd |
| Scorton Creek | 2.7 | 5.2 | 5.8 | 4.6 | 5.1 | 3.2 | 6.3 |

Table A1 - Sediment polynuclear aromatic hydrocarbon concentrations from 8 sites in the Massachusetts Bays

| Station Location | Dibenzo(a,h)anthracene | Fluoranthene | Fluorene | Indeno(1,2,3cd)pyrene | Naphthalene | Phenanthrene | Pyrene | Total PAH (ug/kg) | Total PAH (ppm) |
|------------------|------------------------|--------------|----------|-----------------------|-------------|--------------|--------|-------------------|-----------------|
| Weymouth | 4 | 55 | 3 | 2.6 | 2.4 | 27 | 53 | 305.6 | 0.3056 |
| Weymouth | 18 | 110 | 6.5 | 59 | 3.3 | 60 | 110 | 720.6 | 0.7206 |
| Weymouth | 17 | 98 | 5.8 | 56 | 5.4 | 55 | 100 | 653.5 | 0.6535 |
| Neponset - Mya | 19 | 86 | 3.9 | 66 | 6.1 | 39 | 92 | 629.2 | 0.6292 |
| Neponset - Mya | 12 | 84 | 3.6 | 48 | 4 | 38 | 85 | 568.8 | 0.5688 |
| Neponset - Mya | 12 | 74 | 3.1 | 49 | 4.4 | 31 | 72 | 484.7 | 0.4847 |
| Neponset -Fund | 320 | 4000 | 260 | 1100 | 26 | 2300 | 2800 | 19944 | 19.944 |
| Neponset -Fund | 170 | 1100 | 44 | 480 | 35 | 470 | 930 | 6764 | 6.764 |
| Neponset -Fund | 140 | 1300 | 37 | 550 | 26 | 470 | 1100 | 7831 | 7.831 |
| Fort Point Chan. | 1400 | 17000 | 14000 | 5700 | 780 | 11000 | 14000 | 109830 | 109.83 |
| Fort Point Chan. | 1100 | 12000 | 660 | 4300 | 290 | 6000 | 9100 | 61930 | 61.93 |
| Fort Point Chan. | 150 | 2600 | 130 | 960 | 65 | 1500 | 2200 | 14430 | 14.43 |
| Chelsea | 110 | 860 | 41 | 390 | 53 | 370 | 950 | 6139 | 6.139 |
| Chelsea | 28 | 280 | 15 | 100 | 19 | 120 | 210 | 1518.6 | 1.5186 |
| Island End | 4600 | 50000 | 3400 | 15000 | 870 | 9700 | 30000 | 227370 | 227.37 |
| Island End | 9 | 70000 | 3800 | 4000 | 1300 | 11000 | 40000 | 273309 | 273.309 |
| Island End | 2000 | 200000 | 13000 | 40000 | 1600 | 70000 | 80000 | 753600 | 753.6 |
| Saugus River | 120 | 1300 | 26 | 500 | 13 | 460 | 1100 | 6944 | 6.944 |
| Saugus River | 200 | 2200 | 48 | 900 | 40 | 780 | 1600 | 11693 | 11.693 |
| Wellfleet | nd | nd | nd | nd | nd | nd | nd | 0 | 0 |
| Wellfleet | nd | nd | nd | nd | nd | nd | nd | 0 | 0 |
| Wellfleet | nd | nd | nd | nd | nd | nd | nd | 0 | 0 |
| Scorton Creek | nd | nd | nd | nd | nd | nd | nd | 0 | 0 |
| Scorton Creek | nd | nd | nd | nd | nd | nd | nd | 0 | 0 |
| Scorton Creek | p | 13 | nd | 3.5 | nd | 11 | 11 | 71.4 | 0.0714 |

Table A2

Fundulus heteroclitus histology data - 1992

| Stn | ID # | Intestine | Renal tubules | Sk muscle | Exocrine pancreas | Endocrine pancreas | Gonad | Spleen | Spleen MA's | Comments on histology |
|-----|-----------|-----------|---------------|-----------|-------------------|--------------------|-------|--------|-------------|------------------------------------|
| 1 | 92D- 1220 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| 1 | 92D- 1221 | 0 | x | x | 0 | 0 | 0 | 0 | 4 | |
| 1 | 92D- 1222 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | |
| 1 | 92D- 1223 | 0 | x | x | 0 | 0 | 0 | 0 | x | Peritoneal granulomatous tissue |
| 1 | 92D- 1224 | 0 | 0 | x | 0 | 0 | x | 2 | 0 | |
| 1 | 92D- 1225 | 0 | x | x | 0 | x | 0 | x | x | |
| 1 | 92D- 1226 | | | | | | | | | |
| 1 | 92D- 1227 | 0 | x | x | 0 | 0 | x | 0 | 0 | Metazoan parasite in gut lumen |
| 1 | 92D- 1228 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| 1 | 92D- 1229 | 0 | 0 | 0 | 0 | x | 0 | 0 | 1 | |
| 1 | 92D- 1230 | 0 | x | x | 0 | x | 0 | 0 | 0 | |
| 1 | 92D- 1231 | 0 | x | x | 0 | x | 0 | 0 | 0 | |
| 1 | 92D- 1232 | 0 | x | x | 0 | x | 0 | 0 | 0 | |
| 1 | 92D- 1233 | 0 | x | x | 0 | x | x | 2 | 0 | |
| 1 | 92D- 1234 | 0 | 0 | x | 0 | x | 0 | 0 | 0 | |
| 1 | 92D- 1235 | 0 | x | x | 0 | x | x | x | x | |
| 1 | 92D- 1236 | 0 | 0 | 0 | 0 | x | 0 | x | x | |
| 1 | 92D- 1237 | 0 | 0 | 0 | 0 | 0 | 0 | x | x | |
| 1 | 92D- 1238 | 0 | x | x | 0 | x | x | 0 | 0 | |
| 1 | 92D- 1239 | 0 | 0 | 0 | 0 | x | 0 | x | x | |
| 2 | 92D- 1240 | 0 | x | x | 0 | x | 0 | 0 | 0 | Patches of hepatocyte regeneration |
| 2 | 92D- 1241 | 0 | 0 | x | 0 | x | 0 | 0 | 0 | |
| 2 | 92D- 1242 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | |
| 2 | 92D- 1243 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | |
| 2 | 92D- 1244 | 0 | 0 | 0 | 0 | 0 | 0 | x | x | Basophilic hepatocytes |
| 2 | 92D- 1245 | 0 | x | x | 0 | x | 0 | 0 | 0 | Basophilic hepatocytes |
| 2 | 92D- 1246 | | 0 | 0 | 0 | x | 0 | 0 | 0 | Basophilic hepatocytes |
| 2 | 92D- 1247 | 0 | 0 | 0 | 0 | x | x | x | x | |
| 2 | 92D- 1248 | 0 | 0 | x | 0 | x | 0 | 0 | 0 | |
| 2 | 92D- 1249 | 0 | x | x | 0 | x | 0 | 0 | 1 | |
| 2 | 92D- 1250 | 0 | 0 | 0 | 0 | x | 0 | x | x | |
| 2 | 92D- 1251 | 0 | 0 | 0 | 0 | x | x | 0 | 1 | Metazoa bile duct |
| 2 | 92D- 1252 | 0 | x | 0 | 0 | x | 0 | 0 | 0 | |
| 2 | 92D- 1253 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 | |
| 2 | 92D- 1254 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 | |
| 2 | 92D- 1255 | 0 | 0 | 0 | 0 | x | 0 | 0 | 1 | |

Table A2

Fundulus heteroclitus histology data - 1992

| Sm | ID# | Sex | TL | SL | Wet Wt. | Liver Wt. | Fin (0-4) | Liver (0-2) | LN2 Liv. Wt. | Sex (histo) | Liver MA's | Liver coccidia | Liver necrosis | Clear cells | Megalocytosis | Cardiac nematodes | Cardiac muscle | Gill Trem. | Gill hyperpl |
|----|-----------|-----|-----|----|---------|-----------|-----------|-------------|--------------|-------------|------------|----------------|----------------|-------------|---------------|-------------------|----------------|------------|--------------|
| 2 | 92D- 1256 | F | 77 | 63 | 5.8 | 0.23 | 0 | 0 | | F | 0 | 0 | 0 | | | x | x | 3 | 2 |
| 2 | 92D- 1257 | | 75 | 64 | 5.6 | 0.19 | 0 | 0 | | F | 0 | 0 | 0 | | | x | x | 0 | 1 |
| 2 | 92D- 1258 | M | 70 | 58 | 4.3 | 0.12 | 0 | 0 | | M | 0 | 0 | 0 | | | x | 0 | 1 | 1 |
| 2 | 92D- 1259 | | 70 | 58 | 4.1 | 0.14 | 0 | 0 | | F | 0 | 0 | 0 | | | x | x | 0 | 2 |
| 3 | 92D- 1260 | F | 102 | 89 | 13.4 | 0.53 | 0 | 0 | 0.15 | F | 0 | 0 | 0 | | | x | 0 | 0 | 2 |
| 3 | 92D- 1261 | F | 85 | 70 | 8.8 | 0.39 | 0 | 0 | 0.15 | | | | | | | | | | |
| 3 | 92D- 1262 | F | 95 | 80 | 10 | 0.38 | 0 | 0 | 0.14 | F | 0 | 0 | 0 | | | x | x | 0 | 3 |
| 3 | 92D- 1263 | F | 117 | 95 | 17.3 | 0.64 | 0 | 0 | 0.14 | F | 0 | 0 | 0 | | | x | 0 | 0 | 2 |
| 3 | 92D- 1264 | M | 94 | 80 | 12.4 | 0.46 | 0 | 0 | 0.13 | x | 0 | 0 | 0 | | | x | 0 | 0 | 2 |
| 3 | 92D- 1265 | F | 95 | 83 | 12.6 | 0.54 | 0 | 0 | | F | 0 | 0 | 0 | | | x | 0 | 0 | 2 |
| 3 | 92D- 1266 | | 92 | 75 | 11.1 | 0.51 | 0 | 0 | | F | 0 | 0 | 0 | | | x | 0 | 0 | 3 |
| 3 | 92D- 1267 | F | 83 | 72 | 9.3 | 0.35 | 0 | 0 | | M | 0 | 0 | 0 | | | 0 | 0 | x | x |
| 3 | 92D- 1268 | F | 85 | 73 | 7.9 | 0.23 | 0 | 0 | | x | 0 | 0 | 0 | | | x | 0 | x | x |
| 3 | 92D- 1269 | M | 80 | 70 | 7.6 | 0.27 | 0 | 0 | | M | 0 | 0 | 0 | | | 0 | 0 | 0 | 3 |
| 3 | 92D- 1270 | M | 81 | 70 | 7.4 | 0.25 | 0 | 0 | | M | 0 | 0 | 0 | | | x | 0 | 0 | 0 |
| 3 | 92D- 1271 | F | 85 | 71 | 7.9 | 0.19 | 0 | 0 | | F | 0 | 0 | 0 | | | x | x | 1 | 0 |
| 3 | 92D- 1272 | | 91 | 78 | 8.6 | 0.58 | 0 | 0 | | x | 0 | 0 | 0 | | | 0 | 0 | 0 | 2 |
| 3 | 92D- 1273 | F | 82 | 67 | 7.4 | 0.4 | 0 | 0 | | F | 0 | 0 | 0 | | | x | 0 | x | x |
| 3 | 92D- 1274 | F | 88 | 73 | 10 | 0.38 | 0 | 0 | | F | 0 | 0 | 0 | | | x | 0 | 0 | 2 |
| 3 | 92D- 1275 | F | 83 | 70 | 7.5 | 0.24 | 0 | 0 | | F | 0 | 0 | 0 | | | 0 | 0 | 0 | 2 |
| 3 | 92D- 1276 | M | 89 | 74 | 9.9 | 0.3 | 0 | 0 | | x | 0 | 0 | 0 | | | 0 | 0 | 0 | 0 |
| 3 | 92D- 1277 | F | 80 | 69 | 7.3 | 0.27 | 0 | 0 | | x | 0 | 0 | 0 | | | x | 0 | 0 | 1 |
| 3 | 92D- 1278 | F | 73 | 62 | 5.6 | 0.2 | 0 | 0 | | F | 0 | 0 | 0 | | | x | x | 0 | 2 |
| 3 | 92D- 1279 | M | 78 | 67 | 6.4 | 0.22 | 0 | 0 | | M | 0 | 0 | 0 | | | x | 0 | 0 | 2 |
| 4 | 92D- 1280 | | 48 | 40 | 1.2 | | | | | M | 0 | 0 | 0 | | | x | x | 0 | 0 |
| 4 | 92D- 1281 | | 42 | 38 | 1 | | | | | M | 0 | 0 | 0 | | | x | x | x | x |
| 4 | 92D- 1282 | | 52 | 43 | 1.6 | | | | | F | 0 | 0 | 0 | | | 0 | 0 | x | x |
| 4 | 92D- 1283 | | 55 | 46 | 2 | | | | | F | 0 | 0 | 0 | 1 | | x | 0 | x | x |
| 4 | 92D- 1284 | | 50 | 43 | 1.8 | | | | | F | 0 | 0 | 0 | 2 | | x | x | x | x |
| 4 | 92D- 1285 | | 50 | 43 | 1.8 | | | | | F | 0 | 0 | 0 | 0 | | x | x | x | x |
| 4 | 92D- 1286 | | 50 | 42 | 1.5 | | | | | | | | | | | | | | |
| 4 | 92D- 1287 | | 42 | 36 | 0.8 | | | | | M | 0 | 0 | 0 | 0 | | x | x | x | x |
| 4 | 92D- 1288 | | 53 | 46 | 2 | | | | | x | 0 | 0 | 0 | 0 | | x | x | x | x |
| 4 | 92D- 1289 | | 50 | 43 | 1.6 | | | | | M | 0 | 0 | 0 | 0 | | x | x | x | x |
| 4 | 92D- 1290 | | 44 | 35 | 1.2 | | | | | F | 0 | 0 | 0 | 0 | | x | x | x | x |

