Exxon Valdez Oil Spill
State/Federal Natural Resource Damage Assessment Final Report

Fish Histopathology Damage Assessment after the *Exxon Valdez* Oil Spill

Technical Services Study Number 2
Final Report

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October 1998

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Study History: The project effort was initiated in 1991 as a contract between the Alaska Dept. of Fish and Game (ADF&G) and the Regents of the University of California, Davis campus (UCD), laboratory of Dr. David E. Hinton. The contract originally designated Dr. Hinton's laboratory to provide fish histopathology support services for natural resource damage assessment studies that ADF&G was conducting after the *Exxon Valdez* oil spill. Tissues were collected by ADF&G personnel and sent under blind code to UCD for analysis. After preliminary results were reported in quarterly reports, the potential exposure history of each fish was revealed, data reanalyzed, and a summary histopathology report was submitted in May 1993, by Marty, G.D., Okihiro, M.S., and Hinton, D.E., under the title Fish Histopathology Report On: Exxon Valdez Oil Spill. This work contributed to several publications:

Brown, E.D., T.T. Baker, J.E. Hose, R.M. Kocan, G.D. Marty, M.D. McGurk, B.L. Norcross, and J. Short. 1996. Injury to the early life history stages of Pacific herring in Prince William Sound after the Exxon Valdez oil spill. American Fisheries Society Symposium 18:448-462.

Hose, J.E., M.D. McGurk, G.D. Marty, D.E. Hinton, E.D. Brown, and T.T. Baker. 1996. Sublethal effects of the Exxon Valdez oil spill on herring embryos and larvae: morphological, cytogenetic, and histopathological assessments, 1989-1991. Canadian Journal of Fisheries and Aquatic Sciences 53:2355-2365.

Kocan, R.M., G.D. Marty, M.S. Okihiro, E.D. Brown, and T.T. Baker. 1996. Reproductive success and histopathology of individual Prince William Sound herring 3 years after the Exxon Valdez oil spill. Canadian Journal of Fisheries and Aquatic Sciences 53:2388-2393.

Marty, G. D., J.E. Hose, M.D. McGurk, E.D. Brown, and D.E. Hinton. 1997. Histopathology and cytogenetic evaluation of Pacific herring larvae exposed to petroleum hydrocarbons in the laboratory or in Prince William Sound, Alaska, after the Exxon Valdez oil spill. Canadian Journal of Fisheries and Aquatic Sciences 54:1846-1857.

Marty, G.D., M.S. Okihiro, E.D. Brown, D. Hanes, and D.E. Hinton. 1999. Histopathology of adult Pacific herring in Prince William Sound, Alaska, after the Exxon Valdez oil spill. Canadian Journal of Fisheries and Aquatic Sciences 56:419-426.

Marty, G.D., A. Hoffmann, M.S. Okihiro, K. Hepler, and D. Hanes. In review. Histopathology and bile hydrocarbon analysis of demersal rockfish in Prince William Sound, Alaska, after the *Exxon Valdez* oil spill. Canadian Journal of Fisheries and Aquatic Sciences.

Moles, A.D., S.D. Rice, and M.S. Okihiro. 1993. Herring parasite and tissue alterations following the Exxon Valdez oil spill. 1993 International Oil Spill Conference (Prevention, Preparedness, Response). United States Coast Guard, American Petroleum Institute, and U.S. Environmental Protection Agency, Tampa, Florida. 325-328 pp.

Wiedmer, M., M.J. Fink, J.J. Stegeman, R. Smolowitz, G.D. Marty, and D.E. Hinton. 1996. Cytochrome P450 induction and histopathology in pre-emergent pink salmon from oiled

streams in Prince William Sound, Alaska. American Fisheries Society Symposium 18:509-517.

Abstract: Tissue samples from 4 fish species were examined for microscopic lesions after the *Exxon Valdez* oil spill: 1) Dolly Varden char *Salvelinus malma* adults (1990 only); 2) Pacific herring *Clupea harengus* larvae (1989 and 1990) and adults (1989 - 1992); 3) several rockfish *Sebastes* spp. adults (1990 and 1991); and 4) pink salmon *Oncorhynchus gorbuscha* larvae (1989 - 1991) and adults (1990). For each group of fish, samples from both oiled and reference sites were examined. In Dolly Varden char, hepatic lipidosis and megalocytosis were the major histopathologic markers separating exposed from reference sites. In adult herring in 1989, hepatocellular necrosis occurred in fish from exposed sites only. In larval herring in 1989, ascites prevalence was significantly greater in fish from oiled sites. Adult pink salmon had no lesions significantly related to oil exposure. Larval and juvenile pink salmon had few lesions; those most likely related to oil exposure were renal tubular necrosis and vascular thrombosis. In rockfish species from 1990 and 1991, hepatocellular lipidosis and macrophage aggregates in the liver, spleen, and kidney were the major histopathologic markers separating exposed from reference sites; based on lack of documented exposure, however, these probably resulted from site differences other than oil exposure.

Key Words: *Clupea pallasi*, Dolly Varden char, *Exxon Valdez* oil spill, histopathology, *Oncorhynchus gorbuscha*, Pacific herring, pink salmon, Prince William Sound, rockfish, *Salvelinus malma, Sebastes* spp.

Project Data: *Description of Data* - Results from histopathological analysis of approximately 7500 tissues are reported as lesion scores (none = 0, mild = 1, moderate = 2, and severe = 3) and descriptive comments. When available, age, weight, length, capture site, and capture date are also included. *Format* - Data are stored in spreadsheet format, SuperCalc 5.5b for DOS. *Custodian* - Contact Gary D. Marty, VM:APC, University of California, 1 Shields Avenue, Davis, CA 95616 (work phone: 530-754-8062, fax: 530-752-7690, or e-mail at gdmarty@ucdavis.edu). *Availability* - Copies of all data can be made available on a CD-ROM for the cost of duplication.

Citation:

Marty, G.D., M.S. Okihiro, and D.E. Hinton. 2000. Fish histopathology damage assessment after the *Exxon Valdez* oil spill, *Exxon Valdez* Oil Spill State/Federal Natural Resource Damage Assessment Final Report (Technical Services Study Number 2), Alaska Department of Fish and Game, Habitat and Restoration Division, Anchorage, Alaska.

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Executive Summary

Introduction

On March 24, 1989, the *Exxon Valdez* ran aground on Bligh Reef and spilled 11 million gallons of Prudhoe Bay crude oil into Prince William Sound, Alaska. Several fish species were likely to be impacted by oil contamination. Damage assessment efforts were focused on commercial and sport fish species from which some background fisheries information was available. Several species and life stages were studied: pink salmon *Oncorhynchus gorbuscha* larvae and adults, Pacific herring *Clupea pallasi* larvae and adults, Dolly Varden char *Salvelinus malma* adults, and several species of rockfish *Sebastes* spp. (adults). All field studies were done on fish from contaminated and references sites in Prince William Sound. Also, Pacific herring larvae and adults were exposed to crude oil in the laboratory. This project examined fish tissues from these studies for histopathological lesions related to the spill. Because most tissues were collected in 1990 and subsequent years (more than one year after the spill), organs chosen for histopathologic examination were those most likely to contain chronic or residual rather than acute lesions. The liver, kidney, spleen, and nares (olfactory organ) were thought most likely to contain chronic lesions related to oil and were sampled from most fish in 1990. Because of funding limitations, organs less likely to have chronic lesions were usually not sampled; these included heart and gill (gill lesions would have been more likely in 1989).

This contract was designed to provide histopathology support for several other projects, and it was not originally designated as an independent project. We had almost no input on the design of projects for which we provided histopathologic support. In most cases, we first learned what tissues needed analysis when they were delivered to our laboratory. Sample size and quality of tissue received were highly variable.

Objectives

The primary objective was to determine the nature and prevalence of microscopic changes in fish tissues sampled from oiled and reference sites in Prince William Sound, Alaska. The basic assumption was that differences in microscopic lesions between oiled and reference sites were a result of oil exposure. For Pacific herring larvae and adults, analysis also included tissues from laboratory study where the amount of crude oil exposure was known. A supporting objective was to provide references, related to histopathologic analysis of larval and adult fish, that would supplement oil spill litigation (and oil spill research).

Methods

Tissues received at the University of California, Davis, were usually labeled by site of origin, but the potential exposure history of each site was not known. Samples were assigned a random number for processing and blind examination. Tissues were trimmed, processed into paraffin, sectioned at 5 μm, and stained with hematoxylin and eosin. Histopathological lesions in each organ were semiquantitatively ranked as none (0), mild (1), moderate (2), or severe (3). After scoring was completed and preliminary report was submitted to the Alaska Department of Fish

and Game, the exposure history of each site was revealed. Data were then subjected to statistical analysis, which involved summarizing the data using principal components analysis followed by analysis of variance (ANOVA) or multivariate analysis of variance (MANOVA) of the scale values derived from principal components analysis. When the appropriate data were available, the analysis could test for gender and age differences.

To determine if oil had a significant effect on exposed fish, several questions were considered; greatest confidence occurred when the following were met: 1) sites were separable by visually scanning lesion scores; 2) site separation was confirmed with statistical analysis; and 3), lesions important for separating sites were consistent with the peer-reviewed scientific literature or laboratory experiments that were part of this project.

Two laboratory studies were conducted with Pacific herring. In one experiment, Pacific herring eggs were exposed to an oil-water dispersion of Prudhoe Bay crude oil (initial concentrations of 0.0, 0.10, 0.24, 0.48, and 2.41 mg/L) and sampled for histopathology < 24 hours after hatching. In the other experiment, adult Pacific herring were exposed to water soluble fraction of Alaska North Slope crude oil via the water column as water-soluble fraction (0.36 or 0.72 mg crude oil/L seawater) or via force-fed gelatin capsules (control, low, and high dose groups). Fish were sampled at days 0, 1, 2, 4, 7, and 10 days after initiation of exposure. After 10 days, some fish were transferred to clean water for 3 and 7 day depuration. Tissue samples were analyzed for hydrocarbon uptake, fluorescent aromatic compounds (in bile), mixed function oxidase activity (liver only), and microscopic lesions.

Results and Discussion

Rockfish (*Sebastes spp.*) sampled from oiled sites in Prince William Sound, Alaska, USA, had biliary hydrocarbons consistent with exposure to *Exxon Valdez* oil in 1989, but not in 1990 or 1991. Microscopic lesions in rockfish from oiled sites were significantly different from fish from reference sites in 1991, but not in 1990. Increased scores for pigmented macrophage aggregates were significantly related to age in 1990 and 1991. Hepatocellular megalocytosis and sinusoidal fibrosis, most common in quillback rockfish, occurred at oiled and reference sites. Hepatocellular lipidosis, most common in yelloweye rockfish from oiled sites, was more consistent with lipid storage than with pathologic change. These results provide evidence that demersal rockfish species were exposed to significant concentrations of *Exxon Valdez* oil in 1989, but differences in microscopic lesions in 1990 and 1991 were probably not related to previous oil exposure.

In Dolly Varden char sampled in June 1990, fatty liver (hepatic lipidosis) and enlarged liver cells (megalocytosis) were the major histopathologic markers separating exposed from reference sites. Samples from fall 1990 no longer had differences in hepatic lipidosis and megalocytosis, but fish from reference sites had significantly greater inflammatory lesions in the nares.

In adult Pacific herring sampled in 1989, and in the laboratory exposure, the major acute lesion was death of liver cells (hepatocellular necrosis), and moderate to severe necrosis appeared in fish from exposed sites only. Later experiments provided evidence that liver cell death was a result of expression of viral hemorrhagic septicemia virus in oil-exposed fish. Dead cells are ingested by a

type of white blood cell in the tissues (macrophages), and macrophages may accumulate in distinctive pigmented aggregates. In 1990, macrophage aggregates were the major histopathologic marker separating reference from exposed sites, but recent study provided evidence that these differences were probably a result of age differences between the sites (pigmented macrophage aggregates are normally more abundant in older fish, and fish from the exposed site were significantly older than fish from the reference site). Samples from 1991 were not significantly different based on exposure history of the site of capture. In 1992, microscopic lesions were associated with decreased reproductive success but not with exposure history of the site of capture.

In newly hatched Pacific herring larvae that were sampled from Prince William Sound as eggs in 1989, and then hatched in clean water in the laboratory, preliminary examination with a dissecting microscope revealed cranial and eye masses; histopathologic examination, however, revealed that the masses were not tumors. In Pacific herring sampled as larvae from Prince William Sound, up to 2 months after the spill, fish from oiled sites had excess fluid in the abdominal cavity and around the heart. Laboratory study confirmed that these lesions were consistent with oil exposure. Larvae from oiled sites were shorter, had ingested less food, and had slower growth (oiled, 0.07-0.10 mm/d; reference, 0.15-0.18 mm/d). Larvae from oiled sites had higher prevalence of excess abdominal fluid (16%, oiled; 1%, reference). In the laboratory experiment, effects were statistically significant at the 0.48 mg/L dose (Dunnett's procedure, $P < 0.05$). Lesions included increased abdominal fluid, liver cell vacuolar change, and degeneration or death of 3 types of cells: muscle cells, retinal (eye) cells, and developing brain cells. Lesions in field-sampled larvae were consistent with higher mortality rates documented in larvae from oiled sites.

Adult pink salmon had several lesions including macrophage aggregates, hepatocellular megalocytosis, and parasites, but none were clearly related to oil exposure. Lesions in larval and juvenile pink salmon included muscle cell necrosis, necrosis of kidney tubules, and vascular blockage (thrombosis), but these could not be definitively related to oil exposure.

In rockfish sampled in 1990 and 1991, fatty liver (hepatocellular lipidosis) and macrophage aggregates in the liver, spleen, and kidney were the major histopathologic markers separating exposed from reference sites. In other studies, both types of lesions have been associated with exposure to a variety of toxicants, including oil. However, rockfish bile had no evidence of oil exposure in 1990 or 1991, and tissue differences in 1991 were probably a result of site differences other than oil.

Conclusions

Fish in Prince William Sound were significantly affected by the spill. Pacific herring larvae from oiled bays grew slower and many had severe distension of their abdominal cavity. Pacific herring adults from oiled areas had liver lesions that were consistent with expression of a potentially deadly virus. Dolly Varden char and rockfish from oiled sites had liver lesions that fish from reference sites did not have. Only for pink salmon were significant affects not detected in fish from oiled areas.

Project Introduction

On March 24, 1989, the *Exxon Valdez* ran aground on Bligh Reef and spilled 11 million gallons of Prudhoe Bay crude oil into Prince William Sound, Alaska. Several fish species were likely to be impacted by oil contamination. Unfortunately, initial response and damage assessment efforts were poorly organized and fragmented, and too few fish tissues were sampled for histopathology in 1989. In 1990, David Hinton was contacted to coordinate histopathologic analysis of fish tissues collected from Prince William Sound, and he assisted with collection of pink salmon samples in 1990. Because most tissues were to be collected in 1990 and subsequent years (more than one year after the spill), organs chosen for histopathologic examination were those most likely to contain chronic or residual rather than acute lesions. The liver, kidney, spleen, and nares (olfactory organ) were thought most likely to contain chronic lesions related to oil and were sampled from most fish in 1990. Because of funding limitations, organs less likely to have chronic lesions were usually not sampled; these included heart and gill (gill lesions would have been more likely in 1989). Tissues were shipped to the University of California, Davis, beginning in September 1990, but analysis was delayed until after this contract (IHP-91-033) was finally approved on February 21, 1991.

Note that this contract was designed to provide histopathology support for several other projects, and it was not originally designated as an independent project. We had almost no input on the design of projects for which we provided histopathologic support. Indeed, in most cases we first learned what tissues needed analysis when they were delivered to our laboratory. Sample size and quality of tissue received were highly variable. Because this report is primarily to document results, individual chapters do not have an "Introduction" section. Results that have already been published are included by reference, along with the text of the published abstract. For a comprehensive literature review of fish histopathology after oil exposure, see Chapter XI.

Project Objectives

The primary objective was to determine the nature and prevalence of microscopic changes in fish tissues sampled from oiled and reference sites in Prince William Sound, Alaska. The basic assumption was that differences in microscopic lesions between oiled and reference sites were a result of oil exposure. For Pacific herring larvae and adults, analysis also included tissues from laboratory study where the amount of crude oil exposure was known. A supporting objective was to provide references, related to histopathologic analysis of larval and adult fish, that would supplement oil spill litigation (and oil spill research).

Project Statistical Analysis

Statistical Consultant - Neil Willits, Senior Statistician, Division of Statistics, 380 Kerr Hall, University of California, Davis, 95616

Tissues were logged in by sample group (e.g., rockfish samples collected in 1990). Each fish was assigned a unique number (processing code) used for embedding, cutting, and histopathologic analysis. Tissues were processed routinely in paraffin and stained with hematoxylin and Eosin

(HE). Slides were read in ascending numerical order based on the assigned random number (i.e., blind study). Lesions were subjectively ranked using a 4 point scale: none (0), mild (1), moderate (2), or severe (3); some tissues were not present (NP) for analysis. To optimize precision of results in adult tissues, all specimens of a given organ were read and scored before any specimens of the next organ were scored. After lesion scores were recorded in spreadsheet format and sorted by site of origin, significance of findings was determined as follows:

1) visually scan data to identify lesions that occurred in different frequency and/or severity at different sites;

2) speculate on exposure history of each site in a progress report;

3) determine actual exposure history of each site; revealed by the Alaska Dept. of Fish and Game after lesion scores had been submitted.

4) analyze the data statistically to determine if impressions of the histopathologist were quantifiable.

In Oil Spill studies, several types of analysis have been recommended (Cox et al. 1979). The fish histopathology data were analyzed using Principal components analysis (PCA) followed by analysis of variance (ANOVA) or multivariate analysis of variance (MANOVA) of the scale values derived from PCA. This type of analysis has several advantages:

1) accounts for the presence AND severity of lesions;
2) identifies sources of variability;
3) identifies the most significant lesions;
4) determines the significance of oiled vs. reference site differences with a single P value.

PCA has 2 disadvantages. First, it cannot handle missing values; for example, if the nares were missing from a fish, then the fish was lost from analysis even if lesions in the liver, spleen, and kidney from that fish were scored. And second, interpretation required caution. The underlying assumption for all oiled vs. reference site comparisons was that fish from the various sample sites differed only in their potential exposure to oil. Often, variables such as age and sex could be accounted for using ANOVA, but other important variables might have been missed. To determine if oil had a significant effect on exposed fish, several questions were considered; greatest confidence occurred when the following were met: 1) sites were separable by visually scanning lesion scores; 2) site separation was confirmed with statistical analysis; and 3), lesions important for separating sites had previously been associated with oil or toxicant exposure in the peer-reviewed scientific literature or in known-exposure experiments. The use of semiquantitative histopathology, in the hands of trained pathologists, has been shown to be both accurate and cost effective when critically compared with more laborious, but quantifiable, morphometric techniques (Hyde et al. 1992a).

Summary of tissues received and status of results

HISTOPATHOLOGY SAMPLES:	STATUS:
Rockfish, 1990, 1991	Oil-related differences in microscopic lesions were more significant in 1991 than in 1990. Results were summarized in a manuscript submitted for publication in the peer-reviewed literature (Marty et al. In review); Chapter I has the title and abstract.
Dolly Varden char, fall and spring samples from 1990	Oil-related differences in microscopic lesions were subtle but significant. Lesions were not specific. Results are reported in Chapter II, and they will not be prepared for publication in the peer-reviewed literature.
Pacific herring larvae from egg incubation experiments; 1989, 1990	Results have been published (Hose et al. 1996); chapter III has the title and abstract.
Pacific herring larvae trawled from Prince William Sound, 1989	Results have been published (Marty et al. 1997b); chapter IV has the title and abstract.
Pacific herring adults, 1989, 1990, 1991	Results have been published (Marty et al. 1999); chapter V has the title and abstract.
Pacific herring adults, 1991 (NMFS, Auke Bay Laboratory, oil exposure study)	Results described in Chapter VI, but they will not be published. Results from a better study have been published (Carls et al. 1998).
Pacific herring adults, 1992 reproduction study	Results have been published (Kocan et al. 1996); chapter VII has the title and abstract.
Pink salmon larvae, 1989, 1990, and 1991; response samples	Results have been published (Weidmer et al. 1996); chapter VIII has the title and abstract.
Pink salmon eggs/larvae, 1990 and 1991; damage assessment samples	The potential exposure history of some of these samples was never revealed. Of those that were revealed, we found no oil-related histologic lesions. Results are reported in Chapter IX, and they will not be prepared for publication in the peer-reviewed literature.
Pink salmon adults - 1990	No microscopic lesions could be attributed to oil exposure. Results are reported in Chapter X, and they will not be prepared for publication in the peer-reviewed literature.

CHAPTER 1 - Histopathology and bile hydrocarbon analysis of demersal rockfish in Prince William Sound, Alaska, after the *Exxon Valdez* oil spill.

Citation:

Marty, G.D., A. Hoffmann, M.S. Okihiro, K. Hepler, and D. Hanes. In review. Histopathology and bile hydrocarbon analysis of demersal rockfish in Prince William Sound, Alaska, after the *Exxon Valdez* oil spill. Canadian Journal of Fisheries and Aquatic Sciences.

Abstract: Rockfish (*Sebastes spp.*) sampled from oiled sites in Prince William Sound, Alaska, USA, had biliary hydrocarbons consistent with exposure to *Exxon Valdez* oil in 1989, but not in 1990 or 1991. Microscopic lesions in rockfish from oiled sites were significantly different from fish from reference sites in 1991, but not in 1990. Liver, kidney, and spleen of copper rockfish (*Sebastes caurinus*), quillback rockfish (*S. maliger*), and yelloweye rockfish (*S. ruberrimus*) from two oiled and two reference sites were examined for microscopic lesions in 1990 (n = 50) and 1991 (n = 107). Increased scores for pigmented macrophage aggregates were significantly related to age in 1990 and 1991. Hepatocellular megalocytosis and sinusoidal fibrosis, most common in quillback rockfish, occurred at oiled and reference sites. Hepatocellular lipidosis, most common in yelloweye rockfish from oiled sites, was more consistent with lipid storage than with pathologic change. We conclude that demersal rockfish species were exposed to significant concentrations of *Exxon Valdez* oil in 1989, but differences in microscopic lesions in 1990 and 1991 were probably not related to previous oil exposure.

CHAPTER 2 - Histopathology of Dolly Varden char in Prince William Sound, Alaska, USA, after the *Exxon Valdez* oil spill.

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Methods

Jars of formalin containing tissues from 60 spring/summer-sampled and 72 fall-sampled 1990 adult Dolly Varden Char were received at the University of California, Davis. Liver, spleen, kidney, and nares were submitted for each of the 72 fall-sampled fish, but only liver was submitted from the spring/summer-sampled fish. All fish were assigned a random number (processing code, see Tables I-1 and I-2) and all tissues were processed routinely in paraffin and stained with hematoxylin and Eosin. Slides were read in ascending numerical order based on the assigned random number (i.e., blind study); spring/summer livers were read at the same time as the fall livers (i.e., the random numbers were generated for the 2 samples as if they were one group). Lesions were subjectively ranked using a 4 point scale: none (0), mild (1), moderate (2), or severe (3); some tissues were not present (NP) for examination. To optimize precision of results, all specimens of a given organ (e.g., all 132 livers) were read and scored before any specimens of the next organ were scored. Basic historical/site data and significant lesion scores are listed in Tables I-1 and I-2.

To detect cellular expression of cytochrome P450IA (CYP1A), a serial section of each tissue was sent to the Wood Hole Oceanographic Institute, laboratory of Dr. John Stegeman. Immunohistochemical evaluation was done by the indirect horseradish peroxidase technique using the mouse monoclonal antibody (Mab) 1-12-3 as the primary antibody (1:30,000) as described previously (Park et al. 1986, Smolowitz et al. 1991). Monoclonal antibody 1-12-3 is specific for CYP1A protein product in all vertebrate species examined to date (Stegeman and Hahn 1995, Guiney et al. 1997). At least 1 section from each organ was examined immunohistochemically. Positive and negative control sections were livers from known induced winter flounder *Pleuronectes americanus* and livers from known uninduced scup *Stenotomus chrysops*. Sections from every third section were incubated with a nonspecific mouse Mab (IgG) substituted for Mab 1-12-3. All sections were processed under identical conditions, including incubation times with the primary antibodies. Staining intensity and occurrence of CYP1A expression were evaluated histologically for each tissue and scored on a 5-point scale: negative (0), very mild (1), mild (2), moderate (3), or strong (4). CYP1A results were not used in the statistical analysis.

Results

In the liver, normal hepatocytes were laden with glycogen. Hepatocellular megalocytosis was uncommon, and when present, was usually mild. A few fish had nuclear pseudo-inclusions, probably a result of finger-like depressions of nuclear margins with "apparent" inclusion of the

cytoplasm in the nucleus. Several livers had increased numbers of mitotic figures. A few fish had scattered, individual, dead hepatocytes (apoptosis). Many fish had small numbers of widely scattered macrophage aggregates. Aggregates of lymphocytes were rare. Hepatocellular glycogen depletion was common, and a few fish from oiled sites had mild to severe hepatocellular lipidosis. A few fish were infected with *Ichthyophonus hoferi*, and a few fish had trematodes in their biliary system.

In the kidney, some fish had mild vacuolation of renal tubular epithelium. In the interstitium, small to moderate numbers of macrophage aggregates were common. The aggregates were usually associated with heavy deposits of melanin (melanomacrophage centers). *Ichthyophonus hoferi* was rare.

In the spleen, some fish had small numbers of macrophage aggregates containing large amounts of melanin. Fish had no other significant lesions in the spleen.

Normal olfactory nares were composed of multiple, regular, plates or lamellae covered with stratified olfactory epithelium. Lamellar tips and side branches were lined by squamous epithelium and often infiltrated by small numbers of lymphocytes. Nares commonly had inflammatory changes. The majority of nares had mild to moderate lymphocytic infiltration of the squamous portion of the olfactory epithelium. Many nares had severe neutrophilic inflammation centered in the lamina propria of the olfactory lamellae. Neutrophilic infiltration resulted in marked thickening of inflamed lamellae and was often mixed with large numbers of eosinophilic granular leukocytes (EGLs) and lesser numbers of mononuclear inflammatory cells. Some lamellae contained small foci of hemorrhage. Some fish had small to moderate numbers of globular leukocytes in either the olfactory epithelium or within the lamina propria. Globular leukocytes had characteristic large, refractile, brightly eosinophilic, intracytoplasmic granules (they may represent a type of EGL). A few fish had macrophages mixed in with the other inflammatory cells. Some fish had mild mucous cell hyperplasia. In a few fish, the ciliated, stratified, columnar epithelium appeared thickened. Several fish had refractile, eosinophilic, intranuclear inclusions in olfactory epithelial cells. The inclusion were concentrated within the squamous portion of the epithelium (lamellar tips, side branches, and base). Some nuclei contained multiple inclusions.

Final comment on histopathologic lesions: Lesions in some Dolly Varden were similar to those observed in the other fish species (e.g., pink salmon, rockfish, and Pacific herring), but the prevalence in affected fish was much lower and, in general, the lesions tended to be much milder. The only exception to this was in the nares. The nares of many Dolly Varden had moderate to severe mixed inflammation involving primarily eosinophilic granular leukocytes (EGLs) and neutrophils. The lesions are compatible with exposure to infectious agents. There was no evidence of large protozoan or metazoan parasites, but bacterial or viral pathogens are still a possibility.

Statistics: For general details about the types of statistical analysis used, see Project Statistical Analysis on page vii.

Statistical Consultant - Neil Willits, Senior Statistician, Division of Statistics, 254 Kerr Hall, University of California, Davis, 95616

After lesion scores were recorded in spreadsheet format (Tables II-1 and II-2) and sorted by site of origin, lesions were visually scanned. Because the lesions were generally mild, speculation on the exposure history (i.e., oiled vs. reference) of fish from each site was tenuous. However, qualitatively scanning the scores from the June 1990 samples revealed some differences, and the speculated exposure history was correct in every case (Table II-1). For the Fall 1990 samples, differences were not discernable by visually scanning the data, and speculation of exposure history was not done.

June (Spring) 1990 Samples

Exposure-related differences were significant in the first principal component (ANOVA), and principal components analysis revealed that hepatic lipidosis and megalocytosis were most important in the first principal component. Note that these are the same lesions that were used to speculate on exposure status in Table II-1. Oil-related differences were not significant for the second, third, or fourth principal components. Sex differences were not significant for any principal component, and length differences were significant only for the third component. Tests for overall effects were significant for oiled vs. reference comparisons but not for gender. In a separate analysis, overall length differences were not significant ($P = 0.2709$, Wilks' Lambda, Pillai's Trace, Hotelling-Lawley Trace, and Roy's Greatest Root; data not shown).

To determine if differences within oiled or reference sites were significant, the analysis was repeated to include nested site effects. Results were similar: 1) for the first principal component, oiled vs. reference comparisons and differences within oiled sites were significant; 2) for the second through fourth principal components, differences were not significant; 3) tests for overall effects of oiled vs. reference were not significant ($P = 0.1049$; Wilks' Lambda, Pillai's Trace, Hotelling-Lawley Trace, and Roy's Greatest Root; data not shown); 4) site (within oiled or reference) differences were significant; and 5) sex differences were not significant.

Fall 1990 Samples

Overall differences in lesions scores were significant with respect to exposure history (MANOVA). Also, oiled vs. reference differences were significant for the first principal component (ANOVA), but not for the second, third, or fourth components. Lesions most important in the first principal component included splenic macrophages, nares neutrophils, eosinophilic granular leukocytes, and single cell necrosis. Note that these lesions are different from the lesions that were used to speculate on exposure status in the spring samples (Table II-1). In general, the inflammatory nares lesions were more severe in fish from the reference sites than in fish from the oiled sites (see summary section at the end of Table II-3). Sex and length differences were not significant for any principal component.

To determine if differences within oiled or reference sites were significant, the analysis was repeated to include nested site effects. Results were similar: 1) for the first principal component,

oiled vs. reference effects were significant, but differences within oiled or reference sites were not significant; 2) for the second, third, and fourth principal components, differences were not significant; and 3) tests for overall effects were significant for oiled vs. reference ($P = 0.019$), but not for site within oiled or reference groups.

Discussion

For the Spring 1990 samples, hepatic lipidosis and megalocytosis were used to separate oiled from reference sites in the first principal component. Hepatocellular lipidosis has been associated with oil exposure (McCain et al. 1978, Eurell and Haensly 1981, Fletcher et al. 1982, Solangi and Overstreet 1982, Khan and Kiceniuk 1984), but megalocytosis has not been associated with crude oil exposure. Therefore, evidence is weak that differences in lesion scores between oiled and reference sites are a result of oil exposure. Also, the overall results (MANOVA) were not significant. Note that a preliminary analysis of the results, in which length effects were not included, found significant differences related to exposure history of the site of capture ($P = 0.02$). Ages of fish were not available, but the length of spring-sampled fish were not statistically related to lesion scores. Significant site differences resulted in part from more severe oil-associated lesions in fish from Eshamy creek compared with Green Island (both sites were oiled), but both sites had more oil-associated lesions than the 2 reference sites.

For the fall samples, at least one lesion that was used to separate oiled from reference sites—single cell necrosis of olfactory epithelium—has been associated with oil exposure (Solangi and Overstreet 1982), but lesions in that study were more severe in exposed fish. By comparison, nares lesions (including inflammation in the nares) in Dolly Varden from Prince William Sound were more severe in fish from reference sites. One possible explanation is that the olfactory nares are normally exposed and respond to antigens in the environment; lack of inflammation might be a result of immunosuppression in the fish or decreased numbers/types of antigens in the environment (i.e., oil-associated decrease in microbiota). Both of these explanations, however, are speculative, and it seems improbable that lack of olfactory lesions was related to oil exposure. For fall samples, increased inflammation in the nares of fish from reference sites was more likely a site effect. Lesion scores were not related to either site or length differences.

Table II-1. Hepatic lesion scores in Dolly Varden char sampled in June 1990 from Prince William Sound, Alaska. Each numeral represents a score from an individual fish from the named site. Lesions were scored as none (0), mild (1), moderate (2), or severe (3).

	Liver lesion scores		Exposure History	
Site	Lipidosis	Megalocytosis	Speculated[a]	Actual[b]
Makaka Creek	0 0 0 0 0 0 0 0 0 0 0 0	0 0 0 0 0 0 0 0 0 0 0 0	reference	reference
Boswell Bay	0 0 0 0 0 0 0 0 0 0 0 0	0 0 0 0 0 0 0 0 0 0 0 1	reference	reference
Rocky Bay	0 0 0 0 0 0 0 0 0 0 0 0	0 0 0 1 0 0 0 0 0 0 0 0	reference	reference
Green Island	0 0 1 0 0 0 0 0 0 0 1 0	0 0 0 1 0 0 0 0 0 0 0 0	oiled?	oiled
Eshamy Creek	2 0 1 0 0 0 0 3 2 1 3 0	0 0 1 0 0 0 0 1 0 2 1 1	oiled	oiled

[a]Speculated exposure history was reported on 5-4-92.
[b]Actual exposure history was revealed by Kelly Hepler on 5-18-92 and 11-20-92.

Table II-2. Histopathologic findings in Dolly Varden char adults sampled from Prince William Sound in spring/summer 1990.

Key to table symbols:
Proc. code = random (processing) number generated by Dr. Hinton's laboratory
ADF&G AWL # = numbers reported by the Alaska Dept. of Fish and Game
OS = oiled status; oiled (O), or control/clean (C)
MFO = mixed function oxidase; ranked as negative (0), very mild (1), mild (2), mod (3), or strong (4)
note: MFO values were determined for hepatocytes, bile ductules, and hepatic endothelium, but scores here are the maximum score for any of the 3 sites (usually hepatocytes).
ND = MFO determination was not done
Lesion scores = none (0), mild (1), moderate (2), severe (3), or not present "."
LIVER:

glycogen depletion (GLY)	lipidosis (LIP)
macrophage aggregates (MA)	lymphocytes (LY)
single cell necrosis (SCN)	hepatocellular karyomegaly (MEG)
sinusoidal fibrosis (FIB)	

#	Hinton Fish #	Proc. Code	Sex	ADF&G AWL #	Location	OS	MFO	Length (mm)	Date collected	Liver GLY	LIP	MA	SCN	LY	MEG	FIB
1	38	V 1	F	211974-2	Makaka Creek	C	0	246	13 Jun 90	3	0	0	0	0	0	0
2	47	V 7	?	211974-11	Makaka Creek	C	0	361	13 Jun 90	3	0	1	0	0	0	0
3	37	V 16	M	211974-1	Makaka Creek	C	2	312	13 Jun 90	3	0	0	0	0	0	0
4	40	V 19	F	211974-4	Makaka Creek	C	3	346	13 Jun 90	3	0	1	0	1	0	0
5	41	V 37	F	211974-5	Makaka Creek	C	3	246	13 Jun 90	3	0	1	0	0	0	0
6	42	V 63	F	211974-6	Makaka Creek	C	ND	309	13 Jun 90	3	0	0	0	0	0	0
7	43	V 75	F	211974-7	Makaka Creek	C	ND	300	13 Jun 90	3	0	1	0	1	0	0
8	44	V 76	M	211974-8	Makaka Creek	C	ND	401	13 Jun 90	3	0	1	0	0	0	0
9	48	V 90	?	211974-12	Makaka Creek	C	ND	211	13 Jun 90	3	0	1	1	0	0	0
10	46	V 98	?	211974-10	Makaka Creek	C	ND	273	13 Jun 90	3	0	0	0	0	0	0
11	45	V 103	?	211974-9	Makaka Creek	C	ND	314	13 Jun 90	3	0	0	0	0	0	0
12	39	V 133	F	211974-3	Makaka Creek	C	ND	261	13 Jun 90	3	0	0	0	0	0	0
13	54	V 12	M	211747-6	Rocky Bay Weir	C	0	245	13 Jun 90	3	0	0	0	0	0	0
14	58	V 17	M	211747-10	Rocky Bay Weir	C	3	323	13 Jun 90	3	0	1	0	0	0	0
15	57	V 29	M	211747-9	Rocky Bay Weir	C	3	330	13 Jun 90	3	0	0	0	0	0	0
16	59	V 31	F	211747-11	Rocky Bay Weir	C	0	335	13 Jun 90	3	0	0	0	0	1	0
17	51	V 57	F	211747-3	Rocky Bay Weir	C	ND	315	13 Jun 90	3	0	1	0	0	0	0
18	50	V 68	F	211747-2	Rocky Bay Weir	C	ND	310	13 Jun 90	3	0	0	0	0	0	0
19	53	V 73	M	211747-5	Rocky Bay Weir	C	ND	247	13 Jun 90	3	0	1	0	0	0	0
20	60	V 113	F	211747-12	Rocky Bay Weir	C	ND	352	13 Jun 90	3	0	1	0	0	0	0
21	55	V 115	M	211747-7	Rocky Bay Weir	C	ND	241	13 Jun 90	3	0	1	0	0	0	0
22	52	V 123	F	211747-4	Rocky Bay Weir	C	ND	245	13 Jun 90	3	0	0	0	0	0	0
23	49	V 132	M	211747-1	Rocky Bay Weir	C	ND	337	13 Jun 90	3	0	0	0	0	0	0
24	56	V 136	F	211747-8	Rocky Bay Weir	C	ND	305	13 Jun 90	3	0	1	0	0	0	0
25	35	V 5	F	219328-11	Boswell Bay W.	C	0	312	14 Jun 90	3	0	1	0	0	0	0
26	36	V 26	F	219328-12	Boswell Bay W.	C	0	318	14 Jun 90	3	0	0	0	0	0	0
27	30	V 27	F	219328-6	Boswell Bay W.	C	0	384	14 Jun 90	3	0	1	0	0	0	0
28	33	V 41	F	219328-9	Boswell Bay W.	C	0	292	14 Jun 90	3	0	0	0	0	0	0
29	26	V 43	F	219328-2	Boswell Bay W.	C	0	321	14 Jun 90	3	0	1	0	0	0	0
30	27	V 77	F	219328-3	Boswell Bay W.	C	ND	277	14 Jun 90	3	0	1	0	0	0	0
31	28	V 83	F	219328-4	Boswell Bay W.	C	ND	448	14 Jun 90	3	0	1	0	0	0	0
32	34	V 87	F	219328-10	Boswell Bay W.	C	ND	335	14 Jun 90	3	0	1	0	0	0	0
33	32	V 97	F	219328-8	Boswell Bay W.	C	ND	315	14 Jun 90	3	0	1	0	1	0	0
34	25	V 100	F	219328-1	Boswell Bay W.	C	ND	332	14 Jun 90	3	0	0	0	0	0	0
35	31	V 101	M	219328-7	Boswell Bay W.	C	ND	269	14 Jun 90	3	0	1	0	0	0	0
36	29	V 129	M	219328-5	Boswell Bay W.	C	ND	278	14 Jun 90	3	0	0	0	0	1	0
37	4	V 23	M	none	Eshamy Cr. Weir	O	0	204	14 Jun 90	3	2	0	0	0	0	0
38	9	V 35	F	none	Eshamy Cr. Weir	O	0	244	14 Jun 90	3	0	1	0	0	0	0
39	1	V 47	M	none	Eshamy Cr. Weir	O	0	186	14 Jun 90	3	1	0	2	0	1	0
40	12	V 49	M	none	Eshamy Cr. Weir	O	0	294	14 Jun 90	3	0	0	0	1	0	0

#	Hinton Fish #	Proc. Code	Sex	ADF&G AWL #	Location	OS	MFO	Length (mm)	Date collected	Liver GLY	LIP	MA	SCN	LY	MEG	FIB
41	6	V 58	M	none	Eshamy Cr. Weir	O	ND	217	14 Jun 90	3	0	0	0	1	0	0
42	2	V 64	F	none	Eshamy Cr. Weir	O	ND	195	14 Jun 90	3	0	0	1	1	0	0
43	5	V 80	M	none	Eshamy Cr. Weir	O	ND	210	14 Jun 90	3	0	2	0	0	0	0
44	7	V 105	M	none	Eshamy Cr. Weir	O	ND	220	14 Jun 90	2	3	0	0	0	1	0
45	8	V 119	F	none	Eshamy Cr. Weir	O	ND	239	14 Jun 90	3	2	0	0	0	0	0
46	10	V 120	F	none	Eshamy Cr. Weir	O	ND	274	14 Jun 90	3	1	0	0	1	2	0
47	11	V 124	F	none	Eshamy Cr. Weir	O	ND	291	14 Jun 90	3	3	0	0	0	1	0
48	3	V 135	F	none	Eshamy Cr. Weir	O	ND	200	14 Jun 90	3	0	0	1	0	1	0
49	24	V 2	F	214805	Green Isl. Weir	O	ND	?	21 Jun 90	3	0	1	0	0	0	0
50	19	V 18	F	214807	Green Isl. Weir	O	3	215	15 Jun 90	3	0	0	0	0	0	0
51	23	V 22	F	214805	Green Isl. Weir	O	3	234	21 Jun 90	3	1	0	0	0	0	0
52	16	V 42	F	214813	Green Isl. Weir	O	0	212	13 Jun 90	3	0	1	0	0	1	0
53	14	V 86	?	214813	Green Isl. Weir	O	ND	205	13 Jun 90	3	0	0	0	0	0	0
54	18	V 91	F	214810	Green Isl. Weir	O	ND	206	14 Jun 90	3	0	0	0	0	0	0
55	17	V 104	F	214810	Green Isl. Weir	O	ND	227	14 Jun 90	3	0	1	0	0	0	0
56	13	V 109	F	214813	Green Isl. Weir	O	ND	297	13 Jun 90	3	0	1	0	0	0	0
57	21	V 127	F	214805	Green Isl. Weir	O	ND	192	21 Jun 90	3	0	0	0	0	0	0
58	15	V 128	F	214813	Green Isl. Weir	O	ND	206	13 Jun 90	3	0	0	0	0	0	0
59	22	V 131	F	214805	Green Isl. Weir	O	ND	195	21 Jun 90	3	1	0	0	0	0	0
60	20	V 134	F	214807	Green Isl. Weir	O	ND	245	15 Jun 90	3	0	1	0	0	0	0

Exposure status	Site	Length mean	Length std. dev.	Liver lesions (Mean scores) GLY	LIP	MA	SCN	LY	MEG	FIB
reference	Makaka	298	52	3	0	.5	.08	.17	0	0
reference	Rocky Bay	299	40	3	0	.5	0	0	.08	0
reference	Boswell Bay	323	48	3	0	.67	0	.08	.08	0
oiled	Eshamy Creek	231	36	2.9	1	.25	.33	.33	.5	0
oiled	Green Island	221	28	3	.17	.42	0	0	.08	0

Table II-3. Histopathologic findings in Dolly Varden char adults sampled from Prince William Sound in fall 1990.