Table A2

Fundulus heteroclitus histology data - 1992

| Stn | ID # | Intestine | Renal tubules | Sk muscle | Exocrine pancreas | Endocrine pancreas | Gonad | Spleen | Spleen MA's | Comments on histology |
|-----|-----------|-----------|---------------|-----------|-------------------|--------------------|-------|--------|-------------|--|
| 2 | 92D- 1256 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 | |
| 2 | 92D- 1257 | 0 | 0 | 0 | 0 | x | 0 | x | x | |
| 2 | 92D- 1258 | 0 | 0 | 0 | 0 | x | 0 | x | x | Small hepatic granuloma |
| 2 | 92D- 1259 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 | Intestinal helminth |
| 3 | 92D- 1260 | 0 | x | 0 | 0 | x | 0 | x | x | |
| 3 | 92D- 1261 | | | | | | | | | |
| 3 | 92D- 1262 | | 0 | 0 | 0 | x | 0 | 0 | 0 | Encysted masses of unicellular parasites in gill |
| 3 | 92D- 1263 | 0 | 0 | 0 | 0 | x | 0 | 0 | 1 | |
| 3 | 92D- 1264 | 0 | 0 | 0 | 0 | x | x | x | x | |
| 3 | 92D- 1265 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 | |
| 3 | 92D- 1266 | 0 | 0 | 0 | 0 | x | 0 | 0 | 2 | Encysted masses of unicellular parasites in gill |
| 3 | 92D- 1267 | 0 | 0 | 0 | 0 | x | 0 | 0 | 2 | |
| 3 | 92D- 1268 | 0 | 0 | 0 | 0 | x | x | 0 | 3 | |
| 3 | 92D- 1269 | 0 | 0 | x | 0 | x | 0 | 0 | | Unicellular parasites in gill, metazoa in liver |
| 3 | 92D- 1270 | 0 | 0 | 0 | 0 | x | 0 | 0 | 2 | Encysted masses of unicellular parasites in gill |
| 3 | 92D- 1271 | 0 | 0 | 0 | 0 | x | 0 | 0 | 2 | |
| 3 | 92D- 1272 | 0 | 0 | 0 | 0 | 0 | x | 0 | 0 | Metazoan in intrahepatic vessel |
| 3 | 92D- 1273 | 0 | 0 | 0 | 0 | x | 0 | 0 | 2 | |
| 3 | 92D- 1274 | 0 | 0 | 0 | 0 | x | 0 | 0 | 1 | |
| 3 | 92D- 1275 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 | |
| 3 | 92D- 1276 | 0 | 0 | x | 0 | 0 | x | 0 | 0 | |
| 3 | 92D- 1277 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 | |
| 3 | 92D- 1278 | 0 | x | x | 0 | 0 | 0 | 0 | 0 | |
| 3 | 92D- 1279 | 0 | 0 | 0 | 0 | x | 0 | 0 | 1 | Metazoans in intrahepatic vessels |
| 4 | 92D- 1280 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 | |
| 4 | 92D- 1281 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 | |
| 4 | 92D- 1282 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 | |
| 4 | 92D- 1283 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 | |
| 4 | 92D- 1284 | 0 | 0 | 0 | 0 | x | 0 | x | x | Gas gland in section |
| 4 | 92D- 1285 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 | |
| 4 | 92D- 1286 | | | | | | | | | Inadequate section to read |
| 4 | 92D- 1287 | 0 | 0 | 0 | 0 | x | 0 | x | x | |
| 4 | 92D- 1288 | 0 | 0 | 0 | 0 | x | x | x | x | |
| 4 | 92D- 1289 | 0 | 0 | 0 | 0 | x | 0 | x | x | |
| 4 | 92D- 1290 | 0 | 0 | 0 | 0 | x | 0 | x | x | |

Table A2

Fundulus heteroclitus histology data - 1992

| Stn | ID# | Sex | TL | SL | Wet Wt. | Liver Wt. | Fin (0-4) | Liver (0-2) | LN2 Liv. Wt. | Sex (histo) | Liver MA's | Liver coccidia | Liver necrosis | Clear cells | Megalocytosis | Cardiac nematodes | Cardiac muscle | Gill Trem. | Gill hyperpl |
|-----|-----------|-----|-----|----|---------|-----------|-----------|-------------|--------------|-------------|------------|----------------|----------------|-------------|---------------|-------------------|----------------|------------|--------------|
| 4 | 92D- 1291 | | 45 | 36 | 1.2 | | | | | M | 0 | 0 | 0 | 0 | | x | x | 0 | 0 |
| 4 | 92D- 1292 | | 52 | 43 | 1.7 | | | | | F | 0 | 0 | 0 | 0 | | x | 0 | x | x |
| 4 | 92D- 1293 | | 48 | 40 | 1.2 | | | | | F | 0 | 0 | 0 | 0 | | x | x | x | x |
| 4 | 92D- 1294 | | 45 | 40 | 1.3 | | | | | F | 0 | 0 | 0 | 0 | | x | x | x | x |
| 4 | 92D- 1295 | | 48 | 39 | 1.5 | | | | | F | 0 | 0 | 0 | 0 | | x | x | 0 | 2 |
| 4 | 92D- 1296 | | 50 | 44 | 1.5 | | | | | M | 0 | 0 | 0 | 0 | | x | x | x | x |
| 4 | 92D- 1297 | | 40 | 31 | 1 | | | | | F | 0 | 0 | 0 | 0 | | x | 0 | x | x |
| 4 | 92D- 1298 | | 51 | 45 | 1.9 | | | | | M | 0 | 0 | 0 | 1 | | x | 0 | x | x |
| 4 | 92D- 1299 | | 49 | 43 | 1.3 | | | | | F | 0 | 0 | 0 | 0 | | x | 0 | x | x |
| 4 | 92D- 1300 | | 45 | 33 | 1.2 | | | | 0.05 | | | | | | | | | | |
| 4 | 92D- 1301 | | 53 | 43 | 1.5 | | | | 0.03 | | | | | | | | | | |
| 4 | 92D- 1302 | | 53 | 45 | 1.7 | | | | 0.06 | | | | | | | | | | |
| 4 | 92D- 1303 | | 45 | 38 | 1.1 | | | | 0.04 | | | | | | | | | | |
| 4 | 92D- 1304 | | 41 | 39 | 0.9 | | | | 0.01 | | | | | | | | | | |
| 5 | 92D- 1310 | F | 100 | 87 | 14.7 | 0.72 | 0 | 0 | 0.18 | F | 0 | 0 | 0 | 0 | | 0 | 0 | 0 | 0 |
| 5 | 92D- 1311 | F | 103 | 89 | 17.2 | 0.55 | 0 | 0 | 0.16 | F | 0 | 0 | 0 | 0 | | 0 | 0 | 0 | 0 |
| 5 | 92D- 1312 | F | 103 | 89 | 17.2 | 0.55 | 0 | 0 | 0.16 | F | 0 | 0 | 0 | 0 | | 0 | 0 | 0 | 0 |
| 5 | 92D- 1313 | M | 90 | 77 | 9.9 | 0.27 | 0 | 0 | 0.13 | M | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 1 |
| 5 | 92D- 1314 | F | 99 | 85 | 13.7 | 0.56 | 0 | 0 | 0.17 | F | 0 | 0 | 0 | 0 | 0 | x | 0 | 0 | 1 |
| 5 | 92D- 1315 | F | 70 | 61 | 4.34 | 0.09 | 0 | 0 | | F | 0 | 0 | 0 | 0 | 0 | x | x | 0 | 0 |
| 5 | 92D- 1316 | F | 83 | 72 | 7.1 | 0.18 | 0 | 0 | | x | 1 | 0 | 0 | 0 | 1 | x | 0 | 0 | 0 |
| 5 | 92D- 1317 | F | 88 | 74 | 10.5 | 0.33 | 0 | 0 | | x | 0 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 |
| 5 | 92D- 1318 | F | 73 | 64 | 6.19 | 0.15 | 0 | 0 | | x | 0 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 |
| 5 | 92D- 1319 | F | 95 | 82 | 11 | 0.45 | 0 | 0 | | F | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 5 | 92D- 1320 | F | 78 | 68 | 5.95 | 0.18 | 0 | 0 | | F | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 5 | 92D- 1321 | F | 95 | 83 | 13.2 | 0.58 | 0 | 0 | | x | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 5 | 92D- 1322 | M | 93 | 78 | 11.8 | 0.44 | 0 | 0 | | M | 0 | 0 | 0 | 0 | 0 | x | x | 0 | 0 |
| 5 | 92D- 1323 | F | 93 | 77 | 11.4 | 0.4 | 0 | 0 | | F | 0 | 0 | 0 | 0 | 0 | x | x | 0 | 2 |
| 5 | 92D- 1324 | M | 83 | 68 | 7.91 | 0.27 | 0 | 0 | | M | 0 | 0 | 0 | 0 | 0 | x | x | 0 | 0 |
| 5 | 92D- 1325 | F | 87 | 73 | 9.22 | 0.16 | 0 | 0 | | F | 0 | 0 | 0 | 0 | 0 | x | 0 | 0 | 2 |
| 5 | 92D- 1326 | F | 97 | 83 | 11.9 | 0.35 | 0 | 0 | | F | 0 | 0 | 0 | 0 | 0 | x | 0 | 0 | 1 |
| 5 | 92D- 1327 | F | 86 | 72 | 8.23 | 0.21 | 0 | 0 | | F | 0 | 0 | 0 | 0 | 0 | x | 0 | 0 | 1 |
| 5 | 92D- 1328 | F | 85 | 72 | 8.5 | 0.24 | 0 | 0 | | F | 0 | 0 | 0 | 0 | 0 | x | 0 | 0 | 1 |
| 5 | 92D- 1329 | F | 73 | 64 | 5.11 | 0.06 | 0 | 0 | | F | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 6 | 92D- 1501 | F | 104 | 90 | 14.8 | 0.35 | 0 | 0 | 0.1 | x | 0 | 2 | 0 | 0 | 0 | x | 0 | 0 | 2 |

Table A2

Fundulus heteroclitus histology data - 1992

| Site | ID # | Intestine | Renal tubules | Sk muscle | Exocrine pancreas | Endocrine pancreas | Gonad | Spleen | Spleen MA's | Comments on histology |
|------|-----------|-----------|---------------|-----------|-------------------|--------------------|-------|--------|-------------|---|
| 4 | 92D- 1291 | 0 | 0 | 0 | 0 | x | 0 | x | x | |
| 4 | 92D- 1292 | 0 | 0 | 0 | 0 | x | 0 | x | x | |
| 4 | 92D- 1293 | 0 | 0 | 0 | 0 | x | 0 | x | x | |
| 4 | 92D- 1294 | 0 | 0 | 0 | 0 | x | 0 | x | x | |
| 4 | 92D- 1295 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 | |
| 4 | 92D- 1296 | 0 | 0 | 0 | 0 | x | 0 | x | x | |
| 4 | 92D- 1297 | 0 | 0 | 0 | 0 | x | 0 | x | x | |
| 4 | 92D- 1298 | 0 | 0 | 0 | 0 | x | 0 | x | x | |
| 4 | 92D- 1299 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 | |
| 4 | 92D- 1300 | | | | | | | | | |
| 4 | 92D- 1301 | | | | | | | | | |
| 4 | 92D- 1302 | | | | | | | | | |
| 4 | 92D- 1303 | | | | | | | | | |
| 4 | 92D- 1304 | | | | | | | | | |
| 5 | 92D- 1310 | 0 | 0 | 0 | 0 | x | 0 | 0 | 1 | |
| 5 | 92D- 1311 | 0 | 0 | 0 | 0 | x | 0 | 0 | 1 | |
| 5 | 92D- 1312 | 0 | 0 | 0 | 0 | x | 0 | 0 | 2 | |
| 5 | 92D- 1313 | 0 | 0 | 0 | 0 | x | 0 | x | x | Megalocytic hepatocytes, unicellular gill parasites |
| 5 | 92D- 1314 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 | |
| 5 | 92D- 1315 | 0 | 0 | 0 | 0 | x | 0 | 0 | 1 | |
| 5 | 92D- 1316 | 0 | 0 | 0 | 0 | 0 | x | 0 | 0 | Basophilic hepatocytes |
| 5 | 92D- 1317 | 0 | x | 0 | 0 | x | x | 0 | 0 | |
| 5 | 92D- 1318 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 | |
| 5 | 92D- 1319 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 | |
| 5 | 92D- 1320 | 0 | x | 0 | 0 | 0 | 0 | 0 | 0 | |
| 5 | 92D- 1321 | 0 | x | 0 | 0 | x | x | x | x | Encysted masses of unicellular parasites in gill |
| 5 | 92D- 1322 | 0 | 0 | 0 | 0 | 0 | 0 | x | x | Encysted masses of unicellular parasites in gill |
| 5 | 92D- 1323 | 0 | 0 | 0 | 0 | x | 0 | 0 | 1 | |
| 5 | 92D- 1324 | 0 | 0 | 0 | 0 | x | 0 | x | x | |
| 5 | 92D- 1325 | 0 | 0 | 0 | 0 | x | 0 | x | x | |
| 5 | 92D- 1326 | 0 | 0 | 0 | 0 | x | 0 | x | x | |
| 5 | 92D- 1327 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| 5 | 92D- 1328 | 0 | 0 | 0 | 0 | x | x | x | | |
| 5 | 92D- 1329 | 0 | 0 | 0 | 0 | x | 0 | x | x | |
| 6 | 92D- 1501 | 0 | 0 | 0 | 0 | x | x | x | x | |

Table A2

Fundulus heteroclitus histology data - 1992

| Stn | ID # | Sex | TL | SL | Wet Wt. | Liver Wt. | Fin (0-4) | Liver (0-2) | LN2 Liv. Wt. | Sex (histo) | Liver MA's | Liver coccidia | Liver necrosis | Clear cells | Megalocytosis | Cardiac nematodes | Cardiac muscle | Gill Trem. | Gill hyperpl |
|-----|-----------|-----|-----|----|---------|-----------|-----------|-------------|--------------|-------------|------------|----------------|----------------|-------------|---------------|-------------------|----------------|------------|--------------|
| 6 | 92D- 1502 | F | 9.8 | 82 | 13.3 | 0.43 | 0 | 0 | 0.13 | x | 0 | 3 | 0 | 0 | 0 | x | 0 | 0 | 1 |
| 6 | 92D- 1503 | F | 105 | 94 | 16.7 | 0.49 | 0 | 0 | 0.13 | x | 0 | 3 | 0 | 0 | 0 | x | 0 | 0 | 1 |
| 6 | 92D- 1504 | F | 95 | 84 | 12.2 | 0.51 | 0 | 0 | 0.17 | x | 0 | 3 | 0 | 0 | 0 | x | 0 | 0 | 0 |
| 6 | 92D- 1505 | F | 105 | 90 | 13.3 | 0.35 | 0 | 0 | 0.35 | x | 0 | 3 | 0 | 0 | 0 | x | 0 | 1 | 1 |
| 6 | 92D- 1506 | F | 97 | 80 | 12.6 | 0.48 | 0 | 0 | 0.07 | x | 0 | 2 | 2 | 0 | 0 | x | 0 | x | x |
| 6 | 92D- 1507 | F | 103 | 92 | 14.6 | 0.53 | 0 | 0 | | x | 0 | 4 | 1 | 0 | 0 | 0 | 0 | 1 | 1 |
| 6 | 92D- 1508 | F | 105 | 92 | 14.4 | 0.41 | 0 | 0 | | x | 0 | 3 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 6 | 92D- 1509 | F | 95 | 80 | 11.2 | 0.32 | 0 | 0 | | x | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 6 | 92D- 1510 | F | 88 | 78 | 9.25 | 0.26 | 0 | 0 | | x | 0 | 1 | 0 | 0 | 0 | x | 0 | 0 | 0 |
| 6 | 92D- 1511 | M | 90 | 76 | 9.9 | 0.37 | 0 | 0 | 0.13 | x | 0 | 1 | 0 | 0 | 0 | x | 0 | 1 | 0 |
| 6 | 92D- 1512 | M | 93 | 83 | 10.5 | 0.25 | 0 | 0 | 0.1 | x | 0 | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 6 | 92D- 1513 | M | 87 | 73 | 8.46 | 0.21 | 0 | 0 | 0.08 | x | 0 | 2 | 0 | 0 | 0 | x | 0 | x | x |
| 6 | 92D- 1514 | M | 96 | 80 | 11 | 0.32 | 0 | 0 | 0.12 | x | 0 | 1 | 0 | 0 | 0 | 0 | 0 | x | x |
| 6 | 92D- 1515 | M | 91 | 75 | 9.47 | 0.24 | 0 | 0 | 0.13 | x | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| 6 | 92D- 1516 | M | 84 | 71 | 8.94 | 0.33 | 0 | 0 | | x | 0 | 1 | 0 | 0 | 0 | 0 | 0 | x | x |
| 6 | 92D- 1517 | M | 92 | 79 | 10.3 | 0.77 | 0 | 0 | | x | 0 | 2 | 0 | 0 | 0 | x | 0 | 0 | 0 |
| 6 | 92D- 1518 | M | 78 | 68 | 6.05 | 0.12 | 0 | 0 | | x | 0 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 |
| 6 | 92D- 1519 | M | 88 | 74 | 8.6 | 0.22 | 0 | 0 | | x | 0 | 0 | 0 | 0 | 0 | 0 | 0 | x | x |
| 6 | 92D- 1520 | M | 88 | 74 | 8.06 | 0.17 | 0 | 0 | 0 | M | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 7 | 92D- 1660 | | 27 | 23 | 0.17 | | 0 | 0 | | M | 0 | 0 | 0 | 0 | 0 | x | x | x | x |
| 7 | 92D- 1661 | | 32 | 25 | 0.25 | | 0 | 0 | | F | 0 | 0 | 0 | 0 | 0 | x | 0 | x | x |
| 7 | 92D- 1662 | | 40 | 33 | 0.6 | | 0 | 0 | | M | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 7 | 92D- 1663 | | 47 | 40 | 1.05 | | 0 | 0 | | F | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 7 | 92D- 1664 | | 29 | 25 | 0.25 | | 0 | 0 | | x | 0 | 0 | 0 | 0 | 0 | x | x | x | x |
| 7 | 92D- 1665 | | 35 | 28 | 0.4 | | 0 | 0 | | F | 0 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 |
| 7 | 92D- 1666 | | 27 | 23 | 0.2 | | 0 | 0 | | F | 0 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 |
| 7 | 92D- 1667 | | 34 | 29 | 0.44 | | 0 | 0 | | F | 0 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 |
| 7 | 92D- 1668 | | 37 | 30 | 0.66 | | 0 | 0 | | F | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 7 | 92D- 1669 | | 30 | 26 | 0.33 | | 0 | 0 | | F | 0 | 0 | 0 | 0 | 0 | x | x | 0 | 0 |
| 7 | 92D- 1670 | | 33 | 28 | 0.39 | | 0 | 0 | | F | 0 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 |
| 7 | 92D- 1671 | | 33 | 28 | 0.39 | | 0 | 0 | | M | 0 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 |
| 7 | 92D- 1672 | | 40 | 32 | 0.68 | | 0 | 0 | | F | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 7 | 92D- 1673 | | 30 | 26 | 0.26 | | 0 | 0 | | F | 0 | 0 | 0 | 0 | 0 | x | x | x | x |
| 7 | 92D- 1674 | | 33 | 29 | 0.33 | | 0 | 0 | | M | 0 | 0 | 0 | 0 | 0 | x | x | x | x |
| 7 | 92D- 1675 | | 27 | 23 | 0.23 | | 0 | 0 | | F | 0 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 |
| 7 | 92D- 1676 | | 31 | 27 | 0.28 | | 0 | 0 | | M | 0 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 |

Table A2

Fundulus heteroclitus histology data - 1992

| Site | ID # | Intestine | Renal tubules | Sk muscle | Exocrine pancreas | Endocrine pancreas | Gonad | Spleen | Spleen MA's | Comments on histology |
|------|-----------|-----------|---------------|-----------|-------------------|--------------------|-------|--------|-------------|---|
| 6 | 92D- 1502 | 0 | 0 | 0 | 0 | x | x | x | x | |
| 6 | 92D- 1503 | 0 | 0 | 0 | 0 | 0 | x | x | x | |
| 6 | 92D- 1504 | 0 | 0 | 0 | 0 | x | x | x | x | Unicellular parasites in gill and gut |
| 6 | 92D- 1505 | 0 | 0 | 0 | 0 | x | x | x | x | |
| 6 | 92D- 1506 | 0 | 0 | x | 0 | x | x | x | x | |
| 6 | 92D- 1507 | 0 | 0 | 0 | 0 | x | x | x | x | |
| 6 | 92D- 1508 | 0 | 0 | 0 | 0 | x | x | x | x | |
| 6 | 92D- 1509 | 0 | 0 | 0 | 0 | x | x | x | x | |
| 6 | 92D- 1510 | 0 | 0 | 0 | 0 | x | x | x | x | |
| 6 | 92D- 1511 | 0 | x | 0 | 0 | x | x | x | x | |
| 6 | 92D- 1512 | 0 | 0 | 0 | 0 | x | x | x | x | |
| 6 | 92D- 1513 | 0 | 0 | 0 | 0 | x | x | x | x | |
| 6 | 92D- 1514 | 0 | 0 | 0 | 0 | x | x | x | x | |
| 6 | 92D- 1515 | 0 | 0 | 0 | 0 | x | x | x | x | |
| 6 | 92D- 1516 | 0 | 0 | 0 | 0 | x | x | x | x | |
| 6 | 92D- 1517 | 0 | 0 | 0 | 0 | 0 | x | x | x | |
| 6 | 92D- 1518 | 0 | 0 | 0 | 0 | x | x | x | x | |
| 6 | 92D- 1519 | 0 | 0 | 0 | 0 | x | x | x | x | |
| 6 | 92D- 1520 | 0 | 0 | 0 | 0 | x | x | x | x | |
| 7 | 92D- 1660 | 0 | 0 | 0 | 0 | x | 0 | x | x | Mild karyomegaly in liver |
| 7 | 92D- 1661 | 0 | 0 | 0 | 0 | x | 0 | x | x | |
| 7 | 92D- 1662 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 | |
| 7 | 92D- 1663 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 | |
| 7 | 92D- 1664 | 3 | 0 | 0 | 0 | x | x | x | x | Many pyknotic inflamm. cells in intestine |
| 7 | 92D- 1665 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| 7 | 92D- 1666 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 | |
| 7 | 92D- 1667 | 2 | 0 | 0 | 0 | x | 0 | 0 | 0 | Many pyknotic inflamm. cells in intestine |
| 7 | 92D- 1668 | 2 | 0 | 0 | 0 | x | 0 | 0 | 0 | midline section |
| 7 | 92D- 1669 | 1 | 0 | 0 | 0 | x | 0 | 0 | 0 | Some pyknotic inflamm. cells in intestine |
| 7 | 92D- 1670 | 1 | 0 | 0 | 0 | x | 0 | 0 | 0 | Some pyknotic inflamm. cells in intestine |
| 7 | 92D- 1671 | 2 | 0 | 0 | 0 | x | 0 | 0 | 0 | Some pyknotic inflamm. cells in intestine |
| 7 | 92D- 1672 | 2 | 0 | 0 | 0 | x | 0 | 0 | 0 | Some pyknotic inflamm. cells in intestine |
| 7 | 92D- 1673 | 2 | 0 | 0 | 0 | x | 0 | 0 | 0 | Some pyknotic inflamm. cells in intestine |
| 7 | 92D- 1674 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | Some pyknotic inflamm. cells in intestine |
| 7 | 92D- 1675 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | Some pyknotic inflamm. cells in intestine |
| 7 | 92D- 1676 | 2 | 0 | 0 | 0 | x | 0 | x | x | Some pyknotic inflamm. cells in intestine |