Key to table symbols:
Proc. code = random (processing) number generated by Dr. Hinton's laboratory
ADF&G AWL # and Fish # = numbers reported by the Alaska Dept. of Fish and Game
Site - Bos. (= Boswell Bay), Rocky (= Rocky Bay), Eshamy (=Eshamy Lake), and Green (=Green Island)
OS = oiled status; oiled (O) or control/clean (C)
MFO = mixed function oxidase; ranked as negative (0), very mild (1), mild (2), mod (3), or strong (4)
note: MFO values were determined for hepatocytes, bile ductules, and hepatic endothelium, but scores here are the maximum score for any of the 3 sites (usually hepatocytes).
ND = MFO determination was not done
Length = fork length (mm)

Lesion scores = none (0), mild (1), moderate (2), severe (3), or not present "."

LIVER:
- glycogen depletion (GLY)
- lipidosis (LIP)
- macrophage aggregates (MA)
- lymphocytes (LY)
- single cell necrosis (SCN)
- hepatocellular karyomegaly (MEG)
- sinusoidal fibrosis (FIB)

SPLEEN (SPL):
- melanomacrophage centers (MA)

KIDNEY:
- melanomacrophage centers (MA)
- lymphocytes (LY)
- tubular epithelial vacuolar degeneration (VD)
- tubular necrosis (NEC)
- glomerular basement membrane thickening (GBM)

NARES:
- lymphocytes (LY)
- neutrophils (NEU)
- eosinophilic granular leukocytes (EGL)
- macrophages (MAC)
- globular leukocytes (GLO)
- mucous cell hyperplasia (MCH)
- sensory epithelial cell hyperplasia (SEH)
- single cell necrosis (SCN)

#	Proc. Code	Sex	ADF&G AWL #	ADF&G fish#	Site	OS	MFO	L (mm)	Sample Date	Liver GLY	Liver LIP	Liver MA	Liver LY	Liver SCN	Liver MEG	Liver FIB	Spl MA	Kidney MA	Kidney LY	Kidney VD	Kidney NEC	Kidney GBM	Kidney LY	NARES NEU	NARES EGL	NARES MAC	NARES GLO	NARES MCH	NARES SEH	NARES SCN
1	V 110	F	UCD 915	55	Makaka	C	ND	367	23 Oct 90	2	0	0	1	0	0	0	0	2	0	0	0	0	2	3	3	0	0	0	0	1
2	V 72	M	UCD 916	56	Makaka	C	ND	385	23 Oct 90	3	3	0	0	0	0	0	0	2	0	1	0	0	3	3	3	0	0	2	1	1
3	V 44	M	UCD 917	57	Makaka	C	0	376	24 Oct 90	3	3	0	0	0	0	0	0	1	0	0	0	0	2	3	2	1	0	0	0	2
4	V 15	F	UCD 901	41	Bos	C	0	369	16 Oct 90	3	0	0	0	0	2	0	1	1	0	0	0	0	1	0	0	3	0	0	0	1
5	V 30	M	UCD 902	42	Bos	C	0	379	16 Oct 90	3	0	0	0	0	0	0	1	3	0	0	0	0	1	0	0	1	0	0	0	0
6	V 62	M	UCD 903	43	Bos	C	ND	300	16 Oct 90	3	0	0	1	0	0	0	1	2	0	0	0	0	.	.	.	.	.	.	.	.
7	V 36	F	UCD 904	44	Bos	C	0	427	17 Oct 90	3	1	1	0	0	0	0	1	3	0	1	0	0	.	.	.	.	.	.	.	.
8	V 52	M	UCD 905	45	Bos	C	ND	409	17 Oct 90	3	3	0	0	0	0	0	0	1	0	0	0	0	3	3	3	1	0	0	0	2
9	V 67	F	UCD 906	46	Bos	C	ND	380	17 Oct 90	3	0	1	0	0	0	0	1	2	0	0	0	0	2	1	3	0	0	0	0	1
10	V 50	M	UCD 907	47	Bos	C	0	321	17 Oct 90	2	1	0	0	0	0	0	0	2	0	1	0	0	2	3	3	0	0	0	1	1
11	V 14	F	UCD 908	48	Bos	C	0	468	17 Oct 90	3	2	0	0	0	0	0	1	2	0	0	0	0	1	3	0	1	1	0	0	1
12	V 10	M	UCD 909	49	Bos	C	0	343	17 Oct 90	3	0	0	0	0	0	0	0	1	0	0	0	0	1	2	3	0	1	0	0	1
13	V 81	M	UCD 910	50	Bos	C	ND	341	17 Oct 90	3	0	0	0	0	0	0	1	2	0	0	0	0	3	3	3	0	0	0	1	0
14	V 53	F	UCD 911	51	Bos	C	ND	471	17 Oct 90	3	0	1	0	0	0	0	1	3	0	1	0	0	2	2	3	1	0	0	1	2
15	V 60	F	UCD 912	52	Bos	C	ND	365	17 Oct 90	3	0	0	0	0	0	0	0	2	1	0	0	0	3	3	3	0	0	0	2	1
16	V 74	F	UCD 913	53	Bos	C	ND	349	17 Oct 90	3	0	0	0	0	0	0	1	1	0	0	0	0	1	3	3	0	0	0	1	2
17	V 107	M	UCD 914	54	Bos	C	ND	348	17 Oct 90	3	0	1	0	0	0	0	1	2	0	1	0	0	2	2	3	0	0	1	0	0
18	V 89	M	UCD 801	1	Rocky	C	ND	449	9 Oct 90	3	3	0	0	0	1	0	1	2	0	0	0	0	1	3	3	0	1	0	1	1
19	V 85	F	UCD 802	2	Rocky	C	ND	270	9 Oct 90	3	0	1	0	0	0	0	1	2	0	0	0	0	.	.	.	.	.	.	.	.
20	V 137	F	UCD 803	3	Rocky	C	ND	257	9 Oct 90	3	0	0	0	0	0	0	0	2	0	1	0	0	.	.	.	.	.	.	.	.
21	V 125	F	UCD 804	4	Rocky	C	ND	330	9 Oct 90	3	0	0	0	0	0	0	0	2	0	0	0	0	2	1	1	0	1	0	0	0
22	V 38	M	UCD 805	5	Rocky	C	0	342	9 Oct 90	3	0	0	0	0	0	0	2	3	0	0	0	0	3	0	1	2	0	0	0	1

#	Proc. Code	Sex	ADF&G AWL #	fish#	Site	OS	MFO	L (mm)	Sample Date	Liver GLY	LIP	MA	LY	SCN	MEG	FIB	Spl MA	Kidney MA	LY	VD	NEC	GBM	NARES LY	NEU	EGL	MAC	GLO	MCH	SEH	SCN
23	V 71	M	UCD 806	6	Rocky	C	ND	330	9 Oct 90	3	1	0	1	0	0	0	0	2	1	0	0	0	2	2	3	0	0	1	0	2
24	V 48	F	UCD 807	7	Rocky	C	0	335	9 Oct 90	3	2	0	0	0	0	0	0	2	0	0	0	0	1	3	3	1	2	0	0	0
25	V 108	F	UCD 808	8	Rocky	C	ND	291	9 Oct 90	3	2	0	1	0	0	0	1	2	0	0	0	0	3	1	2	0	2	0	1	1
26	V 65	M	UCD 809	9	Rocky	C	ND	277	9 Oct 90	3	2	0	0	0	0	0	0	2	0	0	0	0	2	3	3	0	2	0	2	1
27	V 54	M	UCD 810	10	Rocky	C	ND	361	9 Oct 90	3	0	0	0	0	0	0	0	3	0	0	0	0	2	3	3	0	2	0	0	2
28	V 4	F	UCD 811	11	Rocky	C	0	428	9 Oct 90	3	0	1	0	0	0	0	1	2	0	0	0	0	3	1	3	1	2	1	2	1
29	V 56	F	UCD 812	12	Rocky	C	ND	310	9 Oct 90	3	1	0	0	0	0	0	0	1	0	1	0	0	2	3	2	0	1	0	1	2
30	V 114	F	UCD 813	13	Rocky	C	ND	334	9 Oct 90	3	2	0	0	1	0	0	0	2	0	1	0	0	.	.	.	.	.	.	.	.
31	V 78	F	UCD 814	14	Rocky	C	ND	362	9 Oct 90	3	0	0	0	0	0	0	0	2	0	0	0	0	3	3	3	0	0	0	0	1
32	V 88	M	UCD 815	15	Rocky	C	ND	322	9 Oct 90	3	0	1	0	0	0	0	1	3	0	0	0	0	.	.	.	.	.	.	.	.
33	V 69	F	UCD 816	16	Rocky	C	ND	289	9 Oct 90	3	1	1	0	0	0	0	0	2	0	0	0	0	1	2	3	1	0	0	0	1
34	V 28	M	UCD 817	17	Rocky	C	0	240	9 Oct 90	3	0	0	0	0	0	0	1	2	1	0	0	0	2	3	3	1	1	0	0	1
35	V 33	M	UCD 818	18	Rocky	C	3	293	9 Oct 90	3	0	1	0	0	0	0	0	2	0	0	0	0	2	1	1	0	0	0	0	0
36	V 112	M	UCD 819	19	Rocky	C	ND	292	9 Oct 90	2	0	0	0	1	0	0	0	2	1	1	0	0	3	3	1	1	1	1	0	3
37	V 6	M	UCD 820	20	Rocky	C	0	260	9 Oct 90	3	0	0	1	0	1	0	0	2	1	0	0	0	1	3	2	1	2	0	0	1
38	V 61	F	UCD 918	58	Eshamy	O	ND	250	29 Oct 90	3	0	0	0	0	1	0	0	1	1	0	0	0	3	3	3	0	0	0	0	0
39	V 116	F	UCD 919	59	Eshamy	O	ND	276	29 Oct 90	3	0	0	0	0	0	0	1	2	0	0	0	0	2	1	3	1	1	0	0	0
40	V 82	F	UCD 920	60	Eshamy	O	ND	232	29 Oct 90	3	0	1	0	0	0	0	1	2	0	0	0	0	2	1	2	0	0	0	0	0
41	V 106	F	UCD 921	61	Eshamy	O	ND	240	29 Oct 90	3	0	1	0	0	0	0	1	2	0	1	0	0	1	0	1	0	2	0	0	0
42	V 21	F	UCD 922	62	Eshamy	O	0	228	29 Oct 90	3	0	1	0	0	0	0	1	2	0	0	0	0	1	0	1	0	1	0	0	0
43	V 118	F	UCD 923	63	Eshamy	O	ND	215	29 Oct 90	3	1	0	0	0	1	0	0	1	1	0	0	0	2	1	3	1	1	0	1	0
44	V 130	F	UCD 924	64	Eshamy	O	ND	241	29 Oct 90	3	2	0	0	0	0	0	0	1	0	1	0	0	2	2	3	0	0	0	0	0
45	V 9	F	UCD 925	65	Eshamy	O	0	331	30 Oct 90	3	0	0	0	0	0	0	1	1	0	0	0	0	1	0	0	0	0	0	0	0
46	V 11	F	UCD 926	66	Eshamy	O	3	231	30 Oct 90	3	0	0	0	0	0	0	1	1	0	1	0	0	2	1	0	0	0	0	0	0
47	V 55	F	UCD 927	67	Eshamy	O	ND	209	30 Oct 90	3	0	1	1	0	1	0	1	1	0	1	0	0	2	0	1	0	0	0	0	0
48	V 70	F	UCD 928	68	Eshamy	O	ND	253	30 Oct 90	3	2	1	0	0	0	0	1	3	0	1	1	0	2	0	3	0	1	0	0	0
49	V 93	?	UCD 929	69	Eshamy	O	ND	?	30 Oct 90	3	0	1	0	0	0	0	1	2	0	2	0	0	1	3	2	1	1	0	0	0
50	V 111	F	UCD 930	70	Eshamy	O	ND	188	30 Oct 90	1	0	0	0	0	0	0	0	1	0	1	0	0	1	0	2	0	0	0	0	0
51	V 39	F	UCD 931	71	Eshamy	O	0	255	30 Oct 90	3	0	0	0	0	2	0	0	1	0	0	0	0	2	0	1	0	0	0	1	0
52	V 59	M	UCD 932	72	Eshamy	O	ND	211	30 Oct 90	3	0	1	0	0	0	0	1	3	0	0	0	0	2	0	2	0	1	0	0	0
53	V 84	F	UCD 821	21	Green	O	ND	229	10 Oct 90	2	1	0	0	0	0	0	1	2	0	0	0	0	2	0	1	0	0	0	0	0
54	V 66	M	UCD 822	22	Green	O	ND	214	10 Oct 90	3	0	0	0	0	0	0	0	2	0	0	0	0	2	0	1	0	0	0	0	0
55	V 102	F	UCD 823	23	Green	O	ND	220	10 Oct 90	3	0	0	0	0	0	0	0	2	0	1	0	0	3	0	3	0	1	0	1	2
56	V 32	?	UCD 824	24	Green	O	0	187	11 Oct 90	3	0	2	0	0	0	0	0	1	0	1	0	0	2	0	2	1	0	0	0	1
57	V 94	F	UCD 825	25	Green	O	ND	287	11 Oct 90	2	0	0	0	0	0	0	0	2	0	1	0	0	.	.	.	.	.	.	.	.
58	V 46	F	UCD 826	26	Green	O	0	321	11 Oct 90	3	0	1	0	0	0	0	1	3	0	0	0	0	3	1	1	2	0	0	0	0
59	V 51	F	UCD 827	27	Green	O	3	302	11 Oct 90	3	0	0	1	0	0	0	1	2	0	1	0	0	3	3	2	0	1	0	0	0
60	V 24	F	UCD 828	28	Green	O	0	350	11 Oct 90	3	0	1	0	0	0	0	0	3	0	0	0	0	1	3	2	0	0	0	0	2
61	V 20	F	UCD 829	29	Green	O	0	320	11 Oct 90	3	0	0	0	0	0	0	0	2	0	0	0	0	3	1	1	3	2	0	0	0
62	V 121	F	UCD 830	30	Green	O	ND	436	11 Oct 90	2	0	0	0	0	0	0	1	3	0	1	0	0	1	1	2	0	0	0	0	2
63	V 126	F	UCD 831	31	Green	O	ND	241	11 Oct 90	0	0	0	0	0	0	0	0	2	0	1	0	0	2	1	2	0	0	0	1	1
64	V 92	F	UCD 832	32	Green	O	ND	396	11 Oct 90	3	0	0	0	0	1	0	0	2	0	1	0	0	2	0	3	0	2	0	0	1
65	V 25	M	UCD 833	33	Green	O	0	272	12 Oct 90	3	1	0	0	0	1	0	1	2	0	0	0	0	2	2	2	1	0	0	0	0
66	V 79	M	UCD 834	34	Green	O	ND	363	12 Oct 90	3	2	0	0	0	0	0	1	3	0	0	0	0	1	0	2	0	0	0	0	0
67	V 13	F	UCD 835	35	Green	O	0	340	12 Oct 90	3	0	1	0	0	0	0	1	3	0	0	0	0	2	0	3	0	1	0	0	0

#	Proc. Code	Sex	ADF&G AWL #	fish#	Site	OS	MFO	L (mm)	Sample Date	Liver GLY	LIP	MA	LY	SCN	MEG	FIB	Spl MA	Kidney MA	LY	VD	NEC	GBM	NARES LY	NEU	EGL	MAC	GLO	MCH	SEH	SCN
68	V 34	M	UCD 836	36	Green	O	0	219	12 Oct 90	3	1	0	0	0	1	0	1	2	0	1	0	0	2	0	0	1	0	0	0	0
69	V 3	M	UCD 837	37	Green	O	0	236	12 Oct 90	3	0	0	0	0	0	0	0	1	0	0	0	0	3	1	2	1	0	0	1	1
70	V 95	M	UCD 838	38	Green	O	ND	243	12 Oct 90	0	0	0	0	0	0	0	1	2	0	1	0	0	.	.	.	.	.	.	.	.
71	V 117	M	UCD 839	39	Green	O	ND	196	12 Oct 90	3	1	0	0	0	0	0	1	2	0	1	0	0	2	0	2	0	0	1	0	1
72	V 99	M	UCD 840	40	Green	O	ND	240	12 Oct 90	3	1	0	0	0	0	0	0	1	0	2	0	0	1	1	3	0	0	0	1	1

Exposure	n	Site	Length mean	STD	SE	Mean Lesion Scores: Liver GLY	LIP	MA	LY	SCN	MEG	FIB	Spl MA	Kidney MA	LY	VD	NEC	GBM	NARES LY	NEU	EGL	MAC	GLO	MCH	SEH	SCN
reference	3	Makaka	376	7	4																					
reference	14	Boswell Bay	376	49	13	2.9	.5	.29	.07	0	.14	0	.71	1.9	.07	.29	0	0	1.8	2.1	2.3	.58	.17	.08	.5	1
reference	20	Rocky Bay	319	52	12	3.0	.7	.25	.15	.1	.1	0	.4	2.1	.2	.2	0	0	2.1	2.2	2.3	.5	1.1	.19	.44	1.1
oiled	14	Eshamy	240	33	9	2.9	.33	.47	.07	0	.33	0	.67	1.6	.13	.53	.07	0	1.7	.8	1.8	.2	.53	0	13	0
oiled	20	Green Island	281	68	15	2.6	.35	.25	.05	0	.15	0	.5	2.1	0	.6	0	0	2.1	.78	1.9	.5	.39	.06	.22	.67

CHAPTER 3 - Sublethal effects of the *Exxon Valdez* oil spill on herring embryos and larvae: morphological, cytogenetic, and histopathological assessments, 1989–1991.

Citation:

Hose, J.E., M.D. McGurk, G.D. Marty, D.E. Hinton, E.D. Brown, and T.T. Baker. 1996. Sublethal effects of the *Exxon Valdez* oil spill on herring embryos and larvae: morphological, cytogenetic, and histopathological assessments, 1989-1991. Canadian Journal of Fisheries and Aquatic Sciences 53:2355-2365.

Abstract: Following the *Exxon Valdez* oil spill in Prince William Sound, Alaska, in March 1989, Pacific herring *Clupea pallasi* larvae were evaluated for sublethal damage. From 1989 to 1991, egg masses were collected from oiled and unoiled beaches and incubated to hatch. Newly hatched herring larvae were assessed for morphological deformities, cytogenetic abnormalities, and histopathological lesions. In 1989, herring larvae from oiled areas had significantly more morphological deformities and cytogenetic abnormalities than did larvae from the unoiled location. The extent of morphological and cytogenetic damage was correlated with oil exposure in adjacent native bay mussels. Larvae had no oil-related histopathological lesions. In 1990 and 1991, oil-related developmental and genetic effects were undetectable.

CHAPTER 4 - Histopathology and cytogenetic evaluation of Pacific herring larvae exposed to petroleum hydrocarbons in the laboratory or in Prince William Sound, Alaska, after the *Exxon Valdez* oil spill.

Citation:

Marty, G.D., J.E. Hose, M.D. McGurk, E.D. Brown, and D.E. Hinton. 1997. Histopathology and cytogenetic evaluation of Pacific herring larvae exposed to petroleum hydrocarbons in the laboratory or in Prince William Sound, Alaska, after the *Exxon Valdez* oil spill. Canadian Journal of Fisheries and Aquatic Sciences 54:1846-1857.

Abstract: Following the 1989 *Exxon Valdez* oil spill in Prince William Sound, Alaska, USA, Pacific herring *Clupea pallasi* larvae sampled from oiled sites had ascites, pericardial edema, and genotoxic damage. Laboratory study confirmed that these lesions were consistent with oil exposure. Pacific herring larvae were trawled from 2 oiled and 2 unoiled sites in Prince William Sound in May 1989. Larvae from oiled sites were shorter, had ingested less food, and had slower growth (0.07-0.10 mm/d, oiled; 0.15-0.18 mm/d, unoiled). Larvae from oiled sites had higher prevalence of cytogenetic damage (56-84%, oiled; 32-40%, unoiled) and ascites (16%, oiled; 1%, unoiled). In the laboratory experiment, Pacific herring eggs were exposed to an oil-water dispersion of Prudhoe Bay crude oil (initial concentrations of 0.0, 0.10, 0.24, 0.48, and 2.41 mg/L) and sampled for histopathology < 24 h after hatching. Effects were statistically significant at the 0.48 mg/L dose (Dunnett's procedure, $P < 0.05$). Lesions included ascites, hepatocellular vacuolar change, and degeneration or necrosis of skeletal myocytes, retinal cells, and developing brain cells. Lesions in field-sampled larvae were consistent with higher mortality rates documented in larvae from oiled sites.

CHAPTER 5 - Histopathology of adult Pacific herring in Prince William Sound, Alaska, after the *Exxon Valdez* oil spill.

Citation:

Marty, G.D., M.S. Okihiro, E.D. Brown, D. Hanes, and D.E. Hinton. 1999. Histopathology of adult Pacific herring in Prince William Sound, Alaska, after the *Exxon Valdez* oil spill. Canadian Journal of Fisheries and Aquatic Sciences 56:419-526.

Abstract: Pacific herring *Clupea pallasi* sampled from oiled sites in Prince William Sound, Alaska, USA, 3 weeks after the 1989 *Exxon Valdez* oil spill had multifocal hepatic necrosis and significantly increased tissue concentrations of polynuclear aromatic hydrocarbons (PAH). By comparison, Pacific herring from reference sites in 1989, and from all sites in 1990 and 1991 did not have hepatic necrosis or increased PAH concentrations. Adult Pacific herring were sampled for histopathology of liver, spleen, and kidney from oiled and reference sites in April (1989 and 1991) and October (1990 and 1991). Increased scores for macrophage aggregates contributed to significant differences in 1990, but these differences probably resulted from sampling older fish from the oiled site. Naphthalenes were the predominant PAH in all tissue samples. The development of hepatic necrosis and the predominance of naphthalenes in samples from 1989 is consistent with recent laboratory study in which crude oil exposure resulted in dose-dependent expression of viral hemorrhagic septicemia virus (VHSV). We conclude that Pacific herring were exposed to *Exxon Valdez* oil in 1989, and that development of hepatic necrosis in exposed fish probably was a result of VHSV expression.

CHAPTER 6 - Histopathology of adult Pacific herring exposed to crude oil in the laboratory.

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Methods

Fish collection and exposure

Mature adult Pacific herring *Clupea pallasi* were captured near Juneau, Alaska, by purse seine. Fish were bailed from the purse by water-filled buckets and transferred to live tanks on the ship for transport to the laboratory. Fish were held in floating net pens suspended in Auke Bay until ready for use. Salinity in both live tanks and holding pens was between 15 and 25 ppt, which reduced scale loss. Herring were transferred to holding tanks in the laboratory 3 weeks before dosing. Exposures was either via the water column as water-soluble fraction or via force-fed gelatin capsules (per os).

Adult Pacific herring were exposed to water soluble fraction of Alaska North Slope crude oil using an oil generator. The crude oil had similar composition to *Exxon Valdez* crude oil. Herring were exposed to water soluble fraction in 800-L tanks, 20 to 30 fish per tank, at a flow rate of 4L/min/tank. A generator dripped 10L/min seawater through 2000 pipette tips onto a continuously replenishing 40-cm layer of crude oil. The resulting water soluble fraction was collected from below the slick after all oil droplets had floated out. Stable test concentrations were achieved by diluting the water soluble fraction with seawater before delivery to the test tanks. The 2-d LC50 of 1.2 mg/L was confirmed for this stock of adult herring and used to set concentrations for the sublethal exposures. Two tanks had a water soluble fraction of 0.72 mg crude oil/L seawater (=60% of LC50) and 2 other tanks had a water soluble fraction of 0.36 mg crude oil/L seawater (=20% of LC50). Herring were exposed for 0, 1, 2, and 4 d to the higher dose and for 0, 1, 2, 4, 7, and 10 days to the low dose. In addition, some of the fish exposed for 10 d were transferred to clean water for 3 and 7 d depuration. Ten adult fish were sampled at most of the time intervals, giving a total of nearly 100 fish sampled.

For ingestion exposure, Oregon Moist Pellets were soaked in crude oil and placed in gelatin capsules. Low-dose capsules contained one oil-soaked pellet (for 60 fish), and high-dose capsules contained 4 pellets (for 60 fish). The gelatin capsules were force fed to anesthetized prespawn adult herring. By force feeding the capsules, problems with regurgitation of whole oil capsules and poor feeding by herring in the laboratory and were minimized. Ten fish from each group were sampled at 1, 2, 4, 7, 10, and 15 d post-ingestion. As ingestion controls, 10 fish were force-fed gelatin capsules with no oil and sampled 48 h later.

Ovary, gut [viscera ?], and muscle were sampled for hydrocarbon uptake from the first 3 female fish in each sample of 10 fish; tissues were frozen in glass bottles that were certified hydrocarbon free. Bile was drained from gall bladders into amber vials previously baked hydrocarbon clean;

the bile was frozen and sent to Texas A&M for analysis. After removing a slice of the liver for histology, the remaining liver was transferred to hydrocarbon-free scintillation vials and rushed to a supercooled freezer (-80° C) for storage. Mixed function oxidase determinations were made at Auke Bay Laboratory. The remaining fish was dissected and all tissues for histology were placed in glass jars containing 10% neutral buffered formalin. After 3 months, the tissues were transferred to 70% ethanol for shipment to the University of California, Davis.

Histopathology

Two coolers containing 236 jars of herring tissues were received and logged in by Gary D. Marty on 8-19-91. There was miscommunication on how tissues could be shipped, and formalin had been drained from the jars for several hours before isopropyl alcohol was added. Hence, several of the tissues, particularly the spleen, were often too dry for processing for histology. The following tissues, when present, were trimmed in for each fish: liver, kidney, spleen, skin/muscle, gastrointestinal tract, heart, nares, gonad (ovary or testis), and gill. Unfortunately, many of the liver samples were too small or absent; only 190 of 236 herring (81%) had livers analyzed for histopathologic lesions. All fish were assigned a number at random, and tissues were processed routinely in paraffin and stained with hematoxylin and eosin. Tissues were read blindly in ascending numerical order, using the randomly generated numbers. Lesions were semiquantitatively scored as none (0), mild (1), moderate (2), or severe (3). After lesion scores for all organs were finalized, fish were separated by exposure history for determination of significant lesions.

Results and Discussion

Because of numerous problems with study design, tissue artifact, completeness of sampling, and lack of information on the status of viral hemorrhagic septicemia in these fish, these results will not be prepared for publication. Results from a better, more recent study of Pacific herring exposed to crude oil have been published (Carls et al. 1998). The format of this chapter is retained in the original report format: primarily an outline and tabular presentation of findings. Lesion scores for each fish are listed by organ: liver, kidney, spleen, and heart (Table VI-1), esophagus and stomach (Table VI-2), and gill, ovary, testes, nares, and muscle (Table VI-3). This report contains no figures, but figures of many of the major lesions have recently been published elsewhere (Marty et al. 1998). Basic findings in each organ are outlined below:

I. <u>Liver</u>

A. **Normal Histology**: Normal herring liver is composed of scattered large veins (portal and central veins cannot be differentiated histologically), bile ducts, sinusoids, and hepatocytes arranged in tubular fashion.

B. **Megalocytosis**: Megalocytosis was seen in the livers of a some herring experimentally exposed to oil. Affected hepatocytes had varying degrees of karyomegaly and were somewhat similar to those in 1989 and 1990 wild-caught herring. Enlarged nuclei varied from round to oval to irregular, and nucleoli were prominent. The primary difference between the megalocytosis in these fish was that the megalocytes occurred most often around vessels and in lightly staining hepatocytes (i.e., hepatocytes with

increased amounts of pale basophilic cytoplasm). In only a few fish, megalocytes were in dark-staining cells. Multinucleated syncytial giant cells, some with karyomegalic nuclei, were also seen in a few fish.

Comment: Significant amounts of crush/handling artifact in many of the livers examined made interpretation of the degree of megalocytosis very difficult. The crush/handling artifact appeared to have resulted in the appearance of 2 populations of hepatocytes: "light" and "dark" cells. Based on previous experience, the light cells are probably hepatocytes which were altered by handling. The damaged hepatocytes are assumed to swell up and lose some of their normal cytoplasmic basophilia, thereby becoming paler than the surrounding "normal" "dark" cells. In addition, the majority of "light" cells were perivascular and at the periphery of the sections.

Interpretation of these slides was difficult because the majority of megalocytes occurred in "light" cells. Several interpretations are possible. One possibility is that both the megalocytes and the "light" cells were the result of crush/handling artifact. Some of this seems reasonable (i.e., nuclear swelling results in artificial enlargement of hepatocyte nuclei and the appearance of karyomegaly), but artifactual swelling of this magnitude is not something that we have previously encountered. A second possibility is that these livers may have generalized megalocytosis and artifactual creation of "light" cells simply makes it easier to visualize enlarged nuclei. Nuclei in the "dark" cells were often very difficult to visualize because of increased amount of cytoplasmic basophilia.

In the future, tissues destined for histopathologic analysis must be handled carefully. We understand that the livers of each fish had to be divided up for multiple purposes (e.g., biochemical analyses, P450 enzyme determination, and histopathology), but better results might have been possible if the sections for histopathology were taken first.

C. **Sinusoidal fibrosis**: none

D. **Necrosis**:

1. Coagulative necrosis: The most striking lesion in livers of experimentally exposed herring was mild to severe, multifocal, coagulation necrosis. Necrotic foci were usually randomly distributed and characterized by rounding up of hepatocytes, hypereosinophilia, loss of nuclear profiles, and fragmentation. In a few fish, necrosis was distinctly perivascular.

2. Single cell necrosis: Many livers had single cell necrosis characterized by shrinkage, granular cytoplasm, and nuclear pyknosis of individual hepatocytes.

Comment: Necrosis was most common in the high-dose exposure group at 7 days. In that group, 50% (5 of 10 fish) had moderate to severe focal necrosis, and 90% (9 of 10 fish) had at least mild single cell necrosis. This compares with a 0% (0 of 7) incidence of focal necrosis and a 14.7% (1 of 7) incidence of single cell necrosis at day 1. None of the control or P-control fish had either moderate or severe necrosis, and only 11.7%

(2 of 17) had mild single cell necrosis. Recent study indicates that hepatocellular necrosis is a result of expression of viral hemorrhagic septicemia virus, and expression of the virus may result from oil-induced stress and immunosuppression (Carls et al. 1998). Because viral hemorrhagic septicemia virus was not isolated from Pacific herring until 1993 (Meyers et al. 1994), there was no attempt to isolate the virus from the fish in our experiment (which occurred in 1991).

E. **Inflammation**

1 Macrophage aggregates: common

2. Lymphocytic aggregates: usually only a few

3. Granulomas: Granulomas were primarily associated with *Ichthyophonus hoferi* infection

4. Eosinophilic granular leukocytes: EGL's were associated with granulomas or perivascular connective tissue

F. **Hepatocyte storage disorders**

1. Glycogen depletion: common

2. Lipidosis: usually mild

G. **Bile duct hyperplasia**: not observed

H. **Parasitism**

1. *Ichthyophonus hoferi*: occasionally seen

2. *Goussia clupearum*: occasionally seen.

II. **Kidney**

A. **Normal Histology**: Normal kidney is composed of glomeruli, tubules, intertubular hematopoietic tissue, and perivenular endocrine tissue.

B. **Necrosis**:

1. **Interstitial Necrosis**: Some kidneys had unique necrotizing lesions which had not previously been seen. In contrast to single cell necrosis, which was occasionally observed in tubular epithelium of some of the wild-caught herring, the necrotizing lesions in these fish were centered in the renal interstitium or hematopoietic tissue. The earliest (most acute) lesions were characterized by fragmentation of interstitial cells (probably a combination of hematopoietic and inflammatory cells) and infiltration by variable amounts of acellular, pale, eosinophilic, fibrillar material (probably fibrin). In more advanced (subacute) lesions, there was infiltration and peripheral localization by macrophages along with organization by

fibroblasts in some lesions. In some fish, interstitial necrosis was accompanied by varying degrees of hemorrhage, and interstitial blood vessels occasionally contained fibrin thrombi.

2. **Tubular Necrosis**: A few fish had multifocal to diffuse coagulation necrosis of renal tubules. The necrosis involved entire tubules and was characterized by complete dissolution of tubular epithelium and replacement by casts of granular, pale, eosinophilic debris.

Comment: The necrotizing lesions in the renal interstitium are believed to be related to vascular damage to interstitial capillaries and venules. The assumed pathogenesis is: 1) circulating xenobiotics or infective virus damages small interstitial blood vessels; 2) damaged vessels allow fibrin and protein to leak into the interstitium; 3) local hematopoietic cells die and undergo necrosis as they are flooded by fibrin and protein, and are separated from the blood vessels; 4) attraction of neutrophils, which add their enzymes to the necrotizing process; 5) infiltration by macrophages for cleanup (phagocytosis); and 6) organization by fibroblasts. Severe vascular damage, with major disruption of vascular walls, probably accounts for the hemorrhagic lesions in some fish. Macrophage aggregates might be a residual lesion to interstitial necrosis.

The necrotizing lesions involving tubules are believed to be the result of infarction. This hypothesis is based on the presence of thrombi in some kidneys and the focal severity of the tubular necrosis. Carls et al. (1998) reported fibrin thrombi in spleen, liver, and gill, but kidney was not examined.

B. **Glomeruli**

1. Glomerular Basement Membrane thickening: Some fish had mild to moderate thickening of glomerular basement membrane.

2. Glomerular Thrombi: Many fish had fibrin thrombi trapped within glomerular capillaries. In some fish, thrombi were very common with the majority of glomeruli involved. A few fish had markedly enlarged and severely dysplastic glomeruli with large deposits of fibrin completely filling capillary lumens.

3. Glomerular Fibrosis: A few fish had unusual foci of interstitial scarring which might have been fibrotic glomeruli. These foci were roughly spherical and composed of irregular, papillary strands of collagen mixed with fibroblasts. The papillary strands of connective tissue were separated by what appeared to be remnants endothelial-lined capillaries with little or no blood.

Comment: The thrombi in glomerular capillaries are consistent with the vascular lesions in other parts of the kidney and in other organs. Fish with glomerular thrombi almost invariably also had interstitial necrosis. The proposed pathogenesis of glomerular lesions is: 1) generalized disseminated intravascular coagulation (DIC); 2) trapping of fibrin thrombi in glomerular capillaries; 3) complete filling of glomerular

capillaries with fibrin with resultant enlargement and distortion of architecture; and 4) organization of the lesion by macrophages and fibroblasts, terminating in glomerular fibrosis.

C. **Renal tubular vacuolar degeneration**: rare

D. **Inflammation**
   1. Macrophage aggregates: common

   2. Lymphoid aggregates: rare

E. **Parasitism**: Small to large numbers of the myxosporean *Ortholinea orientalis* were in the distal tubules and collecting ducts of some fish.

III. **Spleen**

A. **Normal Histology**: Normal Pacific herring spleen is composed of a mixture of hematopoietic and lymphoid tissue. Splenic arterioles are usually small and unapparent.

B. **Inflammation**
   1. Macrophage aggregates: common

   2. Lymphoid aggregates: rare

C. **Splenic arterioles**
   1. Vasculitis: In many fish, generalized inflammation was centered over splenic arterioles. Inflamed arterioles had irregular mural thickening with acellular, pale, eosinophilic material mixed with small amounts of karyorrhectic debris. In some arteriolar walls, the infiltrating material was refractile and brightly eosinophilic. Vascular lesions were occasionally associated with the fibrin leakage into the splenic parenchyma. Some fish also had fibrin or fibrinocellular thrombi within splenic vessels of all sizes.

   2. Mural thickening: In some fish, the walls of ensheathed capillaries were markedly thickened by pale, eosinophilic, acellular material similar to that in fish with vasculitis. These fish, however, had minimal or no inflammation and leakage of fibrin. In some fish, distorted splenic arterioles were clustered close together, with apparent collapse and loss of intervening hematopoietic and lymphoid tissue.

Comment: The vascular lesions in the spleen were often difficult to evaluate because of varying degrees of congestion, along with poor fixation and artifactual distortion in some fish. In addition, there is still considerable confusion over the vascular lesions involving the splenic arterioles. Although there did appear to be definitive vasculitis and thrombosis in some spleens, the relationship between the inflammatory lesions and the mural thickening is unclear. Inflammatory lesions in the arterioles may precede mural

thickening, but there is no clear cut evidence for this. The exact composition of the pale eosinophilic material in the arteriolar walls is also unknown.

The splenic vascular lesions seem to be strongly correlated with the vascular lesions in the kidney and it is likely that the pathogenesis is similar. Spleens were scored in only 3 categories (macrophage aggregates, congestion, and vasculitis). Re-evaluation with the use of 2 additional parameters (thrombi and arteriolar wall thickening) may help to better define the difference between the exposed and control groups. Carls et al. (1998) found that splenic thrombosis was significantly related to both viral hemorrhagic septicemia virus and crude oil exposure.

D. **Congestion**: Many fish had varying degrees of splenic congestion. The congestion was often irregular with blebbing and ballooning of the subcapsular space by large pools of blood. In some fish, the congestion was severe and in some areas appeared to be hemorrhagic.

IV. Gastrointestinal Tract

A. Esophagus

1. **Normal Histology**: The esophagus is the second segment of the gastrointestinal tract and is located between the pharynx and glandular stomach. The pharynx in fish has pharyngeal teeth. The esophagus has thick villi, lined by simple columnar epithelium, and crypts or glands which are lined by a simple layer of mucous cells. The lamina propria has large amounts of dense fibrous connective tissue and the tunic muscularis in both the pharynx and esophagus is composed of skeletal muscle.

2. **Necrosis**: Necrosis of skeletal muscle in the tunic muscularis of the esophagus was a common finding and was often severe.

   Comment: The necrosis in the tunica muscularis of the esophagus may have been artifactual and due to excessive handling or clamping of the organ prior to removal from the fish.

B. Stomach

1. **Normal Histology**: The stomach of adult herring is divided into 2 portions; a glandular section and a nonglandular section. The glandular stomach has gastric glands in the lamina propria, whereas the nonglandular stomach is devoid of glands.

2. **Hemorrhage**: Some fish had small to large foci of hemorrhage in the lamina propria or tunica muscularis.

3. **Necrosis**:
   a. Focal necrosis:

(1) Lamina propria: A few fish had large foci of coagulation necrosis in the lamina propria. The necrotic foci were characterized by pallor, hemorrhage, loss of cellular architecture, and loss of glands.

(2) Tunica muscularis: Some fish also had necrotic foci in the tunica muscularis of the glandular and nonglandular stomach. Necrotic foci were in sections of the tunica muscularis where it was composed of smooth muscle and in sections where it was composed of skeletal muscle.

b. Single cell necrosis: Scattered individually necrotic epithelial cells were common in the mucosa. In some fish, the necrosis was concentrated in the basal cell layer.

4. **Fibrosis**: Fibrosis of the superficial aspect of the gastric lamina propria was common. In some fish, fibrosis was diffuse and severe, with complete scarring of the lamina propria. A few fish also had fibrosis involving the tunica muscularis.

5. **Thrombosis**: A few small thrombi were in mesenteric veins attached to the serosa of the stomach.

6. **Mucosal atrophy**: A few fish had marked atrophy of either the superficial mucosal epithelium or the glandular mucosa.

7. **Squamous metaplasia**: One fish had focal squamous metaplasia of the lamina epithelialis of the stomach.

8. **Parasites**: A few fish had trematodes in the gastric lumen or *Ichthyophonus hoferi* in the gastric wall.

C. Intestine: Sections of intestine were examined from some fish. The majority of intestinal samples were severely distorted from a combination of autolysis and (assumed) rough handling. Fibrin thrombi were seen in veins in the lamina propria of a few fish.

V. **Nares**

A. **Normal Histology**:

1. Olfactory lamellae: The nares are composed of rosettes of olfactory lamellae which are lined by stratified layers of sensory epithelium. The sensory epithelium is composed of a mixture of bipolar neurons, sustentacular (support) cells, non-sensory ciliated epithelial cells, basal cells, rodlet cells, mucous cells, and inflammatory cells (lymphocytes and EGLs). The composition of the sensory epithelium varies with location. The lamellar tips are lined by squamous epithelium, and the adjacent lamina propria is infiltrated by small to moderate numbers of lymphocytes. The lateral aspects of the lamellae are lined by primarily

ciliated columnar epithelium, interspersed with scattered clumps ("buds") of squamous mucosa. There appear to be an increased number of mucous cells along the peripheral edges of the lamellae when compared to the center of the lamellae. The lamina propria often contains scattered mononuclear inflammatory cells and EGLs.

2. Olfactory nerves: The olfactory nerves extend from the base of the lamellae all the way up to the lamellar tips. The nerves appear to be unmyelinated within the lamellae and at the base, but then become myelinated when exiting the immediate area.

B. **Necrosis**: Single cell necrosis in the sensory epithelium of the olfactory lamellae was very mild.

C. **Inflammation**: Mild mononuclear inflammation (primarily lymphocytic) was in the lamina propria at the base of lamellae. In many fish, small numbers of EGLs were also mixed with the mononuclear inflammatory cells. A few fish also had small numbers of macrophage aggregates in the lamina propria. In most fish, the inflammatory cells were centered around the olfactory nerves.

D. **Vascular Lesions**: Some fish had mild to moderate hemorrhage in the lamina propria of the lamellae and at the base, adjacent to olfactory nerves. Several fish also had thrombi within olfactory veins at the base of the lamellae. In some, the thrombosis was associated with mild to moderate vasculitis.

Comment: The vascular lesions in the nares were similar to those present in other internal organs (spleen, kidney, stomach) and were consistent with DIC.

E. **Mucous cell hyperplasia**: Some fish had mild to moderate hyperplasia of mucous cells along the lateral aspects of olfactory lamellae.

F. **Parasitism**: In many fish, the olfactory lamellae were infected with an unidentified parasite. The parasite was primarily found within the sensory epithelium near the lamellar tips and appeared to be intracellular. The parasites ranged from 30 to 60 μm in diameter and were roughly spherical with 2 somewhat distinct morphologic appearances. In some fish, the parasites were primarily composed of an amorphous, mucinous cytoplasm with small, single or double "nuclear bodies". In other fish, the parasites were mostly composed of a large, deeply basophilic, finely granular "nuclear body" with a peripheral rim of lighter staining mucinous material (when the peripheral rim of mucinous material was almost non-existent, these forms resembled the rickettsial parasite *Epitheliocystis*. Some sections had parasitic forms intermediate in appearance.

G. **Protein droplets**: A few fish had large numbers of small, eosinophilic, intracytoplasmic droplets within the sensory epithelium. One fish had large, distinct droplets. The eosinophilic droplets were assumed to be protein.

VI. Gonads

A. Ovaries

1. Normal morphology: The vast majority of ovaries had a high percentage of mature, yolked eggs and did not have any significant lesions.

2. postspawning ovaries: A few ovaries had only a small percentage of yolked eggs, most of which were atretic. These ovaries also had numerous postovulatory follicles.

B. Testes

1. Normal morphology: Normal testes had seminiferous tubules packed with mature sperm/spermatids, and had wide separation of intervening stromal connective tissue.

2. Sperm depletion: A few male fish had testes with moderate depletion of sperm which was characterized by small seminiferous tubules and prominent stromal connective tissue.

3. Parasitism: Parasite vacuoles associated with the coccidian *Eimeria sardinae* were common. Vacuoles were consistently 40 to 60 μm in diameter, and were usually within seminiferous tubules packed with spermatids. All vacuoles had large, clear, peripheral clear zones, but internal/central structure varied widely. While some vacuoles were completely empty, the majority contained oocysts or their fragments. In some, the vacuoles were centered around a single round cell (10-15 μm), while others had multiple (3-5) spindle-shaped oocysts with distinct oval nuclei. A few vacuoles contained sporonts characterized by an indistinct, eccentric, basophilic nucleus, and a mucinous cytoplasm with irregular fibrillar strands.

4. Necrosis: One fish had multifocal coagulation necrosis in the testes.

5. Vacuolar degeneration: Many fish had, what appeared to be, mild to moderate vacuolar degeneration of the ductular epithelium in the testes. Some ducts also had scattered individually necrotic cells in the lining epithelium.

   Comment: Alternatively, this "lesion" might be either normal for testes or an artifact of processing.

6. Syncytial giant cells: Some testes had syncytial giant cells within seminiferous tubules. The giant cells appeared to be composed of squamous epithelial cells with up to 20 individual nuclei.

7. Macrophage aggregates: A few testes had small numbers of macrophage aggregates.

VII. **Gills**

A. **Hyperplasia**

1. Squamous cell hyperplasia: rare
2. Mucous cell hyperplasia: none
3. Chloride cell hyperplasia: rare (in 2 fish, both from the 60-4 exposure group and sampled on day 4)

B. **Inflammation**

1. Lymphocytes: rare
2. EGLs: moderate to large numbers of EGLs were often in the connective tissue adjacent to the large vascular sinus centered in the transverse septum bridging the paired rows of gill filaments.
3. Macrophage aggregates: A few fish had small numbers of macrophage aggregates.

C. **Vascular lesions**

1. Lamellar capillary aneurysms: A few fish had small numbers of aneurysms along the tips of gill lamellae.
2. Hemorrhage: Some fish had acute hemorrhages in the connective tissue at the base of gill filaments or in the arch.
3. Thromboses: rare
4. Vasculitis: rare

D. **Parasites**

1. *Ichthyophonus*: rare
2. *Epitheliocystis*-like organism: Parasites, similar to those described in the nares (see p. VI-9 above), were also present in the gills in a few fish.
3. Unidentified micro/myxosporidian parasite: rare

VIII. **Skeletal Muscle**

A. **Hemorrhage**: some fish had acute hemorrhage.
B. **Necrosis**: some fish had focal to multifocal coagulation necrosis.
C. *Ichthyophonus*: uncommon

IX. **Heart**: Heart was included with a few fish, but in general had no significant lesions.

Statistical Analysis

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For general details about the types of statistical analysis used, see the general "Statistical Analysis" section on page vii.

This laboratory study had several differences from field studies on effects of the spill. First, Pacific herring were either exposed to oil or known to have been free of oil at all times. Hence, trying to document significant differences between oiled and control fish was only of moderate interest. And second, several additional organs were sampled here (e.g., heart, gastrointestinal tract, skin, and gonad) that were not sampled in most of the other studies. For consistency, though, the same basic analysis was used here as in other fish groups. Heart, esophagus, and nonglandular stomach were not used in the analysis because only a few fish had scores for these organs. Because ovary and testes were scored separately, and principal components analysis cannot handle missing values, scores for ovary and testes were not included in the analysis. Organs for analysis included liver, spleen, kidney, glandular stomach, gill, nares, and muscle. With these organs, because of missing values, only 70 of 240 (29%) of the herring were used in the analysis.

Exposure differences were significant for the first and fourth principal components, but were not significantly different for the second and third principal components (ANOVA). For the first principal component, splenic vasculitis, renal necrosis, and renal glomerular thrombi were most important lesions. For the fourth principal component, the most important lesions were necrosis and hemorrhage in the muscle, plus lamellar capillary aneurysms in the gill. The most important in the second principal component–macrophage aggregates in liver, kidney, and spleen–also consistently occurred together in analysis of wild-caught herring. For the third component, the most important lesions were necrosis in the glandular stomach and thrombosis in the glandular stomach and nares.

For the first principal component, the High dietary exposure was significantly different from the 20-4, 20-4D, and control exposures; in addition, the 20-4 exposure was significantly different from the low-diet and P-control groups. For the fourth principal component, the P-control group was significantly different from all but the control group. Overall exposure effects were also significant (MANOVA).

Final comment on histopathologic lesions

The necrotizing lesions in the liver were consistent with expression of viral hemorrhagic septicemia virus and were fairly similar to lesions observed in wild herring captured in 1989 following the oil spill (Marty et al. 1999). Note that thrombosis is relatively common in fish with viral hemorrhagic septicemia following oil exposure (this report and Carls et al. 1998), whereas thrombosis is not a prominent lesion in fish developing viral hemorrhagic septicemia without acute oil exposure (Marty et al. 1998).

Table VI-1. Histopathology of liver, kidney, spleen, and heart of Pacific herring adults exposed to crude oil in the Laboratory.

Key to table symbols:
Hinton number = random (processing) number generated by Dr. Hinton's Laboratory
DAY = number of days after initiation of exposure
Lesion scores: none (0), mild (1), moderate (2), severe (3), not present (.)

EXP. = level of exposure in the laboratory
LOW = 1 oil-soaked pellet force-fed/fish
HIGH = 4 oil-soaked pellets force-fed/fish
P control = clean gelatin capsule force-fed
20-4 = 20% LC50 in water soluble fraction (WSF)
20-4D = 20% LC50 in WSF, with depuration
60-4 = 60% LC50 in water soluble fraction
control = clean water

LIVER:
glycogen depletion (GLY)
lipidosis (LIP)
macrophage aggregates (MA)
single cell necrosis (SCN)
focal necrosis (FN)
hepatocellular karyomegaly (MEG)
coccidian parasites (COC)
- *Goussia clupearum*

SPLEEN:
macrophage aggregates (MA)
congestion (CON)
vasculitis (VAS)

KIDNEY:
macrophage aggregates (MA)
lymphocytes (LY)
tubular epithelial vacuolar degen. (VD)
interstitial necrosis (NEC)
glomerular b. mem. thickening (GBM)
glomerular thrombi (GT)
myxosporeans (MYX)

HEART:
macrophage aggregates (MA)
lymphocytes (LY)
necrosis (NEC)

	HINTON	SAMPLE			ALASKA	Liver							HINTON	Spleen			Kidney							Heart		
#	NUMBER	DATE	DAY	EXP.	ID #	GLY	LIP	MAC	SCN	FN	MEG	COC	#	MA	CON	VAS	MA	LY	VD	NEC	GBM	GT	MYX	MA	LY	NEC
1	91HKE 14	4-23-91	1	LOW	1092	3	1	1	1	1	0	0	HKE 14	1	2	3	1	0	0	1	0	0	0	.	.	.
2	91HKE 21	4-23-91	1	LOW	1068	3	0	0	3	2	0	1	HKE 21	0	2	0	1	0	0	3	0	3	0	.	.	.
3	91HKE 44	4-23-91	1	LOW	1096	3	2	1	1	0	0	1	HKE 44	2	3	2	1	0	0	1	0	3	0	.	.	.
4	91HKE 97	4-23-91	1	LOW	1076	3	2	2	0	0	0	0	HKE 97	3	2	0	1	0	0	0	0	0	0	.	.	.
5	91HKE 107	4-23-91	1	LOW	1072	.	.	.	.	.	.	.	HKE 107	.	.	.	0	0	0	0	0	0	0	0	0	0
6	91HKE 118	4-23-91	1	LOW	1054	3	0	1	1	0	0	1	HKE 118	1	2	2	1	0	0	1	0	0	0	.	.	.
7	91HKE 144	4-23-91	1	LOW	1088	.	.	.	.	.	.	.	HKE 144	1	1	0	3	0	0	0	0	0	0	0	0	0
8	91HKE 159	4-23-91	1	LOW	1061	3	0	3	0	0	0	1	HKE 159	3	0	3	3	0	0	2	0	3	0	.	.	.
9	91HKE 197	4-23-91	1	LOW	1080	.	.	.	.	.	.	.	HKE 197	2	2	1	2	0	0	1	0	0	0	0	0	0
10	91HKE 225	4-23-91	1	LOW	1084	3	0	1	0	0	0	1	HKE 225	.	.	.	.	.	.	.	.	.	.	.	.	.
11	91HKE 230	5-7-91	1	LOW	1061	3	0	0	0	0	1	0	HKE 230	0	3	0	1	0	0	0	0	0	0	0	0	0
12	91HKE 37	4-24-91	2	LOW	1191	.	.	.	.	.	.	.	HKE 37	3	0	0	1	0	0	0	0	0	0	.	.	.
13	91HKE 64	4-24-91	2	LOW	1160	3	1	0	1	1	0	1	HKE 64	.	.	.	1	0	0	1	0	2	0	.	.	.
14	91HKE 75	4-24-91	2	LOW	1175	3	0	1	0	0	0	0	HKE 75	1	0	3	1	0	0	1	0	0	2	.	.	.
15	91HKE 101	4-24-91	2	LOW	1179	3	1	1	1	0	0	1	HKE 101	.	.	.	2	0	0	2	0	0	0	.	.	.
16	91HKE 102	4-24-91	2	LOW	1171	3	1	0	1	0	0	0	HKE 102	1	0	0	1	0	0	0	0	0	0	.	.	.
17	91HKE 114	4-24-91	2	LOW	1195	3	0	1	2	1	0	3	HKE 114	0	1	3	0	0	0	2	0	3	0	.	.	.
18	91HKE 156	4-24-91	2	LOW	1167	3	1	1	1	0	0	1	HKE 156	2	0	1	2	0	0	1	0	0	0	.	.	.
19	91HKE 178	4-24-91	2	LOW	1153	.	.	.	.	.	.	.	HKE 178	3	3	3	3	0	0	2	0	0	0	.	.	.
20	91HKE 195	4-24-91	2	LOW	1187	3	2	1	1	0	0	1	HKE 195	1	0	2	1	0	0	1	0	0	0	.	.	.
21	91HKE 215	4-24-91	2	LOW	1183	3	0	3	0	0	0	2	HKE 215	1	0	1	2	0	0	2	0	0	0	.	.	.
22	91HKE 8	4-26-91	4	LOW	1379	3	0	1	0	0	0	1	HKE 8	1	0	0	1	0	0	0	0	0	0	.	.	.
23	91HKE 18	4-26-91	4	LOW	1367	3	1	1	1	1	1	3	HKE 18	2	1	2	1	0	0	1	0	0	1	.	.	.

#	HINTON NUMBER	SAMPLE DATE	DAY	EXP	ALASKA ID #	Liver							HINTON #	Spleen			Kidney							Heart		
						GLY	LIP	MAC	SCN	EN	MEG	COC		MA	CON	VAS	MA	LY	VD	NEC	GBM	GT	MYX	MA	LY	NEC
24	91HKE 33	4-26-91	4	LOW	1371	3	2	0	1	0	0	3	HKE 33	.	.	.	1	0	0	2	0	0	0	.	.	.
25	91HKE 80	4-26-91	4	LOW	1387	3	1	0	2	1	0	2	HKE 80	0	0	3	1	0	0	3	0	3	0	.	.	.
26	91HKE 137	4-26-91	4	LOW	1353	3	0	1	0	1	0	1	HKE 137	0	3	3	2	0	0	3	0	0	0	.	.	.
27	91HKE 139	4-26-91	4	LOW	1395	3	0	2	3	1	0	0	HKE 139	2	0	3	.	.	.	.	.	.	.	.	.	.
28	91HKE 148	4-26-91	4	LOW	1375	.	.	.	.	.	.	.	HKE 148	0	0	2	1	0	0	2	0	0	0	.	.	.
29	91HKE 198	4-26-91	4	LOW	1391	3	1	0	1	0	0	3	HKE 198	0	0	3	0	0	0	2	0	1	0	.	.	.
30	91HKE 210	4-26-91	4	LOW	1360	3	1	1	1	2	0	3	HKE 210	1	0	3	2	0	0	1	0	0	3	.	.	.
31	91HKE 219	4-26-91	4	LOW	1383	3	0	0	3	3	0	0	HKE 219	0	0	3	0	0	0	3	0	3	0	0	0	1
32	91HKE 28	4-29-91	7	LOW	1475	3	0	1	1	0	0	1	HKE 28	1	1	1	1	0	0	1	0	0	0	.	.	.
33	91HKE 36	4-29-91	7	LOW	1460	3	0	1	0	0	1	1	HKE 36	.	.	.	2	0	0	2	0	0	0	.	.	.
34	91HKE 49	4-29-91	7	LOW	1453	3	0	1	1	0	0	3	HKE 49	3	2	1	2	0	0	1	1	0	3	.	.	.
35	91HKE 73	4-29-91	7	LOW	1487	.	.	.	.	.	.	.	HKE 73	1	0	1	1	0	0	2	1	1	0	.	.	.
36	91HKE 84	4-29-91	7	LOW	1471	3	0	2	0	0	0	0	HKE 84	.	.	.	1	0	0	0	0	0	0	.	.	.
37	91HKE 141	4-29-91	7	LOW	1495	3	0	2	3	2	0	0	HKE 141	3	1	0	2	0	0	3	0	3	0	.	.	.
38	91HKE 179	4-29-91	7	LOW	1483	3	0	2	0	0	0	2	HKE 179	1	3	0	2	0	0	0	0	0	0	.	.	.
39	91HKE 185	4-29-91	7	LOW	1479	3	1	1	0	0	0	0	HKE 185	.	.	.	3	0	0	2	0	0	0	.	.	.
40	91HKE 227	4-29-91	7	LOW	1467	3	0	1	1	0	0	1	HKE 227	1	1	1	1	0	0	3	0	0	3	.	.	.
41	91HKE 237	4-29-91	7	LOW	1491	3	0	0	0	0	0	0	HKE 237	0	0	2	0	0	0	3	0	1	0	.	.	.
42	91HKE 3	5-2-91	10	LOW	1591	3	1	1	2	1	0	2	HKE 3	2	2	0	1	0	0	1	0	0	0	.	.	.
43	91HKE 19	5-2-91	10	LOW	1579	3	2	1	1	0	0	2	HKE 19	.	.	.	1	0	0	0	0	0	0	.	.	.
44	91HKE 22	5-2-91	10	LOW	1575	3	0	1	1	0	1	0	HKE 22	1	1	1	1	0	0	0	0	0	3	.	.	.
45	91HKE 29	5-2-91	10	LOW	1553	3	0	0	1	0	0	1	HKE 29	1	1	0	1	0	0	1	0	0	0	.	.	.
46	91HKE 62	5-2-91	10	LOW	1571	3	0	1	0	0	0	1	HKE 62	1	3	0	2	0	0	0	0	0	0	.	.	.
47	91HKE 140	5-2-91	10	LOW	1595	3	0	1	1	0	0	0	HKE 140	2	1	2	2	0	0	1	0	0	3	.	.	.
48	91HKE 155	5-2-91	10	LOW	1567	3	0	0	0	0	0	1	HKE 155	0	0	2	0	0	0	2	0	2	0	0	0	1
49	91HKE 199	5-2-91	10	LOW	1560	3	1	1	1	0	1	1	HKE 199	1	2	2	1	0	0	1	1	0	0	0	0	0
50	91HKE 205	5-2-91	10	LOW	1583	3	0	1	0	0	0	1	HKE 205	1	0	3	1	0	0	0	0	0	0	.	.	.
51	91HKE 216	5-2-91	10	LOW	1587	.	.	.	.	.	.	.	HKE 216	2	0	0	2	0	0	1	0	0	0	.	.	.
52	91HKE 10	5-6-91	14	LOW	1653	3	0	2	3	2	0	1	HKE 10	2	1	2	2	0	0	1	0	0	0	0	0	0
53	91HKE 15	5-6-91	14	LOW	1660	3	1	1	1	0	0	0	HKE 15	1	1	0	1	0	0	0	0	0	0	.	.	.
54	91HKE 34	5-6-91	14	LOW	1691	3	0	1	0	0	0	0	HKE 34	1	2	0	1	0	0	0	0	0	0	.	.	.
55	91HKE 78	5-6-91	14	LOW	1675	3	0	2	1	0	0	0	HKE 78	2	2	2	2	0	0	1	0	0	0	.	.	.
56	91HKE 86	5-6-91	14	LOW	1679	3	0	1	0	0	0	1	HKE 86	0	1	3	0	0	0	1	1	0	0	.	.	.
57	91HKE 108	5-6-91	14	LOW	1671	3	0	1	0	0	0	3	HKE 108	1	2	0	2	0	0	0	0	0	0	.	.	.
58	91HKE 109	5-6-91	14	LOW	1683	3	0	1	0	0	0	1	HKE 109	1	3	0	1	0	0	0	0	0	0	.	.	.
59	91HKE 173	5-6-91	14	LOW	1687	3	1	1	1	0	1	0	HKE 173	1	1	2	1	0	0	1	0	0	0	.	.	.
60	91HKE 176	5-6-91	14	LOW	1667	3	1	0	1	0	0	0	HKE 176	1	1	2	0	0	0	0	0	0	0	.	.	.
61	91HKE 31	4-23-91	1	HIGH	1027	.	.	.	.	.	.	.	HKE 31	.	.	.	0	0	0	0	0	0	0	0	0	0
62	91HKE 45	4-23-91	1	HIGH	1019	3	2	1	0	0	0	0	HKE 45	1	1	1	1	0	0	0	0	1	2	.	.	.
63	91HKE 61	4-23-91	1	HIGH	1035	.	.	.	.	.	.	.	HKE 61	.	.	.	.	.	.	.	.	.	.	.	.	.
64	91HKE 67	4-23-91	1	HIGH	1039	3	0	1	1	0	0	0	HKE 67	.	.	.	0	0	0	2	0	1	0	.	.	.
65	91HKE 81	4-23-91	1	HIGH	1005	3	0	2	0	0	0	0	HKE 81	1	3	0	1	0	0	0	1	0	0	.	.	.
66	91HKE 130	4-23-91	1	HIGH	1043	3	0	2	0	0	0	0	HKE 130	.	.	.	.	.	.	.	.	.	.	.	.	.
67	91HKE 162	4-23-91	1	HIGH	1047	3	0	0	0	0	1	0	HKE 162	0	0	1	0	0	0	0	0	1	0	.	.	.
68	91HKE 165	4-23-91	1	HIGH	1031	3	0	1	0	0	2	1	HKE 165	0	0	2	0	0	0	1	0	0	0	.	.	.

#	HINTON NUMBER	SAMPLE DATE	DAY	EXP	ALASKA ID #	Liver							HINTON #	Spleen			Kidney							Heart		
						GLY	LIP	MAC	SCN	EN	MEG	COC		MA	CON	VAS	MA	LY	VD	NEC	GBM	GT	MYX	MA	LY	NEC
69	91HKE 171	4-23-91	1	HIGH	1023	.	.	.	.	.	.	.	HKE 171	.	.	.	2	0	0	0	0	0	0	.	.	.
70	91HKE 174	4-23-91	1	HIGH	1012	3	1	1	0	0	0	1	HKE 174	0	2	3	1	0	0	2	0	1	0	.	.	.
71	91HKE 47	4-24-91	2	HIGH	1104	3	0	2	3	2	0	0	HKE 47	1	3	0	1	0	0	1	1	0	0	.	.	.
72	91HKE 57	4-24-91	2	HIGH	1126	3	1	1	0	0	0	1	HKE 57	.	.	.	1	0	0	2	0	1	1	.	.	.
73	91HKE 59	4-24-91	2	HIGH	1118	3	0	2	1	0	0	1	HKE 59	2	0	2	2	0	0	2	0	0	0	.	.	.
74	91HKE 66	4-24-91	2	HIGH	1130	3	2	0	2	2	0	1	HKE 66	0	1	2	0	0	0	3	0	3	0	0	0	0
75	91HKE 70	4-24-91	2	HIGH	1146	3	1	2	3	1	0	0	HKE 70	0	1	3	2	0	0	3	0	2	0	.	.	.
76	91HKE 85	4-24-91	2	HIGH	1122	3	2	1	1	0	0	0	HKE 85	1	0	2	1	0	0	1	0	0	0	.	.	.
77	91HKE 126	4-24-91	2	HIGH	1142	3	1	1	0	0	1	1	HKE 126	0	1	2	1	0	0	2	0	0	0	.	.	.
78	91HKE 160	4-24-91	2	HIGH	1138	3	1	1	1	0	0	1	HKE 160	1	1	2	1	0	0	2	0	1	0	.	.	.
79	91HKE 203	4-24-91	2	HIGH	1134	3	0	3	0	0	0	2	HKE 203	3	0	2	3	0	0	2	0	0	0	.	.	.
80	91HKE 228	4-24-91	2	HIGH	1111	3	0	1	1	0	0	0	HKE 228	0	0	3	1	0	0	3	0	3	0	.	.	.
81	91HKE 20	4-26-91	4	HIGH	1322	3	0	2	0	1	0	0	HKE 20	2	2	3	2	0	0	0	0	0	0	.	.	.
82	91HKE 89	4-26-91	4	HIGH	1326	3	1	0	1	1	0	0	HKE 89	.	.	.	1	0	0	3	1	1	0	.	.	.
83	91HKE 95	4-26-91	4	HIGH	1330	3	0	0	0	1	0	3	HKE 95	0	2	3	0	0	0	3	0	1	0	.	.	.
84	91HKE 105	4-26-91	4	HIGH	1311	3	1	1	0	0	0	1	HKE 105	1	1	2	1	0	0	1	0	1	0	.	.	.
85	91HKE 123	4-26-91	4	HIGH	1334	.	.	.	.	.	.	.	HKE 123	1	3	1	1	0	0	0	1	0	0	0	0	0
86	91HKE 145	4-26-91	4	HIGH	1338	3	0	2	1	0	3	3	HKE 145	0	3	3	2	0	0	1	0	0	0	.	.	.
87	91HKE 152	4-26-91	4	HIGH	1346	3	1	2	1	0	0	0	HKE 152	1	0	3	1	0	0	1	0	1	0	.	.	.
88	91HKE 153	4-26-91	4	HIGH	1342	3	0	0	2	2	0	1	HKE 153	.	.	.	0	0	0	3	0	0	0	.	.	.
89	91HKE 167	4-26-91	4	HIGH	1318	3	0	1	0	0	3	0	HKE 167	3	0	3	3	0	0	3	0	3	0	.	.	.
90	91HKE 226	4-26-91	4	HIGH	1304	3	0	3	0	0	0	1	HKE 226	2	2	0	2	0	0	0	0	0	0	.	.	.
91	91HKE 7	4-29-91	7	HIGH	1430	3	0	1	3	3	0	1	HKE 7	2	0	2	2	0	0	2	0	0	0	.	.	.
92	91HKE 25	4-29-91	7	HIGH	1438	3	1	0	2	2	2	2	HKE 25	0	2	2	1	0	0	1	0	0	0	0	0	0
93	91HKE 40	4-29-91	7	HIGH	1446	3	0	1	0	0	0	0	HKE 40	1	2	0	1	0	0	0	0	0	0	.	.	.
94	91HKE 51	4-29-91	7	HIGH	1422	3	0	1	1	1	0	1	HKE 51	0	0	3	1	0	0	2	0	0	0	.	.	.
95	91HKE 154	4-29-91	7	HIGH	1404	3	0	1	1	2	0	0	HKE 154	2	1	2	1	0	0	1	0	0	0	.	.	.
96	91HKE 181	4-29-91	7	HIGH	1434	3	0	1	1	0	1	0	HKE 181	2	1	3	2	0	0	3	0	0	0	.	.	.
97	91HKE 187	4-29-91	7	HIGH	1442	3	1	1	1	2	0	3	HKE 187	0	0	3	0	0	0	1	0	2	0	.	.	.
98	91HKE 204	4-29-91	7	HIGH	1411	3	0	2	1	2	0	1	HKE 204	1	0	3	1	0	0	1	0	0	0	.	.	.
99	91HKE 212	4-29-91	7	HIGH	1426	3	0	1	1	0	0	1	HKE 212	1	1	3	1	0	0	3	0	2	0	.	.	.
100	91HKE 231	4-29-91	7	HIGH	1418	3	0	2	1	1	0	1	HKE 231	2	0	3	2	0	0	3	0	3	0	.	.	.
101	91HKE 72	5-2-91	10	HIGH	1538	3	0	3	0	0	1	0	HKE 72	3	0	0	2	1	0	0	0	0	0	.	.	.
102	91HKE 77	5-2-91	10	HIGH	1511	3	0	3	3	0	2	0	HKE 77	.	.	.	3	0	0	2	1	0	0	.	.	.
103	91HKE 93	5-2-91	10	HIGH	1542	3	0	1	3	0	0	0	HKE 93	2	1	3	1	0	0	3	0	2	0	.	.	.
104	91HKE 104	5-2-91	10	HIGH	1504	3	0	3	0	0	0	2	HKE 104	.	.	.	2	0	0	0	1	0	0	.	.	.
105	91HKE 111	5-2-91	10	HIGH	1522	3	0	0	1	2	0	1	HKE 111	0	3	3	0	0	0	3	0	0	0	0	0	0
106	91HKE 115	5-2-91	10	HIGH	1526	3	1	2	0	0	0	0	HKE 115	2	0	0	3	0	0	0	0	0	0	.	.	.
107	91HKE 136	5-2-91	10	HIGH	1546	3	0	2	0	0	0	0	HKE 136	1	1	0	1	0	0	0	0	0	0	.	.	
108	91HKE 182	5-2-91	10	HIGH	1518	3	0	2	0	0	1	3	HKE 182	2	3	3	2	0	0	2	0	0	0	.	.	
109	91HKE 186	5-2-91	10	HIGH	1530	3	1	1	0	0	1	3	HKE 186	2	1	0	2	0	0	0	0	0	1	.	.	.
110	91HKE 207	5-2-91	10	HIGH	1534	.	.	.	.	.	.	.	HKE 207	3	2	3	0	0	0	3	0	0	0	.	.	.
111	91HKE 26	5-6-91	14	HIGH	1630	.	.	.	.	.	.	.	HKE 26	1	2	0	1	0	0	0	0	0	1	.	.	.
112	91HKE 79	5-6-91	14	HIGH	NO JAR	.	.	.	.	.	.	.	HKE 79	.	.	.	.	.	.	.	.	.	.	.	.	.
113	91HKE 92	5-6-91	14	HIGH	1622	3	0	1	0	0	0	0	HKE 92	0	3	2	1	0	0	2	0	0	0	.	.	.