Table A2

Fundulus heteroclitus histology data - 1992

| Site | ID # | Sex | TL | SL | Wet Wt. | Liver Wt. | Fin (0-4) | Liver (0-2) | LN2 Liv. Wt. | Sex (histo) | Liver MA's | Liver coccidia | Liver necrosis | Clear cells | Megalocytosis | Cardiac nematodes | Cardiac muscle | Gill Trem. | Gill hyperpl |
|------|-----------|-----|----|----|---------|-----------|-----------|-------------|--------------|-------------|------------|----------------|----------------|-------------|---------------|-------------------|----------------|------------|--------------|
| 7 | 92D- 1677 | | 32 | 27 | 0.42 | | 0 | 0 | | F | 0 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 |
| 7 | 92D- 1678 | | 34 | 29 | 0.36 | | 0 | 0 | | M | 0 | 0 | 0 | 0 | 0 | 0 | x | x | 0 |
| 7 | 92D- 1679 | | 23 | 21 | 0.15 | | 0 | 0 | | M | 0 | 0 | 0 | 0 | 0 | x | x | x | x |
| 7 | 92D- 1680 | | 32 | 28 | 0.41 | 0.04 | 0 | 0 | | | | | | | | | | | |
| 7 | 92D- 1681 | | 30 | 28 | 0.26 | 0.01 | 0 | 0 | | | | | | | | | | | |
| 7 | 92D- 1682 | | 33 | 28 | 0.37 | 0.02 | 0 | 0 | | | | | | | | | | | |
| 7 | 92D- 1683 | | 30 | 27 | 0.3 | 0.02 | 0 | 0 | | | | | | | | | | | |
| 7 | 92D- 1684 | | 28 | 25 | 0.24 | 0.01 | 0 | 0 | | | | | | | | | | | |
| 8 | 92D 1685 | F | 73 | 63 | 4.11 | 0.21 | | | | F | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 8 | 92D 1686 | M | 60 | 50 | 2.61 | 0.13 | | | | x | 0 | 0 | 0 | 0 | 0 | x | x | 0 | 0 |
| 8 | 92D 1687 | F | 70 | 59 | 3.31 | 0.12 | | | | x | 0 | 0 | 0 | 0 | 0 | x | x | x | x |
| 8 | 92D 1688 | M | 60 | 50 | 2.02 | 0.08 | | | | M | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 8 | 92D 1689 | F | 60 | 49 | 2.09 | 0.09 | | | | M | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 8 | 92D 1690 | M | 57 | 48 | 2.11 | 0.11 | | | | F | 0 | 0 | 0 | 0 | 0 | x | x | x | x |
| 8 | 92D 1691 | M | 60 | 50 | 2.33 | 0.13 | | | | F | 0 | 0 | 0 | 0 | 0 | x | x | 0 | 0 |
| 8 | 92D 1692 | | 55 | 44 | 1.68 | | | | | M | 0 | 0 | 0 | 0 | 0 | x | x | x | x |
| 8 | 92D 1693 | | 65 | 53 | 2.67 | | | | | M | 0 | 0 | 0 | 0 | 0 | x | x | x | x |
| 8 | 92D 1694 | | | | 2.17 | | | | | M | 0 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 |
| 8 | 92D 1695 | | 60 | 52 | 2.04 | | | | | F | 0 | 0 | 0 | 0 | 0 | x | x | x | x |
| 8 | 92D 1696 | | 60 | 52 | 2.16 | | | | | M | 0 | 0 | 0 | 0 | 0 | x | 0 | 0 | 0 |
| 8 | 92D 1697 | | 50 | 42 | 1.43 | | | | | F | 0 | 0 | 0 | 0 | 0 | x | x | x | x |
| 8 | 92D 1698 | | 56 | 46 | 1.77 | | | | | F | 0 | 0 | 0 | 0 | 0 | x | 0 | 0 | x |
| 8 | 92D 1699 | | 46 | 40 | 1.24 | | | | | F | 0 | 0 | 0 | 0 | 0 | x | 0 | x | x |
| 8 | 92D 1700 | | 63 | 52 | 2.3 | | | | | F | 0 | 0 | 0 | 0 | 0 | x | 0 | x | x |
| 8 | 92D 1701 | | 42 | 36 | 0.9 | | | | | M | 0 | 0 | 0 | 0 | 0 | 0 | 0 | x | x |
| 8 | 92D 1702 | | 52 | 43 | 1.62 | | | | | M | 0 | 0 | 0 | 0 | 0 | x | x | x | x |
| 8 | 92D 1703 | | 50 | 40 | 1.3 | | | | | M | 0 | 0 | 0 | 0 | 0 | x | x | x | x |
| 8 | 92D 1704 | | 48 | 42 | 1.14 | | | | | F | 0 | 0 | 0 | 0 | 0 | x | 0 | x | x |

Table A3

Fish histology - 1993

| Sex | Species | ID # | Cardiac muscle | Gill Par. | Gill hyperpl | Intestine | Renal tubules | Sk muscle | Exocrine pancreas | Endocrine pancreas | Gonad | Spleen | Spleen MA's | Histo. comments |
|-----|---------|--------|----------------|-----------|--------------|-----------|---------------|-----------|-------------------|--------------------|-------|--------|-------------|---|
| 9 | F het | 93 210 | | 0 | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| 9 | F het | 93 211 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| 9 | F het | 93 212 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | G. ac. Rhomboid basophilic hepatocytes |
| 9 | G acu | 93 213 | | | | 0 | | 0 | | | 0 | | | |
| 9 | F het | 93 214 | | | | | | | | | | | | |
| 9 | F het | 93 215 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| 9 | F het | 93 216 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | | 0 | | | |
| 9 | F het | 93 217 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | | 0 | 0 | 0 | |
| 9 | F het | 93 218 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | | 0 | 0 | 0 | |
| 9 | F het | 93 219 | 0 | 1 | 1 | 0 | | 0 | 0 | 0 | 0 | 0 | 0 | |
| 9 | F het | 93 220 | | | | | | | | | | | | |
| 9 | F het | 93 221 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | | | |
| 9 | F het | 93 222 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | 0 | 0 | 0 | |
| 9 | F het | 93 223 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | 0 | 0 | 0 | |
| 9 | F het | 93 224 | | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | | | Clear cell focus in liver. Mucous cell hyperplasia. |
| 9 | F het | 93 225 | 0 | 0 | 0 | 0 | 3 | 0 | 0 | | 0 | 0 | 0 | Renal epithelial vacuolation |
| 9 | F het | 93 226 | 0 | 1 | 0 | 0 | | | 0 | | 0 | 0 | 0 | |
| 9 | G acu | 93 227 | | 0 | 0 | 0 | 0 | 0 | | | | | | Liver very glycogen rich |
| 9 | G acu | 93 228 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | 0 | | | Liver very basophilic. marked contrast to above male. |
| 9 | G acu | 93 229 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | 0 | 0 | 0 | |
| 8 | F het | 93 230 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | | 0 | | | |
| 8 | F het | 93 231 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| 8 | G acu | 93 232 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| 8 | G acu | 93 233 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| 7 | G acu | 93 234 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | | 0 | 0 | |
| 7 | G acu | 93 235 | | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | 0 | 0 | |
| 7 | G acu | 93 236 | | | | 0 | 0 | 0 | 0 | 0 | | | | |
| 7 | G acu | 93 237 | | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| 7 | G acu | 93 238 | | | | | | | | | | | | |
| 7 | G acu | 93 239 | | 0 | 0 | 0 | 0 | 0 | | | | 0 | 0 | |
| 7 | G acu | 93 240 | 0 | 0 | 0 | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | Erosion of gut epithelial villi tips. |
| 7 | G acu | 93 241 | | 0 | 0 | | | | | | | | | |
| 7 | G acu | 93 242 | | | | | | | | | | | | |
| 7 | G acu | 93 243 | | | | 0 | 0 | 0 | 0 | | 0 | 0 | 0 | |
| 7 | G acu | 93 244 | 0 | | | 0 | 0 | 0 | 0 | | 0 | 0 | 0 | |
| 6 | F het | 93 305 | | | 2 | 0 | | 0 | 0 | | 0 | 0 | 0 | |
| 6 | F het | 93 306 | | 0 | 2 | 0 | 0 | 0 | 0 | | 0 | 0 | 0 | |
| 6 | F het | 93 307 | 0 | | | 2 | 0 | 0 | 0 | | 0 | | | Inflamm. cells in gut epithel. |
| 6 | F het | 93 308 | 0 | 0 | 2 | 0 | | 0 | 0 | | 0 | 0 | 0 | |
| 6 | F het | 93 309 | 0 | 0 | 3 | 0 | 0 | 0 | 0 | | 0 | 0 | 0 | |
| 6 | F het | 93 310 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | | 0 | | | |
| 6 | F het | 93 311 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | | 0 | 0 | 0 | |
| 6 | F het | 93 312 | | 0 | 2 | 0 | 0 | 0 | | | 0 | | | |
| 6 | F het | 93 313 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | | | | | |
| 6 | F het | 93 314 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | | 0 | | | Intestinal protozoa |
| 6 | F het | 93 315 | | | | | | | | | | | | |
| 6 | F het | 93 316 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| 6 | F het | 93 317 | 0 | 0 | 0 | 0 | | | | | 0 | | | |
| 6 | F het | 93 318 | 0 | 0 | 2 | 0 | | 0 | 0 | 0 | 0 | | | Intestinal metazoan |
| 6 | F het | 93 319 | | | | | | | | | | | | |
| 6 | F het | 93 320 | 0 | | | | | | | | | | | |
| 6 | F het | 93 321 | 0 | 0 | 1 | 0 | 0 | 0 | | | | | | |
| 6 | F het | 93 322 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | | 0 | 0 | 0 | |