#	HINTON NUMBER	SAMPLE DATE	DAY	EXP	ALASKA ID #	Liver							HINTON #	Spleen			Kidney							Heart		
						GLY	LIP	MAC	SCN	FN	MEG	COC		MA	CON	VAS	MA	LY	VD	NEC	GBM	GT	MYX	MA	LY	NEC
114	91HKE 124	5-6-91	14	HIGH	NO JAR	.	.	.	.	.	.	.	HKE 124	.	.	.	.	.	.	.	.	.	.	.	.	.
115	91HKE 125	5-6-91	14	HIGH	1618	3	1	2	3	0	0	1	HKE 125	0	0	3	1	0	0	3	0	0	0	.	.	.
116	91HKE 161	5-6-91	14	HIGH	1611	3	2	0	1	0	0	0	HKE 161	1	0	3	1	0	0	2	0	0	0	.	.	.
117	91HKE 180	5-6-91	14	HIGH	NO JAR	.	.	.	.	.	.	.	HKE 180	.	.	.	.	.	.	.	.	.	.	.	.	.
118	91HKE 183	5-6-91	14	HIGH	1626	.	.	.	.	.	.	.	HKE 183	.	.	.	1	0	0	1	0	0	0	.	.	.
119	91HKE 229	5-6-91	14	HIGH	1604	3	0	2	0	0	1	3	HKE 229	2	0	1	1	0	0	0	0	0	0	.	.	.
120	91HKE 235	5-6-91	14	HIGH	NO JAR	.	.	.	.	.	.	.	HKE 235	.	.	.	.	.	.	.	.	.	.	.	.	.
121	91HKE 43	5-7-91	1	20-4	11092	3	0	2	0	0	0	2	HKE 43	3	0	0	2	0	0	0	0	0	0	.	.	.
122	91HKE 46	5-7-91	1	20-4	11096	3	0	0	0	0	0	0	HKE 46	0	2	0	0	0	0	1	0	0	3	.	.	.
123	91HKE 54	5-7-91	1	20-4	11061	.	.	.	.	.	.	.	HKE 54	3	0	0	2	0	0	0	0	0	1	.	.	.
124	91HKE 58	5-7-91	1	20-4	11080	.	.	.	.	.	.	.	HKE 58	2	0	0	1	0	0	0	0	0	0	.	.	.
125	91HKE 65	5-7-91	1	20-4	11072	3	.	.	.	.	.	.	HKE 65	0	1	3	0	0	0	0	1	0	0	0	0	1
126	91HKE 83	5-7-91	1	20-4	11088	3	0	1	1	0	0	0	HKE 83	.	.	.	0	0	0	0	0	0	0	.	.	.
127	91HKE 87	5-7-91	1	20-4	11076	3	0	0	1	0	0	0	HKE 87	0	2	0	1	0	0	0	1	0	0	.	.	.
128	91HKE 90	5-7-91	1	20-4	11068	3	0	1	0	0	0	1	HKE 90	.	.	.	1	0	0	0	0	0	0	.	.	.
129	91HKE 120	5-7-91	1	20-4	11084	3	0	2	0	0	0	0	HKE 120	2	2	0	2	0	0	0	0	0	0	.	.	.
130	91HKE 188	5-7-91	1	20-4	11054	3	0	0	1	0	0	0	HKE 188	.	.	.	0	0	0	0	0	1	0	0	0	0
131	91HKE 35	5-8-91	2	20-4	11167	3	1	1	0	0	0	0	HKE 35	1	2	0	1	0	0	0	0	0	0	.	.	.
132	91HKE 41	5-8-91	2	20-4	11183	3	2	1	2	0	0	2	HKE 41	2	3	0	1	0	0	0	0	0	3	.	.	.
133	91HKE 50	5-8-91	2	20-4	11171	3	0	0	3	2	0	0	HKE 50	0	3	2	0	0	0	0	1	0	0	.	.	.
134	91HKE 76	5-8-91	2	20-4	11187	3	0	2	0	0	0	2	HKE 76	1	1	1	1	0	0	1	1	0	0	.	.	.
135	91HKE 138	5-8-91	2	20-4	11191	3	1	1	0	0	0	0	HKE 138	1	1	0	1	0	0	0	0	0	0	.	.	.
136	91HKE 163	5-8-91	2	20-4	11153	.	.	.	.	.	.	.	HKE 163	2	1	0	2	0	0	0	0	0	0	.	.	.
137	91HKE 164	5-8-91	2	20-4	11179	3	0	3	0	0	0	0	HKE 164	3	2	0	3	0	0	0	0	0	0	.	.	.
138	91HKE 184	5-8-91	2	20-4	11195	3	1	0	0	0	0	3	HKE 184	0	0	0	1	0	0	0	0	0	0	.	.	.
139	91HKE 214	5-8-91	2	20-4	11160	3	0	2	0	0	0	2	HKE 214	.	.	.	1	0	0	0	0	2	0	.	.	.
140	91HKE 223	5-8-91	2	20-4	11175	3	0	0	0	0	0	0	HKE 223	0	3	0	1	0	0	0	0	0	0	.	.	.
141	91HKE 11	5-10-91	4	20-4	11353	3	0	2	2	0	0	0	HKE 11	3	0	2	3	0	0	0	0	0	2	.	.	.
142	91HKE 13	5-10-91	4	20-4	11375	.	.	.	.	.	.	.	HKE 13	1	0	0	1	0	0	0	0	0	0	.	.	.
143	91HKE 48	5-10-91	4	20-4	11387	3	0	2	0	0	0	0	HKE 48	0	3	0	1	0	0	0	1	0	0	.	.	.
144	91HKE 63	5-10-91	4	20-4	11379	3	0	1	0	0	0	2	HKE 63	2	1	2	2	0	0	1	0	0	0	.	.	.
145	91HKE 117	5-10-91	4	20-4	11383	3	0	0	0	0	0	1	HKE 117	0	3	1	1	0	0	0	0	0	0	.	.	.
146	91HKE 122	5-10-91	4	20-4	11395	.	.	.	.	.	.	.	HKE 122	3	0	1	3	0	0	0	0	0	0	.	.	.
147	91HKE 157	5-10-91	4	20-4	11360	3	0	0	0	0	0	0	HKE 157	0	1	0	0	0	0	0	0	0	3	.	.	.
148	91HKE 166	5-10-91	4	20-4	11371	.	.	.	.	.	.	.	HKE 166	1	3	0	2	0	0	0	0	0	0	.	.	.
149	91HKE 224	5-10-91	4	20-4	11391	.	.	.	.	.	.	.	HKE 224	.	.	.	.	.	.	.	.	.	.	.	.	.
150	91HKE 232	5-10-91	4	20-4	11367	3	2	0	1	0	0	2	HKE 232	0	2	2	.	.	.	.	.	.	.	.	.	.
151	91HKE 6	5-13-91	7	20-4	11479	.	.	.	.	.	.	.	HKE 6	3	3	0	2	0	0	0	0	0	2	.	.	.
152	91HKE 17	5-13-91	7	20-4	11475	3	0	0	3	0	0	1	HKE 17	1	1	0	0	0	0	0	0	0	0	.	.	.
153	91HKE 27	5-13-91	7	20-4	11495	.	.	.	.	.	.	.	HKE 27	2	2	0	2	0	0	0	0	0	0	.	.	.
154	91HKE 39	5-13-91	7	20-4	11467	3	0	0	1	0	0	0	HKE 39	0	1	0	0	0	0	0	0	0	0	.	.	.
155	91HKE 69	5-13-91	7	20-4	11487	3	0	3	1	0	0	0	HKE 69	2	2	0	2	0	0	0	0	0	0	.	.	.
156	91HKE 112	5-13-91	7	20-4	11483	3	0	3	0	0	0	0	HKE 112	3	3	0	3	0	0	0	0	0	0	.	.	.
157	91HKE 127	5-13-91	7	20-4	11491	3	0	1	0	0	0	1	HKE 127	1	1	3	1	0	0	3	0	0	0	.	.	.
158	91HKE 169	5-13-91	7	20-4	11460	3	0	0	3	3	0	2	HKE 169	0	0	3	1	0	0	3	0	0	0	.	.	.

#	HINTON NUMBER	SAMPLE DATE	DAY	EXP	ALASKA ID #	Liver GLY	LIP	MAC	SCN	EN	MEG	COC	HINTON #	Spleen MA	CON	VAS	Kidney MA	LY	VD	NEC	GBM	GT	MYX	Heart MA	LY	NEC
159	91HKE 177	5-13-91	7	20-4	11471	3	0	0	0	0	1	2	HKE 177	0	3	0	1	0	0	0	0	0	0	0	0	0
160	91HKE 196	5-13-91	7	20-4	11453	3	0	1	1	0	0	2	HKE 196	1	3	0	1	0	0	0	1	0	0	.	.	.
161	91HKE 2	5-16-91	10	20-4	11560	3	1	1	0	0	0	1	HKE 2	1	0	0	1	0	0	0	0	0	0	.	.	.
162	91HKE 38	5-16-91	10	20-4	11591	.	.	.	.	.	.	.	HKE 38	0	2	0	1	0	0	0	0	0	0	.	.	.
163	91HKE 53	5-16-91	10	20-4	11553	3	1	1	0	0	0	1	HKE 53	2	0	1	2	0	0	0	0	0	0	.	.	.
164	91HKE 55	5-16-91	10	20-4	11567	.	.	.	.	.	.	.	HKE 55	1	0	0	1	0	0	0	0	0	0	0	0	1
165	91HKE 68	5-16-91	10	20-4	11583	.	.	.	.	.	.	.	HKE 68	.	.	.	1	0	0	0	0	0	0	0	0	0
166	91HKE 147	5-16-91	10	20-4	11579	3	0	3	0	0	0	0	HKE 147	1	3	0	1	0	0	0	0	0	0	.	.	.
167	91HKE 168	5-16-91	10	20-4	11587	3	0	2	0	0	0	0	HKE 168	3	2	2	2	0	0	0	0	0	0	.	.	.
168	91HKE 192	5-16-91	10	20-4	11571	3	0	2	0	0	0	0	HKE 192	1	1	0	2	0	0	0	0	0	0	.	.	.
169	91HKE 221	5-16-91	10	20-4	11595	3	1	0	0	0	0	0	HKE 221	0	3	0	0	0	0	0	0	0	0	0	0	0
170	91HKE 238	5-16-91	10	20-4	11575	3	0	3	0	0	0	0	HKE 238	.	.	.	1	0	0	0	0	0	0	.	.	.
171	91HKE 23	5-13-91	7	20-4D	11404	3	1	1	1	0	0	2	HKE 23	1	2	1	1	0	0	3	0	0	3	.	.	.
172	91HKE 52	5-13-91	7	20-4D	11430	3	1	2	0	0	0	0	HKE 52	2	2	0	2	0	0	0	0	0	0	.	.	.
173	91HKE 94	5-13-91	7	20-4D	11434	3	0	2	0	0	0	0	HKE 94	2	2	0	2	0	0	0	0	0	0	0	0	0
174	91HKE 106	5-13-91	7	20-4D	11422	3	0	0	0	0	0	3	HKE 106	0	3	3	0	0	0	0	0	0	0	.	.	.
175	91HKE 116	5-13-91	7	20-4D	11418	3	0	1	0	0	0	1	HKE 116	1	1	0	1	0	0	0	0	0	0	.	.	.
176	91HKE 119	5-13-91	7	20-4D	11411	3	0	1	0	0	0	0	HKE 119	1	1	2	0	0	0	0	0	0	0	.	.	.
177	91HKE 134	5-13-91	7	20-4D	11442	3	0	2	0	0	0	0	HKE 134	3	3	0	2	0	0	0	0	0	0	.	.	.
178	91HKE 170	5-13-91	7	20-4D	11446	.	.	.	.	.	.	.	HKE 170	0	3	0	0	0	0	0	0	0	0	.	.	.
179	91HKE 172	5-13-91	7	20-4D	11438	.	.	.	.	.	.	.	HKE 172	.	.	.	1	0	0	0	0	0	0	.	.	.
180	91HKE 233	5-13-91	7	20-4D	11426	3	0	1	0	0	0	1	HKE 233	0	1	0	2	0	0	0	0	0	1	.	.	.
181	91HKE 4	5-17-91	11	20-4D	11534	3	0	1	0	0	0	1	HKE 4	1	2	0	1	0	0	0	0	0	0	.	.	.
182	91HKE 9	5-17-91	11	20-4D	11526	.	.	.	.	.	.	.	HKE 9	3	0	0	1	1	0	0	2	0	0	.	.	.
183	91HKE 56	5-17-91	11	20-4D	11518	3	1	1	0	0	1	0	HKE 56	2	3	0	1	0	0	0	0	0	0	.	.	.
184	91HKE 71	5-17-91	11	20-4D	11542	3	0	2	0	0	0	0	HKE 71	2	2	0	2	0	0	0	0	0	0	.	.	.
185	91HKE 113	5-17-91	11	20-4D	11511	3	0	0	1	0	0	1	HKE 113	0	3	0	0	0	0	1	0	0	0	.	.	.
186	91HKE 143	5-17-91	11	20-4D	11504	.	.	.	.	.	.	.	HKE 143	0	0	0	1	0	0	0	0	0	0	.	.	.
187	91HKE 175	5-17-91	11	20-4D	11546	3	0	2	0	0	0	0	HKE 175	2	2	0	2	0	0	0	0	0	0	.	.	.
188	91HKE 191	5-17-91	11	20-4D	11538	3	0	1	0	0	1	1	HKE 191	0	3	0	1	0	0	0	0	0	0	.	.	.
189	91HKE 209	5-17-91	11	20-4D	11522	3	0	2	0	0	0	1	HKE 209	3	0	0	2	0	0	0	0	0	0	.	.	.
190	91HKE 213	5-17-91	11	20-4D	11530	3	0	1	0	0	0	0	HKE 213	1	0	0	1	0	0	0	0	0	0	.	.	.
191	91HKE 32	5-7-91	1	60-4	11043	.	.	.	.	.	.	.	HKE 32	2	3	0	2	0	0	0	0	0	0	.	.	.
192	91HKE 98	5-7-91	1	60-4	11039	3	1	2	1	0	0	0	HKE 98	.	.	.	1	0	0	0	0	0	0	.	.	.
193	91HKE 135	5-7-91	1	60-4	11031	3	0	0	0	0	0	1	HKE 135	0	0	0	0	0	0	0	0	0	0	0	0	2
194	91HKE 149	5-7-91	1	60-4	11023	3	0	1	0	1	1	1	HKE 149	2	0	0	1	0	0	0	0	0	0	.	.	.
195	91HKE 189	5-7-91	1	60-4	11012	.	.	.	.	.	.	.	HKE 189	1	0	0	1	0	0	0	0	0	0	.	.	.
196	91HKE 206	5-7-91	1	60-4	11027	3	0	1	1	0	0	0	HKE 206	1	0	1	1	0	0	1	0	0	0	.	.	.
197	91HKE 218	5-7-91	1	60-4	11019	.	.	.	.	.	.	.	HKE 218	1	0	0	2	0	0	0	0	0	0	.	.	.
198	91HKE 234	5-7-91	1	60-4	11005	3	1	1	0	0	0	2	HKE 234	1	2	1	1	0	0	0	0	0	0	.	.	.
199	91HKE 239	5-7-91	1	60-4	11035	3	1	2	0	0	1	0	HKE 239	.	.	.	2	0	0	0	0	0	0	0	0	1
200	91HKE 240	5-7-91	1	60-4	11047	3	0	2	0	0	0	1	HKE 240	2	0	0	2	0	0	0	0	0	0	0	0	0
201	91HKE 30	5-8-91	2	60-4	11118	3	1	1	1	0	0	0	HKE 30	1	.	.	1	0	0	2	0	0	0	.	.	.
202	91HKE 74	5-8-91	2	60-4	11138	3	1	0	1	0	0	1	HKE 74	0	2	0	0	0	0	0	0	0	0	.	.	.
203	91HKE 100	5-8-91	2	60-4	11126	3	1	1	2	0	0	0	HKE 100	.	.	.	2	0	0	0	0	0	0	.	.	.

#	HINTON NUMBER	SAMPLE DATE	DAY	EXP	ALASKA ID #	Liver							HINTON #	Spleen			Kidney							Heart		
						GLY	LIP	MAC	SCN	EN	MEG	COC		MA	CON	VAS	MA	LY	VD	NEC	GBM	GT	MYX	MA	LY	NEC
204	91HKE 146	5-8-91	2	60-4	11111	3	0	2	1	0	1	1	HKE 146	2	0	0	3	0	0	0	0	0	0	0	0	0
205	91HKE 190	5-8-91	2	60-4	11104	3	0	3	3	0	0	0	HKE 190	2	0	3	2	0	0	3	0	0	0	.	.	.
206	91HKE 200	5-8-91	2	60-4	11134	.	.	.	.	.	.	.	HKE 200	1	2	0	2	0	0	0	0	0	0	.	.	.
207	91HKE 202	5-8-91	2	60-4	11130	.	.	.	.	.	.	.	HKE 202	2	1	0	2	0	0	0	0	0	0	.	.	.
208	91HKE 208	5-8-91	2	60-4	11146	3	0	0	3	0	0	3	HKE 208	.	.	.	1	0	0	2	0	0	0	.	.	.
209	91HKE 217	5-8-91	2	60-4	11142	3	0	0	0	0	0	1	HKE 217	0	3	0	1	0	0	0	0	0	0	.	.	.
210	91HKE 222	5-8-91	2	60-4	11122	3	0	2	2	1	0	1	HKE 222	.	.	.	2	0	0	1	0	0	0	.	.	.
211	91HKE 1	5-10-91	4	60-4	11338	3	0	1	0	0	0	0	HKE 1	.	.	.	1	0	0	2	0	0	0	0	0	0
212	91HKE 5	5-10-91	4	60-4	11334	.	.	.	.	.	.	.	HKE 5	2	.	.	.	.	.	.	.	.	.	0	0	0
213	91HKE 16	5-10-91	4	60-4	11311	3	1	2	3	0	0	0	HKE 16	1	3	1	1	0	0	1	0	0	0	.	.	.
214	91HKE 24	5-10-91	4	60-4	11304	.	.	.	.	.	.	.	HKE 24	0	3	2	.	.	.	.	.	.	.	.	.	.
215	91HKE 82	5-10-91	4	60-4	11326	3	0	3	0	0	0	0	HKE 82	3	0	0	3	0	0	0	0	0	2	.	.	.
216	91HKE 96	5-10-91	4	60-4	11346	3	0	0	1	1	0	1	HKE 96	0	0	3	0	0	0	0	0	0	0	.	.	.
217	91HKE 110	5-10-91	4	60-4	11318	3	0	1	0	0	0	0	HKE 110	1	1	1	1	0	0	0	0	0	0	.	.	.
218	91HKE 132	5-10-91	4	60-4	11322	3	0	2	0	0	0	1	HKE 132	3	2	0	3	0	0	0	0	0	0	.	.	.
219	91HKE 158	5-10-91	4	60-4	11330	.	.	.	.	.	.	.	HKE 158	0	0	3	1	0	0	3	0	0	0	.	.	.
220	91HKE 193	5-10-91	4	60-4	11342	3	0	3	0	0	0	0	HKE 193	2	1	1	3	0	0	0	0	0	0	.	.	.
221	91HKE 12	4-25-91	3	CONTROL	1279	3	1	1	0	1	0	1	HKE 12	0	2	2	1	0	0	1	0	0	0	.	.	.
222	91HKE 88	4-25-91	3	CONTROL	1271	3	0	3	0	0	0	3	HKE 88	2	3	0	3	0	0	0	0	0	0	.	.	.
223	91HKE 91	4-25-91	3	CONTROL	1287	3	2	1	0	0	0	0	HKE 91	2	2	0	2	0	0	0	0	0	0	.	.	.
224	91HKE 99	4-25-91	3	CONTROL	1260	.	.	.	.	.	.	.	HKE 99	0	1	0	0	0	0	0	0	0	0	.	.	.
225	91HKE 121	4-25-91	3	CONTROL	1267	.	.	.	.	.	.	.	HKE 121	0	3	1	1	0	0	1	0	0	1	.	.	.
226	91HKE 133	4-25-91	3	CONTROL	1291	.	.	.	.	.	.	.	HKE 133	1	3	3	2	0	0	2	0	0	0	0	0	1
227	91HKE 194	4-25-91	3	CONTROL	1283	3	0	1	0	0	1	0	HKE 194	0	2	0	1	0	0	0	0	0	0	.	.	.
228	91HKE 211	4-25-91	3	CONTROL	1275	3	2	1	1	1	0	0	HKE 211	0	0	3	0	0	0	1	1	2	0	.	.	.
229	91HKE 220	4-25-91	3	CONTROL	1295	3	0	1	0	0	1	0	HKE 220	1	2	0	1	0	0	0	0	0	0	.	.	.
230	91HKE 236	4-25-91	3	CONTROL	1253	3	0	2	0	0	1	0	HKE 236	3	0	2	1	0	0	1	0	0	0	.	.	.
231	91HKE 42	4-25-91	3	P CONTROL	1242	3	1	1	0	0	1	2	HKE 42	2	2	2	2	0	0	1	0	0	0	.	.	.
232	91HKE 60	4-25-91	3	P CONTROL	1234	3	1	1	0	0	0	3	HKE 60	2	1	2	1	0	0	1	0	0	0	0	1	0
233	91HKE 103	4-25-91	3	P CONTROL	1238	3	0	0	1	0	0	0	HKE 103	0	3	3	0	0	0	1	0	0	0	.	.	.
234	91HKE 128	4-25-91	3	P CONTROL	1218	3	0	1	0	0	0	0	HKE 128	1	1	2	1	0	0	1	0	3	0	.	.	.
235	91HKE 129	4-25-91	3	P CONTROL	1211	3	0	2	0	0	0	1	HKE 129	3	1	3	3	0	0	2	0	0	0	.	.	.
236	91HKE 131	4-25-91	3	P CONTROL	1230	3	0	0	0	0	0	1	HKE 131	0	0	0	0	0	0	0	0	0	0	.	.	.
237	91HKE 142	4-25-91	3	P CONTROL	1204	3	0	1	0	0	0	0	HKE 142	3	1	3	2	0	0	2	0	0	0	.	.	.
238	91HKE 150	4-25-91	3	P CONTROL	1246	3	1	2	0	0	0	0	HKE 150	.	.	3	1	0	0	2	0	0	0	0	0	0
239	91HKE 151	4-25-91	3	P CONTROL	1222	3	0	2	0	0	0	0	HKE 151	.	.	.	2	0	0	1	0	0	0	.	.	.
240	91HKE 201	4-25-91	3	P CONTROL	1226	3	0	1	0	0	1	0	HKE 201	2	1	0	1	0	0	0	0	0	0	.	.	.

SUMMARY STATISTICS:

		Mean Lesion Score							Mean Lesion Scores												
		Liver							Spleen			Kidney							Heart		
DAY	Experiment	GLY	LIP	MAC	SCN	FN	MEG	COC	Day	Exp	MA	CON	VAS	MA	LY	VD	NEC	GBM	GT	MYX	MA	LY	NEC
1	LOW	3	.63	1.1	.75	.38	.13	.63	1	LO	1.4	1.9	1.2	1.4	0	0	.9	0	.9	0	0	0	0
2	LOW	3	.75	1	.88	.25	0	1.1	2	LO	1.5	.5	1.6	1.4	0	0	1.2	0	.5	.2	.	.	.
4	LOW	3	.67	.67	1.3	1	.11	1.8	4	LO	.67	.44	2.4	1	0	0	1.9	0	.78	.44	0	0	1
7	LOW	3	.11	1.2	.67	.22	.11	.89	7	LO	1.4	1.1	.86	1.5	0	0	1.7	.2	.5	.6	.	.	.
10	LOW	3	.44	.78	.78	.11	.22	1	10	LO	1.2	1.1	1.1	1.2	0	0	.7	.1	.2	.6	0	0	.5
14	LOW	3	.33	1.1	.78	.22	.11	.67	14	LO	1.1	1.6	1.2	1.1	0	0	.44	.11	0	0	0	0	0
1	HIGH	3	.43	1.1	.14	0	.43	.29	1	HI	.4	1.2	1.4	.63	0	0	.63	.13	.5	.25	0	0	0
2	HIGH	3	.8	1.4	1.2	.5	.1	.7	2	HI	.89	.78	2	1.3	0	0	2.1	.1	1	.1	0	0	0
4	HIGH	3	.33	1.2	.56	.56	.67	1	4	HI	1.3	1.6	2.3	1.3	0	0	1.5	.2	.7	0	0	0	0
7	HIGH	3	.2	1.1	1.2	1.3	.3	1	7	HI	1.1	.7	2.4	1.2	0	0	1.7	0	.7	0	0	0	0
10	HIGH	3	.22	1.9	.78	.22	.56	1	10	HI	1.9	1.4	1.5	1.6	.1	0	1.3	.2	.2	.1	0	0	0
14	HIGH	3	.75	1.3	1	0	.25	1	14	HI	.8	1	1.8	1	0	0	1.3	0	0	.17	.	.	.
1	20-4	3	0	.86	.43	0	0	.43	1	20	1.4	1	.43	.9	0	0	.1	.2	.1	.4	0	0	.5
2	20-4	3	.56	1.1	.56	.22	0	1	2	20	1.1	1.8	.33	1.2	0	0	.1	.2	.2	.3	.	.	.
4	20-4	3	.33	.83	.5	0	0	.83	4	20	1.1	1.4	.89	1.6	0	0	.13	.13	0	.63	.	.	.
7	20-4	3	0	1	1.1	.38	.13	1	7	20	1.3	1.9	.6	1.3	0	0	.6	.1	0	.2	0	0	0
10	20-4	3	.43	1.7	0	0	0	.29	10	20	1.1	1.4	.38	1.2	0	0	0	0	0	0	0	0	.33
7	20-4D	3	.25	1.3	.13	0	0	.88	7	4d	1.1	2	.67	1.1	0	0	.3	0	0	.4	0	0	0
11	20-4D	3	.13	1.3	.13	0	.25	.5	11	4d	1.4	1.5	0	1.2	.1	0	.1	.2	0	0	.	.	.
1	60-4	3	.43	1.3	.29	.14	.29	.71	1	60	1.3	.63	.25	1.3	0	0	.1	0	0	0	0	0	1
2	60-4	3	.38	1.1	1.6	.13	.13	.88	2	60	1.1	1.3	.5	1.6	0	0	.8	0	0	0	0	0	0
4	60-4	3	.14	1.7	.57	.14	0	.29	4	60	1.3	1.3	1.4	1.6	0	0	.75	0	0	.25	0	0	0
3	CONTROL	3	.71	1.4	.14	.29	.43	.57	3	C	.9	1.8	1.1	1.2	0	0	.6	.1	.2	.1	0	0	1
3	P CONTROL	3	.3	1.1	.1	0	.2	.7	3	PC	1.6	1.3	2	1.2	0	0	1.1	0	.3	0	0	.5	0

Table VI-2. Histopathology of esophagus and stomach in Pacific herring adults exposed to crude oil in the Laboratory.
Key to table symbols:

Hinton number = random (processing) number generated by Dr. Hinton's Laboratory
Lesion scores: none (0), mild (1), moderate (2), severe (3), not present (.)
Exp. = same as for Table VI-1 above

ESOPHAGUS:
- mucosal atrophy (ATR)
- eosinophilic granular leukocytes (EGL)
- hemorrhage (HEM)
- necrosis (NEC)
- fibrosis (FIB)
- thrombosis (THB)

STOMACH:
- mucosal atrophy (ATR)
- eosinophilic granular leukocytes (EGL)
- hemorrhage (HEM)
- necrosis (NEC)
- fibrosis (FIB)
- thrombosis (THB)

	ESOPHAGUS										NONGLANDULAR STOMACH											GLANDULAR STOMACH								
	Tunica mucosae					T. muscularis					Tunica mucosae					T. muscularis						Tunica mucosae					T. muscularis			
#	ATR	EGL	HEM	NEC	FIB	HEM	NEC	FIB	THB	Exp.	ATR	EGL	HEM	NEC	FIB	HEM	NEC	FIB	THB	HINTON #		ATR	EGL	HEM	NEC	FIB	HEM	NEC	FIB	THB
1	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE	14	0	3	0	0	1	0	0	0	0
2	0	2	0	0	3	0	1	0	0	LOW	.	.	.	.	.	.	.	.	.	HKE	21	.	.	.	.	.	.	.	.	.
3	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE	44	0	3	0	0	1	0	0	0	0
4	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE	97	0	3	0	0	1	0	1	0	0
5	.	.	.	.	.	.	.	.	.	LOW	0	1	0	0	2	0	0	0	0	HKE	107	0	3	0	0	1	0	0	0	0
6	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE	118	.	.	.	.	.	.	.	.	.
7	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE	144	0	3	0	0	1	0	0	0	0
8	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE	159	0	2	0	0	2	0	0	0	0
9	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE	197	0	3	0	0	1	0	0	0	0
10	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE	225	0	3	0	0	2	0	0	0	0
11	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE	230	0	3	0	1	2	0	0	0	0
12	1	2	0	0	2	0	0	0	0	LOW	.	.	.	.	.	.	.	.	.	HKE	37	.	.	.	.	.	.	.	.	.
13	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE	64	0	2	0	0	2	0	0	0	0
14	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE	75	1	2	0	0	3	0	0	0	0
15	0	3	0	0	2	0	0	0	0	LOW	.	.	.	.	.	.	.	.	.	HKE	101	.	.	.	.	.	.	.	.	.
16	.	.	.	.	.	.	.	.	.	LOW	0	2	0	0	2	0	0	0	0	HKE	102	0	2	0	0	1	0	0	0	0
17	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE	114	.	.	.	.	.	.	.	.	.
18	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE	156	1	2	0	0	2	1	1	0	0
19	.	.	.	.	.	.	.	.	.	LOW	0	0	0	0	3	0	0	0	0	HKE	178	.	.	.	.	.	.	.	.	.
20	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE	195	0	1	0	0	1	0	0	0	0
21	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE	215	1	2	0	0	2	0	0	0	0
22	0	3	0	0	2	0	0	0	0	LOW	.	.	.	.	.	.	.	.	.	HKE	8	.	.	.	.	.	.	.	.	.

	ESOPHAGUS										NONGLANDULAR STOMACH											GLANDULAR STOMACH									
	Tunica mucosae					T. muscularis					Tunica mucosae					T. muscularis						Tunica mucosae					T. muscularis			
#	ATR	EGL	HEM	NEC	FIB	HEM	NEC	FIB	THB	Exp.	ATR	EGL	HEM	NEC	FIB	HEM	NEC	FIB	THB	HINTON #	ATR	EGL	HEM	NEC	FIB	HEM	NEC	FIB	THB
23	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 18	0	2	0	1	2	0	0	0	0
24	.	.	.	.	.	.	.	.	.	LOW	0	2	0	0	3	0	1	0	0	HKE 33	.	.	.	.	.	.	.	.	.
25	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 80	0	1	0	0	1	0	0	0	0
26	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 137	.	.	.	.	.	.	.	.	.
27	.	.	.	.	.	.	.	.	.	LOW	0	1	0	0	3	0	0	0	0	HKE 139	0	2	0	0	1	0	0	0	0
28	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 148	0	2	0	0	2	0	0	0	0
29	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 198	.	.	.	.	.	.	.	.	.
30	0	2	0	0	2	0	0	0	0	LOW	.	.	.	.	.	.	.	.	.	HKE 210	.	.	.	.	.	.	.	.	.
31	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 219	0	3	0	0	2	0	0	0	0
32	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 28	0	1	0	1	3	0	1	0	0
33	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 36	0	1	0	1	3	0	0	0	0
34	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 49	0	3	0	1	3	0	1	0	0
35	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 73	0	2	0	0	2	0	0	0	0
36	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 84	0	2	0	0	1	0	0	0	0
37	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 141	0	1	0	0	2	0	0	0	0
38	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 179	0	3	0	0	2	0	0	0	0
39	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 185	0	3	0	0	3	0	0	0	1
40	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 227	0	1	0	0	1	0	0	0	0
41	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 237	0	2	0	0	2	0	0	0	0
42	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 3	0	3	0	1	3	0	0	0	1
43	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 19	0	3	0	0	2	0	1	0	0
44	.	.	.	.	.	.	.	.	.	LOW	0	0	0	0	1	0	0	0	0	HKE 22	.	.	.	.	.	.	.	.	.
45	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 29	2	1	0	0	3	0	1	0	0
46	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 62	0	3	0	0	2	0	2	0	0
47	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 140	0	3	0	1	2	0	0	0	0
48	0	3	0	0	3	0	0	0	0	LOW	.	.	.	.	.	.	.	.	.	HKE 155	.	.	.	.	.	.	.	.	.
49	0	2	0	0	3	0	0	0	0	LOW	.	.	.	.	.	.	.	.	.	HKE 199	.	.	.	.	.	.	.	.	.
50	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 205	0	2	0	0	3	0	0	0	0
51	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 216	0	2	0	0	2	0	0	0	0
52	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 10	0	3	0	0	3	0	0	0	0
53	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 15	0	2	0	0	0	0	1	0	0
54	0	2	0	1	3	0	0	0	0	LOW	.	.	.	.	.	.	.	.	.	HKE 34	0	2	0	0	1	0	0	0	0
55	0	1	0	0	2	0	2	0	0	LOW	.	.	.	.	.	.	.	.	.	HKE 78	0	3	0	0	1	0	0	0	0
56	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 86	0	3	0	1	3	0	1	0	0
57	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 108	0	2	0	0	2	0	0	0	0
58	0	3	0	0	1	0	1	0	0	LOW	.	.	.	.	.	.	.	.	.	HKE 109	.	.	.	.	.	.	.	.	.
59	0	1	0	0	3	0	1	0	0	LOW	.	.	.	.	.	.	.	.	.	HKE 173	.	.	.	.	.	.	.	.	.
60	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	HKE 176	0	3	0	0	2	0	0	0	0
61	.	.	.	.	.	.	.	.	.	HIGH	0	1	0	0	3	0	3	1	0	HKE 31	0	1	0	2	3	0	3	0	0
62	.	.	.	.	.	.	.	.	.	HIGH	0	0	0	0	1	0	0	0	0	HKE 45	.	.	.	.	.	.	.	.	.
63	.	.	.	.	.	.	.	.	.	HIGH	0	2	0	0	3	0	0	0	0	HKE 61	.	.	.	.	.	.	.	.	.
64	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 67	0	2	0	0	2	0	0	0	0

	ESOPHAGUS										NONGLANDULAR STOMACH											GLANDULAR STOMACH									
	Tunica mucosae					T. muscularis					Tunica mucosae					T. muscularis						Tunica mucosae					T. muscularis			
#	ATR	EGL	HEM	NEC	FIB	HEM	NEC	FIB	THB	Exp.	ATR	EGL	HEM	NEC	FIB	HEM	NEC	FIB	THB	HINTON #	ATR	EGL	HEM	NEC	FIB	HEM	NEC	FIB	THB
65	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 81	3	1	0	0	3	0	3	0	0
66	.	.	.	.	.	.	.	.	.	HIGH	0	0	0	0	2	0	0	0	0	HKE 130	.	.	.	.	.	.	.	.	.
67	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 162	0	2	0	0	1	0	0	0	0
68	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 165	0	1	0	0	1	0	0	0	0
69	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 171	0	1	0	0	1	0	3	0	0
70	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 174	2	1	0	0	3	0	1	0	0
71	.	.	.	.	.	.	.	.	.	HIGH	0	1	0	0	2	0	0	0	0	HKE 47	.	.	.	.	.	.	.	.	.
72	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 57	0	3	0	0	1	0	0	0	0
73	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 59	1	3	0	0	3	0	1	0	0
74	1	2	0	0	3	0	0	0	0	HIGH	.	.	.	.	.	.	.	.	.	HKE 66	.	.	.	.	.	.	.	.	.
75	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 70	0	2	0	0	3	0	0	0	0
76	1	1	0	0	2	0	1	0	0	HIGH	.	.	.	.	.	.	.	.	.	HKE 85	.	.	.	.	.	.	.	.	.
77	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 126	0	2	0	0	2	0	0	0	0
78	2	1	0	3	3	1	0	0	0	HIGH	.	.	.	.	.	.	.	.	.	HKE 160	.	.	.	.	.	.	.	.	.
79	.	.	.	.	.	.	.	.	.	HIGH	0	0	0	0	1	0	0	0	0	HKE 203	.	.	.	.	.	.	.	.	.
80	0	1	0	0	3	0	2	0	0	HIGH	.	.	.	.	.	.	.	.	.	HKE 228	.	.	.	.	.	.	.	.	.
81	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 20	1	2	1	0	3	0	0	0	0
82	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 89	0	1	0	0	2	0	1	0	0
83	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 95	1	3	0	0	1	0	0	0	0
84	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 105	2	2	0	0	3	0	0	0	0
85	0	3	0	0	3	0	0	0	0	HIGH	.	.	.	.	.	.	.	.	.	HKE 123	.	.	.	.	.	.	.	.	.
86	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 145	1	2	0	0	3	0	0	0	0
87	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 152	0	3	3	0	3	3	0	0	0
88	0	3	0	0	3	0	0	0	0	HIGH	.	.	.	.	.	.	.	.	.	HKE 153	.	.	.	.	.	.	.	.	.
89	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 167	0	3	0	0	3	0	0	0	0
90	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 226	0	2	0	0	1	0	0	0	0
91	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 7	0	2	0	0	3	0	0	0	0
92	2	2	0	0	3	0	1	0	0	HIGH	.	.	.	.	.	.	.	.	.	HKE 25	.	.	.	.	.	.	.	.	.
93	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 40	0	1	0	3	2	0	3	1	0
94	1	1	3	0	3	0	0	0	0	HIGH	.	.	.	.	.	.	.	.	.	HKE 51	.	.	.	.	.	.	.	.	.
95	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 154	0	2	0	0	3	0	0	0	0
96	3	2	1	0	3	0	0	0	0	HIGH	.	.	.	.	.	.	.	.	.	HKE 181	.	.	.	.	.	.	.	.	.
97	2	3	0	0	3	0	1	0	0	HIGH	.	.	.	.	.	.	.	.	.	HKE 187	.	.	.	.	.	.	.	.	.
98	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 204	0	1	0	0	3	0	1	0	0
99	0	2	1	0	2	0	0	0	0	HIGH	.	.	.	.	.	.	.	.	.	HKE 212	.	.	.	.	.	.	.	.	.
100	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 231	0	2	0	0	2	0	0	0	0
101	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 72	0	3	0	1	1	0	0	0	0
102	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 77	0	3	0	1	2	0	0	0	0
103	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 93	0	2	0	0	2	0	1	0	0
104	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 104	0	3	0	0	2	0	1	0	0
105	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 111	1	1	1	1	2	1	3	0	0
106	1	2	0	0	1	0	0	0	0	HIGH	.	.	.	.	.	.	.	.	.	HKE 115	.	.	.	.	.	.	.	.	.

	ESOPHAGUS										NONGLANDULAR STOMACH										HINTON	GLANDULAR STOMACH								
	Tunica mucosae					T. muscularis					Tunica mucosae					T. muscularis						Tunica mucosae					T. muscularis			
#	ATR	EGL	HEM	NEC	FIB	HEM	NEC	FIB	THB	Exp.	ATR	EGL	HEM	NEC	FIB	HEM	NEC	FIB	THB	#	ATR	EGL	HEM	NEC	FIB	HEM	NEC	FIB	THB
107	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 136	0	1	0	1	2	0	0	0	0
108	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 182	1	1	0	0	3	0	0	0	0
109	0	2	0	0	3	0	0	0	0	HIGH	.	.	.	.	.	.	.	.	.	HKE 186	.	.	.	.	.	.	.	.	.
110	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 207	0	3	0	0	2	0	0	0	0
111	0	2	0	0	2	0	2	0	0	HIGH	.	.	.	.	.	.	.	.	.	HKE 26	.	.	.	.	.	.	.	.	.
112	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 79	.	.	.	.	.	.	.	.	.
113	0	2	0	0	3	0	0	0	0	HIGH	.	.	.	.	.	.	.	.	.	HKE 92	.	.	.	.	.	.	.	.	.
114	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 124	.	.	.	.	.	.	.	.	.
115	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 125	0	2	0	0	2	0	0	0	0
116	.	.	.	.	.	.	.	.	.	HIGH	2	1	2	3	3	1	2	0	0	HKE 161	.	.	.	.	.	.	.	.	.
117	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 180	.	.	.	.	.	.	.	.	.
118	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 183	0	1	0	0	3	0	1	0	0
119	0	1	0	0	2	0	0	0	0	HIGH	.	.	.	.	.	.	.	.	.	HKE 229	.	.	.	.	.	.	.	.	.
120	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	HKE 235	.	.	.	.	.	.	.	.	.
121	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 43	0	2	0	1	1	0	3	0	0
122	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 46	0	2	0	1	2	0	1	0	0
123	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 54	.	.	.	.	.	.	.	.	.
124	0	1	0	0	3	0	0	0	0	20-4	.	.	.	.	.	.	.	.	.	HKE 58	0	2	0	0	2	0	1	0	0
125	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 65	0	1	0	0	2	0	3	0	0
126	1	0	0	0	1	0	3	0	0	20-4	.	.	.	.	.	.	.	.	.	HKE 83	0	3	0	0	3	0	0	0	0
127	0	0	0	0	2	0	2	0	0	20-4	.	.	.	.	.	.	.	.	.	HKE 87	.	.	.	.	.	.	.	.	.
128	0	2	0	0	3	0	0	0	0	20-4	.	.	.	.	.	.	.	.	.	HKE 90	.	.	.	.	.	.	.	.	.
129	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 120	0	2	0	0	2	0	1	0	0
130	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 188	0	1	0	0	2	0	0	0	0
131	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 35	0	3	0	2	1	0	2	1	1
132	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 41	0	2	0	0	3	0	2	0	0
133	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 50	0	1	0	0	1	0	3	0	0
134	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 76	0	0	0	0	1	0	0	0	0
135	0	1	0	0	3	0	0	0	0	20-4	0	1	0	0	2	0	0	0	0	HKE 138	.	.	.	.	.	.	.	.	.
136	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 163	0	2	0	0	2	0	1	0	0
137	0	1	0	0	2	0	3	0	0	20-4	.	.	.	.	.	.	.	.	.	HKE 164	.	.	.	.	.	.	.	.	.
138	.	.	.	.	.	.	.	.	.	20-4	0	0	0	0	2	0	0	0	0	HKE 184	0	2	0	0	2	0	0	0	0
139	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 214	0	1	0	1	2	0	0	0	0
140	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 223	0	2	0	0	3	0	1	0	0
141	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 11	0	1	0	0	2	0	2	0	0
142	.	.	.	.	.	.	.	.	.	20-4	0	1	0	0	1	0	0	0	0	HKE 13	.	.	.	.	.	.	.	.	.
143	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 48	0	1	0	0	3	0	2	0	0
144	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 63	.	.	.	.	.	.	.	.	.
145	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 117	0	2	0	0	1	0	0	0	0
146	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 122	0	3	0	0	3	0	1	0	0
147	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 157	0	2	0	0	2	0	0	0	0
148	0	2	0	0	2	0	0	0	0	20-4	.	.	.	.	.	.	.	.	.	HKE 166	.	.	.	.	.	.	.	.	.

	ESOPHAGUS										NONGLANDULAR STOMACH										HINTON	GLANDULAR STOMACH								
	Tunica mucosae					T. muscularis					Tunica mucosae					T. muscularis						Tunica mucosae					T. muscularis			
#	ATR	EGL	HEM	NEC	FIB	HEM	NEC	FIB	THB	Exp.	ATR	EGL	HEM	NEC	FIB	HEM	NEC	FIB	THB	#	ATR	EGL	HEM	NEC	FIB	HEM	NEC	FIB	THB
149	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 224	.	.	.	.	.	.	.	.	.
150	0	1	0	0	2	0	1	0	0	20-4	.	.	.	.	.	.	.	.	.	HKE 232	.	.	.	.	.	.	.	.	.
151	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 6	0	1	0	1	2	0	0	0	0
152	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 17	0	2	0	0	3	0	2	0	0
153	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 27	0	2	0	1	2	0	2	0	0
154	0	2	0	1	2	0	0	0	0	20-4	.	.	.	.	.	.	.	.	.	HKE 39	.	.	.	.	.	.	.	.	.
155	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 69	0	1	0	0	2	0	0	0	0
156	0	1	0	0	2	0	0	0	0	20-4	.	.	.	.	.	.	.	.	.	HKE 112	.	.	.	.	.	.	.	.	.
157	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 127	0	1	0	0	1	0	0	0	0
158	.	.	.	.	.	.	.	.	.	20-4	0	1	0	0	2	0	0	0	0	HKE 169	.	.	.	.	.	.	.	.	.
159	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 177	0	1	0	1	2	0	3	0	0
160	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 196	0	1	0	0	2	0	3	0	0
161	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 2	0	1	0	0	3	0	0	0	0
162	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 38	0	1	0	0	3	0	1	0	0
163	0	1	0	1	3	0	1	0	0	20-4	.	.	.	.	.	.	.	.	.	HKE 53	0	1	0	1	2	0	1	0	0
164	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 55	0	2	0	0	1	0	0	0	0
165	0	3	0	0	3	0	3	0	0	20-4	.	.	.	.	.	.	.	.	.	HKE 68	0	3	0	0	2	0	3	0	0
166	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 147	0	3	0	0	1	0	0	0	0
167	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 168	0	2	0	0	1	0	0	0	0
168	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 192	0	3	0	0	1	0	0	0	0
169	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 221	0	3	0	0	1	0	1	0	0
170	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	HKE 238	0	3	0	0	2	0	1	0	0
171	.	.	.	.	.	.	.	.	.	20-4D	.	.	.	.	.	.	.	.	.	HKE 23	0	2	0	0	1	0	0	0	0
172	.	.	.	.	.	.	.	.	.	20-4D	0	2	0	0	3	0	0	0	0	HKE 52	0	3	0	1	2	0	1	0	0
173	.	.	.	.	.	.	.	.	.	20-4D	.	.	.	.	.	.	.	.	.	HKE 94	0	3	0	0	1	0	0	0	0
174	.	.	.	.	.	.	.	.	.	20-4D	.	.	.	.	.	.	.	.	.	HKE 106	0	1	0	0	1	0	0	0	0
175	.	.	.	.	.	.	.	.	.	20-4D	.	.	.	.	.	.	.	.	.	HKE 116	.	.	.	.	.	.	.	.	.
176	.	.	.	.	.	.	.	.	.	20-4D	.	.	.	.	.	.	.	.	.	HKE 119	0	2	0	0	3	0	0	0	0
177	.	.	.	.	.	.	.	.	.	20-4D	.	.	.	.	.	.	.	.	.	HKE 134	.	.	.	.	.	.	.	.	.
178	.	.	.	.	.	.	.	.	.	20-4D	.	.	.	.	.	.	.	.	.	HKE 170	0	2	0	0	2	0	0	0	0
179	.	.	.	.	.	.	.	.	.	20-4D	.	.	.	.	.	.	.	.	.	HKE 172	0	3	0	0	1	0	1	0	0
180	.	.	.	.	.	.	.	.	.	20-4D	.	.	.	.	.	.	.	.	.	HKE 233	0	1	0	0	2	0	1	0	0
181	.	.	.	.	.	.	.	.	.	20-4D	.	.	.	.	.	.	.	.	.	HKE 4	0	1	0	0	1	0	2	0	0
182	.	.	.	.	.	.	.	.	.	20-4D	.	.	.	.	.	.	.	.	.	HKE 9	.	.	.	.	.	.	.	.	.
183	.	.	.	.	.	.	.	.	.	20-4D	.	.	.	.	.	.	.	.	.	HKE 56	0	1	0	0	3	0	0	0	0
184	.	.	.	.	.	.	.	.	.	20-4D	.	.	.	.	.	.	.	.	.	HKE 71	0	2	0	0	2	0	0	0	0
185	.	.	.	.	.	.	.	.	.	20-4D	.	.	.	.	.	.	.	.	.	HKE 113	0	1	0	0	2	0	0	0	0
186	0	2	0	1	3	0	0	0	0	20-4D	.	.	.	.	.	.	.	.	.	HKE 143	.	.	.	.	.	.	.	.	.
187	.	.	.	.	.	.	.	.	.	20-4D	.	.	.	.	.	.	.	.	.	HKE 175	0	1	0	0	3	0	2	0	0
188	.	.	.	.	.	.	.	.	.	20-4D	.	.	.	.	.	.	.	.	.	HKE 191	0	2	0	0	3	1	2	0	0
189	.	.	.	.	.	.	.	.	.	20-4D	.	.	.	.	.	.	.	.	.	HKE 209	0	1	0	0	3	0	0	0	0
190	0	1	0	0	3	0	1	0	0	20-4D	.	.	.	.	.	.	.	.	.	HKE 213	.	.	.	.	.	.	.	.	.

	ESOPHAGUS										NONGLANDULAR STOMACH											GLANDULAR STOMACH									
	Tunica mucosae					T. muscularis					Tunica mucosae					T. muscularis						Tunica mucosae					T. muscularis				
#	ATR	EGL	HEM	NEC	FIB	HEM	NEC	FIB	THB	Exp.	ATR	EGL	HEM	NEC	FIB	HEM	NEC	FIB	THB	HINTON #	ATR	EGL	HEM	NEC	FIB	HEM	NEC	FIB	THB
191	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	HKE 32	0	1	0	0	2	0	2	0	0
192	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	HKE 98	0	1	0	1	1	0	2	0	0
193	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	HKE 135	.	.	.	.	.	.	.	.	.
194	0	1	0	0	3	0	3	0	0	60-4	.	.	.	.	.	.	.	.	.	HKE 149	0	1	0	0	1	0	2	0	0
195	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	HKE 189	0	1	0	0	2	0	0	0	0
196	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	HKE 206	0	1	0	2	3	0	1	0	0
197	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	HKE 218	.	.	.	.	.	.	.	.	.
198	0	1	0	0	3	0	0	0	0	60-4	.	.	.	.	.	.	.	.	.	HKE 234	.	.	.	.	.	.	.	.	.
199	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	HKE 239	0	1	0	0	1	0	0	0	0
200	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	HKE 240	0	1	0	0	3	0	2	0	0
201	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	HKE 30	0	1	0	1	2	0	3	0	0
202	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	HKE 74	0	3	0	0	1	0	0	0	0
203	0	0	0	1	2	0	2	0	0	60-4	.	.	.	.	.	.	.	.	.	HKE 100	0	1	0	1	1	0	1	0	0
204	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	HKE 146	0	1	0	0	1	0	0	0	0
205	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	HKE 190	0	3	0	1	2	0	2	0	0
206	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	HKE 200	0	2	0	0	3	0	3	0	0
207	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	HKE 202	0	1	0	0	2	0	3	0	0
208	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	HKE 208	0	1	0	0	2	0	0	0	0
209	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	HKE 217	0	1	0	0	3	0	3	0	0
210	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	HKE 222	0	1	0	0	2	0	3	0	0
211	.	.	.	.	.	.	.	.	.	60-4	0	1	0	0	3	0	1	0	0	HKE 1	.	.	.	.	.	.	.	.	.
212	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	HKE 5	0	1	0	0	2	0	3	0	0
213	0	1	0	0	1	0	3	0	0	60-4	.	.	.	.	.	.	.	.	.	HKE 16	.	.	.	.	.	.	.	.	.
214	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	HKE 24	0	1	0	1	2	0	2	0	0
215	0	3	0	0	2	0	3	0	0	60-4	.	.	.	.	.	.	.	.	.	HKE 82	.	.	.	.	.	.	.	.	.
216	0	1	0	0	1	0	3	0	0	60-4	.	.	.	.	.	.	.	.	.	HKE 96	.	.	.	.	.	.	.	.	.
217	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	HKE 110	0	2	0	0	2	0	1	0	0
218	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	HKE 132	0	3	0	0	1	0	0	0	0
219	0	3	1	1	3	0	1	0	0	60-4	.	.	.	.	.	.	.	.	.	HKE 158	0	3	0	0	1	0	0	0	0
220	.	.	.	.	.	.	.	.	.	60-4	0	0	0	0	2	0	0	0	0	HKE 193	.	.	.	.	.	.	.	.	.
221	.	.	.	.	.	.	.	.	.	CONTROL	.	.	.	.	.	.	.	.	.	HKE 12	0	3	0	0	1	0	0	0	0
222	.	.	.	.	.	.	.	.	.	CONTROL	.	.	.	.	.	.	.	.	.	HKE 88	.	.	.	.	.	.	.	.	.
223	.	.	.	.	.	.	.	.	.	CONTROL	.	.	.	.	.	.	.	.	.	HKE 91	0	2	0	0	1	0	0	0	0
224	.	.	.	.	.	.	.	.	.	CONTROL	.	.	.	.	.	.	.	.	.	HKE 99	0	1	0	0	2	0	1	0	0
225	0	3	0	0	1	0	0	0	0	CONTROL	.	.	.	.	.	.	.	.	.	HKE 121	.	.	.	.	.	.	.	.	.
226	.	.	.	.	.	.	.	.	.	CONTROL	0	1	0	0	1	0	0	0	0	HKE 133	0	3	0	0	2	0	0	0	0
227	.	.	.	.	.	.	.	.	.	CONTROL	.	.	.	.	.	.	.	.	.	HKE 194	0	2	0	0	1	0	0	0	0
228	.	.	.	.	.	.	.	.	.	CONTROL	.	.	.	.	.	.	.	.	.	HKE 211	0	2	0	0	3	0	0	0	0
229	.	.	.	.	.	.	.	.	.	CONTROL	.	.	.	.	.	.	.	.	.	HKE 220	0	3	0	0	3	0	0	0	0
230	0	1	0	0	3	0	0	0	0	CONTROL	.	.	.	.	.	.	.	.	.	HKE 236	.	.	.	.	.	.	.	.	.
231	.	.	.	.	.	.	.	.	.	P CONTROL	.	.	.	.	.	.	.	.	.	HKE 42	0	1	0	0	1	0	0	0	0
232	.	.	.	.	.	.	.	.	.	P CONTROL	.	.	.	.	.	.	.	.	.	HKE 60	0	3	0	0	1	0	0	0	0

	ESOPHAGUS											NONGLANDULAR STOMACH										HINTON	GLANDULAR STOMACH									
	Tunica mucosae					T. muscularis						Tunica mucosae					T. muscularis						Tunica mucosae					T. muscularis				
#	ATR	EGL	HEM	NEC	FIB	HEM	NEC	FIB	THB	Exp.		ATR	EGL	HEM	NEC	FIB	HEM	NEC	FIB	THB	#	ATR	EGL	HEM	NEC	FIB	HEM	NEC	FIB	THB	
233	.	.	.	.	.	.	.	.	.	P	CONTROL	.	.	.	.	.	.	.	.	.	HKE 103	0	2	0	0	3	0	0	0	0	
234	0	1	0	0	1	0	0	0	0	P	CONTROL	.	.	.	.	.	.	.	.	.	HKE 128	.	.	.	.	.	.	.	.	.	
235	0	2	0	0	2	0	0	0	0	P	CONTROL	.	.	.	.	.	.	.	.	.	HKE 129	.	.	.	.	.	.	.	.	.	
236	.	.	.	.	.	.	.	.	.	P	CONTROL	0	2	0	0	2	0	0	0	0	HKE 131	0	2	0	0	2	0	0	0	0	
237	.	.	.	.	.	.	.	.	.	P	CONTROL	.	.	.	.	.	.	.	.	.	HKE 142	0	3	0	0	2	0	0	0	0	
238	.	.	.	.	.	.	.	.	.	P	CONTROL	.	.	.	.	.	.	.	.	.	HKE 150	0	3	0	0	1	0	0	0	0	
239	.	.	.	.	.	.	.	.	.	P	CONTROL	.	.	.	.	.	.	.	.	.	HKE 151	0	3	0	0	2	0	0	0	0	
240	0	3	0	0	2	0	0	0	0	P	CONTROL	.	.	.	.	.	.	.	.	.	HKE 201	.	.	.	.	.	.	.	.	.	

SUMMARY STATISTICS:

	Mean Lesion Scores										Mean Lesion Scores										Mean Lesion Scores									
	ESOPHAGUS										NONGLANDULAR STOMACH										GLANDULAR STOMACH									
	Tunica mucosae					T. muscularis					Tunica mucosae					T. muscularis					Tunica mucosae					T. muscularis				
Day	ATR	EGL	HEM	NEC	FIB	HEM	NEC	FIB	THB	Exp	ATR	EGL	HEM	NEC	FIB	HEM	NEC	FIB	THB	EXP.	ATR	EGL	HEM	NEC	FIB	HEM	NEC	FIB	THB
1	0	2	0	0	3	0	1	0	0	LO	0	1	0	0	2	0	0	0	0	LOW	0	2.9	0	.11	1.3	0	.11	0	0
2	.5	2.5	0	0	2	0	0	0	0	LO	0	1	0	0	2.5	0	0	0	0	LOW	.5	1.8	0	0	1.8	.17	.17	0	0
4	0	2.5	0	0	2	0	0	0	0	LO	0	1.5	0	0	3	0	.5	0	0	LOW	0	2	0	.2	1.6	0	0	0	0
7	.	.	.	.	.	.	.	.	.	LO	.	.	.	.	.	.	.	.	.	LOW	0	1.9	0	.3	2.2	0	.2	0	.1
10	0	2.5	0	0	3	0	0	0	0	LO	0	0	0	0	1	0	0	0	0	LOW	.29	2.4	0	.29	2.4	0	.57	0	.14
14	0	1.8	0	.25	2.3	0	1	0	0	LO	.	.	.	.	.	.	.	.	.	LOW	0	2.6	0	.14	1.7	0	.29	0	0
1	.	.	.	.	.	.	.	.	.	HI	0	.75	0	0	2.3	0	.75	.25	0	HIGH	.71	1.3	0	.29	2	0	1.4	0	0
2	1	1.3	0	.75	2.8	.25	.75	0	0	HI	0	.5	0	0	1.5	0	0	0	0	HIGH	.25	2.5	0	0	2.3	0	.25	0	0
4	0	3	0	0	3	0	0	0	0	HI	.	.	.	.	.	.	.	.	.	HIGH	.63	2.3	.5	0	2.4	.38	.13	0	0
7	1.6	2	1	0	2.8	0	.4	0	0	HI	.	.	.	.	.	.	.	.	.	HIGH	0	1.6	0	.6	2.6	0	.8	.2	0
10	.5	2	0	0	2	0	0	0	0	HI	.	.	.	.	.	.	.	.	.	HIGH	.25	2.1	.13	.5	2	.13	.63	0	0
14	0	1.7	0	0	2.3	0	.67	0	0	HI	2	1	2	3	3	1	2	0	0	HIGH	0	1.5	0	0	2.5	0	.5	0	0
1	.25	.75	0	0	2.3	0	1.3	0	0	20	.	.	.	.	.	.	.	.	.	20-4	0	1.9	0	.29	2	0	1.3	0	0
2	0	1	0	0	2.5	0	1.5	0	0	20	0	.5	0	0	2	0	0	0	0	20-4	0	1.6	0	.38	1.9	0	1.1	.13	.13
4	0	1.5	0	0	2	0	.5	0	0	20	0	1	0	0	1	0	0	0	0	20-4	0	1.8	0	0	2.2	0	1	0	0
7	0	1.5	0	.5	2	0	0	0	0	20	0	1	0	0	2	0	0	0	0	20-4	0	1.3	0	.43	2	0	1.4	0	0
10	0	2	0	.5	3	0	2	0	0	20	.	.	.	.	.	.	.	.	.	20-4	0	2.2	0	.1	1.7	0	.7	0	0
7	.	.	.	.	.	.	.	.	.	4d	0	2	0	0	3	0	0	0	0	20-4D	0	2.1	0	.13	1.6	0	.38	0	0
11	0	1.5	0	.5	3	0	.5	0	0	4d	.	.	.	.	.	.	.	.	.	20-4D	0	1.3	0	0	2.4	.14	.86	0	0
1	0	1	0	0	3	0	1.5	0	0	60	.	.	.	.	.	.	.	.	.	60-4	0	1	0	.43	1.9	0	1.3	0	0
2	0	0	0	1	2	0	2	0	0	60	.	.	.	.	.	.	.	.	.	60-4	0	1.5	0	.3	1.9	0	1.8	0	0
4	0	2	.25	.25	1.8	0	2.5	0	0	60	0	.5	0	0	2.5	0	.5	0	0	60-4	0	2	0	.2	1.6	0	1.2	0	0
3	0	2	0	0	2	0	0	0	0	C	0	1	0	0	1	0	0	0	0	CONTROL	0	2.3	0	0	1.9	0	.14	0	0
3	0	2	0	0	1.7	0	0	0	0	PC	0	2	0	0	2	0	0	0	0	P CONTROL	0	2.4	0	0	1.7	0	0	0	0

Table VI-3. Histopathology of gill, ovary, testes, nares, and skeletal muscle in Pacific herring adults exposed to crude oil in the Laboratory.

Key to table symbols:
Hinton number = random (processing) number generated by Dr. Hinton's Laboratory
Lesion scores: none (0), mild (1), moderate (2), severe (3), not present (.)
Exp. = same as for Table VI-1 above

GILL:
- squamous cell hyperplasia (SCH)
- lymphocytes (LYM)
- eosinophilic granular leukocytes (EGL)
- macrophage aggregates (MA)
- lamellar capillary anuerysms (LCA)
- hemorrhage (HEM)
- thrombosis (THB)
- vasculitis (VAS)
- *Epitheliocystis*-like organism (EPI)
- Ichthyophonus (ICH)

OVARY:
- percent yolked eggs (%Y)
- oocyte atresia (OA)
- macrophage aggregates (MA)
- thrombosis (THB)

TESTES:
- sperm depletion (DEP)
- interstitial vacuolization (IVZ)
- vacuolar degeneration of spermatogonia (VD)
- macrophage aggregates (MA)
- thrombosis (THB)

NARES:
- single cell necrosis (SCN)
- mucous cell hyperplasia (MCH)
- lymphocytes (LYM)
- eosinophilic granular leukocytes (EGL)
- hemorrhage (HEM)
- thrombosis (THB)

MUSCLE:
- necrosis (NEC)
- hemorrhage (HEM)

	GILL											OVARY				TESTES						NARES						HINTON	Muscle	
#	SCH	LYM	EGL	MA	LCA	HEM	THB	VAS	EPI	ICH	Exp.	%Y	OA	MA	THB	DEP	IVZ	VD	SCN	MA	THB	SCN	MCH	LYM	EGL	HEM	THB	#	NEC	HEM
1	0	0	2	0	0	2	0	0	0	0	LOW	.	.	.	.	3	0	2	0	0	0	.	.	.	.	.	.	HKE 14	.	.
2	0	0	0	0	0	0	0	0	0	0	LOW	.	.	.	.	0	1	1	0	0	1	0	0	1	1	0	1	HKE 21	.	.
3	0	0	0	0	0	2	0	0	0	0	LOW	.	.	.	.	.	.	.	.	.	.	0	0	0	1	1	0	HKE 44	.	.
4	.	.	..	.	.	.	.	.	.	.	LOW	.	.	.	.	3	0	1	0	1	0	0	0	1	1	0	0	HKE 97	.	.
5	0	1	1	0	0	0	0	1	1	0	LOW	.	.	.	.	0	1	0	0	0	0	.	.	.	.	.	.	HKE 107	.	.
6	0	1	0	0	0	0	0	0	0	0	LOW	100	0	0	0	.	.	.	.	.	.	0	2	1	0	0	0	HKE 118	.	.
7	.	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	0	1	1	0	0	0	0	0	0	0	0	0	HKE 144	.	.
8	0	3	0	0	0	0	0	0	0	3	LOW	.	.	.	.	0	1	0	0	0	0	0	0	0	0	0	0	HKE 159	0	0
9	0	0	1	0	0	0	0	0	0	0	LOW	.	.	.	.	0	1	1	0	0	0	0	0	0	0	1	0	HKE 197	.	.
10	0	0	0	0	0	0	0	0	0	0	LOW	.	.	.	.	0	1	0	0	0	0	.	.	.	.	.	.	HKE 225	.	.
11	0	.	.	.	.	.	.	.	.	.	LOW	75	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 230	0	0
12	0	0	0	0	0	1	0	0	0	0	LOW	95	0	0	0	.	.	.	.	.	.	0	0	1	1	0	0	HKE 37	0	0
13	0	0	0	0	0	0	0	0	0	0	LOW	95	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 64	0	0
14	0	0	0	0	0	0	0	0	0	0	LOW	95	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 75	0	0
15	0	0	3	0	0	0	0	0	0	0	LOW	95	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 101	0	0
16	0	0	0	0	0	0	0	0	0	0	LOW	95	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 102	0	0
17	0	0	0	0	0	0	0	0	0	0	LOW	.	.	.	.	.	.	.	.	.	.	0	2	1	0	0	1	HKE 114	0	0
18	0	0	2	0	0	0	0	0	0	0	LOW	95	0	0	0	.	.	.	.	.	.	0	1	0	0	1	0	HKE 156	0	0
19	0	0	0	0	0	0	0	0	0	0	LOW	.	.	.	.	0	1	0	0	0	0	0	0	0	0	0	0	HKE 178	0	2
20	0	0	0	0	0	0	0	0	0	0	LOW	75	0	0	0	.	.	.	.	.	.	.	.	.	.	.	.	HKE 195	0	0
21	0	0	0	0	0	0	0	0	0	0	LOW	.	.	.	.	0	1	0	0	0	0	0	0	0	0	0	0	HKE 215	0	0
22	0	0	0	0	0	0	0	0	0	0	LOW	.	.	.	.	.	.	.	.	.	.	0	1	0	1	0	0	HKE 8	0	0
23	0	0	1	0	0	2	0	0	0	0	LOW	.	.	.	.	0	1	0	0	0	0	0	0	0	0	1	0	HKE 18	0	0

	GILL											OVARY				TESTES						NARES						HINTON	Muecle	
#	SCH	LYM	EGL	MA	LCA	HEM	THB	VAS	EPI	ICH	Exp.	%Y	OA	MA	THB	DEP	IVZ	VD	SCN	MA	THB	SCN	MCH	LYM	EGL	HEM	THB	#	NEC	HEM
24	.	.	.	.	.	.	.	.	.	.	LOW	90	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 33	0	0
25	0	0	0	0	0	1	0	0	0	0	LOW	95	0	0	0	.	.	.	.	.	.	0	1	0	0	0	0	HKE 80	0	0
26	0	2	0	0	0	0	0	0	0	0	LOW	95	0	0	0	.	.	.	.	.	.	0	0	1	0	0	0	HKE 137	0	0
27	.	.	.	.	.	.	.	.	.	.	LOW	95	0	0	0	.	.	.	.	.	.	0	1	0	0	0	0	HKE 139	0	0
28	.	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	0	0	1	1	0	0	0	0	0	0	0	0	HKE 148	0	0
29	0	1	1	0	0	1	0	0	1	0	LOW	95	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 198	0	1
30	.	.	.	.	.	.	.	.	.	.	LOW	10	0	0	0	.	.	.	.	.	.	0	1	0	0	0	0	HKE 210	0	0
31	0	0	0	0	0	0	0	0	0	0	LOW	.	.	.	.	.	.	.	.	.	.	1	1	0	0	0	0	HKE 219	0	1
32	0	0	0	0	0	1	0	0	0	0	LOW	.	.	.	.	0	2	0	0	1	0	0	3	0	0	0	0	HKE 28	0	0
33	0	0	0	0	0	0	0	0	0	0	LOW	95	1	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 36	0	0
34	0	1	0	0	0	2	0	0	0	0	LOW	.	.	.	.	0	0	0	0	0	0	0	1	0	0	0	0	HKE 49	0	0
35	.	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	0	1	0	0	0	0	.	.	.	.	.	.	HKE 73	0	0
36	0	0	0	0	0	0	0	0	0	0	LOW	.	.	.	.	.	.	.	.	.	.	0	1	0	0	0	0	HKE 84	0	0
37	0	0	0	0	0	0	0	0	0	0	LOW	.	.	.	.	0	0	0	0	0	0	0	0	0	0	0	0	HKE 141	0	0
38	.	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	.	0	2	2	1	0	0	HKE 179	0	0
39	0	1	1	0	0	0	0	0	0	0	LOW	95	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 185	0	1
40	0	0	0	0	0	0	0	1	0	0	LOW	.	.	.	.	.	.	.	.	.	.	0	1	0	0	1	0	HKE 227	0	0
41	.	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	.	0	1	0	0	0	0	HKE 237	0	0
42	0	0	2	0	1	0	0	0	0	0	LOW	.	.	.	.	0	1	1	0	0	1	0	0	2	1	0	2	HKE 3	0	0
43	0	0	0	0	0	0	0	0	0	0	LOW	.	.	.	.	.	.	.	.	.	.	0	3	0	0	0	0	HKE 19	0	0
44	0	0	0	0	0	2	0	0	0	0	LOW	.	.	.	.	0	1	0	0	0	0	0	1	1	0	0	0	HKE 22	0	0
45	0	0	0	0	0	0	0	0	0	0	LOW	.	.	.	.	.	.	.	.	.	.	0	0	0	0	2	0	HKE 29	0	0
46	0	0	0	0	0	0	0	0	0	0	LOW	.	.	.	.	0	2	1	0	0	0	0	0	0	0	0	0	HKE 62	0	0
47	.	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	1	3	2	1	0	0	0	0	0	0	0	0	HKE 140	0	0
48	0	1	0	0	0	0	0	0	0	0	LOW	100	0	0	0	.	.	.	.	.	.	.	.	.	.	.	.	HKE 155	0	0
49	0	0	1	0	0	0	0	0	0	0	LOW	95	0	0	0	.	.	.	.	.	.	.	.	.	.	.	.	HKE 199	0	0
50	.	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	0	2	2	0	0	0	0	1	0	0	0	0	HKE 205	0	0
51	0	2	0	0	0	0	0	0	0	0	LOW	.	.	.	.	1	2	1	0	0	0	0	0	1	0	0	0	HKE 216	0	0
52	0	1	1	0	0	0	1	0	1	0	LOW	90	0	0	0	.	.	.	.	.	.	0	2	1	0	0	0	HKE 10	0	0
53	0	1	1	0	0	0	0	0	1	0	LOW	.	.	.	.	.	.	.	.	.	.	0	1	1	0	2	0	HKE 15	0	0
54	0	0	2	0	0	0	0	0	0	0	LOW	.	.	.	.	0	1	0	0	0	0	0	0	0	0	1	0	HKE 34	0	0
55	.	.	.	.	.	.	.	.	.	.	LOW	.	.	.	.	.	.	.	.	.	.	0	0	0	0	2	0	HKE 78	.	.
56	0	0	0	0	0	0	0	0	0	0	LOW	.	.	.	.	0	1	2	0	0	1	0	0	0	0	0	1	HKE 86	0	0
57	0	0	0	0	2	0	0	0	0	0	LOW	.	.	.	.	0	1	2	1	0	0	0	0	0	0	0	0	HKE 108	0	0
58	0	1	0	0	0	0	0	0	0	0	LOW	.	.	.	.	.	.	.	.	.	.	0	0	0	0	0	0	HKE 109	0	0
59	0	1	1	0	0	0	0	0	0	0	LOW	.	.	.	.	0	0	1	0	0	0	0	0	0	0	0	0	HKE 173	0	0
60	0	0	0	0	0	1	0	0	0	0	LOW	.	.	.	.	0	1	1	0	0	0	0	2	0	0	1	0	HKE 176	0	0
61	.	.	.	.	.	.	.	.	.	.	HIGH	90	0	0	0	.	.	.	.	.	.	0	2	0	0	0	0	HKE 31	.	.
62	0	0	0	0	0	0	0	0	0	0	HIGH	95	0	0	0	.	.	.	.	.	.	.	.	.	.	.	.	HKE 45	.	.
63	0	0	0	0	.	.	.	.	.	.	HIGH	.	.	.	.	0	1	1	0	0	0	0	1	0	0	0	0	HKE 61	.	.
64	.	.	.	.	.	.	.	.	.	2	HIGH	.	.	.	.	.	.	.	.	.	.	0	0	1	1	0	0	HKE 67	.	.
65	0	0	0	0	0	0	0	0	0	0	HIGH	.	.	.	.	.	.	.	.	.	.	0	0	0	0	0	0	HKE 81	0	0
66	.	.	.	.	.	.	.	.	.	.	HIGH	95	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 130	.	.
67	.	.	.	.	.	.	.	.	.	.	HIGH	95	0	0	0	.	.	.	.	.	.	.	.	.	.	.	.	HKE 162	.	.
68	.	.	.	.	.	.	.	.	.	.	HIGH	5	0	0	0	.	.	.	.	.	.	0	2	0	0	0	0	HKE 165	.	.