Table A3

Fish histology - 1993

| Sn | Species | ID # | Sex | TL | SL | Wet Wt | Liver Wt | Fin (0-4) | Liver (0-2) | LN2 Liv. Wt | Comments | Sex (histo) | Liver MA's | Liver coccidia | Liver necrosis | Liver neoplasm | Altered hepatic foci | Clear cells | Megalocytosis | Cardiac nematodes |
|----|---------|------|-----|----|-----|--------|----------|-----------|-------------|-------------|----------|--------------------|------------|----------------|----------------|----------------|----------------------|-------------|---------------|-------------------|
| 6 | Fhet | 93 | 323 | F | 77 | 67 | 4.56 | 0.2 | 0 | 0 | 0.12 | F | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 6 | Fhet | 93 | 324 | F | 68 | 56 | 3.71 | 0.06 | 0 | 0 | 0.01 | F | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 |
| 7 | Fhet | 93 | 430 | M | 53 | 44 | 1.44 | 0.09 | 0 | 0 | 0.05 | | | | | | | | | |
| 7 | Fhet | 93 | 431 | M | 78 | 63 | 5.04 | 0.14 | 0 | 0 | 0.08 | | | | | | | | | |
| 7 | Fhet | 93 | 432 | M | 54 | 45 | 1.69 | 0.08 | 0 | 0 | 0.03 | | | | | | | | | |
| 7 | Fhet | 93 | 433 | M | 85 | 72 | 7.25 | 0.26 | 0 | 2 | 0.03 | M | 2 | 0 | 2 | 4 | 4 | 2 | 1 | 0 |
| 7 | Fhet | 93 | 434 | M | 70 | 58 | 3.84 | 0.13 | 0 | 1 | 0.03 | | | | | | | | | |
| 7 | Fhet | 93 | 435 | F | 55 | 48 | 2.04 | 0.12 | 0 | 0 | 0.03 | | | | | | | | | |
| 7 | Fhet | 93 | 436 | F | 57 | 47 | 2.13 | 0.11 | 0 | 0 | 0.05 | | | | | | | | | |
| 7 | Fhet | 93 | 437 | F | 53 | 45 | 1.81 | 0.12 | 0 | 0 | 0.05 | | | | | | | | | |
| 7 | Fhet | 93 | 438 | | 48 | 40 | 1.98 | 0.13 | 0 | 0 | 0.1 | | | | | | | | | |
| 7 | Fhet | 93 | 439 | | | | | | 0 | 0 | | | | | | | | | | |
| 7 | Fhet | 93 | 440 | F | 66 | 58 | 4.02 | 0.26 | 0 | 0 | 0.05 | | | | | | | | | |
| 7 | Gac | 93 | 441 | F | 54 | 47 | 1.3 | 0.1 | 0 | 0 | | | | | | | | | | |
| 7 | Gac | 93 | 442 | F | | 50 | 1.5 | 0.1 | 0 | 0 | 0.03 | | | | | | | | | |
| 7 | Gac | 93 | 443 | | | | 1.57 | 0.07 | 0 | 0 | 0.03 | | | | | | | | | |
| 7 | yx. ac | 93 | 454 | | 60 | 48 | 2.35 | 0.04 | 0 | 0 | 0.02 | | | | | | | | | |
| 7 | Gac | 93 | 455 | | | | 1.26 | 0.08 | 0 | 0 | 0.05 | | | | | | | | | |
| 7 | Gac | 93 | 456 | F | 57 | 51 | 1.26 | 0.08 | 0 | 0 | | | | | | | | | | |
| 7 | Gac | 93 | 457 | F | 52 | 46 | 1.08 | 0.05 | | | 0.02 | | | | | | | | | |
| 7 | Gac | 93 | 458 | F | 53 | 48 | 1.25 | 0.08 | | | 0.05 | | | | | | | | | |
| 7 | Gac | 93 | 459 | F | 51 | 45 | 1.03 | 0.05 | | | 0.01 | | | | | | | | | |
| 7 | Gac | 93 | 460 | F | 55 | 49 | 1.12 | 0.06 | | | 0.03 | | | | | | | | | |
| 7 | Gac | 93 | 461 | F | 52 | 47 | 1.04 | 0.1 | | | 0.05 | | | | | | | | | |
| 7 | Gac | 93 | 462 | F | 50 | 45 | 0.84 | 0.05 | | | 0.01 | | | | | | | | | |
| 7 | Fhet | 93 | 480 | F | 95 | 85 | 14 | 0.55 | 0 | 0 | 0.15 | F | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 7 | Fhet | 93 | 481 | M | 93 | 80 | 9.34 | | 0 | 0 | | | | | | | | | | |
| 7 | Fhet | 93 | 482 | M | 78 | 68 | 5.64 | | 0 | 0 | | DEAD | M | 2 | 0 | 4 | 0 | 2 | 2 | 0 |
| 7 | Fhet | 93 | 483 | F | 80 | 68 | | | 0 | 0 | | DEAD | | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 7 | Fhet | 93 | 484 | F | 85 | 75 | | | 0 | 0 | | DEAD | | | | | | | | |
| 7 | Fhet | 93 | 485 | M | 80 | 68 | 5.3 | | 0 | 0 | | DEAD | | 0 | 0 | 1 | 0 | 2 | 0 | 0 |
| 7 | Fhet | 93 | 486 | F | 85 | 73 | 8.54 | | 0 | 0 | | DEAD | | 2 | 0 | 0 | 4 | 2 | 0 | 0 |
| 7 | Fhet | 93 | 487 | F | 80 | 60 | 6.28 | | 0 | 0 | | DEAD | | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 7 | Fhet | 93 | 488 | F | 100 | 86 | 14.33 | | 0 | 0 | | DEAD | | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 7 | Fhet | 93 | 489 | F | 90 | 80 | 10.34 | | 0 | 0 | | DEAD | | 0 | 0 | 0 | 0 | 2 | 0 | 0 |
| 7 | Fhet | 93 | 490 | F | 80 | 70 | 8.15 | | 0 | 0 | | DEAD | | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 7 | Fhet | 93 | 491 | M | 75 | 63 | 4.95 | | 0 | 0 | | DEAD | | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 7 | Fhet | 93 | 492 | M | 81 | 71 | 7.02 | | 0 | 2 | | DEAD - 1mm lesions | | 2 | 0 | 0 | 0 | 0 | 0 | 0 |
| 7 | Fhet | 93 | 493 | F | 70 | 60 | 5.24 | | 0 | 0 | | DEAD | | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 7 | Fhet | 93 | 494 | F | 67 | 57 | 4.87 | | 0 | 0 | | DEAD | | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 7 | Fhet | 93 | 495 | F | 71 | 60 | 5.07 | | 0 | 0 | | DEAD | | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 7 | Fhet | 93 | 496 | F | 65 | 55 | 3.6 | | 0 | 0 | | DEAD | | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 7 | Fhet | 93 | 497 | F | 65 | 55 | 4.11 | | 0 | 0 | | DEAD | | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 7 | Fhet | 93 | 498 | F | 65 | 55 | 3.86 | | 0 | 0 | | DEAD | | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 7 | Fhet | 93 | 499 | F | 80 | 70 | 7.56 | 0.54 | 0 | 0 | 0.17 | | | | | | | | | |
| 7 | Fhet | 93 | 500 | M | 78 | 68 | 5.27 | 0.13 | 0 | 1 | | | M | 2 | 0 | 0 | 0 | 2 | 0 | 0 |
| 7 | Fhet | 93 | 501 | M | 78 | 68 | 6.1 | | 0 | 2 | | 1-4mm fr | M | 2 | 0 | 0 | 4 | 4 | 0 | 0 |
| 7 | Fhet | 93 | 502 | M | 72 | 60 | 4.31 | | 0 | 1 | | 1mm foci | M | 1 | 0 | 0 | 0 | 1 | 0 | 0 |
| 7 | Fhet | 93 | 503 | F | 59 | 71 | 3.94 | 0.22 | 0 | 0 | 0.1 | | | | | | | | | |
| 7 | Fhet | 93 | 504 | M | 75 | 66 | 5.38 | 0.16 | 0 | 0 | 0.07 | | | | | | | | | |
| 7 | Fhet | 93 | 505 | F | 66 | 56 | 3.81 | 0.19 | 0 | 0 | 0.11 | F | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 7 | Fhet | 93 | 506 | F | 85 | 75 | 8.18 | 0.45 | 0 | 0 | 0.13 | F | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 7 | Fhet | 93 | 507 | M | 80 | 70 | 6.08 | | 0 | 1 | | M | 1 | 0 | 0 | 0 | 4 | 1 | 1 | |

Table A3

Fish histology - 1993

| Site | Species | ID # | Cardiac muscle | Gill Par. | Gill hyperpl | Intestine | Renal tubules | Sk muscle | Exocrine pancreas | Endocrine pancreas | Gonad | Spleen | Spleen MA's | Histo. comments |
|------|---------|------|----------------|-----------|--------------|-----------|---------------|-----------|-------------------|--------------------|-------|--------|-------------|--------------------|
| 7 | Fhet | 93 | 508 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | | | |
| 7 | Fhet | 93 | 509 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | | | | |
| 7 | Fhet | 93 | 510 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | |
| 7 | Fhet | 93 | 511 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | | |
| 7 | Fhet | 93 | 512 | 0 | 0 | 3 | 0 | | | | | | | |
| 7 | Fhet | 93 | 513 | 0 | 0 | 0 | 0 | 0 | 0 | | 0 | 0 | 1 | |
| 7 | Fhet | 93 | 514 | 0 | 0 | 0 | 0 | | 0 | | | 0 | 0 | |
| 7 | Fhet | 93 | 515 | | 0 | 2 | 0 | 0 | 0 | | | 0 | 0 | |
| 7 | Fhet | 93 | 516 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | |
| 7 | Fhet | 93 | 517 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| 7 | Fhet | 93 | 518 | 0 | 0 | 1 | 0 | 0 | 0 | | | | | ? Apoptotic bodies |
| 7 | Fhet | 93 | 519 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | |
| 7 | Fhet | 93 | 520 | 0 | 0 | 2 | 0 | 0 | 0 | | 1 | 0 | 1 | |
| 7 | Fhet | 93 | 521 | 0 | | | 0 | 0 | 0 | 0 | | 0 | 1 | |
| 7 | Fhet | 93 | 522 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | | |
| 7 | Fhet | 93 | 523 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| 7 | Fhet | 93 | 524 | | | | 0 | 0 | 0 | 0 | | 0 | 2 | |
| 7 | Fhet | 93 | 525 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | | | | |
| 7 | Fhet | 93 | 526 | | | | 0 | 0 | 0 | 0 | | | | |
| 7 | Fhet | 93 | 527 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | | | | |
| 7 | Fhet | 93 | 528 | 0 | 0 | 0 | 0 | 0 | 0 | | 0 | 0 | 0 | |

Table A4

Key for fish histology

Filename: Fhetkey

Abbreviations used in files named Fhetpath.92 and Fhetpath.93

| Column title | Unit | Explanation | |
|---------------------------|------------|--|-----|
| Station | | Number - see Table 1, Figure 1 and Copies of portions of nautical charts. | |
| Species | | Fhet = Fundulus heteroclitus, or mummichog, or killifish. Gac = Gasterosteus aculeatus, or 3-spined stickleback | |
| Year | | Year collected | |
| ID # | | WHOI Histological Archive accession number | |
| Sex | | M= Male, F=Female. Determined by visual inspection | |
| TL | mm | Total Length (Nose tip to tip of caudal fin) | |
| SL | mm | Standard Length (Nose tip to root of caudal fin rays) | |
| Wet Wt. | g. | Weight of whole animal | |
| Liver Wt. | g. | Weight of liver after removal from animal | |
| Fin | Index 0-4 | Degree of Fin erosion | |
| Liver | Index 0-2 | Visible liver abnormality. 0=Normal, 1= mild, 2= severe | |
| LN2 Liv. Wt. | g. | Weight of liver frozen for biochemistry, if any. | |
| Comments | | Remarks about dissection, if any | |
| Sex (histo) | | Gender as determined by gonad histology, if present | |
| Liver MA's | Index 0-4 | Liver Macrophage Aggregations | LV1 |
| Liver coccidia | Index 0-4 | Liver coccidian parasites | LV2 |
| Liver necrosis | Index 0-4 | Necrotic cell death | LV3 |
| Liver neoplasm | Index 0-4 | Lesion(s) meeting standard criteria for neoplasia | LV4 |
| Tinctorially altered foci | Index 0-4 | Foci of cells of altered staining - usually regarded as preneoplastic | LV5 |
| Clear cells | Index 0-4 | Isolated nests of clear cells, smaller than the above tinctorially altered foci | |
| Megalocytosis | Index 0-4 | Oversized hepatocytes | LV6 |
| Cardiac nematodes | Index 0-4 | Nematodes present in heart chambers | |
| Cardiac muscle | Index 0-4 | Abnormality in cardiac muscle. | |
| Gill Par. | Index 0-4 | Parasites in the gill | GL1 |
| Gill hyperpl | Index 0-4 | Hyperplasia of gill epithelia | GL2 |
| Intestine | Index 0-4 | Abnormality in the intestine | |
| Renal tubules | Index 0-4 | Abnormality in kidney tubules | |
| Sk muscle | Index 0-4 | Abnormality in skeletal muscle | |
| Exocrine pancreas | Index 0-4 | Abnormality in exocrine pancreas | |
| Endocrine pancreas | Index 0-4 | Abnormality in endocrine pancreas | |
| Gonad | Index 0-4 | Abnormality in gonad | |
| Spleen | Index 0-4 | Abnormality in spleen | |
| Spleen MA's | Index 0-4 | Spleen macrophage aggregations | SP1 |
| Histo. comments | | Comments concerning histology | |
| | Index 0-4: | 0=absent, 1=mild, 2=moderate, 3=severe, 4=extreme | |

Missing data points reflect absence of that tissue from available histological slide,
or in the case of dissection data, the fish was too small to dissect.

Table A5

Fundulus P4501A and EROD data

| ID | STN | 1A normalized %/mg | Chemilum. P4501A /mg | Colorim. P4501A/mg | EROD pmol/min/mg |
|------|-----|-----------------------|-------------------------|-----------------------|---------------------|
| 1224 | 1 | | | | 361.59 |
| 1222 | 1 | 48.049 | 10.27 | 1.170 | 353.33 |
| 1223 | 1 | 34.735 | 8.61 | 0.693 | 231.87 |
| 1221 | 1 | 60.275 | 11.93 | 1.590 | 640.48 |
| 1220 | 1 | 33.920 | 6.28 | 0.951 | 348.46 |
| 1243 | 2 | 38.19 | 7.70 | | 419.63 |
| 1240 | 2 | 26.16 | 5.27 | | 289.49 |
| 1241 | 2 | 20.31 | 4.09 | | 247.82 |
| 1264 | 3 | 82.487 | 13.10 | 2.590 | 763.06 |
| 1263 | 3 | 62.537 | 11.38 | 1.778 | 891.35 |
| 1262 | 3 | 51.246 | 9.65 | 1.415 | 412.13 |
| 1261 | 3 | 39.534 | 7.99 | 1.021 | 408.07 |
| 1260 | 3 | 66.237 | 14.25 | 1.600 | 717.16 |
| 1300 | 4 | 97.232 | 20.16 | 2.446 | 709.19 |
| 1302 | 4 | 38.397 | 4.97 | 1.35 | 255.75 |
| 1312 | 5 | 25.11 | 5.06 | | 304.99 |
| 1310 | 5 | 28.88 | 5.82 | | 332.62 |
| 1311 | 5 | 13.97 | 2.82 | | 154.57 |
| 1314 | 5 | 29.48 | 5.94 | | 417.72 |
| 307 | 6 | 19.653 | | 0.51 | 97.23 |
| 305 | 6 | 21.390 | | 0.55 | 203.17 |
| 317 | 6 | 0.775 | 0.31 | 0.00 | 94.15 |
| 315 | 6 | 3.398 | | 0.09 | 151.48 |
| 314 | 6 | 0.000 | | 0.00 | 12.4 |
| 311 | 6 | | | | 203.37 |
| 316 | 6 | 2.542 | 0.48 | 0.070 | |
| 320 | 6 | 5.685 | 1.67 | 0.080 | |
| 1512 | 6 | 11.839 | 2.64 | 0.274 | |
| 1513 | 6 | 23.273 | 6.05 | 0.428 | |
| 1515 | 6 | 5.685 | | 0.147 | 289.21 |
| 1514 | 6 | 28.009 | | 0.725 | 418.72 |
| 1511 | 6 | 17.435 | | 0.452 | 339.67 |
| 1501 | 6 | 23.387 | 4.86 | 0.587 | 992.65 |
| 1502 | 6 | 10.571 | 2.07 | 0.281 | 918.21 |
| 1505 | 6 | 11.851 | | 0.307 | 363.02 |
| 1504 | 6 | 5.478 | | 0.142 | 282.76 |
| 1503 | 6 | 19.988 | | 0.518 | 194.72 |
| 313 | 6 | 4.942 | 1.00 | 0.127 | 447.46 |
| 430 | 7 | 68.033 | 12.98 | 1.856 | 331.64 |
| 433 | 7 | 66.208 | 12.44 | 1.831 | 343.65 |
| 511 | 7 | 63.308 | 11.20 | 1.840 | 448.77 |
| 480 | 7 | 1.451 | 0.04 | 0.070 | 128.96 |
| 499 | 7 | 37.523 | 5.17 | 1.280 | 365.82 |
| 506 | 7 | 2.989 | 0.14 | 0.137 | 64.67 |
| 1680 | 7 | | | | 753.17 |
| 1688 | 8 | 42.32 | 8.53 | | 312.46 |
| 1685 | 8 | 30.18 | 6.08 | | 244.22 |
| 1686 | 8 | 35.64 | 7.18 | | 379 |
| 1687 | 8 | 31.71 | 6.39 | | 346.61 |

Table A7

1992, Mya, Station 2

| Cell/tissue | Animal number, 92-1124 | | 1125 | | 1126 | | 1127 | | 1128 | | 1129 | | 1130 | | 1131 | | 1132 | | 1133 | | 1134 | | 1135 | | 1136 | | 1137 | | 1138 | | 1139 | | 1140 | | 1141 | | 1142 | | 1143 | | | | | | | | | | | | | | | | | | | | | |
|------------------|------------------------|---|------|---|------|---|------|---|------|---|------|---|------|---|------|---|------|---|------|---|------|---|------|---|------|---|------|---|------|---|------|---|------|---|------|---|------|---|------|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | f | f | m | f | f | f | m | f | f | f | f | m | f | f | f | f | f | f | f | f | f | m | m | m | m | m | m | m | m | m | m | m | m | m | m | m | m | m | f | f | | | | | | | | | | | | | | | | | | | | |
| leukemia index | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | | | | | | | | | | | | | | | | | |
| gonad | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| male/female | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| stage | 2 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | | | | | | | | | | | | |
| inflammation | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | | | | | | | | | | |
| parasites | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | | | | | | | | |
| neoplasia | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | | | | | | | |
| gill | 3 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | | | | | | | | |
| parasites* | 3 | 1 | 0 | 3 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | | | | | |
| inflammation | 3 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | | | | | | |
| mucus cells # | 1 | 1 | 2 | 4 | 0 | 2 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | | | | | | |
| hyperplasia | 3 | 4 | 5 | 5 | 3 | 3 | 2 | 4 | 2 | 3 | 3 | 3 | 2 | 2 | 4 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | | | | | | | |
| kidney | 3 | 4 | 4 | 4 | 3 | 2 | 2 | 3 | 2 | 2 | 2 | 2 | 2 | 2 | 3 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | | | | | | | |
| brown cell # | 0 | 3 | 0 | 1 | 3 | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | | | | |
| parasites* | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | | | |
| gr./brown debrec | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | | | | |
| hyperplasia | 3 | 4 | 5 | 5 | 3 | 3 | 2 | 4 | 2 | 3 | 3 | 3 | 2 | 2 | 4 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | | | | | |
| heart | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | | |
| sac hyperplasia | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | | |
| inflammation | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | | |
| other abnorm* | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | | |
| neural tissue | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| foot | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| inflammation | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| other | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| style sac | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| intestine | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| epithelium | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| parasite* | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| inflammation | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| necrosis* | 0 | 0 | 0 | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |

Table A9
Cells/tissue

1992, Mya, Station 4

| Cells/tissue | Animal number, 92- | | 1175 | | 1176 | | 1177 | | 1178 | | 1179 | | 1180 | | 1181 | | 1182 | | 1183 | | 1184 | | 1185 | | 1186 | | 1187 | | 1188 | | 1189 | | 1190 | | 1191 | | |
|-------------------|--------------------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---|--|
| | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | | |
| leukemia index | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| gonad | f | f | f | m | f | f | m | m | m | m | f | f | m | m | f | f | m | m | m | m | m | m | m | f | f | f | f | f | m | m | m | f | f | m | m | | |
| male/female | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| stage | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | act gam | | |
| inflammation | 2 | 1 | 1 | 0 | 0 | 4 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 4 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| parasites | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| neoplasia | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| gill | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| parasites* | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| inflammation | 2 | 0 | 1 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | | |
| mucus cells # | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | | |
| hyperplasia | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| kidney | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| brown cell # | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | | |
| parasites | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| gr./brown debris | 1 | 2 | 2 | 0 | 1 | 1 | 1 | 1 | 0 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | | |
| hyperplasia | 3 | 4 | 4 | 3 | 3.5 | 2 | 4 | 2 | 4 | 2 | 2 | 2 | 2 | 2 | 3.5 | 3 | 2 | 3 | 2 | 3 | 2 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | | |
| heart | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| sac hyperplasia | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| inflammation | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| colon | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| mucile | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| inflammation | 2 | 0 | 0 | 0 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | | |
| other abnorm | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| neural tissue | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| foot | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| inflammation | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| other | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| style sac | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| intestine | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| epithelium | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| parasite | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| inflammation | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| necrosis | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| hyperplasia | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| brown cell # | 3 | 3 | 2 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | |
| lumen | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| parasite | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| other abnorm | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| digestive gland | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| absorptive cells | 5 | 5 | 4 | 5 | 4 | 5 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 5 | 4 | 5 | 4 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | | |
| vacuolation | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| height | 5 | 5 | 4 | 5 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 5 | 4 | 5 | 4 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | | |
| reserve cells | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| occurrence | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 5 | 5 | 5 | 4 | 4 | 4 | 4 | 4 | 5 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | | |
| parasites | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| brown cells in ct | 3 | 2 | 2 | 3 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 1 | 1 | 2 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | | |
| mucile edge | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| inflammation | 0 | 0 | 0 | 0 | 2 | nt | | |
| mucus cell # | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| green gland | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| abnorm | 3 | 3 | 3 | 3 | 4 | 3 | 4 | 3 | 3 | 2 | nt | 2 | nt | 2 | 2 | 3 | 4 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | | |
| adductor muscle | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| abnormality* | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| siphon | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| inflammation* | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | |
| hyperplasia | 0 | 0 | 0 | 0 | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |

Table A13

1993, Mya Pathology, Station 8

| Organ/cells examined | 93-190 | 93-191 | 93-192 | 93-193 | 93-194 | 93-195 | 93-196 | 93-197 | 93-198 | 93-199 | 93-200 | 93-201 | 93-202 | 93-203 | 93-204 | 93-205 | 93-206 | 93-207 | 93-208 | 93-209 | |
|-----------------------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--|
| leukemia | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | |
| gonad | | | | | | | | | | | | | | | | | | | | | |
| inflammation | 3 | 0 | 0 | 0 | 0 | 1 | 2 | 0 | 2 | 3 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 3 | |
| parasite | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| neoplasia | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| gill | | | | | | | | | | | | | | | | | | | | | |
| parasite | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 2 | 0 | 0 | 0 | 0 | 2 | |
| inflammation | 1 | 3 | 1 | 1 | 1 | 2 | 2 | 1 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | 3 | 2 | 2 | 3 | |
| hyperplasia/papilloma | 2 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 2 | 2 | 1 | 0 | 0 | 0 | 2 | 2 | 0 | 0 | 0 | 1 | |
| kidney | | | | | | | | | | | | | | | | | | | | | |
| brown cells | 4 | 2 | 2 | 2 | 2 | 3 | 2 | 2 | 3 | 4 | 2 | 2 | 2 | 2 | 1 | 2 | 3 | 2 | nt | 4 | |
| parasite | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | nt | 0 | |
| gr/br debris | 1 | 0 | 0 | 1 | 0 | 1 | 1 | 1 | 1 | 0 | 1 | 2 | 1 | 1 | 1 | 1 | 3 | 2 | nt | 1 | |
| hyperplasia | 2 | 3 | 2 | 3 | 3 | 3 | 3 | 2 | 3 | 3 | 2 | 2 | 3 | 3 | 3 | 3 | 2 | 3 | nt | 2 | |
| mantle | | | | | | | | | | | | | | | | | | | | | |
| inflammation | 1,4 | 1 | 1 | 1 | 1 | nt | 0 | 2 | 1 | 1 | 3 | 0 | 2 | 1 | 1 | 2 | 1 | 1 | 1 | 2 | |
| digestive gland | | | | | | | | | | | | | | | | | | | | | |
| brown cells in ct | 3 | 2 | 1 | 1 | 2 | 1 | 1 | 1 | 1 | 3 | 1 | 1 | 2 | 1 | 1 | 2 | 2 | 1 | 1 | nt | |
| green gland | | | | | | | | | | | | | | | | | | | | | |
| abnorm. | 3 | 2 | 2 | 2 | 3 | 2 | 2 | 2 | 2 | 3 | 2 | 2 | 3 | 3 | 2 | 3 | 2 | 3 | 2 | 3 | |