	GILL											OVARY				TESTES						NARES						HINTON	Muscle	
#	SCH	LYM	EGL	MA	LCA	HEM	THB	VAS	EPI	ICH	Exp.	%Y	OA	MA	THB	DEP	IVZ	VD	SCN	MA	THB	SCN	MCH	LYM	EGL	HEM	THB	#	NEC	HEM
69	.	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	0	1	1	0	0	0	.	.	.	.	.	.	HKE 171	.	.
70	.	.	.	.	.	.	.	.	.	.	HIGH	95	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 174	.	.
71	0	0	0	0	0	0	0	0	0	0	HIGH	95	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 47	0	0
72	0	0	0	0	0	0	0	0	0	0	HIGH	95	0	0	0	.	.	.	.	.	.	0	1	0	1	0	0	HKE 57	0	0
73	0	0	0	0	0	0	0	0	0	0	HIGH	95	0	0	0	.	.	.	.	.	.	0	1	0	0	0	0	HKE 59	0	0
74	.	.	.	.	.	.	.	.	.	.	HIGH	95	0	0	0	.	.	.	.	.	.	0	0	0	0	1	0	HKE 66	0	0
75	0	0	1	0	0	0	0	0	0	0	HIGH	.	.	.	.	0	1	0	0	0	0	0	0	0	0	0	0	HKE 70	0	0
76	0	0	0	0	0	0	0	0	0	0	HIGH	95	0	0	0	.	.	.	.	.	.	0	3	0	0	0	0	HKE 85	0	0
77	0	0	1	0	0	0	0	0	0	1	HIGH	.	.	.	.	0	1	0	0	0	0	0	0	0	0	0	0	HKE 126	0	0
78	0	0	0	0	0	0	0	0	0	0	HIGH	.	.	.	.	.	.	.	.	.	.	0	2	0	0	0	0	HKE 160	0	0
79	0	2	2	2	0	0	0	0	0	2	HIGH	.	.	.	.	0	1	0	0	0	0	0	0	1	1	0	0	HKE 203	0	0
80	0	1	1	0	0	0	0	0	0	1	HIGH	95	0	0	0	.	.	.	.	.	.	0	1	0	0	3	0	HKE 228	0	1
81	0	0	0	0	0	1	0	0	0	0	HIGH	95	0	0	0	.	.	.	.	.	.	0	0	1	0	0	0	HKE 20	0	0
82	0	0	0	0	1	0	0	0	0	0	HIGH	95	0	0	0	.	.	.	.	.	.	0	1	0	0	1	0	HKE 89	0	0
83	0	1	0	0	0	0	0	0	0	0	HIGH	.	.	.	.	0	2	0	0	0	0	0	0	1	1	0	0	HKE 95	0	0
84	0	0	0	0	0	0	0	0	0	0	HIGH	.	.	.	.	0	0	0	0	0	0	.	.	.	.	.	1	HKE 105	0	0
85	0	2	2	0	0	0	0	1	0	3	HIGH	.	.	.	.	0	1	0	0	0	0	0	1	0	0	0	0	HKE 123	0	0
86	0	1	1	0	0	0	0	0	1	0	HIGH	.	.	.	.	0	1	0	0	0	0	0	1	0	0	0	0	HKE 145	0	0
87	0	0	0	0	0	0	0	0	0	0	HIGH	.	.	.	.	0	1	1	0	0	0	0	0	1	0	0	0	HKE 152	0	1
88	0	2	2	0	0	0	0	0	0	0	HIGH	95	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 153	0	2
89	0	0	0	0	0	0	0	0	0	0	HIGH	95	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 167	0	1
90	0	1	0	1	0	0	0	0	0	0	HIGH	95	0	0	0	.	.	.	.	.	.	0	2	1	0	0	0	HKE 226	0	0
91	0	0	0	0	0	0	0	0	0	0	HIGH	95	0	0	0	.	.	.	.	.	.	.	.	.	.	.	.	HKE 7	0	0
92	0	0	0	0	0	0	0	0	0	0	HIGH	.	.	.	.	0	1	0	0	0	0	0	0	0	0	0	0	HKE 25	0	0
93	.	.	.	.	.	.	.	.	.	.	HIGH	95	0	0	0	.	.	.	.	.	.	0	1	0	0	0	0	HKE 40	0	0
94	0	0	1	0	0	0	0	0	0	0	HIGH	95	0	0	0	.	.	.	.	.	.	0	0	0	0	1	0	HKE 51	0	0
95	0	0	1	0	0	0	0	0	0	0	HIGH	100	0	0	0	.	.	.	.	.	.	0	0	0	0	1	1	HKE 154	0	0
96	0	2	2	0	0	0	0	0	0	1	HIGH	.	.	.	.	0	0	0	0	0	0	1	2	2	1	1	2	HKE 181	0	1
97	0	0	0	0	0	0	1	0	0	0	HIGH	10	0	0	0	.	.	.	.	.	.	.	.	.	.	.	.	HKE 187	1	0
98	0	0	1	0	0	0	0	0	0	0	HIGH	95	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 204	0	0
99	0	1	1	0	0	0	0	0	0	0	HIGH	.	.	.	.	0	0	0	0	0	0	0	0	0	0	0	0	HKE 212	0	0
100	0	0	0	0	0	2	0	0	0	0	HIGH	.	.	.	.	0	0	0	0	0	0	0	2	0	0	1	0	HKE 231	0	0
101	0	0	0	0	0	0	0	0	0	0	HIGH	.	.	.	.	1	0	0	1	2	0	0	2	1	0	0	0	HKE 72	0	0
102	0	3	2	0	0	0	0	0	0	0	HIGH	100	0	0	0	.	.	.	.	.	.	0	2	0	0	0	0	HKE 77	0	0
103	0	1	1	0	0	0	0	0	0	0	HIGH	5	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 93	0	0
104	0	1	1	0	1	0	0	0	0	0	HIGH	.	.	.	.	0	1	0	0	0	0	0	0	0	0	0	0	HKE 104	0	0
105	0	0	0	0	0	0	0	0	0	0	HIGH	.	.	.	.	0	3	0	0	0	0	0	0	0	0	0	0	HKE 111	0	0
106	.	.	.	.	.	.	.	.	.	.	HIGH	95	0	0	0	.	.	.	.	.	.	.	.	.	.	.	.	HKE 115	0	0
107	0	0	1	0	0	0	0	0	1	0	HIGH	.	.	.	.	.	.	.	.	.	.	0	2	1	0	0	0	HKE 136	0	0
108	0	2	1	1	0	0	0	0	1	0	HIGH	.	.	.	.	0	1	0	0	0	0	0	2	1	0	0	0	HKE 182	0	2
109	0	1	1	0	0	0	0	0	0	0	HIGH	95	0	0	0	.	.	.	.	.	.	0	0	0	0	1	0	HKE 186	0	0
110	0	1	2	0	0	0	0	0	0	0	HIGH	95	0	0	0	.	.	.	.	.	.	0	0	0	0	1	0	HKE 207	0	0
111	0	0	0	0	0	0	0	0	0	0	HIGH	.	.	.	.	0	1	1	0	0	0	0	0	0	0	0	0	HKE 26	0	0
112	.	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	HKE 79	.	.
113	1	0	0	0	0	0	0	0	0	0	HIGH	.	.	.	.	0	0	2	1	0	0	0	0	1	0	0	0	HKE 92	0	0

	GILL											OVARY				TESTES						NARES						HINTON	Muscle	
#	SCH	LYM	EGL	MA	LCA	HEM	THB	VAS	EPI	ICH	Exp.	%Y	OA	MA	THB	DEP	IVZ	VD	SCN	MA	THB	SCN	MCH	LYM	EGL	HEM	THB	#	NEC	HEM
114	.	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	HKE 124	.	.
115	.	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	0	1	1	0	0	0	.	.	.	.	.	.	HKE 125	0	1
116	0	1	0	0	0	2	0	0	0	0	HIGH	.	.	.	.	.	.	.	.	.	.	0	2	0	0	0	0	HKE 161	0	0
117	.	.	.	.	.	.	.	.	.	.	HIGH	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	HKE 180	.	.
118	.	.	.	.	.	.	.	.	.	.	*HIGH*	.	.	.	.	0	1	1	0	0	0	.	.	.	.	.	.	HKE 183	0	0
119	0	0	0	0	2	0	0	0	0	0	HIGH	100	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 229	0	0
120	0	0	0	0	0	0	0	0	0	2	HIGH	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	HKE 235	.	.
121	0	2	3	0	0	0	0	0	1	2	20-4	.	.	.	.	0	1	1	0	0	0	0	2	1	1	0	0	HKE 43	0	0
122	0	0	0	0	0	3	0	0	0	0	20-4	.	.	.	.	1	1	2	1	0	0	0	1	1	1	0	0	HKE 46	0	0
123	0	0	0	0	0	2	0	0	0	0	20-4	5	2	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 54	0	0
124	0	0	0	0	0	0	0	0	0	0	20-4	.	.	.	.	0	0	0	0	0	0	0	0	0	0	0	0	HKE 58	0	0
125	.	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	.	0	1	0	1	0	0	HKE 65	0	0
126	0	0	0	0	0	0	0	0	0	0	20-4	.	.	.	.	1	1	1	0	0	0	0	1	1	0	0	0	HKE 83	0	0
127	0	0	0	0	0	0	0	0	0	0	20-4	.	.	.	.	.	.	.	.	.	.	0	2	1	0	0	0	HKE 87	0	0
128	1	0	2	0	0	0	0	0	1	0	20-4	95	0	0	0	.	.	.	.	.	.	0	0	0	0	1	0	HKE 90	0	0
129	.	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	.	0	1	0	0	0	0	HKE 120	0	0
130	0	1	0	0	0	0	0	0	0	0	20-4	50	0	0	0	.	.	.	.	.	.	1	1	0	0	0	0	HKE 188	0	0
131	0	0	0	0	0	0	0	0	0	0	20-4	90	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 35	0	0
132	0	0	0	0	0	0	0	0	0	0	20-4	.	.	.	.	0	1	0	0	0	0	0	0	0	0	0	0	HKE 41	0	0
133	.	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	0	2	1	1	0	0	0	0	1	1	0	0	HKE 50	0	0
134	.	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	0	2	0	0	0	0	0	0	0	0	0	0	HKE 76	0	0
135	.	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	0	0	2	1	0	0	0	1	0	0	0	0	HKE 138	0	0
136	0	1	1	0	0	0	0	0	0	0	20-4	95	2	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 163	0	0
137	0	1	0	0	0	0	0	0	0	0	20-4	.	.	.	.	.	.	.	.	.	.	0	1	0	0	0	0	HKE 164	0	0
138	.	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	0	1	2	0	0	0	0	0	1	0	1	0	HKE 184	0	1
139	0	0	0	0	0	0	0	0	0	0	20-4	90	0	0	0	.	.	.	.	.	.	.	.	.	.	.	.	HKE 214	0	0
140	.	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	HKE 223	0	0
141	0	0	0	0	0	0	0	0	0	0	20-4	90	0	0	0	.	.	.	.	.	.	1	0	1	0	0	0	HKE 11	0	0
142	0	0	0	0	0	0	0	0	0	0	20-4	.	.	.	.	0	0	2	3	0	0	0	0	1	0	0	0	*HKE 13*	0	0
143	0	2	1	0	0	0	0	0	0	0	20-4	.	.	.	.	0	1	1	0	0	0	.	.	.	.	.	.	HKE 48	0	0
144	.	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	0	1	1	0	0	0	1	0	0	0	0	0	HKE 63	0	0
145	0	2	0	0	0	0	0	0	0	0	20-4	.	.	.	.	.	.	.	.	.	.	0	0	0	0	0	0	HKE 117	0	0
146	0	1	1	0	0	0	0	0	0	0	20-4	.	.	.	.	.	.	.	.	.	.	0	0	1	1	0	0	HKE 122	0	0
147	.	.	.	.	.	.	.	.	.	.	20-4	100	0	0	0	.	.	.	.	.	.	0	0	1	0	0	0	HKE 157	0	0
148	.	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	0	2	0	0	0	0	0	0	0	0	0	0	HKE 166	0	0
149	.	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	HKE 224	0	0
150	0	0	0	0	0	0	0	0	0	0	20-4	.	.	.	.	.	.	.	.	.	.	0	2	0	0	0	0	*HKE 232*	0	0
151	0	0	2	0	0	0	0	0	0	0	20-4	.	.	.	.	.	.	.	.	.	.	1	1	1	0	0	0	HKE 6	0	0
152	0	0	0	0	0	0	0	0	0	1	20-4	.	.	.	.	.	.	.	.	.	.	0	3	1	0	0	0	HKE 17	0	0
153	0	0	0	0	0	0	0	0	0	0	20-4	.	.	.	.	.	.	.	.	.	.	0	0	0	0	0	0	HKE 27	0	0
154	0	0	0	0	0	0	0	0	0	0	20-4	95	0	0	0	.	.	.	.	.	.	0	1	1	0	0	0	HKE 39	0	0
155	0	0	2	0	0	0	0	0	0	0	20-4	.	.	.	.	.	.	.	.	.	.	0	0	0	0	0	0	HKE 69	0	0
156	0	2	0	0	0	0	0	0	0	0	20-4	.	.	.	.	0	0	1	0	0	0	0	0	0	0	0	0	HKE 112	0	0
157	0	1	0	0	0	0	0	0	0	0	20-4	.	.	.	.	0	1	2	1	0	0	0	1	0	0	0	0	HKE 127	0	0
158	.	.	.	.	.	.	.	.	.	.	20-4	5	0	0	0	.	.	.	.	.	.	0	1	0	0	0	0	*HKE 169*	0	0

	GILL											OVARY				TESTES							NARES							HINTON	Muscle	
#	SCH	LYM	EGL	MA	LCA	HEM	THB	VAS	EPI	ICH	Exp.	%Y	OA	MA	THB	DEP	IVZ	VD	SCN	MA	THB	SCN	MCH	LYM	EGL	HEM	THB	#	NEC	HEM
159	.	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	.	0	0	0	0	0	0	HKE 177	0	0
160	0	1	0	1	0	0	0	0	0	0	20-4	20	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 196	0	0
161	.	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	1	0	2	3	0	0	0	0	1	0	0	0	HKE 2	1	0
162	0	0	0	0	0	0	0	0	0	0	20-4	.	.	.	.	1	1	3	1	0	0	0	1	1	0	0	0	HKE 38	0	0
163	0	2	2	0	0	0	0	0	0	0	20-4	95	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 53	0	0
164	0	0	0	0	0	0	0	0	0	0	20-4	0	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 55	0	0
165	0	0	0	0	0	0	0	0	0	0	20-4	.	.	.	.	0	0	1	1	0	0	0	0	0	0	0	0	HKE 68	0	0
166	0	0	0	0	1	0	0	0	0	0	20-4	.	.	.	.	0	1	0	0	0	0	0	0	0	0	0	0	HKE 147	0	0
167	.	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	.	.	.	.	.	.	0	0	1	0	0	0	HKE 168	0	0
168	.	.	.	.	.	.	.	.	.	.	20-4	.	.	.	.	0	0	0	0	0	0	.	.	.	.	.	.	HKE 192	0	0
169	0	1	1	0	0	0	0	0	0	0	20-4	0	0	0	0	.	.	.	.	.	.	0	3	1	0	0	0	HKE 221	0	0
170	.	.	.	.	.	.	.	.	.	.	20-4	0	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 238	0	0
171	.	.	.	.	.	.	.	.	.	.	20-4D	95	0	0	0	.	.	.	.	.	.	0	1	1	0	0	0	HKE 23	0	0
172	0	0	0	0	0	0	0	0	0	0	20-4D	.	.	.	.	1	0	1	1	0	0	0	0	1	1	0	0	HKE 52	0	0
173	.	2	1	.	.	.	.	.	.	.	20-4D	.	.	.	.	.	.	.	.	.	.	0	0	0	0	0	0	HKE 94	0	0
174	0	1	1	0	0	0	0	0	0	0	20-4D	.	.	.	.	0	1	0	0	0	0	0	0	0	0	0	0	HKE 106	0	0
175	0	0	0	0	0	0	0	0	0	0	20-4D	80	0	0	0	.	.	.	.	.	.	0	0	1	0	0	0	HKE 116	0	0
176	0	0	0	0	0	0	0	0	0	0	20-4D	95	0	0	0	.	.	.	.	.	.	0	0	0	0	1	0	HKE 119	0	0
177	0	0	0	0	0	0	0	0	0	0	20-4D	.	.	.	.	0	1	0	0	0	0	0	1	1	0	0	0	HKE 134	0	0
178	0	2	1	0	0	0	0	1	0	0	20-4D	.	.	.	.	0	1	2	1	0	0	0	1	0	0	1	0	HKE 170	0	0
179	.	.	.	.	.	.	.	.	.	.	20-4D	100	0	0	0	.	.	.	.	.	.	.	.	.	.	.	.	HKE 172	0	0
180	.	.	.	.	.	.	.	.	.	.	20-4D	.	.	.	.	0	1	2	1	0	0	0	1	2	0	0	0	HKE 233	0	0
181	0	0	0	0	0	0	0	0	0	0	20-4D	.	.	.	.	0	2	0	0	0	0	.	.	.	.	.	.	HKE 4	0	0
182	0	0	0	0	1	3	0	0	2	0	20-4D	.	.	.	.	0	0	1	0	0	0	0	0	1	0	1	0	HKE 9	0	0
183	0	0	0	0	0	0	0	0	0	0	20-4D	80	0	0	0	.	.	.	.	.	.	0	0	1	0	0	0	HKE 56	0	0
184	0	0	0	0	0	0	0	0	1	0	20-4D	.	.	.	.	1	1	2	1	0	0	0	0	1	0	0	0	HKE 71	0	0
185	.	.	.	.	.	.	.	.	.	.	20-4D	100	0	0	0	.	.	.	.	.	.	0	0	1	1	0	0	HKE 113	0	0
186	0	3	0	0	0	0	0	1	1	0	20-4D	95	0	0	0	.	.	.	.	.	.	0	0	1	0	2	2	HKE 143	0	0
187	0	2	0	0	0	0	0	0	0	0	20-4D	.	.	.	.	.	.	.	.	.	.	0	1	0	0	0	0	HKE 175	0	0
188	.	.	.	.	.	.	.	.	.	.	20-4D	.	.	.	.	0	1	2	0	0	0	0	1	1	0	0	0	HKE 191	0	1
189	0	0	0	0	1	0	0	0	0	0	20-4D	.	.	.	.	0	2	2	0	0	0	0	0	0	0	0	0	HKE 209	0	0
190	.	.	.	.	.	.	.	.	.	.	20-4D	.	.	.	.	2	1	1	0	0	0	0	0	1	0	0	0	HKE 213	0	0
191	.	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	0	1	0	0	0	0	0	2	1	0	0	0	HKE 32	0	0
192	.	.	.	.	.	.	.	.	.	.	60-4	95	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 98	0	0
193	.	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	.	0	1	1	0	0	0	HKE 135	0	0
194	.	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	0	1	2	1	0	0	0	1	0	0	0	0	HKE 149	0	3
195	.	.	.	.	.	.	.	.	.	.	60-4	0	0	0	0	.	.	.	.	.	.	0	0	0	0	3	0	HKE 189	0	0
196	.	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	.	0	0	0	0	0	0	HKE 206	0	0
197	.	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	.	0	2	.	.	0	0	HKE 218	0	0
198	.	.	.	.	.	.	.	.	.	.	60-4	95	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 234	0	0
199	.	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	0	2	0	0	0	0	0	0	0	0	0	0	HKE 239	0	0
200	.	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	0	0	0	0	0	0	0	0	0	0	0	0	HKE 240	3	3
201	0	0	1	0	0	0	0	0	0	0	60-4	100	0	0	0	.	.	.	.	.	.	1	1	1	0	2	0	HKE 30	0	0
202	.	.	.	.	.	.	.	.	.	1	60-4	.	.	.	.	0	1	2	0	0	0	0	0	1	0	0	0	HKE 74	0	1
203	0	1	0	0	0	0	0	0	0	0	60-4	.	.	.	.	1	3	1	0	0	0	0	0	0	0	0	0	HKE 100	0	0

	GILL											OVARY				TESTES						NARES						HINTON	Muscle	
#	SCH	LYM	EGL	MA	LCA	HEM	THB	VAS	EPI	ICH	Exp.	%Y	OA	MA	THB	DEP	IVZ	VD	SCN	MA	THB	SCN	MCH	LYM	EGL	HEM	THB	#	NEC	HEM
204	.	.	.	.	.	.	.	.	.	.	60-4	100	0	0	0	.	.	.	.	.	.	0	2	0	0	0	0	HKE 146	0	0
205	0	1	1	0	0	0	0	0	0	0	60-4	95	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 190	0	0
206	0	2	0	0	0	0	0	0	0	0	60-4	.	.	.	.	.	.	.	.	.	.	0	1	1	0	0	0	HKE 200	0	0
207	0	2	1	0	0	0	0	0	0	0	60-4	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	HKE 202	0	0
208	.	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	0	1	1	0	0	0	0	2	0	0	1	1	HKE 208	0	1
209	0	0	0	0	0	0	0	0	0	0	60-4	.	.	.	.	0	2	2	1	0	0	0	0	0	0	0	0	HKE 217	0	0
210	.	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	0	1	1	0	0	0	0	0	0	0	2	1	HKE 222	0	0
211	0	2	0	0	0	0	0	0	0	0	60-4	.	.	.	.	0	1	0	1	0	0	2	0	1	0	0	0	HKE 1	0	0
212	.	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	0	1	0	0	0	0	.	.	.	.	.	.	HKE 5	0	0
213	.	.	.	.	.	.	.	.	.	.	60-4	95	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 16	0	0
214	0	1	0	0	0	0	0	0	0	0	60-4	90	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 24	0	0
215	.	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	0	0	0	0	0	0	0	0	0	0	0	0	HKE 82	0	0
216	.	.	.	.	.	.	.	.	.	1	60-4	.	.	.	.	.	.	.	.	.	.	0	2	1	1	1	0	HKE 96	0	0
217	.	.	.	.	.	.	.	.	.	.	60-4	95	0	0	0	.	.	.	.	.	.	.	.	.	.	.	.	HKE 110	0	0
218	.	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	0	1	1	0	0	0	0	1	0	0	0	0	HKE 132	0	0
219	.	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	HKE 158	.	.
220	.	.	.	.	.	.	.	.	.	.	60-4	.	.	.	.	.	.	.	.	.	.	0	2	0	0	0	0	HKE 193	0	0
221	0	0	3	0	0	3	0	0	1	0	CONTROL	.	.	.	.	.	.	.	.	.	.	0	0	0	0	1	0	HKE 12	0	0
222	0	0	0	0	0	0	0	0	0	0	CONTROL	.	.	.	.	0	1	0	0	0	0	0	0	0	0	0	0	HKE 88	0	0
223	0	2	1	0	0	0	0	2	0	0	CONTROL	95	0	0	0	.	.	.	.	.	.	0	0	1	0	0	0	HKE 91	0	0
224	0	0	0	0	1	0	1	1	0	0	CONTROL	.	.	.	.	.	.	.	.	.	.	0	0	0	0	0	0	HKE 99	0	0
225	0	0	2	0	0	2	0	0	0	0	CONTROL	.	.	.	.	0	1	0	0	0	0	0	2	0	0	0	0	HKE 121	0	0
226	0	2	2	0	0	2	0	0	0	1	CONTROL	95	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 133	0	0
227	0	1	0	0	0	0	0	0	0	0	CONTROL	.	.	.	.	.	.	.	.	.	.	0	0	0	0	0	0	HKE 194	0	0
228	0	0	0	0	0	0	0	1	0	0	CONTROL	95	0	0	0	.	.	.	.	.	.	.	.	.	.	.	.	HKE 211	0	1
229	0	3	1	1	2	0	0	0	0	0	CONTROL	.	.	.	.	0	1	1	0	0	0	0	0	0	0	0	0	HKE 220	0	0
230	0	1	0	0	0	0	0	0	1	0	CONTROL	95	0	0	0	.	.	.	.	.	.	0	0	0	0	0	1	HKE 236	0	0
0	0	0	0	0	0	0	0	0	0	P	CONTROL	95	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 42	1	0 1
232	0	0	0	0	0	0	0	0	0	0	P CONTROL	95	0	0	0	.	.	.	.	.	.	0	2	1	0	0	0	HKE 60	0	0
233	0	0	0	0	0	0	0	0	0	0	P CONTROL	95	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 103	0	0
234	0	0	1	0	2	0	0	0	0	0	P CONTROL	95	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 128	0	0
235	0	0	0	0	0	0	0	0	0	0	P CONTROL	.	.	.	.	0	1	1	0	0	0	0	0	0	0	0	0	HKE 129	0	0
236	0	2	1	0	0	0	0	0	0	1	P CONTROL	80	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 131	0	0
237	0	2	1	0	2	2	0	0	0	0	P CONTROL	95	0	0	0	.	.	.	.	.	.	0	0	0	0	0	0	HKE 142	3	2
238	0	0	0	0	0	0	0	0	0	1	P CONTROL	.	.	.	.	.	.	.	.	.	.	0	0	1	1	0	0	HKE 150	0	0
239	0	0	0	0	0	0	0	0	0	0	P CONTROL	95	0	0	0	.	.	.	.	.	.	0	0	1	1	1	0	HKE 151	0	0
240	.	.	.	.	.	.	.	.	.	.	P CONTROL	.	.	.	.	1	1	0	0	0	0	0	1	0	0	0	0	HKE 201	0	0

SUMMARY STATISTICS:

	Mean Lesion Scores											Mean Lesion Scores																Mean Score			
	GILL											OVARY				TESTES					NARES							MUSCLE		
Day	SCH	LYM	EGL	MA	LCA	HEM	THB	VAS	EPI	ICH	EXP.	%Y	OA	MA	THB	DEP	IVZ	VD	SCN	MA	THB	SCN	MCH	LYM	EGL	HEM	THB	Day	Exp	NEC	HEM
1	0	.63	.5	0	0	.5	0	.13	.13	.38	LOW	88.	0	0	0	.75	.75	.75	0	.13	.13	0	.25	.38	.38	.25	.13	1	LO	0	0
2	0	0	.5	0	0	.1	0	0	0	0	LOW	92.	0	0	0	0	1	0	0	0	0	0	.33	.22	.11	.11	.11	2	LO	0	.2
4	0	.5	.33	0	0	.67	0	0	.17	0	LOW	80	0	0	0	0	.5	.5	.5	0	0	.1	.5	.1	.1	.1	0	4	LO	0	.2
7	0	.29	.14	0	0	.43	0	.14	0	0	LOW	95	.5	0	0	0	.75	0	0	.25	0	0	1	.22	.11	.11	0	7	LO	0	.1
10	0	.38	.38	0	.13	.25	0	0	0	0	LOW	98.	0	0	0	.33	1.8	1.2	.17	0	.17	0	.63	.5	.13	.25	.25	10	LO	0	0
14	0	.5	.63	0	.25	.13	.13	0	.25	0	LOW	90	0	0	0	0	.8	1.2	.2	0	.2	0	.56	.22	0	.67	.11	14	LO	0	0
1	0	0	0	0	0	0	0	0	0	.67	HIGH	79.	0	0	0	0	1	1	0	0	0	0	.71	.14	.14	0	0	1	HI	0	0
2	0	.33	.56	.22	0	0	0	0	0	.44	HIGH	95	0	0	0	0	1	0	0	0	0	0	.8	.1	.2	.4	0	2	HI	0	.1
4	0	.7	.5	.1	.1	.1	0	.1	.1	.3	HIGH	95	0	0	0	0	1	.2	0	0	0	0	.56	.44	.11	.11	.1	4	HI	0	.4
7	0	.33	.67	0	0	.22	.11	0	0	.11	HIGH	82.	0	0	0	0	.25	0	0	0	0	.13	.63	.25	.13	.5	.38	7	HI	.1	1
10	0	1	1	.11	.11	0	0	0	.22	0	HIGH	78	0	0	0	.25	1.3	0	.25	.5	0	0	.89	.33	0	.22	0	10	HI	0	2
14	.2.	.2	0	0	.4	.4	0	0	0	.4	HIGH	100	0	0	0	0	.75	1.3	.25	0	0	0	.5	.25	0	0	0	14	HI	0	17
1	.13	.38	.63	0	0	.63	0	0	.25	.25	20-4	50	.67	0	0	.5	.75	1	.25	0	0	.1	.9	.4	.3	.1	0	1	20	0	0
2	0	.4	.2	0	0	0	0	0	0	0	20-4	92.	.67	0	0	0	1.2	1	.4	0	0	0	.25	.25	.13	.13	0	2	20	0	1
4	0	.83	.33	0	0	0	0	0	0	0	20-4	95	0	0	0	0	1	1	.75	0	0	.25	.25	.5	.13	0	0	4	20	0	0
7	0	.5	.5	.13	0	0	0	0	0	.13	20-4	40	0	0	0	0	.5	1.5	.5	0	0	.1	.7	.3	0	0	0	7	20	0	0
10	0	.5	.5	0	.17	0	0	0	0	0	20-4	24.	0	0	0	.4	.4	1.2	1	0	0	0	.44	.44	0	0	0	10	20	.1	0
7	0	.71	.43	0	0	0	0	.17	0	0	20-4D	93.	0	0	0	.2	.8	1	.6	0	0	0	.44	.67	.11	.22	0	7	4d	0	0
11	0	.71	0	0	.29	.43	0	.14	.57	0	20-4D	92.	0	0	0	.5	1.2	1.3	.17	0	0	0	.22	.78	.11	.33	.22	11	4d	0	1
1	.	.	.	.	.	.	.	.	.	.	60-4	63.	0	0	0	0	1	.5	.25	0	0	0	.6	.22	0	.3	0	1	60	.3	6
2	0	1	.5	0	0	0	0	0	0	.14	60-4	98.	0	0	0	.2	1.6	1.4	.2	0	0	.11	.67	.33	0	.56	.22	2	60	0	2
4	0	0	0	0	0	0	0	0	0	.33	60-4	93.	0	0	0	0	.75	.25	.25	0	0	.29	.71	.29	.14	.14	0	4	60	0	0
3	0	.9	.9	.1	.3	.7	.1	.4	.2	.1	CONTROL	95	0	0	0	0	1	.33	0	0	0	0	.22	.11	0	.11	.11	3	C	0	1
3	0	.44	.33	0	.44	.22	0	0	0	.22	P-CONTROL	93.	0	0	0	.5	1	.5	0	0	0	0	.3	.3	.2	.1	0	3	PC	.4	3

CHAPTER 7 - Reproductive success and histopathology of individual Prince William Sound herring 3 years after the *Exxon Valdez* oil spill.

Citation:

Kocan, R.M., G.D. Marty, M.S. Okihiro, E.D. Brown, and T.T. Baker. 1996. Reproductive success and histopathology of individual Prince William Sound herring 3 years after the *Exxon Valdez* oil spill. Canadian Journal of Fisheries and Aquatic Sciences 53:2388-2393.

Abstract: Adult Pacific herring *Clupea pallasi* collected in 1992 from a site previously oiled by the *Exxon Valdez* oil spill exhibited a lower percent hatch and produced fewer morphologically normal larvae than fish from a previously unoiled site. Possible explanations for these reproductive differences include: 1) exposure to residual oil; 2) homing of previously oil-injured fish; 3) homing of different strains of herring; 4) physical or chemical characteristics of each exposure site unrelated to oil. Differences in microscopic tissue lesions were also observed and were marginally significant between sites. Granulomatous inflammation occurred only in females from previously oiled sites, and this plus increased splenic congestion were negatively correlated to production of normal larvae. Scores for macrophage aggregates in spleen, liver, and kidney were greater in fish from previously oiled sites, particularly in males, but differences were related to age rather than exposure history. Because most of the lesions related to reproductive success were acute or subacute, differences in tissue damage could not be directly related to previous oil exposure.

CHAPTER 8 - Cytochrome P450 induction and histopathology in pre-emergent pink salmon from oiled streams in Prince William Sound, Alaska.

Citation:

Wiedmer, M., M.J. Fink, J.J. Stegeman, R. Smolowitz, G.D. Marty, and D.E. Hinton. 1996. Cytochrome P450 induction and histopathology in pre-emergent pink salmon from oiled streams in Prince William Sound, Alaska. American Fisheries Society Symposium 18:509-517.

Abstract: The March 1989 *Exxon Valdez* oil spill contaminated intertidal pink salmon *Oncorhynchus gorbuscha* spawning areas in Prince William Sound and the Gulf of Alaska. To determine if 8- to 26-month old oil remaining in some spawning areas produced physiological responses in developing pink salmon eggs and alevins, we conducted an initial assessment of cytochrome P-4501A induction and histopathologic lesion occurrence in preemergent pink salmon collected from oiled spawning substrates. Egg and alevin samples were collected from 4 oiled and 5 reference sites in Prince William Sound, Alaska, between December 1989 and May 1991. Immunohistochemical staining for cytochrome P-4501A was increased in alevins from 13 of 16 samples from oiled sites, but was not increased in any of the 7 samples from the reference sites. Cytochrome P-4501A induction was not detected in egg samples from either oiled or control sites. Persistent P-4501A staining through the end of the study was evidence for chronic exposure of 2 year-classes of pink salmon to hydrocarbon contamination. Histopathologic lesions were more frequent in alevins from oiled sites, but differences were not statistically significant, and lesion occurrence seemed dependent on developmental stage. These results provide evidence that pink salmon alevins developing in heavily oiled sites were exposed to hydrocarbons more than 2 years after the initial spill and that the hydrocarbons induced detectable physiological changes. Results of this study were used to develop appropriate treatments for oiled anadromous fish streams.

CHAPTER 9 - Histopathology in pink salmon larvae and juveniles from Prince William Sound, Alaska, damage assessment samples.

G.D. Marty and D.E. Hinton

Methods

Pre-emergent pink salmon larvae were sampled from 23 different sites in 1989 and shipped to the University of California, Davis (received September 15, 1991). Random numbers were generated for up to 32 fish from each site, for a total of 732 assigned random numbers. According to Ken Chalk, Oil Spill Studies coordinator, Commercial Fisheries, Alaska Dept. of Fish and Game, the larvae had been fixed in Bouin's, held for about 2.5 years, and then were transferred to 70% ethanol just before shipment to Davis. Morphologic detail is often lost when tissues are left in Bouin's for more than 2 weeks (ideal fixation in Bouin's is $\leq$48 hours), so we were concerned that the larvae would not be suitable for histopathology or MFO analysis. Therefore, a test run of larvae from 4 sites–2 oiled and 2 reference–was conducted. Thirty-two larvae were randomly selected from sample number **7** (stream # 630, Whale Bay, reference), **12** (stream # 678, Sleepy Bay, oiled), **13** (stream # 663, Shelter Bay, oiled), and **16** (stream # 695, Port Audrey, reference). Each larva was measured (total length) and then embedded in lateral recumbency with the left side down. For histopathologic analysis, 4 to 7 step sections were cut at intervals through each larva. Near the center of each larvae, 5 sections were saved and sent to Woods Hole Oceanographic Institute, laboratory of Dr. John Stegeman, for analysis of cytochrome P4501A (one H&E and 4 unstained sections per larva; sent 4-10-92).

Slides were read in numerical order, using the random accession numbers, so that all slides were read blindly. After histopathological analysis revealed potential differences between the groups based on exposure, larvae from the remaining 19 sites were sectioned and examined for histopathologic lesions. Because larvae from the test run (4 sites) failed to react with immunohistochemical reagents, additional sections were not analyzed for cytochrome P4501A.

Each larva was first scanned at low power (4x objective) for major organs: gonad, retina, brain, heart, gill, skin, skeletal muscle, kidney, gastrointestinal tract, yolk, liver, and spleen. The gonad, when present, was further classified as undifferentiated (or unable to classify) or female (ovary). Although immature ova were fairly easy to identify, active spermatogenesis was not observed in other gonads; hence, gonads not clearly identified as ovaries were classified as undifferentiated.

The extent of liver glycogen was ranked and scored as minimum (no obvious hepatocellular vacuoles, score = 1), moderate (volume of hepatocellular vacuoles less than nuclear volume, score = 2), or abundant (volume of hepatocellular vacuoles greater than nuclear volume, score = 3). Yolk stores were ranked as abundant/eosinophilic (score = 3), minimal (score = 2, for yolks with about equal amounts of eosinophilic protein and pale peripheral tissues), pale (score = 1, when only the pale peripheral yolk-sac tissues remained), or absent (score = 0, for no yolk sac in the sections). Lesions, other comments, pathologist's initials, and date(s) examined were also recorded.

Lesions were ranked and scored as none (0), mild (1), moderate (2), or severe (3) in relation to other similar lesions. The types of lesions looked for included:

1) Epidermal atrophy (EA) - EA was characterized by thinning of the epidermis and absence of mucous cells. Subsequent study found that epidermal atrophy is a normal physiological change in pink salmon larvae that occurs when they emerge from the gravel substrate (Marty et al. 1997a).

2) Myofiber degeneration and necrosis (MDN) - A subtle lesion in a few larvae, MDN was characterized by swelling of affected myofibers, with hypersegmentation and coagulation of the cytoplasm. Nuclei varied from pyknotic to karyorrhectic. Occasional central nuclei indicated attempts at regeneration.

3) Individual hepatocellular necrosis/apoptosis (IHN) - Only one of 738 fish in this study had Individual hepatocellular necrosis, but it was more common in other pink salmon samples (Wiedmer et al. 1996). Affected livers appeared vacuolated as a result of necrosis or apoptosis of individual hepatocytes. The spaces once occupied by hepatocytes were filled with fluid or a single macrophage.

4) Vacuolar degeneration of gastric glands (VDGG) - None of the fish in this study had VDGG. In Wiedmer et al. (1996), however, many larger fish (post-emergent pink salmon) had gastric glands in which epithelial cell cytoplasm contained large, clear, irregular vacuoles characteristic of hydropic degeneration. Nuclei of affected cells were usually normal. This is probably a normal physiologic change associated with feeding.

5) Epidermal cell necrosis or apoptosis (ECN) - ECN occurred exclusively on the ventral epidermis near the midline and usually just anterior to the anus. Characteristic ECN features included cytoplasmic vacuolation, nuclear pyknosis, occasional intracytoplasmic eosinophilic inclusions. Subsequent study found that Epidermal cell apoptosis is a normal physiological change in pink salmon larvae that occurs along the ventral midline as the yolk is absorbed (Marty et al. 1997a).

6) Gastrointestinal food (GIF) - The amount of food (e.g., arthropods or other invertebrates) in the gastrointestinal tract was ranked as none (0), min (1), moderate (2), or abundant (3); contents that were unlikely to be food particles were scored with a "?" mark. For the 1989 group, composed entirely of pre-emergent larvae, none had more than a minimal amount of food.

7) Collection trauma (CT) - Acute lesions likely associated with collection trauma (CT) were ranked as other lesions (0 - 3). The most frequent lesions associated with trauma were hemorrhage in many different organs and rupture of the yolk.

8) Saponified fat (SPF) - Normally, lipid is lost during routine processing to paraffin blocks, and fat cells are transparent when stained with hematoxylin and eosin. Many of the larvae had fat cells with cytoplasmic staining that varied from none (score = 0), mild (faint wispy

basophilic cytoplasm in a few adipocytes, score = 1), moderate (several adipocytes had uniform yellow to black cytoplasm, score = 2), to severe (nearly all adipocytes had uniform black cytoplasm, score = 3). This change was thought to represent an artifact associated with the abnormally long storage in Bouin's fixative, but the changes were scored to determine if oil exposure might have predisposed to development of the "lesion."

For quality control, each larval section, particularly the intestinal tract, was examined for extent of autolysis: 1) minimum (min), all cell membranes were intact; 2) mild, a few cells on the tips of the villi were affected; 3) moderate (mod), at least one section of intestine had transmural autolysis; and 4) severe (sev), for more than focal transmural autolysis. Next, sectioning artifact was ranked as none, mild, moderate, or severe. All sections had at least mild sectioning artifact (as expected with paraffin embedding), and other rankings were based on the extent that artifacts made interpretation of tissue sections difficult.

Statistics -

Statistical Consultant - Neil Willits, Senior Statistician, Division of Statistics, 254 Kerr Hall, University of California, Davis, 95616

For general details about the types of statistical analysis used, see "Project Statistical Analysis" (p. vii). Two additional analyses were done using the scale values derived from principal components analysis: 1) ANOVA was conducted on scale values for autolysis, artifact, yolk, saponified fat, epidermal atrophy, gastrointestinal food, and collection trauma to determine which, if any, contributed most to oiled vs. reference differences; and 2), to determine if epidermal atrophy and hepatocellular glycogen were related to yolk stores, analyses were done using both parametric (Pearson) and nonparametric (Kendall and Spearman) correlation coefficients.

Results and Discussion

Lesion scores for each fish are listed in Table IX-1. Autolysis was minimal in all but 6 fish (0.8%, 6 of 728), indicating that the tissues were generally of excellent quality for histopathologic examination. Sectioning artifact was mild or better in 88% of the fish (640 of 728), moderate in 12% (84 of 728), and severe in only 0.3% (2 of 728); i.e., despite excessively long fixation in Bouin's, suitable sections were still obtained. Nearly all important organs were included in the sections from each fish. For example, the liver was examined in 99.4% (722 of 728) of the larvae.

An interesting finding in this study was that about 43% of pink salmon larvae feed before they emerge and while they still retain abundant yolk stores. These larvae fed exclusively on invertebrates. The presence of food in the gastrointestinal tract did not correlate with the amount of hepatic glycogen or exposure to oil.

Larvae from most sites had some degree of epidermal cell apoptosis. These changes occurred with about equal frequency among all sample groups with abundant yolk stores. None of the 32 fish from Port Audrey (i.e., the longer fish that had minimal yolk stores) had epidermal cell apoptosis.

One larva from Sleepy Bay (oiled site, 89PSL545) had a focus of hyperplasia along the ventral midline that was about 500 μm wide and 200 μm high. The focus, tentatively classified as fibrous hyperplasia of the stratum compactum, was composed of irregular fibroblasts arranged into whirls. The overlying epithelium was slightly raised but not ulcerated, and had epidermal cell apoptosis. The lesion may be a hamartoma (i.e., excess production of a normal tissue) or a focus of connective tissue that failed to resorb properly when the yolk stores were depleted.

Myodegeneration and necrosis was infrequent, and in all cases was associated with, and probably caused by, trauma during the collection process. Degenerative muscle fibers often contained hemorrhage, but were never infiltrated by inflammatory cells. Hemorrhage was common throughout the body, including the cerebral ventricles, body cavity, periorbital connective tissue, and skeletal muscle. Other lesions most likely associated with collection trauma included: 1) rupture of the yolk into the body cavity, which was often accompanied by anterior infiltration of yolk material into cranial connective tissues; and 2), herniation of the yolk through the ventral body wall. Differences in traumatic lesions were independent of differences in yolk stores and total length among the groups. Although the preliminary report on pink salmon from 4 sites suggested a relationship between collection trauma and oil exposure, results from larvae from all 23 sites indicate that differences in collection-associated trauma were due to chance alone. None of the other pink salmon sample groups we analyzed had either the sample size or the completeness of sections to assess traumatic injury; further, the other pink salmon larvae--most from 1990 or 1991 (Wiedmer et al. 1996)--were collected after most acute effects of the 1989 oil spill were gone.

Statistics -

Very few values were missing, so 713 of the 728 fish (98%) were used in statistical analysis. With principal components analysis, The correlation matrix revealed that yolk stores and hepatocellular glycogen were highly correlated ($R^2 = 0.59$); i.e., a fish with depleted yolk stores often had minimal hepatic glycogen.

For the first principal component, differences were significant for oiled status, site, and length. From individual scale values, glycogen and yolk stores were most important. When mean scores were compared (summary page at the end of Table IX-1), glycogen and yolk scores were similar in oiled and reference sites, but scores from lightly oiled sites were slightly higher. Because glycogen and yolk stores were not lower in larvae from oiled sites, differences in scores were more likely due to site and length differences rather than to oil exposure.