Table A14

1993, Mya Pathology, Station 9

| Organs/cells examined | 93-170 | 93-171 | 93-172 | 93-173 | 93-174 | 93-175 | 93-176 | 93-177 | 93-178 | 93-179 | 93-180 | 93-181 | 93-182 | 93-183 | 93-184 | 93-185 | 93-186 | 93-187 | 93-188 | 93-189 |
|-----------------------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|
| leukemia | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| gonad | | | | | | | | | | | | | | | | | | | | |
| inflammation | 2 | 0 | 0 | 0 | 3 | 3 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 3 | 0 |
| parasite | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| neoplasia | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| gill | | | | | | | | | | | | | | | | | | | | |
| parasite | 1 | 0 | 0 | 1 | 1 | 1 | 1 | 1 | 0 | 0 | 1 | 0 | 1 | 0 | 1 | 1 | 0 | 0 | 1 | 0 |
| inflammation | 2 | 1 | 2 | 3 | 1 | 2 | 2 | 2 | 1 | 1 | 1 | 2 | 1 | 1 | 2 | 2 | 2 | 1 | 1 | 1 |
| hyperplasia/papilloma | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| kidney | | | | | | | | | | | | | | | | | | | | |
| brown cells | nt | 3 | nt | nt | 2 | nt | nt | 3 | 1 | 4 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | nt | 3 | 2 |
| parasite | nt | 0 | nt | nt | 0 | nt | nt | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | nt | 0 | 0 |
| gr/br debris | nt | 0 | nt | nt | 1 | nt | nt | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 2 | 0 | 1 | nt | 1 | 1 |
| hyperplasia | nt | 4 | nt | nt | 2 | nt | nt | 3 | 2 | 5 | 2 | 3 | 3 | 2 | 5 | 3 | 4 | nt | 4 | 4 |
| manile | | | | | | | | | | | | | | | | | | | | |
| inflammation | 1 | 1 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 2 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| digestive gland | | | | | | | | | | | | | | | | | | | | |
| brown cells in ct | 2 | 2 | 1 | 1 | 2 | 1 | 1 | 1 | 1 | 3 | 1 | 1 | 1 | 1 | 2 | 2 | 2 | 1 | 2 | 1 |
| green gland | | | | | | | | | | | | | | | | | | | | |
| abnorm. | 3 | 3 | 3 | 3 | 2 | 3 | 3 | 2 | 3 | 4 | 3 | 2 | 3 | 3 | 3 | 3 | 3 | 2 | 2 | 3 |

Table A15

Filename: Bivalve.key

RATING KEY

nt = tissue not present in any section examined from this animal.

0 = not present in sections

1 = rarely present in sections

2 = mildly present in sections

3 = moderately present in sections

4 = strongly present in sections

5 = very strongly present in sections

TISSUE KEY FOR MYA

Gonad

inflammation - Inflammation consisting of brown cells and low but variable numbers of hemocytes are present within tubular lumens predominately. Occasionally, inflammation is also present in peritubular sinuoids.

parasite- Parasites in all cases consisted of encysted metacercaria in the connective tissue of sinusoids surrounding the tubules.

Gill

inflammation- Inflammation consisted of brown cells in the sinusoids unless noted otherwise.

hyperplasia- Hyperplasia consisted of foci of variable number and size characterized by proliferation of epithelium and often underlying connective tissue of gill plica (larger areas are equivalent to papillomas as published in the literature). Ratings for hyperplasia are not based on the occurrence of possible branchial tumors.

Kidney

brown cell #- Refers to the number of brown cells (and usually low but variable numbers of hemocytes in the kidney epithelium)

green/brown debree - Foci of green/brown crystal-like debree is present predominately in the lumens of kidney tubules and is also present occasionally in adjacent cells.

parasite- Three types of possible parasites were seen in tissue sections in this study. Type 1 parasites are characterized by foci of tufts of syncytial-like cells usually associated with a large (up to 50 micron diameter) round egg-like mass nested on the luminal side of the hyperplastic cell tuft, identity not known. Type 2 parasites are oblong holotrich ciliates with blunted ends (approximately 25 x 10 microns). Type 3 organisms (approximately 10 microns in diameter) are characterized by a translucent corrugated capsule containing a 5 micron cell body with a single nucleus (diatom) (They are included with the parasite category for convenience. Their role in any pathology is questionable.)

Table A15

Mantle

inflammation- Inflammation consist of brown cells in the sinusoids unless state otherwise.

Style Sac

All ratings refer to the occurrence of encysted metacercaria in the connective tissue of sinusoids surrounding the sac unless otherwise noted.

Intestinal epithelium

parasite - Refers to the occurrence of encysted metacercaria in connective tissue surrounding the intestine unless otherwise noted.

hyperplasia - Refers to increase in epithelial cell number (subjective evaluation).

brown cells - Refers to the number of brown cells present within the epithelium.

Digestive Gland

parasites - Refers to the occurrence of Chlamydial-like organisms in the epithelial cells.

Brown cells in ct- Refers to the number of brown cells present in the surrounding connective tissues of the sinusoids.

Green gland

abnormalities- The number refers to a subjective evaluation of the green gland which takes into account the number of cells lining the tubules, granularity of those cells and amount of sluffed cells in lumens.

Siphon

parasite - Refers to the occurrence of encysted metacercaria in the connective tissue.

TISSUE KEY FOR MYTILUS

Gonad - Trematodal infections were noted in the gonad tubules of many animals. Parasitic castration and eventual rupture of tubules associated with localized inflammation were occasional seen as noted in the results.

Table A 16

92 *Mya* STATION 1, * INFORMATION

Gill, parasites - Unidentified ciliates were present in water tubules of the gills of all animals rated.

Kidney, parasites-
#s1101 and 1112 - contained type 1 parasites
#1109 - contained type 2 parasites

Siphon, hyperplasia- Focal epithelial hyperplasia was associated with the area of inflammation (#1108-2).

92 *Mya* STATION 2, * INFORMATION

Gill, parasites- Predominately unidentified ciliates in #s 1124, 1125, 1128, 1129, 1130, 1131, 1134, 1135, 1138, 1139, 1141, 1142. The following animals also show encysted metacercaria (see key): #s 1125, 1134, 1135, 1140.

Kidney, parasites - Type 1: #s 1128, 1129, 1133, 1134, 1135. Type 2: #s 1125, 1127, 1138, 1139.

Mantle, other abnormality- Both animals show hyperplastic mantle cavity epithelium.

Intestinal epithelium, parasite - #1134 focally shows remnants of a large multicellular parasite present within the tubular lumen.

Intestinal epithelium, necrosis- associated with parasitic focus in #1134

Intestinal epithelium, hyperplasia- associated with parasitic focus in #1134

Siphon, inflammation - (#1139) Focally moderate subepithelial inflammation.

Siphon, hyperplasia - (#1139) Epithelial hyperplasia associated with inflammation.

Siphon, parasite - (#1139) Possible protistan parasite associated with inflammation.

92 *Mya* STATION 3, * INFORMATION

Gonad, neoplasia - Sheets of monomorphic large (15 micron) cells with large oval to irregular nuclei and moderate amounts of basophilic cytoplasm are present in sinusoids surrounding some tubules. Mitotic figures are noted. No origin from gonad tissue is identified (#s 1153, 1157, 1160, 1165, 1168).

Gill, parasites - Ciliates seen in water tubules lumens of all animals rated. Animal #1165 also contains dinoflagellates in the water tubular lumens.

Gill, hyperplasia - Multifocally within gill sinusoids and rarely associated with hyperplastic epithelium are sheets of tumor cells as described for the gonad. (Animal #s 1153, 1157, 1160, 1168).

Kidney, parasites - Type 2 parasites seen in #s 1151, 1152, 1153. Type 3 organisms seen in #s 1164, 1165, 1169.

Heart, sac hyperplasia - neoplastic cells present in the lumen of the heart in #1168.

Table A 16 (continued)

Mantle, other - neoplastic cells present in the sinusoids of rated animals.

Neural tissue - Ganglions adjacent to renal tissue in each rated animal (#s 1150 and 1151) shows inflammation and brown cell accumulation within the ganglion. In one animal, central chromatolysis (necrosis) of a neuron is noted (#1151).

Foot, other - foci of sheets of neoplastic cells are present in sinusoids (#1153).

92 *Mya* STATION 4, * INFORMATION

Gill, parasites - Ciliates present in water tubules of all rated animals.

Adductor muscle, abnormality - Focal brown cell accumulation, possibly associated with intracellular bacteria. May have resulted from needle puncture during bleeding (#1180).

Siphon, inflammation - Focal subepithelial inflammation. No cause seen (#1184).

92 *Mya* STATION 5, * INFORMATION

Gill, inflammation- In #1197, a focally extensive growth was characterized by trabeculae of numerous eosinophilic granular cells separated by sinusoidal spaces containing numerous hemocytes. The growth appears to originate from the plica of a gill but does not appear to be invasive. No mitotic figures were present. Possible infection/parasite.

Gill, parasites- Encysted metacercaria were present in sinusoidal connective tissue of all identified animals. In #1199, unidentified ciliates were also rarely seen in water tubules.

Heart, inflammation- In #1209, two cells containing myxosporidal-like organisms were seen.

Foot, inflammation- In #1212, there is a focal area of epithelial ulceration associated with underlying inflammation.

Foot, other- In #1213, encysted metacercaria were present in the foot tissues.

92 *Mya* STATION 8, * INFORMATION

Gill, parasite - Ciliates in water tubules are present in #1711. Possible dinoflagellate-like organism present in water tubule of #1712. Encysted metacercaria present in #1713.

Kidney, parasites - In #1711, dinoflagellates are present in the kidney lumens. Type 1 organisms are present in #s 1117 (one focus) and 1726. A type 3 organism is present in #1117. A type 2 organism is present in #1728.

Mantle, other abnormality - Encysted metacercaria are present (#1711).

Intestine, lumen, parasite - Possible nematode in present in the lumen (#1721).

Table A17

Mya Hematology Data

| ID | Cells/field | # of fields | Total cells | # of hn cells | %Hn | |
|----|-------------|-------------|-------------|---------------|-----|--------|
| 92 | 1100 | | | | 0 | |
| 92 | 1101 | | | | 0 | |
| 92 | 1102 | | | | 0 | |
| 92 | 1103 | | | | 0 | |
| 92 | 1104 | | | | 0 | |
| 92 | 1105 | | | | 0 | |
| 92 | 1106 | | | | 0 | |
| 92 | 1107 | | | | 0 | |
| 92 | 1108 | | | | 0 | |
| 92 | 1109 | | | | 0 | |
| 92 | 1110 | | | | 0 | |
| 92 | 1111 | | | | 0 | |
| 92 | 1112 | | | | 0 | |
| 92 | 1113 | | | | 0 | |
| 92 | 1114 | | | | 0 | |
| 92 | 1115 | | | | 0 | |
| 92 | 1116 | | | | 0 | |
| 92 | 1117 | | | | 0 | |
| 92 | 1118 | | | | 0 | |
| 92 | 1119 | | | | 0 | |
| 92 | 1120 | | | | 0 | |
| 92 | 1121 | | | | 0 | |
| 92 | 1122 | | | | 0 | |
| 92 | 1123 | | | | 0 | |
| 92 | 1124 | | | | 0 | |
| 92 | 1125 | | | | 0 | |
| 92 | 1126 | | | | 0 | |
| 92 | 1127 | | | | 0 | |
| 92 | 1128 | | | | 0 | |
| 92 | 1129 | | | | 0 | |
| 92 | 1130 | | | | 0 | |
| 92 | 1131 | | | | 0 | |
| 92 | 1132 | | | | 0 | |
| 92 | 1133 | | | | 0 | |
| 92 | 1134 | | | | 0 | |
| 92 | 1135 | | | | 0 | |
| 92 | 1136 | | | | 0 | |
| 92 | 1137 | | | | 0 | |
| 92 | 1138 | 212 | 22 | 4664 | 1 | 0.0214 |
| 92 | 1139 | | | | 0 | |
| 92 | 1140 | | | | 0 | |
| 92 | 1141 | | | | 0 | |
| 92 | 1142 | | | | 0 | |
| 92 | 1143 | | | | 0 | |
| 92 | 1144 | | | | 0 | |
| 92 | 1145 | | | | 0 | |
| 92 | 1146 | | | | 0 | |
| 92 | 1147 | | | | 0 | |
| 92 | 1148 | | | | 0 | |
| 92 | 1149 | | | | 0 | |
| 92 | 1150 | | | | 0 | |
| 92 | 1151 | | | | 0 | |
| 92 | 1152 | | | | 0 | |
| 92 | 1153 | | | | 0 | |

Table A17

Mya Hematology Data

| ID | Cells/field | # of fields | Total cells | # of hn cells | %Hn | |
|----|-------------|-------------|-------------|---------------|-----|---------|
| 92 | 1154 | | | | 0 | |
| 92 | 1155 | | | | 0 | |
| 92 | 1156 | | | | 0 | |
| 92 | 1157 | | | | 0 | |
| 92 | 1158 | | | | 0 | |
| 92 | 1159 | | | | 0 | |
| 92 | 1160 | | | | 0 | |
| 92 | 1161 | | | | 0 | |
| 92 | 1162 | | | | 0 | |
| 92 | 1163 | | | | 0 | |
| 92 | 1164 | | | | 0 | |
| 92 | 1165 | | | | 0 | |
| 92 | 1166 | | | | 0 | |
| 92 | 1167 | | | | 0 | |
| 92 | 1168 | | | | 0 | |
| 92 | 1169 | | | | 0 | |
| 92 | 1170 | | | | 0 | |
| 92 | 1171 | | | | 0 | |
| 92 | 1172 | | | | 0 | |
| 92 | 1173 | | | | 0 | |
| 92 | 1174 | | | | 0 | |
| 92 | 1175 | | | | 0 | |
| 92 | 1176 | | | | 0 | |
| 92 | 1177 | 600 | 20 | 12000 | 70 | 0.5833 |
| 92 | 1178 | 200 | 41 | 8200 | 25 | 0.3409 |
| 92 | 1179 | 425 | 1 | 725 | 719 | 99.1724 |
| 92 | 1180 | | | | 0 | |
| 92 | 1181 | | | | 0 | |
| 92 | 1182 | | | | 0 | |
| 92 | 1183 | 204 | 2 | 408 | 130 | 31.8627 |
| 92 | 1184 | | | | 0 | |
| 92 | 1185 | | | | 0 | |
| 92 | 1186 | | | | 0 | |
| 92 | 1187 | | | | 0 | |
| 92 | 1188 | | | | 0 | |
| 92 | 1189 | | | | 0 | |
| 92 | 1190 | | | | 0 | |
| 92 | 1191 | | | | 0 | |
| 92 | 1192 | | | | 0 | |
| 92 | 1193 | | | | 0 | |
| 92 | 1194 | | | | 0 | |
| 92 | 1195 | | | | 0 | |
| 92 | 1196 | | | | 0 | |
| 92 | 1197 | | | | 0 | |
| 92 | 1198 | | | | 0 | |
| 92 | 1199 | | | | 0 | |
| 92 | 1200 | | | | 0 | |
| 92 | 1201 | | | | 0 | |
| 92 | 1202 | | | | 0 | |
| 92 | 1203 | | | | 0 | |
| 92 | 1204 | | | | 0 | |
| 92 | 1205 | | | | 0 | |
| 92 | 1206 | | | | 0 | |
| 92 | 1207 | | | | 0 | |

Table A17

Mya Hematology Data

| ID | Cells/field | # of fields | Total cells | # of hn cells | %Hn | |
|----|-------------|-------------|-------------|---------------|-----|--------|
| 92 | 1208 | | | | 0 | |
| 92 | 1209 | | | | 0 | |
| 92 | 1210 | | | | 0 | |
| 92 | 1211 | | | | 0 | |
| 92 | 1212 | | | | 0 | |
| 92 | 1213 | | | | 0 | |
| 92 | 1214 | | | | 0 | |
| 92 | 1215 | | | | 0 | |
| 92 | 1216 | | | | 0 | |
| 92 | 1217 | | | | 0 | |
| 92 | 1218 | | | | 0 | |
| 92 | 1219 | | | | 0 | |
| 92 | 1220 | | | | 0 | |
| 92 | 1221 | | | | 0 | |
| 92 | 1222 | | | | 0 | |
| 92 | 1223 | | | | 0 | |
| 92 | 1224 | | | | 0 | |
| 92 | 1225 | | | | 0 | |
| 92 | 1226 | | | | 0 | |
| 92 | 1227 | | | | 0 | |
| 92 | 1228 | | | | 0 | |
| 92 | 1229 | | | | 0 | |
| 92 | 1230 | 480 | 61 | 29280 | 7 | 0.0239 |
| 92 | 1231 | | | 100000 | 8 | 0.008 |
| 92 | 1232 | | | | 0 | |
| 92 | 1233 | | | | 0 | |
| 92 | 1234 | | | | 0 | |
| 92 | 1235 | | | | 0 | |
| 92 | 1236 | | | | 0 | |
| 92 | 1237 | | | | 0 | |
| 92 | 1238 | | | | 0 | |
| 92 | 1710 | | | | 0 | |
| 92 | 1711 | | | | 0 | |
| 92 | 1712 | | | | 0 | |
| 92 | 1713 | | | | 0 | |
| 92 | 1714 | | | | 0 | |
| 92 | 1715 | | | | 0 | |
| 92 | 1716 | | | | 0 | |
| 92 | 1717 | | | | 0 | |
| 92 | 1718 | | | | 0 | |
| 92 | 1719 | | | | 0 | |
| 92 | 1720 | | | | 0 | |
| 92 | 1721 | | | | 0 | |
| 92 | 1722 | 368 | 16 | 5888 | 103 | 1.7493 |
| 92 | 1723 | 456 | 40 | 18240 | 7 | 0.0384 |
| 92 | 1724 | | | | 0 | |
| 92 | 1725 | | | | 0 | |
| 92 | 1726 | | | | 0 | |
| 92 | 1727 | | | | 0 | |
| 92 | 1728 | | | | 0 | |
| 92 | 1729 | | | | 0 | |

Table A.18

1993, Mytilus Pathology, Station 7

| Organs/cells examined | 93-285 | 93-286 | 93-287 | 93-288 | 93-289 | 93-290 | 93-291 | 93-292 | 93-293 | 93-294 | 93-295 | 93-296 | 93-297 | 93-298 | 93-299 | 93-300 | 93-301 | 93-302 | 93-303 | 93-304 |
|-----------------------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|
| leukemia | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| gonad | | | | | | | | | | | | | | | | | | | | |
| inflammation | 0 | 2 | 0 | 0 | 0 | 2 | 0 | 0 | 4 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| parasite | 0 | 0 | 0 | 0 | 0 | 3 | 0 | 0 | 4 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| neoplasia | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| stage | 2 | 2 | 2 | 3 | 3 | 2 | 2 | 3 | 1 | 2 | 2 | 3 | 3 | 1 | 1 | 4 | 4 | 4 | 2 | 2 |
| gill | | | | | | | | | | | | | | | | | | | | |
| parasite | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| inflammation | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| hyperplasia/papilloma | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| kidney | | | | | | | | | | | | | | | | | | | | |
| granularity of cells | 4 | 2 | 2 | 1 | 2 | 1 | 2 | 2 | 1 | 3 | 3 | 2 | 2 | 1 | 1 | 3 | 1 | 1 | 1 | 1 |
| parasite | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | nt | 0 | 0 |
| hyperplasia | 3 | 4 | 3 | 2 | 2 | 3 | 3 | 2 | 3 | 3 | 4 | 3 | 3 | 3 | 3 | 3 | 2 | 2 | 3 | 2 |
| cells in lumen | 2 | 3 | 3 | 2 | 2 | 1 | 2 | 1 | 1 | 0 | 1 | 2 | 1 | 0 | 0 | 0 | 1 | nt | 0 | 0 |
| brown cells in epith | 2 | 3 | 2 | 2 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 2 | 2 | 1 | 0 | 1 | 2 | nt | 1 | 1 |
| digestive gland | | | | | | | | | | | | | | | | | | | | |
| brown cells in ct | 2 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 |
| parasite | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| green gland | | | | | | | | | | | | | | | | | | | | |
| abnorm. | nt | 3 | nt | 2 | nt | 2 | nt | nt | nt | nt | 2 | nt | 2 | nt | nt | nt | 2 | nt | 2 | 2 |

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Table A.19

| Organs/cells examined | 93-245 | 93-246 | 93-247 | 93-248 | 93-249 | 93-250 | 93-251 | 93-252 | 93-253 | 93-254 | 93-255 | 93-256 | 93-257 | 93-258 | 93-259 | 93-260 | 93-261 | 93-262 | 93-263 | 93-264 |
|-----------------------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|
| leukemia | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| gonad | | | | | | | | | | | | | | | | | | | | |
| inflammation | 1 | 0 | 0 | 2 | 2 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 4 | 2 | 0 | 2 | 0 |
| parasitic | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| neoplasia | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| stage | 2 | 2 | 3 | 2 | 3 | 3 | 2 | 4 | 2 | 3 | 2 | 2 | 4 | 2 | 4 | 4 | 2 | 2 | 3 | 2 |
| gill | | | | | | | | | | | | | | | | | | | | |
| parasitic | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| inflammation | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| hyperplasia/papilloma | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| kidney | | | | | nt | | | | | | | | | | | | | | | |
| granularity of cells | 3 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | nt | 3 | 3 | 2 | nt | 2 | 3 | 3 | 2 | 1 | 3 | 3 |
| parasitic | 0 | 0 | 0 | 0 | nt | 0 | 0 | 0 | nt | 0 | 0 | 0 | nt | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| hyperplasia | 2 | 3 | 2 | 3 | nt | 3 | 3 | 2 | nt | 2 | 2 | 2 | nt | 2 | 2 | 2 | 2 | 1 | 3 | 2 |
| cells in lumen | 2 | 0 | 0 | 3 | nt | 3 | 2 | 2 | nt | 0 | 3 | 2 | nt | 1 | 1 | 1 | 0 | 2 | 0 | 1 |
| brown cells in epith | 0 | 2 | 0 | 0 | nt | 3 | 2 | 1 | nt | 0 | 0 | 2 | nt | 0 | 2 | 0 | 0 | 2 | 2 | 0 |
| digestive gland | | | | | | | | | | | | | | | | | | | | |
| brown cells in ct | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 1 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| parasitic | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| green gland | | | | | | | | | | | | | | | | | | | | |
| abnorm. | ns | 2 | 2 | 2 | 2 | 2 | 3 | 2 | 3 | 2 | nt | 2 | 3 | nt | 2 | 2 | nt | 2 | 3 | 2 |