For the second principal component, differences were again significant for oiled status, site, and length. From individual scale values, artifact, collection trauma, and saponification of fat contributed most to variability. When mean scores were compared (summary page at the end of Table IX-1), none of these scored items were progressively greater when reference, lightly oiled, and oiled sites were compared. Therefore, differences in scores were more likely due to site and length differences rather than to oil exposure.

For the third principal component, differences were significant for site effects, but were not significant for oiled status or length effects. From individual scale values, gastrointestinal food contributed most to variability; this provides evidence that food availability and/or feeding in pre-emergent larvae was controlled more by site differences than by oil exposure. For the fourth principal component, no differences were significant.

Combining the first 4 principal components, overall differences were significant for oiled status, site, and length effects. When individual lesions were compared with ANOVA, several comparisons were significant: artifact for oil status and site; yolk for length and site; saponified fat for oil status and site; gastrointestinal food for site; and collection trauma for site. No differences were significant for epidermal atrophy. As yolk stores decreased, scores for epidermal atrophy increased and scores for hepatocellular glycogen decreased; these correlations were highly significant, but they seemed to be independent of oiled status. More likely, decreased epidermal thickness and decreased hepatocellular glycogen occurs as a physiologic change after endogenous energy (yolk) stores are depleted.

Conclusions -

For those differences that were significant for oil status, actual lesion scores tended to be similar in fish from oiled and reference sites, but different in fish from lightly oiled sites. If differences had truly been related to oil exposure, then scores should have been greatest in fish from oiled sites. Hence, it is unlikely that the lesions in these fish were related to oil exposure.

Table IX-1. Histopathologic findings in pre-emergent pink salmon larvae sampled from Prince William Sound in 1990.

NOTE: sample numbers 7,12,13, and 16 were read first as part of a preliminary screen;
(each sample # has ≤32 larvae)
Abbreviations used:
Proc. # = random number (processing number) generated by Dr. Hinton's laboratory
Sample # and Jar # = numbers submitted with each sample from ADF&G
min = minimal; mod = moderate; sev = severe; NE or "." = not examined, because organ was not present
OS = oiled status; oiled (O) or control/clean (C)
MFO = mixed function oxidase; not done on this group of fish
Atly = autolysis; ranked as min (1), mild (2), mod (3), or sev (4)
art = sectioning artifact; ranked as none (0), mild (1), mod (2), or sev (3)
sex = gonad; listed as male (M), female (F), unknown/undifferentiated (U), absent (A)
GLY = hepatic glycogen; ranked as min (1), mod (2), abundant (3), or hepatocytes not present (.)
YOLK = status of yolk stores; ranked as none (0), pale (1), min (2), or eosinophilic/abundant (3)
Lesion scores: none (0), mild (1), moderate (2), severe (3), or tissue not present (.)
EA = epidermal atrophy
MDN = myofiber degeneration and/or necrosis
IHN = individual hepatocellular necrosis
VDGG = vacuolar degeneration of gastric gland epithelial cells
NG (no good) - the intestine is present, but the stomach (gastric glands) are absent
ECN = epidermal cell necrosis (apoptosis) or inflammation; sometimes with eosinophilic cytoplasmic inclusions
GIF = gastrointestinal food (i.e., recognizable food particles in gi tract)
amount ranked as none (0), min (1), moderate (2), abundant (3), or unsure (?)
CT = collection trauma;
SPF = saponified fat (adipose tissue)

#	Proc. #	TL (mm)	S#	Jar number	Sample date	Stream #	Stream name	Location	OS	Atly	art	sex	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	Proc. SPF	Proc. #
1	89PSL 20	29	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	F	3	3	0	0	0	0	1	1	0	1	20
2	89PSL 89	29	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	F	3	3	0	0	0	0	2	0	0	1	89
3	89PSL 95	30	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	U	3	3	0	0	0	0	0	1	0	1	95
4	89PSL 105	28	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	F	3	3	0	0	0	0	1	1	0	2	105
5	89PSL 167	30	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	U	3	3	0	0	0	0	1	0	0	1	167
6	89PSL 226	30	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	F	3	3	0	0	0	0	0	0	0	1	226
7	89PSL 241	29	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	F	3	3	0	0	0	0	1	1	0	1	241
8	89PSL 244	28	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	F	3	3	0	0	0	0	1	0	0	1	244
9	89PSL 273	30	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	F	3	3	0	0	0	0	1	1	1	1	273
10	89PSL 275	ND	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	U	3	3	0	0	0	0	0	1	0	1	275
11	89PSL 286	30	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	U	3	3	0	0	0	0	1	1	0	0	286
12	89PSL 307	29	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	F	3	3	0	0	0	0	0	0	1	1	307
13	89PSL 317	31	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	U	3	3	0	0	0	0	0	0	0	1	317
14	89PSL 323	30	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	F	3	3	0	0	0	0	2	0	0	1	323
15	89PSL 334	ND	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	U	3	3	0	0	0	0	0	0	0	1	334
16	89PSL 384	ND	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	F	3	3	0	0	0	0	0	0	1	1	384
17	89PSL 386	29	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	U	3	3	0	0	0	0	2	0	0	1	386
18	89PSL 408	30	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	F	3	3	0	0	0	0	0	0	1	1	408
19	89PSL 444	30	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	U	3	3	0	0	0	0	1	1	2	1	444
20	89PSL 479	30	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	F	3	3	0	0	0	0	1	1	0	1	479
21	89PSL 495	30	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	U	3	0	0	0	0	0	1	1	0	1	495
22	89PSL 559	30	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	U	3	3	0	0	0	NG	1	1	0	1	559
23	89PSL 589	30	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	F	3	3	0	0	0	0	0	1	1	1	589
24	89PSL 607	30	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	U	3	3	0	0	0	NG	0	0	0	1	607
25	89PSL 629	30	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	F	3	3	0	0	0	0	0	0	1	1	629
26	89PSL 634	30	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	F	3	3	0	0	0	0	0	0	0	1	634
27	89PSL 638	30	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	F	3	3	0	0	0	0	1	0	0	1	638
28	89PSL 682	31	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	F	3	3	0	0	0	0	1	0	1	1	682
29	89PSL 694	31	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	U	3	3	0	0	0	0	1	1	0	0	694
30	89PSL 695	ND	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	U	A	3	0	0	A	0	2	0	0	1	695
31	89PSL 701	29	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	U	3	3	0	0	0	0	1	0	1	1	701
32	89PSL 714	ND	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	U	3	3	0	0	0	0	0	1	0	1	714

Stats: n = 27
ave. 30.
std. .75
SE = .14

Statistics:															
n =	32	32		31	32	32	32	31	30	32	32	32	32		
ave	1	1		3	2.9	0	0	0	0	.72	.44	.31	.97		
std	0	0		0	.52	0	0	0	0	.67	.50	.53	.30		
SE	0	0		0	.09	0	0	0	0	.12	.09	.09	.05		
										n = 14		9			
									frequency =		.44	.28			

#	Proc. #	TL (mm)	S#	Jar number	Sample date	Stream #	Stream name	Location	OS	Atly	art	sex	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	SPF	Proc. #
33	89PSL 30	33	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	F	3	2	0	0	0	0	0	1	1	1	30
34	89PSL 100	31	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	F	2	2	0	0	0	0	0	0	0	1	100
35	89PSL 146	32	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	F	2	1	0	0	0	NG	0	0	0	2	146
36	89PSL 184	ND	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	F	2	2	0	0	0	0	0	1	0	0	184
37	89PSL 190	33	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	A	3	1	0	0	0	0	0	0	1	2	190
38	89PSL 200	32	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	F	2	2	0	0	0	0	0	1	0	0	200
39	89PSL 202	32	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	U	3	2	0	0	0	0	0	0	0	2	202
40	89PSL 222	29	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	U	1	2	0	0	0	0	0	1	0	1	222
41	89PSL 267	32	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	U	3	1	0	0	0	0	0	1	1	0	267
42	89PSL 284	31	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	F	1	0	0	0	0	0	0	1	0	0	284
43	89PSL 298	33	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	U	1	2	0	0	0	0	0	0	0	0	298
44	89PSL 322	33	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	U	2	2	0	0	0	0	0	1	0	0	322
45	89PSL 339	32	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	U	1	2	0	0	0	0	0	0	0	0	339
46	89PSL 341	ND	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	F	2	2	0	0	0	0	0	0	0	1	341
47	89PSL 385	33	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	F	2	2	0	0	0	0	0	1	0	1	385
48	89PSL 400	31	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	F	2	2	0	0	0	0	0	0	3	1	400
49	89PSL 448	32	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	F	1	2	0	0	0	0	0	0	1	0	448
50	89PSL 460	32	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	F	2	1	0	0	0	0	0	0	0	0	460
51	89PSL 533	ND	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	F	2	1	0	2	0	0	0	0	2	0	533
52	89PSL 541	32	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	F	1	1	0	0	0	0	0	1	0	0	541
53	89PSL 549	33	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	U	1	1	0	0	0	0	0	1	0	0	549
54	89PSL 556	33	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	F	1	1	0	0	0	0	0	1	0	1	556
55	89PSL 575	31	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	U	1	1	0	0	0	0	0	0	0	0	575
56	89PSL 593	32	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	2	1	F	1	2	0	0	0	0	0	1	1	0	593
57	89PSL 603	32	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	U	1	2	0	0	0	0	0	1	0	1	603
58	89PSL 609	32	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	F	1	1	0	0	0	0	0	0	0	0	609
59	89PSL 649	32	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	F	1	1	0	0	0	0	0	0	0	0	649
60	89PSL 653	32	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	F	1	1	0	1	0	0	0	1	0	0	653
61	89PSL 656	31	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	F	1	1	0	0	0	0	0	1	0	0	656
62	89PSL 686	32	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	U	2	2	0	0	0	0	0	0	0	0	686
63	89PSL 704	ND	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	U	1	1	0	0	0	0	0	1	0	1	704
64	89PSL 728	32	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1	1	U	2	2	0	0	0	0	0	1	0	1	728
Stats:	n =	28						Statistics:	n =	32	32		32	32	32	32	32	31	32	32	32	32	
	ave.	32.							ave	1.0	1		1.6	1.5	0	.09	0	0	0	.53	.31	.5	
	std.	.87							std	.17	0		.70	.56	0	.38	0	0	0	.50	.68	.66	
	SE =	.16							SE	.03	0		.12	.10	0	.07	0	0	0	.09	.12	.12	
																			n =	17	7		
																			frequency =		.53	.22	

#	Proc. #	TL (mm)	S#	Jar number	Sample date	Stream #	Stream name	Location	OS	Atly	art	sex	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	SPF	Proc. #
65	89PSL 43	30	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	F	3	3	0	0	0	0	1	0	0	0	43
66	89PSL 46	29	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	F	3	3	0	0	0	0	0	1	0	1	46
67	89PSL 54	29	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	F	3	3	0	0	0	0	0	1	0	0	54
68	89PSL 58	28	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	F	3	3	0	0	0	0	0	1	0	0	58
69	89PSL 65	29	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	U	2	3	0	0	0	0	1	0	0	2	65
70	89PSL 79	28	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	F	3	3	0	2	0	0	1	0	2	2	79
71	89PSL 83	28	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	U	3	3	0	0	0	0	1	1	0	0	83
72	89PSL 87	29	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	U	3	3	0	0	0	0	1	1	1	1	87
73	89PSL 90	29	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	U	3	3	0	0	0	0	0	0	2	1	90
74	89PSL 120	31	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	U	3	3	0	0	0	0	1	1	0	1	120
75	89PSL 180	28	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	F	3	3	0	0	0	0	0	1	2	2	180
76	89PSL 188	30	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	A	3	3	0	0	0	NG	1	0	0	2	188
77	89PSL 234	28	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	U	3	3	0	0	0	0	1	0	3	0	234
78	89PSL 290	30	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	A	3	3	0	0	0	0	1	0	2	0	290
79	89PSL 318	30	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	F	3	3	0	0	0	NG	0	1	0	0	318
80	89PSL 345	28	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	U	3	3	0	0	0	0	1	1	0	1	345
81	89PSL 360	30	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	U	3	3	0	0	0	0	1	1	0	1	360
82	89PSL 366	29	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	U	3	3	0	0	0	0	2	0	0	0	366
83	89PSL 380	29	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	U	3	3	0	0	0	0	1	1	0	0	380
84	89PSL 382	29	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	F	3	3	0	0	0	0	1	0	2	1	382
85	89PSL 392	28	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	U	3	3	0	0	0	0	1	0	0	0	392
86	89PSL 397	29	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	F	3	3	0	0	0	0	1	0	0	0	397
87	89PSL 496	28	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	U	3	3	0	0	0	0	1	0	1	0	496
88	89PSL 502	29	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	U	3	3	0	0	0	0	1	1	1	0	502
89	89PSL 548	29	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	U	2	3	0	0	0	NG	1	0	1	0	548
90	89PSL 560	29	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	U	3	3	0	0	0	0	0	0	1	1	560
91	89PSL 635	29	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	U	3	3	0	0	0	0	1	0	0	0	635
92	89PSL 685	30	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	U	3	3	0	0	0	0	1	0	0	0	685
93	89PSL 697	31	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	F	3	3	0	0	0	0	1	1	1	1	697
94	89PSL 703	30	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	U	3	3	0	0	0	0	1	0	1	0	703
95	89PSL 718	27	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	U	3	3	0	0	0	0	1	1	0	0	718
96	89PSL 729	29	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	F	3	3	0	0	0	0	2	1	3	0	729
Stats:	n =	32						Statistics:	n =	32	32		32	32	32	32	32	29	32	32	32	32	
	ave.	29.							ave	1	1		2.9	3	0	.06	0	0	.84	.47	.72	.53	
	std.	.92							std	0	0		.24	0	0	.35	0	0	.51	.50	.94	.71	
	SE =	.16							SE	0	0		.04	0	0	.06	0	0	.09	.09	.17	.12	
																			n =	15	14		
																			frequency =		.47	.44	

#	Proc. #	TL (mm)	S#	Jar number	Sample date	Stream #	Stream name	Location	OS	Atly	art	sex	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	SPF	Proc. #
97	89PSL 26	29	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	U	3	3	0	0	0	NG	1	0	1	1	26
98	89PSL 92	29	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	U	2	2	0	0	0	0	1	1	0	1	92
99	89PSL 124	30	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	F	3	3	0	0	0	0	2	1	1	1	124
100	89PSL 125	30	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	U	3	3	0	0	0	0	1	1	0	1	125
101	89PSL 140	30	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	F	3	3	0	0	0	0	1	0	0	1	140
102	89PSL 161	30	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	U	3	3	0	0	0	0	1	0	0	1	161
103	89PSL 183	29	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	F	3	3	0	0	0	0	0	1	1	1	183
104	89PSL 229	30	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	U	3	3	0	0	0	0	1	1	0	1	229
105	89PSL 235	30	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	A	3	3	0	0	0	0	1	1	1	1	235
106	89PSL 256	29	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	F	3	3	0	0	0	0	1	0	1	1	256
107	89PSL 280	30	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	F	3	3	0	0	0	0	1	1	0	1	280
108	89PSL 292	30	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	F	3	3	0	0	0	0	1	0	0	1	292
109	89PSL 315	30	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	F	3	3	0	0	0	0	2	0	1	1	315
110	89PSL 321	30	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	F	3	3	0	0	0	0	2	0	0	1	321
111	89PSL 333	29	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	F	3	3	0	0	0	0	1	1	0	1	333
112	89PSL 364	30	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	F	3	3	0	0	0	0	1	1	0	1	364
113	89PSL 425	29	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	U	3	3	0	0	0	0	1	0	0	1	425
114	89PSL 429	30	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	F	3	3	0	0	0	0	1	0	1	1	429
115	89PSL 447	31	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	F	3	3	0	0	0	0	1	0	2	1	447
116	89PSL 452	30	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	F	3	3	0	0	0	0	1	1	0	1	452
117	89PSL 540	30	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	U	3	3	0	0	0	0	1	0	0	1	540
118	89PSL 544	30	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	U	3	3	0	0	0	0	2	1	0	1	544
119	89PSL 545	31	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	U	3	3	0	0	0	0	1	1	0	1	545
120	89PSL 558	29	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	U	3	3	0	0	0	0	1	1	0	1	558
121	89PSL 564	32	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	F	3	3	0	1	0	0	1	1	1	1	564
122	89PSL 581	31	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	2	U	3	3	0	0	0	0	1	0	0	1	581
123	89PSL 614	30	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	U	3	3	0	0	0	0	1	1	1	1	614
124	89PSL 663	29	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	F	3	3	0	0	0	0	1	0	0	1	663
125	89PSL 696	31	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	F	3	3	0	0	0	0	1	1	1	1	696
126	89PSL 719	30	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	F	3	3	0	0	0	0	2	0	0	1	719
127	89PSL 725	30	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	F	3	3	0	0	0	0	1	0	2	1	725
128	89PSL 727	30	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1	F	3	3	0	1	0	0	1	0	1	1	727
Stats:	n =	32						Statistics:	n =	32	32		32	32	32	32	32	31	32	32	32	32	
	ave.	30.							ave	1	1.0		3.0	3.0	0	.06	0	0	1.1	.5	.47	1	
	std.	.70							std	0	.17		.17	.17	0	.24	0	0	.41	.5	.61	0	
	SE =	.12							SE	0	.03		.03	.03	0	.04	0	0	.07	.09	.11	0	
																			n =	16	13		
																			frequency =	.5	.41		

#	Proc. #	TL (mm)	S#	Jar number	Sample date	Stream #	Stream name	Location	OS	Atly	art	sex	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	SPF	Proc. #
129	89PSL 42	31	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	F	3	3	0	0	0	0	1	1	2	1	42
130	89PSL 60	29	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	F	3	3	0	0	0	0	1	0	2	0	60
131	89PSL 103	31	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	F	3	3	0	0	0	0	0	0	2	1	103
132	89PSL 128	32	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	F	3	2	0	0	0	0	0	1	0	1	128
133	89PSL 129	31	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	F	3	3	0	0	0	0	1	1	1	1	129
134	89PSL 131	32	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	F	3	2	0	0	0	0	0	0	2	1	131
135	89PSL 133	31	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	U	3	3	0	0	0	0	1	0	1	1	133
136	89PSL 142	32	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	U	2	2	0	0	0	0	0	0	1	1	142
137	89PSL 150	32	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	F	3	2	0	0	0	0	0	0	0	1	150
138	89PSL 151	31	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	F	2	2	0	0	0	0	0	1	0	0	151
139	89PSL 201	31	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	U	3	3	0	0	0	0	1	1	1	1	201
140	89PSL 220	30	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	U	3	3	0	0	0	0	1	1	2	1	220
141	89PSL 260	33	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	F	3	2	0	0	0	0	1	1	0	1	260
142	89PSL 261	32	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	U	3	3	0	0	0	0	1	0	0	1	261
143	89PSL 264	30	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	U	3	3	0	0	0	0	1	1	1	1	264
144	89PSL 299	32	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	U	3	2	0	0	0	0	1	0	0	0	299
145	89PSL 327	31	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	U	3	3	0	0	0	0	1	1	0	1	327
146	89PSL 332	32	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	U	3	3	0	0	0	0	1	0	0	1	332
147	89PSL 352	31	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	U	3	3	0	0	0	0	1	1	0	1	352
148	89PSL 415	30	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	U	3	3	0	0	0	0	1	0	0	1	415
149	89PSL 419	31	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	2	U	3	3	0	0	0	0	1	1	3	1	419
150	89PSL 426	32	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	F	3	3	0	0	0	0	1	0	2	1	426
151	89PSL 467	32	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	U	3	3	0	0	0	0	1	0	0	1	467
152	89PSL 497	31	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	U	3	3	0	0	0	0	0	0	0	1	497
153	89PSL 515	31	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	U	3	3	0	0	0	0	1	0	1	1	515
154	89PSL 554	31	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	F	3	3	0	0	0	0	1	1	1	1	554
155	89PSL 566	32	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	F	3	3	0	0	0	0	1	0	0	1	566
156	89PSL 594	31	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	F	3	3	0	0	0	0	1	0	1	1	594
157	89PSL 613	31	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	F	3	3	0	0	0	0	0	1	0	1	613
158	89PSL 676	31	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	U	3	3	0	0	0	0	0	0	2	1	676
159	89PSL 691	32	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	U	3	3	0	0	0	0	0	1	0	1	691
160	89PSL 706	31	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1	F	3	3	0	0	0	0	1	1	2	1	706
Stats:	n =	32						Statistics:	n =	32	32		32	32	32	32	32	32	32	32	32	32	
	ave.	31.							ave	1	1.0		2.9	2.8	0	0	0	0	.69	.47	.84	.91	
	std.	.79							std	0	.17		.24	.41	0	0	0	0	.46	.50	.91	.29	
	SE =	.14							SE	0	.03		.04	.07	0	0	0	0	.08	.09	.16	.05	
																				n = 15	17		
																			frequency =		.47	.53	

#	Proc. #	TL (mm)	S#	Jar number	Sample date	Stream #	Stream name	Location	OS	Atly	art	sex	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	SPF	Proc. #
161	89PSL 8	32	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	U	3	3	0	0	0	0	0	0	0	1	8
162	89PSL 18	33	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	F	3	2	0	0	0	0	0	1	1	1	18
163	89PSL 33	32	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	U	3	3	0	0	0	0	0	1	0	1	33
164	89PSL 80	31	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	U	3	3	0	0	0	0	0	0	2	1	80
165	89PSL 137	32	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	U	3	3	0	0	0	0	1	0	0	1	137
166	89PSL 139	31	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	U	3	3	0	0	0	0	1	1	0	1	139
167	89PSL 148	30	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	U	3	3	0	0	0	0	1	0	0	1	148
168	89PSL 198	32	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	F	3	3	0	0	0	0	1	1	1	1	198
169	89PSL 210	32	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	F	3	3	0	0	0	0	1	1	0	1	210
170	89PSL 219	32	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	U	2	2	0	0	0	0	1	0	0	1	219
171	89PSL 257	33	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	F	3	3	0	0	0	0	1	1	0	1	257
172	89PSL 270	31	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	F	3	3	0	0	0	0	0	1	0	0	270
173	89PSL 289	32	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	F	3	3	0	0	0	0	1	1	0	1	289
174	89PSL 291	31	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	F	3	3	0	0	0	0	1	1	0	1	291
175	89PSL 303	31	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	U	3	2	0	0	0	0	1	1	2	1	303
176	89PSL 368	31	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	F	3	3	0	0	0	0	1	1	0	0	368
177	89PSL 378	31	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	F	3	3	0	0	0	0	0	0	1	1	378
178	89PSL 395	32	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	F	3	3	0	0	0	0	0	1	0	1	395
179	89PSL 401	32	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	2	U	3	3	0	0	0	0	1	1	0	1	401
180	89PSL 438	31	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	U	3	3	0	0	0	0	1	0	0	1	438
181	89PSL 455	32	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	U	3	3	0	0	0	0	1	1	0	1	455
182	89PSL 470	32	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	F	3	3	0	0	0	0	0	1	2	1	470
183	89PSL 474	32	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	F	3	3	0	0	0	0	1	1	0	1	474
184	89PSL 480	32	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	U	3	3	0	0	0	0	1	0	0	1	480
185	89PSL 506	31	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	U	3	3	0	0	0	0	0	0	0	1	506
186	89PSL 538	31	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	F	3	3	0	0	0	0	1	2	0	1	538
187	89PSL 555	31	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	F	3	3	0	0	0	0	1	0	0	1	555
188	89PSL 595	32	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	U	3	3	0	0	0	0	0	1	0	1	595
189	89PSL 597	31	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	F	3	3	0	0	0	0	1	1	0	1	597
190	89PSL 598	32	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	U	3	3	0	0	0	0	1	1	0	1	598
191	89PSL 650	31	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	U	3	3	0	0	0	0	1	0	1	1	650
192	89PSL 671	32	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1	U	3	3	0	0	0	0	1	1	0	1	671

Stats:																							
	n =	32																					
	ave.	32.																					
	std.	.65																					
	SE =	.12																					

Statistics:																						
	n =	32	32		32	32	32	32	32	32	32	32	32	32								
	ave	1	1.0		3.0	2.9	0	0	0	0	.69	.69	.31	.94								
	std	0	.17		.17	.29	0	0	0	0	.46	.53	.63	.24								
	SE	0	.03		.03	.05	0	0	0	0	.08	.09	.11	.04								
													n =	22	7							
													frequency =	.69	.22							

#	Proc. #	TL (mm)	S#	Jar number	Sample date	Stream #	Stream name	Location	OS	Atly	art	sex	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	SPF	Proc. #
193	89PSL 13	32	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	F	3	2	0	0	0	0	1	1	1	1	13
194	89PSL 16	32	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	F	2	2	0	0	0	0	1	0	0	1	16
195	89PSL 24	32	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	U	3	2	0	0	0	0	0	1	0	1	24
196	89PSL 48	33	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	F	2	2	0	0	0	0	0	1	0	1	48
197	89PSL 63	32	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	2	F	2	1	1	0	0	0	0	1	2	0	63
198	89PSL 110	29	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	U	1	2	0	0	0	0	1	0	0	1	110
199	89PSL 117	33	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	U	2	2	0	0	0	0	0	0	2	1	117
200	89PSL 122	32	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	F	2	2	0	0	0	0	1	0	0	1	122
201	89PSL 224	33	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	F	1	1	0	0	0	0	1	0	1	2	224
202	89PSL 243	32	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	U	3	3	0	0	0	0	1	1	2	1	243
203	89PSL 246	32	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	F	2	2	0	0	0	0	1	0	0	1	246
204	89PSL 251	32	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	F	2	1	0	0	0	0	0	0	0	1	251
205	89PSL 266	32	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	U	3	2	0	0	0	0	1	1	1	1	266
206	89PSL 268	32	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	F	3	2	0	0	0	0	1	1	0	1	268
207	89PSL 285	32	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	F	3	2	0	0	0	0	1	1	0	2	285
208	89PSL 309	31	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	F	3	3	0	0	0	0	1	0	0	1	309
209	89PSL 325	32	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	U	2	2	0	0	0	0	0	1	3	2	325
210	89PSL 329	33	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	2	U	1	1	1	0	0	0	1	1	1	0	329
211	89PSL 373	32	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	U	2	2	0	0	0	0	0	0	1	1	373
212	89PSL 407	30	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	F	1	1	1	0	0	0	0	0	1	1	407
213	89PSL 413	32	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	U	2	2	0	0	0	0	1	1	2	1	413
214	89PSL 459	31	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	F	3	3	0	0	0	0	1	1	0	1	459
215	89PSL 504	32	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	U	1	1	0	0	0	0	1	0	1	1	504
216	89PSL 507	31	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	F	3	3	0	0	0	0	1	1	0	1	507
217	89PSL 510	32	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	2	F	2	2	0	0	0	0	1	1	0	1	510
218	89PSL 514	32	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	F	1	1	0	0	0	0	1	0	2	0	514
219	89PSL 523	32	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	F	2	1	0	0	0	0	1	0	0	1	523
220	89PSL 547	33	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	F	2	3	0	0	0	0	1	1	3	1	547
221	89PSL 569	32	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	F	2	1	0	0	0	0	1	0	1	1	569
222	89PSL 619	32	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	2	1	F	2	1	0	0	0	0	1	1	0	1	619
223	89PSL 681	32	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	F	3	3	0	0	0	0	1	1	1	1	681
224	89PSL 715	28	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1	1	U	1	2	1	0	0	0	0	0	2	0	715
Stats:	n =	32						Statistics:	n =	32	32		32	32	32	32	32	32	32	32	32	32	
	ave.	32.							ave	1.0	1.1		2.1	1.9	.13	0	0	0	.72	.53	.84	.97	
	std.	1.1							std	.17	.29		.72	.70	.33	0	0	0	.45	.50	.94	.47	
	SE =	.19							SE	.03	.05		.13	.12	.06	0	0	0	.08	.09	.17	.08	
																			n =	17	17		
																			frequency =		.53	.53	

#	Proc. #	TL (mm)	S#	Jar number	Sample date	Stream #	Stream name	Location	OS	Atly	art	sex	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	SPF	Proc. #
225	89PSL 35	31	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	2	U	3	3	0	0	0	0	1	0	0	1	35
226	89PSL 41	31	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	U	3	3	0	0	0	0	2	0	2	1	41
227	89PSL 50	30	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	U	3	3	0	0	0	0	1	0	1	1	50
228	89PSL 76	31	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	U	3	3	0	0	0	0	1	1	0	1	76
229	89PSL 138	32	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	F	3	3	0	0	0	0	1	1	0	1	138
230	89PSL 163	33	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	U	3	3	0	0	0	0	0	0	0	0	163
231	89PSL 164	31	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	U	3	3	0	0	0	0	1	0	0	1	164
232	89PSL 214	32	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	U	3	3	0	0	0	0	1	0	0	1	214
233	89PSL 223	31	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	U	3	3	0	0	0	0	1	1	1	1	223
234	89PSL 262	32	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	F	3	3	0	0	0	0	1	0	0	1	262
235	89PSL 271	30	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	U	3	3	0	0	0	0	2	1	3	0	271
236	89PSL 272	31	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	F	3	3	0	0	0	0	0	1	0	1	272
237	89PSL 281	32	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	F	3	3	0	0	0	0	0	0	0	1	281
238	89PSL 282	32	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	U	3	3	0	0	0	0	1	1	0	0	282
239	89PSL 294	25	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	U	3	3	0	0	0	0	1	1	2	1	294
240	89PSL 308	30	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	U	3	3	0	0	0	0	1	0	0	0	308
241	89PSL 351	30	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	U	3	3	0	0	0	0	1	0	1	1	351
242	89PSL 374	32	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	U	3	3	0	0	0	0	1	0	0	1	374
243	89PSL 391	31	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	F	3	3	0	0	0	0	0	1	0	0	391
244	89PSL 402	31	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	F	3	3	0	0	0	0	1	0	1	1	402
245	89PSL 412	31	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	2	U	3	3	0	0	0	0	0	1	0	1	412
246	89PSL 436	31	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	F	3	3	0	0	0	0	1	0	0	1	436
247	89PSL 446	32	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	U	3	3	0	0	0	0	1	1	3	1	446
248	89PSL 471	31	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	2	F	3	3	0	0	0	0	1	1	0	0	471
249	89PSL 490	31	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	F	3	3	0	0	0	0	1	1	0	1	490
250	89PSL 588	30	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	F	3	3	0	0	0	0	1	0	2	1	588
251	89PSL 615	30	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	U	3	3	0	0	0	0	1	0	1	1	615
252	89PSL 692	31	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	F	3	3	0	0	0	0	0	0	0	0	692
253	89PSL 693	30	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	F	3	3	0	0	0	0	1	1	0	1	693
254	89PSL 711	31	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	U	3	3	0	0	0	0	1	0	2	1	711
255	89PSL 723	32	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	F	3	2	0	0	0	0	1	1	0	1	723
256	89PSL 724	31	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	0	1	1	F	3	3	0	0	0	0	1	0	0	1	724

Stats: n = 32
ave. 31.
std. 1.3
SE = .23

Statistics:

n =	32	32		32	32	32	32	32	32	32	32	32	32	32	
ave	1	1.1		3	3.0	0	0	0	0	.88	.44	.59	.78		
std	0	.29		0	.17	0	0	0	0	.48	.50	.93	.41		
SE	0	.05		0	.03	0	0	0	0	.09	.09	.16	.07		
												n =	14	11	
												frequency =	.44	.34	

#	Proc. #	TL (mm)	S#	Jar number	Sample date	Stream #	Stream name	Location	OS	Atly	art	sex	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	SPF	Proc. #
257	89PSL 4	31	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	1	U	3	3	0	0	0	0	0	0	3	0	4
258	89PSL 9	29	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	2	F	3	3	0	0	0	0	0	1	0	0	9
259	89PSL 71	30	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	1	F	3	3	0	0	0	0	1	1	1	0	71
260	89PSL 175	30	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	1	F	3	3	0	0	0	0	1	0	0	0	175
261	89PSL 191	30	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	1	U	3	3	0	0	0	0	1	0	2	0	191
262	89PSL 213	30	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	1	U	3	3	0	0	0	0	1	0	2	0	213
263	89PSL 269	30	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	1	U	3	3	0	0	0	0	1	0	0	0	269
264	89PSL 277	30	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	1	F	3	3	0	0	0	0	1	1	1	0	277
265	89PSL 342	29	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	2	F	3	3	0	0	0	0	1	1	1	0	342
266	89PSL 347	31	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	1	F	3	3	0	0	0	0	1	0	1	0	347
267	89PSL 359	30	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	1	F	3	3	0	0	0	0	1	1	0	0	359
268	89PSL 361	31	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	2	F	3	3	0	0	0	0	1	1	1	0	361
269	89PSL 387	29	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	1	F	3	3	0	0	0	0	1	0	1	0	387
270	89PSL 398	30	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	2	1	F	3	3	0	0	0	NG	0	1	2	0	398
271	89PSL 403	29	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	2	F	3	3	0	0	0	0	1	1	2	0	403
272	89PSL 443	29	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	1	F	3	3	0	0	0	0	1	1	1	0	443
273	89PSL 445	29	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	1	F	3	3	0	0	0	0	1	0	1	0	445
274	89PSL 453	30	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	2	F	3	3	0	0	0	0	1	0	2	0	453
275	89PSL 466	30	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	1	U	3	3	0	0	0	0	1	0	1	0	466
276	89PSL 477	30	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	2	2	U	1	3	0	0	1	0	0	1	0	0	477
277	89PSL 491	30	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	1	F	3	3	0	0	0	0	1	0	1	0	491
278	89PSL 501	29	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	2	U	3	3	0	0	0	0	1	0	1	1	501
279	89PSL 505	30	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	2	U	3	3	0	0	0	0	1	1	1	0	505
280	89PSL 516	29	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	1	U	3	3	0	0	0	0	1	0	0	0	516
281	89PSL 518	29	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	1	F	3	3	0	0	0	0	1	1	1	0	518
282	89PSL 567	31	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	1	F	3	3	0	0	0	0	1	0	1	0	567
283	89PSL 610	31	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	1	F	3	3	0	0	0	0	0	0	1	0	610
284	89PSL 633	31	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	1	U	3	3	0	0	0	0	1	0	0	0	633
285	89PSL 648	28	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	1	U	3	3	0	0	0	NG	1	0	0	0	648
286	89PSL 709	30	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	3	F	3	3	0	0	0	0	1	1	0	0	709
287	89PSL 716	31	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	1	U	3	3	0	0	0	0	0	1	1	0	716
288	89PSL 720	30	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1	2	F	3	3	0	0	0	0	0	1	2	0	720

Stats: n = 32
ave. 30.
std. .78
SE = .14

Statistics:

	OS	Atly	art	sex	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	SPF
n =	32	32			32	32	32	32	32	30	32	32	32	32
ave	1.1	1.3			2.9	3	0	0	.03	0	.78	.47	.97	.03
std	.24	.54			.35	0	0	0	.17	0	.41	.50	.77	.17
SE	.04	.09			.06	0	0	0	.03	0	.07	.09	.14	.03
											n =	15	23	
											frequency =	.47	.72	

#	Proc. #	TL (mm)	S#	Jar number	Sample date	Stream #	Stream name	Location	OS	Atly	art	sex	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	SPF	Proc. #
289	89PSL 28	23	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	1	F	3	3	0	0	0	NG	1	1	1	0	28
290	89PSL 36	24	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	1	U	.	3	0	0	0	0	1	0	0	0	36
291	89PSL 49	24	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	1	U	3	3	0	0	0	NG	1	0	1	0	49
292	89PSL 73	23	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	2	U	2	3	0	0	0	NG	0	0	3	0	73
293	89PSL 84	24	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	1	U	3	3	0	0	0	NG	0	0	0	0	84
294	89PSL 123	25	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	1	U	3	3	0	0	0	NG	1	0	0	1	123
295	89PSL 145	24	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	2	U	2	3	0	1	0	NG	1	2	0	0	145
296	89PSL 152	24	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	1	U	3	3	0	0	0	0	0	0	1	1	152
297	89PSL 153	24	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	1	U	3	3	0	0	0	NG	0	0	0	0	153
298	89PSL 179	24	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	1	U	3	3	0	0	0	NG	1	0	2	1	179
299	89PSL 185	25	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	1	A	3	3	0	0	0	0	1	0	1	0	185
300	89PSL 227	24	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	2	F	2	3	0	0	0	0	1	0	0	1	227
301	89PSL 253	22	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	2	U	3	3	0	0	0	0	1	0	2	0	253
302	89PSL 254	23	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	2	U	2	3	0	0	0	0	1	1	2	0	254
303	89PSL 358	22	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	1	U	3	3	0	0	0	NG	0	0	0	1	358
304	89PSL 363	23	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	1	U	2	3	0	0	0	0	0	1	1	0	363
305	89PSL 399	22	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	1	U	3	3	0	0	0	0	1	0	3	1	399
306	89PSL 418	24	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	1	F	2	3	0	0	0	0	0	1	0	1	418
307	89PSL 522	25	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	2	F	3	3	0	0	0	0	1	0	0	1	522
308	89PSL 536	22	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	2	U	2	3	0	0	0	0	1	0	0	1	536
309	89PSL 539	23	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	1	U	2	3	0	0	0	0	1	0	0	0	539
310	89PSL 546	21	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	1	U	2	3	0	1	0	0	0	0	2	0	546
311	89PSL 562	23	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	1	F	2	3	0	0	0	0	0	0	0	1	562
312	89PSL 601	23	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	1	U	3	3	0	0	0	0	0	1	0	1	601
313	89PSL 612	25	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	2	A	3	3	0	0	0	0	0	0	3	0	612
314	89PSL 620	24	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	2	U	2	3	0	0	0	0	0	0	0	1	620
315	89PSL 627	23	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	2	U	2	3	0	1	0	0	0	0	2	0	627
316	89PSL 662	25	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	2	F	3	3	0	0	0	0	0	0	1	1	662
317	89PSL 705	24	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	1	U	2	3	0	0	0	0	0	0	0	1	705
318	89PSL 721	23	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	1	U	2	3	0	0	0	0	0	0	2	0	721
319	89PSL 734	23	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	2	U	3	3	0	0	0	0	1	0	2	0	734
320	89PSL 735	24	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	1	U	2	3	0	0	0	0	0	0	0	1	735
Stats:	n =	32						Statistics:	n =	32	32		31	32	32	32	32	23	32	32	32	32	
	ave.	24.							ave	1	1.4		2.5	3	0	.09	0	0	.47	.22	.91	.47	
	std.	1							std	0	.48		.50	0	0	.29	0	0	.50	.48	1.0	.50	
	SE =	.18							SE	0	.09		.09	0	0	.05	0	0	.09	.09	.18	.09	
																			n =	7	16		
																			frequency =	.22	.5		

#	Proc. #	TL (mm)	S#	Jar number	Sample date	Stream #	Stream name	Location	OS	Atly	art	sex	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	SPF	Proc. #
321	89PSL 7	28	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	2	U	3	3	0	0	0	0	0	1	0	1	7
322	89PSL 25	31	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	F	3	3	0	0	0	0	0	1	0	1	25
323	89PSL 51	28	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	F	2	3	0	0	0	0	1	0	2	1	51
324	89PSL 141	32	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	F	3	3	0	0	0	0	0	1	3	0	141
325	89PSL 154	27	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	U	3	3	0	0	0	0	0	0	0	1	154
326	89PSL 181	31	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	F	3	3	0	0	0	0	1	1	0	1	181
327	89PSL 204	32	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	2	U	3	3	0	0	0	0	1	0	1	1	204
328	89PSL 212	31	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	2	F	3	3	0	0	0	0	1	0	2	1	212
329	89PSL 231	27	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	2	U	1	3	0	0	0	0	1	0	2	1	231
330	89PSL 237	33	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	F	3	3	0	0	0	0	0	1	0	1	237
331	89PSL 301	32	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	F	3	3	0	0	0	0	0	1	0	1	301
332	89PSL 336	28	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	U	3	3	0	0	0	0	1	0	0	1	336
333	89PSL 362	32	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	F	3	3	0	0	0	0	1	1	1	1	362
334	89PSL 375	27	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	U	3	3	0	0	0	0	1	1	1	1	375
335	89PSL 396	27	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	U	3	3	0	0	0	0	1	1	0	1	396
336	89PSL 414	31	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	U	3	3	0	0	0	0	0	0	0	1	414
337	89PSL 432	31	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	F	3	3	0	0	0	0	1	1	2	1	432
338	89PSL 442	28	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	U	3	3	0	0	0	0	1	1	0	1	442
339	89PSL 469	31	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	F	3	3	0	0	0	0	1	0	0	1	469
340	89PSL 481	29	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	U	2	3	0	0	0	0	0	0	2	1	481
341	89PSL 498	28	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	F	3	3	0	0	0	NG	0	0	0	1	498
342	89PSL 537	27	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	F	3	3	0	0	0	0	1	0	0	1	537
343	89PSL 573	28	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	U	2	3	0	0	0	0	1	0	0	1	573
344	89PSL 582	28	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	F	3	3	0	0	0	0	1	0	0	1	582
345	89PSL 604	30	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	F	3	3	0	0	0	0	1	0	0	1	604
346	89PSL 632	27	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	U	3	0	0	0	0	0	1	0	3	1	632
347	89PSL 640	28	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	U	3	3	0	0	0	0	1	1	1	1	640
348	89PSL 644	32	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	U	3	3	0	0	0	0	0	1	0	1	644
349	89PSL 687	27	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	F	3	3	0	0	0	0	0	0	0	1	687
350	89PSL 707	28	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	F	3	3	0	0	0	0	1	1	0	1	707
351	89PSL 726	33	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	U	3	3	0	0	0	0	1	1	0	1	726
352	89PSL 732	28	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1	F	3	3	0	0	0	0	1	0	0	1	732
Stats:	n =	32						Statistics:	n =	32	32		32	32	32	32	32	31	32	32	32	32	
	ave.	29.							ave	1	1.1		2.8	2.9	0	0	0	0	.66	.47	.63	.97	
	std.	2.1							std	0	.33		.44	.52	0	0	0	0	.47	.50	.96	.17	
	SE =	.36							SE	0	.06		.08	.09	0	0	0	0	.08	.09	.17	.03	
																			n =	15	11		
																			frequency =		.47	.34	

#	Proc. #	TL (mm)	S#	Jar number	Sample date	Stream #	Stream name	Location	OS	Atly	art	sex	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	SPF	Proc. #
353	89PSL 32	30	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	F	1	3	1	0	0	0	0	0	0	0	32
354	89PSL 98	30	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	U	3	3	0	0	0	0	1	1	0	0	98
355	89PSL 135	29	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	U	3	2	0	0	0	0	0	1	0	1	135
356	89PSL 149	30	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	U	3	3	0	0	0	0	1	0	0	1	149
357	89PSL 189	29	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	F	3	3	0	0	0	0	1	1	3	1	189
358	89PSL 206	29	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	F	3	3	0	0	0	0	0	1	0	1	206
359	89PSL 218	30	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	F	3	3	0	0	0	0	1	1	2	1	218
360	89PSL 230	31	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	F	3	3	0	0	0	0	0	0	2	1	230
361	89PSL 239	30	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	U	3	3	1	0	0	0	0	0	2	1	239
362	89PSL 240	30	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	F	3	3	0	0	0	0	0	0	1	1	240
363	89PSL 340	29	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	F	3	3	0	0	0	NG	1	1	0	1	340
364	89PSL 344	29	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	2	U	3	3	0	0	0	0	1	1	0	1	344
365	89PSL 383	30	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	U	3	3	0	0	0	0	0	1	0	1	383
366	89PSL 390	30	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	F	3	3	0	0	0	0	1	0	1	1	390
367	89PSL 404	30	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	2	F	3	3	0	0	0	0	1	1	1	1	404
368	89PSL 411	30	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	U	3	3	0	0	0	0	1	1	1	1	411
369	89PSL 437	30	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	U	3	3	0	0	0	0	1	1	0	1	437
370	89PSL 440	29	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	F	3	3	0	0	0	0	1	1	0	1	440
371	89PSL 486	29	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	F	3	3	0	0	0	0	1	0	1	1	486
372	89PSL 508	31	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	F	3	3	0	0	0	0	0	1	0	1	508
373	89PSL 528	30	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	U	3	3	0	0	0	0	1	1	0	1	528
374	89PSL 568	30	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	F	1	3	0	0	0	0	1	0	1	1	568
375	89PSL 583	31	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	F	3	3	0	0	0	0	1	1	0	1	583
376	89PSL 585	30	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	2	U	3	3	0	0	0	0	1	0	0	1	585
377	89PSL 599	30	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	F	3	3	0	0	0	0	1	0	0	1	599
378	89PSL 611	29	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	F	3	3	0	0	0	0	1	0	0	0	611
379	89PSL 655	30	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	2	U	3	3	0	0	0	0	1	1	1	1	655
380	89PSL 664	31	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	F	3	3	0	0	0	0	1	0	2	1	664
381	89PSL 665	30	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	F	3	3	0	0	0	0	0	0	2	0	665
382	89PSL 666	31	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	F	3	3	0	0	0	0	1	1	1	1	666
383	89PSL 717	30	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	F	3	3	0	0	0	0	1	1	0	1	717
384	89PSL 722	29	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1	U	3	3	0	0	0	0	1	1	0	1	722
Stats:	n =	32						Statistics:	n =	32	32		32	32	32	32	32	31	32	32	32	32	
	ave.	30.							ave	1	1.1		2.9	3.0	.06	0	0	0	.72	.59	.66	.88	
	std.	.65							std	0	.33		.48	.17	.24	0	0	0	.45	.49	.85	.33	
	SE =	.11							SE	0	.06		.09	.03	.04	0	0	0	.08	.09	.15	.06	
																			n =	19	14		
																			frequency =	.59	.44		

#	Proc. #	TL (mm)	S#	Jar number	Sample date	Stream #	Stream name	Location	OS	Atly	art	sex	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	SPF	Proc. #
385	89PSL 12	31	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	2	F	3	3	0	0	0	0	1	1	0	0	12
386	89PSL 57	31	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	2	F	3	3	0	0	0	0	0	0	0	0	57
387	89PSL 66	32	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	2	U	3	3	0	0	0	0	0	0	0	0	66
388	89PSL 70	31	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	1	F	3	3	0	0	0	0	0	0	1	0	70
389	89PSL 88	30	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	1	U	3	3	0	0	0	0	1	0	1	0	88
390	89PSL 91	32	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	1	F	3	3	0	0	0	0	1	0	2	1	91
391	89PSL 99	31	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	2	U	3	3	0	0	0	0	1	0	0	0	99
392	89PSL 121	30	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	1	F	3	3	0	0	0	0	1	0	2	0	121
393	89PSL 126	31	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	2	F	3	3	0	0	0	0	1	0	0	0	126
394	89PSL 160	31	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	1	U	3	2	0	0	0	0	1	0	0	0	160
395	89PSL 194	31	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	2	F	3	3	0	0	0	0	1	0	1	0	194
396	89PSL 203	31	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	1	U	3	3	0	0	0	0	1	0	0	0	203
397	89PSL 211	30	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	2	F	3	3	0	0	0	0	1	1	0	0	211
398	89PSL 236	31	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	1	U	3	3	0	0	0	0	0	1	0	0	236
399	89PSL 252	30	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	1	F	3	3	0	0	0	0	0	1	0	0	252
400	89PSL 255	31	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	1	F	3	3	0	0	0	0	0	0	2	0	255
401	89PSL 287	30	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	2	U	3	3	0	0	0	0	1	1	0	0	287
402	89PSL 348	31	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	1	U	3	3	0	0	0	0	1	0	0	0	348
403	89PSL 421	31	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	1	U	3	3	0	0	0	0	0	1	2	0	421
404	89PSL 457	31	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	2	U	3	3	0	0	0	0	1	1	0	0	457
405	89PSL 461	32	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	1	F	3	3	0	0	0	0	0	1	0	1	461
406	89PSL 464	30	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	2	U	3	3	0	0	0	0	1	1	2	0	464
407	89PSL 472	31	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	1	U	3	3	0	0	0	0	1	0	1	1	472
408	89PSL 535	31	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	1	F	3	3	0	0	0	0	1	1	0	0	535
409	89PSL 550	31	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	2	F	3	3	0	0	0	0	1	0	3	0	550
410	89PSL 561	30	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	2	U	3	3	0	0	0	0	1	0	1	0	561
411	89PSL 574	30	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	1	F	3	3	0	0	0	0	1	0	1	0	574
412	89PSL 642	31	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	1	U	3	3	0	0	0	0	1	0	0	0	642
413	89PSL 660	31	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	1	F	3	3	0	0	0	0	1	1	0	0	660
414	89PSL 678	31	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	1	U	3	3	0	0	0	0	0	1	0	0	678
415	89PSL 690	31	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	1	F	3	3	0	0	0	0	1	0	2	0	690
416	89PSL 733	27	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	2	F	1	3	0	0	0	0	1	1	0	0	733
Stats:	n =	32						Statistics:	n =	32	32		32	32	32	32	32	32	32	32	32	32	
	ave.	31.							ave	1	1.4		2.9	3.0	0	0	0	0	.72	.41	.66	.09	
	std.	.87							std	0	.49		.35	.17	0	0	0	0	.45	.49	.89	.29	
	SE =	.15							SE	0	.09		.06	.03	0	0	0	0	.08	.09	.16	.05	
																			n =	13	13		
																			frequency =	.41	.41		

#	Proc. #	TL (mm)	S#	Jar number	Sample date	Stream #	Stream name	Location	OS	Atly	art	sex	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	SPF	Proc. #
417	89PSL 38	34	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	F	3	3	0	0	0	0	1	1	0	2	38
418	89PSL 53	33	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	U	3	3	0	0	0	0	0	1	0	2	53
419	89PSL 55	31	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	U	3	3	0	0	0	0	1	1	0	1	55
420	89PSL 56	32	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	F	3	3	0	0	0	0	1	1	1	2	56
421	89PSL 68	30	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	U	3	3	0	0	0	0	0	1	0	1	68
422	89PSL 113	30	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	F	3	2	0	0	0	0	1	0	2	1	113
423	89PSL 143	33	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	U	3	3	0	0	0	0	1	0	0	1	143
424	89PSL 168	30	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	2	1	U	3	3	0	0	0	0	1	1	0	2	168
425	89PSL 192	31	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	U	3	2	0	0	0	0	1	1	0	1	192
426	89PSL 209	30	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	2	F	3	3	0	0	0	0	1	1	3	2	209
427	89PSL 221	30	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	U	3	2	0	0	0	0	0	0	0	2	221
428	89PSL 300	30	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	U	2	2	0	0	0	0	1	0	0	1	300
429	89PSL 305	32	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	U	3	3	0	0	0	0	0	1	2	2	305
430	89PSL 310	30	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	F	3	3	0	0	0	0	0	0	0	2	310
431	89PSL 337	31	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	F	3	3	0	0	0	0	0	0	0	1	337
432	89PSL 406	31	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	U	3	2	0	0	0	0	0	0	1	2	406
433	89PSL 420	34	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	U	3	3	0	0	0	0	0	0	0	2	420
434	89PSL 428	31	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	F	3	2	0	0	0	0	0	0	0	1	428
435	89PSL 433	30	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	F	3	3	0	0	0	0	1	1	1	1	433
436	89PSL 462	29	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	U	2	2	1	0	0	0	1	0	0	2	462
437	89PSL 468	31	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	F	3	3	0	0	0	0	0	1	0	2	468
438	89PSL 473	30	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	2	U	3	3	0	0	0	0	1	1	0	2	473
439	89PSL 493	32	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	F	3	3	0	0	0	0	1	0	1	1	493
440	89PSL 517	31	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	F	3	3	0	0	0	0	1	0	0	2	517
441	89PSL 551	32	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	F	3	3	0	0	0	0	0	0	0	2	551
442	89PSL 618	31	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	F	3	3	0	0	0	0	0	1	0	1	618
443	89PSL 630	31	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	F	3	3	0	0	0	0	0	0	0	2	630
444	89PSL 631	30	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	F	3	3	0	0	0	0	0	0	0	2	631
445	89PSL 636	31	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	F	3	3	0	0	0	0	0	1	2	2	636
446	89PSL 647	30	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	2	U	3	3	0	0	0	0	1	0	0	2	647
447	89PSL 677	30	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	U	3	3	0	0	0	0	0	0	1	1	677
448	89PSL 679	30	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1	1	U	3	3	0	0	0	0	1	1	0	2	679
Stats:	n =	32						Statistics:	n =	32	32		32	32	32	32	32	32	32	32	32	32	
	ave.	31.							ave	1.0	1.1		2.9	2.8	.03	0	0	0	.5	.47	.44	1.6	
	std.	1.2							std	.17	.29		.24	.41	.17	0	0	0	.5	.50	.79	.48	
	SE =	.21							SE	.03	.05		.04	.07	.03	0	0	0	.09	.09	.14	.09	
																				n = 15		9	
																			frequency =		.47	.28	

#	Proc. #	TL (mm)	S#	Jar number	Sample date	Stream #	Stream name	Location	OS	Atly	art	sex	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	SPF	Proc. #
449	89PSL 3	29	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	2	F	3	3	0	0	0	0	1	1	2	1	3
450	89PSL 19	31	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	U	3	2	0	0	0	0	0	1	0	1	19
451	89PSL 22	31	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	F	3	3	0	0	0	0	0	0	0	1	22
452	89PSL 29	29	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	F	3	3	0	0	0	0	1	0	0	1	29
453	89PSL 40	30	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	U	3	3	0	0	0	0	1	1	0	1	40
454	89PSL 62	32	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	F	3	3	0	0	0	0	1	1	0	0	62
455	89PSL 77	31	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	F	3	3	0	0	0	0	0	0	1	1	77
456	89PSL 104	29	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	F	3	3	0	0	0	0	0	0	3	1	104
457	89PSL 111	31	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	U	3	3	0	0	0	0	1	1	1	1	111
458	89PSL 115	none	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO	only 29 larvae were sampled; this # not used														115
459	89PSL 155	28	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	2	F	3	3	0	0	0	0	0	0	0	1	155
460	89PSL 182	31	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	F	3	3	0	0	0	0	0	1	2	1	182
461	89PSL 186	none	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO	only 29 larvae were sampled; this # not used														186
462	89PSL 187	31	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	F	3	3	0	0	0	0	0	0	1	1	187
463	89PSL 199	32	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	U	3	3	0	0	0	0	0	0	0	1	199
464	89PSL 205	28	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	U	3	3	0	0	0	0	1	0	3	1	205
465	89PSL 216	30	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	F	3	3	0	0	0	0	0	0	2	1	216
466	89PSL 288	31	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	U	3	3	0	0	0	0	0	1	2	1	288
467	89PSL 302	29	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	F	3	3	0	0	0	0	1	1	0	1	302
468	89PSL 312	30	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	F	3	3	0	0	0	0	1	0	2	1	312
469	89PSL 320	31	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	U	3	3	0	0	0	0	1	1	0	1	320
470	89PSL 376	31	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	U	3	3	0	0	0	0	1	0	2	1	376
471	89PSL 381	none	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO	only 29 larvae were sampled; this # not used														381
472	89PSL 424	32	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	U	3	3	0	0	0	0	0	1	1	1	424
473	89PSL 484	33	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		2	1	F	3	3	0	0	0	0	0	0	1	1	484
474	89PSL 521	28	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	U	3	3	0	0	0	0	1	0	1	1	521
475	89PSL 531	31	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	F	3	3	0	0	0	0	1	0	0	1	531
476	89PSL 553	29	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	U	3	3	0	0	0	0	1	0	0	1	553
477	89PSL 587	31	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	U	3	3	0	0	0	0	0	0	2	1	587
478	89PSL 602	32	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	U	3	3	0	0	0	0	0	0	1	1	602
479	89PSL 688	29	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	U	3	3	0	0	0	0	1	0	0	1	688
480	89PSL 699	28	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS LO		1	1	F	3	3	0	0	0	0	1	0	0	1	699
Stats:	n =	29						Statistics:	n =	29	29		29	29	29	29	29	29	29	29	29	29	
	ave.	30.							ave	1.0	1.1		3	3.0	0	0	0	0	.52	.34	.93	.97	
	std.	1.4							std	.18	.25		0	.18	0	0	0	0	.50	.48	.98	.18	
	SE =	.26							SE	.03	.05		0	.03	0	0	0	0	.09	.09	.18	.03	
																			n =	10	16		
																			frequency =	.34	.55		

#	Proc. #	TL (mm)	S#	Jar number	Sample date	Stream #	Stream name	Location	OS	Atly	art	sex	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	SPF	Proc. #
481	89PSL 10	30	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	2	F	3	3	0	0	0	0	1	1	0	1	10
482	89PSL 15	32	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	F	3	2	0	0	0	0	1	1	0	1	15
483	89PSL 34	27	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	F	3	3	0	0	0	0	1	0	0	1	34
484	89PSL 72	28	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	F	3	3	0	0	0	0	0	0	1	1	72
485	89PSL 78	33	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	U	3	3	0	0	0	0	0	0	0	1	78
486	89PSL 86	30	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	F	3	3	0	0	0	0	0	1	2	1	86
487	89PSL 93	32	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	U	3	3	0	0	0	0	0	0	0	1	93
488	89PSL 108	25	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	U	2	3	0	0	0	0	0	0	0	1	108
489	89PSL 109	32	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	U	3	3	0	0	0	0	1	1	0	1	109
490	89PSL 136	32	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	F	3	3	0	0	0	0	1	1	1	1	136
491	89PSL 173	32	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	F	3	3	0	0	0	0	0	0	0	0	173
492	89PSL 176	32	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	F	3	3	0	0	0	0	0	1	0	1	176
493	89PSL 207	27	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	U	3	3	0	0	0	0	1	0	0	1	207
494	89PSL 249	32	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	F	3	3	0	0	0	0	1	0	1	1	249
495	89PSL 258	32	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	F	3	3	0	0	0	0	0	0	0	1	258
496	89PSL 279	32	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	F	3	3	0	0	0	0	1	0	0	1	279
497	89PSL 283	32	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	F	3	3	0	0	0	0	1	0	2	1	283
498	89PSL 328	32	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	U	3	3	0	0	0	0	2	0	0	1	328
499	89PSL 343	31	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	U	3	3	0	0	0	0	1	1	0	1	343
500	89PSL 350	32	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	U	3	3	0	0	0	0	1	1	0	2	350
501	89PSL 354	33	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	F	3	3	0	0	0	0	0	1	3	1	354
502	89PSL 355	29	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	F	3	3	0	0	0	0	1	1	0	1	355
503	89PSL 409	31	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	U	3	3	0	0	0	0	1	1	0	1	409
504	89PSL 422	33	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	F	3	3	0	0	0	0	0	0	0	1	422
505	89PSL 451	31	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	F	3	3	0	0	0	0	1	0	0	1	451
506	89PSL 456	32	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	F	3	3	0	0	0	0	0	0	2	1	456
507	89PSL 475	31	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	F	3	3	0	0	0	0	0	1	0	1	475
508	89PSL 570	32	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	U	3	3	0	0	0	0	1	0	0	1	570
509	89PSL 580	32	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	U	3	3	0	0	0	0	1	0	1	1	580
510	89PSL 658	32	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	U	3	3	0	0	0	0	0	0	1	1	658
511	89PSL 698	31	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	F	3	3	0	0	0	0	0	1	0	1	698
512	89PSL 712	31	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS LO	1	1	F	3	3	0	0	0	0	0	1	0	1	712
Stats:	n =	32						Statistics:	n =	32	32		32	32	32	32	32	32	32	32	32	32	
	ave.	31.							ave	1	1.0		3.0	3.0	0	0	0	0	.56	.44	.44	1	
	std.	1.9							std	0	.17		.17	.17	0	0	0	0	.56	.50	.79	.25	
	SE =	.33							SE	0	.03		.03	.03	0	0	0	0	.10	.09	.14	.04	
																			n =	14	9		
																			frequency =	.44	.28		

#	Proc. #	TL (mm)	S#	Jar number	Sample date	Stream #	Stream name	Location	OS	Atly	art	sex	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	SPF	Proc. #
513	89PSL 37	31	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1	U	3	2	0	0	0	0	0	1	0	1	37
514	89PSL 47	29	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1	U	3	3	0	0	0	0	1	1	0	1	47
515	89PSL 59	none	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	only 27 larvae were sampled; this # not used													59
516	89PSL 75	32	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1	F	3	3	0	0	0	0	1	0	2	1	75
517	89PSL 85	none	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	only 27 larvae were sampled; this # not used													85
518	89PSL 101	31	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1	U	3	3	0	0	0	0	1	0	0	1	101
519	89PSL 114	30	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	2	F	3	3	0	0	0	0	0	1	2	1	114
520	89PSL 195	32	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1	F	3	3	0	0	0	0	1	0	1	1	195
521	89PSL 215	30	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	2	U	3	3	0	0	0	0	1	0	0	1	215
522	89PSL 228	30	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1	F	3	3	0	0	0	0	1	1	0	1	228
523	89PSL 245	33	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1	U	3	3	0	0	0	0	1	1	0	1	245
524	89PSL 263	30	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1	U	3	3	0	0	0	0	2	0	1	1	263
525	89PSL 372	29	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1	U	3	3	0	0	0	0	1	1	2	1	372
526	89PSL 379	30	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1	F	3	3	0	0	0	0	2	0	1	1	379
527	89PSL 416	30	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1	F	3	3	0	0	0	0	1	0	1	1	416
528	89PSL 434	none	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	only 27 larvae were sampled; this # not used													434
529	89PSL 439	33	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1	U	3	3	0	0	0	0	1	1	2	1	439
530	89PSL 458	32	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1	U	3	3	0	0	0	0	0	0	0	1	458
531	89PSL 483	32	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1	U	3	3	0	0	0	0	1	0	1	1	483
532	89PSL 487	32	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1	F	3	3	0	0	0	0	1	1	1	1	487
533	89PSL 489	30	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1	U	3	3	0	0	0	0	1	0	0	1	489
534	89PSL 513	33	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1	U	3	3	0	0	0	0	1	0	3	1	513
535	89PSL 524	32	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1	U	3	3	0	0	0	0	1	0	0	1	524
536	89PSL 529	none	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	only 27 larvae were sampled; this # not used													529
537	89PSL 591	30	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1	U	3	3	0	0	0	0	1	1	1	1	591
538	89PSL 606	30	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1	F	3	3	0	0	0	0	1	0	0	1	606
539	89PSL 628	33	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1	F	3	3	0	0	0	0	1	1	2	1	628
540	89PSL 645	33	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1	F	3	3	0	0	0	0	0	0	0	1	645
541	89PSL 669	none	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	only 27 larvae were sampled; this # not used													669
542	89PSL 710	32	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1	F	3	3	0	0	0	0	0	0	0	1	710
543	89PSL 713	33	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1	U	3	3	0	0	0	0	0	0	0	1	713
544	89PSL 736	32	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1	U	3	3	0	0	0	0	0	0	2	1	736

Stats: n = 27
ave. 31.
std. 1.3
SE = .25

Statistics:

n =	27	27	27	27	27	27	27	27	27	27	27	27	
ave	1	1.1	3	3.0	0	0	0	0	.81	.37	.81	1	
std	0	.26	0	.19	0	0	0	0	.55	.48	.90	0	
SE	0	.05	0	.04	0	0	0	0	.11	.09	.17	0	
									n =	10	14		
									frequency =	.37	.52		

#	Proc. #	TL (mm)	S#	Jar number	Sample date	Stream #	Stream name	Location	OS	Atly	art	sex	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	SPF	Proc. #
545	89PSL 23	34	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	0	1	1	1	23
546	89PSL 27	32	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	1	0	1	27
547	89PSL 52	31	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	0	1	1	1	52
548	89PSL 69	31	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	0	1	0	1	69
549	89PSL 106	31	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	1	0	0	1	106
550	89PSL 112	31	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	0	1	0	1	112
551	89PSL 116	31	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	1	0	1	116
552	89PSL 119	30	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	F	3	2	0	0	0	0	1	0	0	1	119
553	89PSL 127	31	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	1	1	0	1	127
554	89PSL 233	31	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	1	1	1	233
555	89PSL 293	28	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	2	A	.	0	0	0	.	0	0	0	3	0	293
556	89PSL 297	31	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	U	3	1	0	0	0	0	1	0	3	0	297
557	89PSL 304	32	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	1	1	0	1	304
558	89PSL 306	30	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	1	1	1	306
559	89PSL 314	32	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	1	1	1	314
560	89PSL 335	31	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	1	0	0	1	335
561	89PSL 338	31	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	1	0	3	1	338
562	89PSL 356	32	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	1	1	0	1	356
563	89PSL 371	32	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	1	0	1	371
564	89PSL 417	31	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	1	1	0	1	417
565	89PSL 427	30	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	1	1	0	1	427
566	89PSL 431	30	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	1	0	1	431
567	89PSL 482	32	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	1	0	0	1	482
568	89PSL 485	31	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	0	0	1	2	485
569	89PSL 499	31	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	0	0	0	1	499
570	89PSL 512	33	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	0	1	1	512
571	89PSL 571	30	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	1	0	1	571
572	89PSL 579	33	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	1	0	0	1	579
573	89PSL 623	33	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	0	1	0	1	623
574	89PSL 625	31	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	0	0	1	625
575	89PSL 626	30	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	0	1	1	626
576	89PSL 646	31	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	1	0	0	1	646
Stats:	n =	32						Statistics:	n =	32	32		31	32	31	32	31	32	32	32	32	32	
	ave.	31.							ave	1	1.0		3	2.8	0	0	0	0	.75	.56	.53	.97	
	std.	1.1							std	0	.17		0	.63	0	0	0	0	.43	.50	.90	.30	
	SE =	.20							SE	0	.03		0	.11	0	0	0	0	.08	.09	.16	.05	
																			n =	18	11		
																			frequency =	.56	.34		

#	Proc. #	TL (mm)	S#	Jar number	Sample date	Stream #	Stream name	Location	OS	Atly	art	sex	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	SPF	Proc. #
577	89PSL 1	28	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	0	0	1	2	1
578	89PSL 5	30	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	0	0	1	1	5
579	89PSL 6	30	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	1	1	1	6
580	89PSL 17	30	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	0	0	1	17
581	89PSL 39	32	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	1	1	1	1	39
582	89PSL 82	29	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	1	2	1	82
583	89PSL 96	29	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	0	0	2	96
584	89PSL 132	30	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	2	U	3	3	0	0	0	0	1	1	0	1	132
585	89PSL 158	29	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	1	3	2	158
586	89PSL 169	30	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	0	0	1	169
587	89PSL 177	31	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	0	0	0	1	177
588	89PSL 193	31	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	1	0	1	193
589	89PSL 196	30	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	1	0	3	1	196
590	89PSL 247	30	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	0	3	1	247
591	89PSL 296	30	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	0	1	1	296
592	89PSL 330	30	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	3	U	3	3	0	0	0	NG	0	1	1	1	330
593	89PSL 357	30	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	2	F	3	3	0	0	0	0	1	0	0	2	357
594	89PSL 476	29	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	0	2	1	476
595	89PSL 478	29	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	1	1	3	1	478
596	89PSL 519	30	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	0	0	2	1	519
597	89PSL 530	31	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	2	U	3	3	0	0	0	0	1	0	1	1	530
598	89PSL 532	30	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	0	0	2	532
599	89PSL 576	31	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	1	0	2	1	576
600	89PSL 584	30	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	1	1	0	1	584
601	89PSL 592	30	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	0	0	1	592
602	89PSL 652	29	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	0	0	1	652
603	89PSL 661	30	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	1	0	3	2	661
604	89PSL 668	0	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	0	1	1	668
605	89PSL 675	30	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	0	1	1	675
606	89PSL 684	29	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	F	3	3	0	0	0	0	1	0	0	1	684
607	89PSL 708	29	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	2	U	3	3	0	0	0	0	1	0	2	1	708
608	89PSL 731	31	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1	U	3	3	0	0	0	0	1	0	1	2	731
Stats:	n =	32						Statistics:	n =	32	32		32	32	32	32	32	31	32	32	32	32	
	ave.	29.							ave	1	1.2		3	3	0	0	0	0	.84	.28	1.1	1.2	
	std.	5.3							std	0	.46		0	0	0	0	0	0	.36	.45	1.1	.41	
	SE =	.93							SE	0	.08		0	0	0	0	0	0	.06	.08	.19	.07	
																			n =	9	20		
																			frequency =	.28	.63		

#	Proc. #	TL (mm)	S#	Jar number	Sample date	Stream #	Stream name	Location	OS	Atly	art	sex	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	SPF	Proc. #
609	89PSL 11	32	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	U	3	3	0	0	0	0	0	0	1	1	11
610	89PSL 74	32	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	U	3	3	0	0	0	0	0	0	2	1	74
611	89PSL 157	33	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	U	3	3	0	0	0	0	1	0	0	1	157
612	89PSL 166	32	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	2	U	3	0	0	0	0	0	1	0	1	1	166
613	89PSL 208	32	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	F	3	3	0	0	0	0	1	0	0	1	208
614	89PSL 217	33	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	F	3	3	0	0	0	0	0	1	0	1	217
615	89PSL 232	32	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	F	3	3	0	0	0		1	1	1	1	232
616	89PSL 248	32	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	2	U	2	3	0	0	0	0	1	1	2	1	248
617	89PSL 259	32	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	F	3	3	0	0	0	0	1	0	1	1	259
618	89PSL 276	32	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	F	3	3	0	0	0	0	0	0	0	1	276
619	89PSL 311	31	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	U	3	3	0	0	0	0	1	0	2	1	311
620	89PSL 369	30	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	F	3	3	0	0	0	0	1	1	0	1	369
621	89PSL 370	32	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	U	3	3	0	0	0	0	1	0	0	1	370
622	89PSL 389	32	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	F	3	3	0	0	0	0	0	0	1	1	389
623	89PSL 423	32	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	U	3	3	0	0	0	0	1	0	0	1	423
624	89PSL 430	31	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	F	2	3	0	0	0	0	1	0	0	1	430
625	89PSL 441	31	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	F	3	3	0	0	0	0	1	1	0	1	441
626	89PSL 492	31	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	F	3	3	0	0	0	0	0	0	1	1	492
627	89PSL 500	32	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	U	3	3	0	0	0	0	0	1	0	1	500
628	89PSL 596	31	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	U	2	3	0	0	0	0	1	0	0	1	596
629	89PSL 616	32	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	2	F	3	3	0	0	0	0	1	1	1	1	616
630	89PSL 617	32	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	F	3	3	0	0	0	0	1	1	1	1	617
631	89PSL 624	32	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	F	3	2	0	0	0	0	0	0	1	1	624
632	89PSL 639	30	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	2	U	3	3	0	0	0	0	1	0	2	1	639
633	89PSL 641	31	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	F	3	3	0	0	0	0	0	0	0	1	641
634	89PSL 643	32	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	F	3	3	0	0	0	0	0	0	1	1	643
635	89PSL 651	31	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	U	3	3	0	0	0	0	0	1	0	1	651
636	89PSL 657	32	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	U	3	3	0	0	0	0	1	0	2	1	657
637	89PSL 659	32	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	U	3	3	0	0	0	0	1	0	3	1	659
638	89PSL 674	31	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	F	3	3	0	0	0	0	0	0	2	1	674
639	89PSL 680	32	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	U	3	3	0	0	0	0	0	0	0	1	680
640	89PSL 730	33	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1	U	3	3	0	0	0	0	0	0	0	1	730

Stats:	n =	32
	ave.	32.
	std.	.72
	SE =	.13

Statistics:	n =	32	32	32	32	32	32	32	32	32	32	32
	ave	1	1.1	2.9	2.9	0	0	0	0	.56	.28	.78	1
	std	0	.33	.29	.54	0	0	0	0	.50	.45	.86	0
	SE	0	.06	.05	.10	0	0	0	0	.09	.08	.15	0
										n =	9	17	
										frequency =	.28	.53	

#	Proc. #	TL (mm)	S#	Jar number	Sample date	Stream #	Stream name	Location	OS	Atly	art	sex	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	SPF	Proc. #
641	89PSL 14	30	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	F	3	3	0	0	0	0	1	1	0	1	14
642	89PSL 21	27	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	F	3	3	0	0	0	0	1	1	1	1	21
643	89PSL 44	32	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	2	U	3	3	0	0	0	0	1	1	0	1	44
644	89PSL 97	32	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	3	3	0	0	0	0	1	1	0	1	97
645	89PSL 107	29	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	3	3	0	0	0	0	1	1	0	1	107
646	89PSL 118	30	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	3	3	0	0	0	0	1	1	1	1	118
647	89PSL 144	30	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	3	3	0	0	0	0	1	0	0	1	144
648	89PSL 159	30	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	3	3	0	0	0	0	1	1	0	1	159
649	89PSL 197	33	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	3	3	0	0	0	0	0	0	0	1	197
650	89PSL 225	28	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	F	3	3	0	0	0	0	1	1	0	1	225
651	89PSL 242	30	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	2	F	3	3	0	0	0	0	1	1	0	1	242
652	89PSL 295	32	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	3	3	0	0	0	0	1	0	3	0	295
653	89PSL 326	31	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	2	F	3	3	0	0	0	0	1	1	0	1	326
654	89PSL 353	30	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	3	3	0	0	0	0	1	1	2	1	353
655	89PSL 377	32	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	3	3	0	0	0	0	1	0	1	1	377
656	89PSL 388	29	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	F	3	3	0	0	0	0	1	1	0	1	388
657	89PSL 394	32	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	F	3	3	0	0	0	0	1	1	1	1	394
658	89PSL 410	31	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	2	U	3	3	0	0	0	0	0	0	0	1	410
659	89PSL 435	32	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	3	3	0	0	0	0	0	1	0	1	435
660	89PSL 449	30	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	.	3	0	0	0	0	1	1	3	1	449
661	89PSL 465	30	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	F	3	3	0	0	0	0	1	1	1	1	465
662	89PSL 494	31	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	3	3	0	0	0	0	1	0	0	1	494
663	89PSL 511	29	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	3	3	0	0	0	0	1	0	0	1	511
664	89PSL 520	30	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	F	3	3	0	0	0	0	1	1	0	1	520
665	89PSL 543	32	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	F	3	3	0	0	0	0	1	0	0	1	543
666	89PSL 565	31	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	3	3	0	0	0	0	1	1	1	1	565
667	89PSL 600	30	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	3	3	0	0	0	0	1	1	0	1	600
668	89PSL 605	30	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	2	U	3	3	0	0	0	0	1	0	0	1	605
669	89PSL 621	29	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	2	U	3	3	0	0	0	0	0	0	1	1	621
670	89PSL 622	33	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	3	3	0	0	0	0	0	1	2	1	622
671	89PSL 654	32	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	F	3	3	0	0	0	0	0	1	0	1	654
672	89PSL 702	29	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	2	F	3	3	0	0	0	0	1	1	0	1	702
Stats:	n =	32						Statistics:	n =	32	32		31	32	32	32	32	32	32	32	32	32	
	ave.	31.							ave	1	1.2		3	3	0	0	0	0	.81	.69	.53	.97	
	std.	1.4							std	0	.41		0	0	0	0	0	0	.39	.46	.87	.17	
	SE =	.25							SE	0	.07		0	0	0	0	0	0	.07	.08	.15	.03	
																			n =	22	11		
																			frequency =		.69	.34	

#	Proc. #	TL (mm)	S#	Jar number	Sample date	Stream #	Stream name	Location	OS	Atly	art	sex	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	SPF	Proc. #
673	89PSL 31	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	F	3	3	0	0	0	0	0	1	0	1	31
674	89PSL 45	30	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	F	3	3	0	0	0	0	1	1	0	1	45
675	89PSL 61	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	3	3	0	0	0	0	1	0	0	1	61
676	89PSL 64	30	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	3	3	0	0	0	0	1	1	0	1	64
677	89PSL 67	28	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	2	F	3	3	0	0	0	0	1	1	0	1	67
678	89PSL 81	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	F	3	3	0	0	0	0	0	1	0	1	81
679	89PSL 102	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	F	3	3	0	0	0	0	0	1	0	1	102
680	89PSL 130	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	F	3	3	0	0	0	0	0	0	0	1	130
681	89PSL 156	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	3	3	0	0	0	0	0	0	2	1	156
682	89PSL 162	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	F	3	3	0	0	0	0	1	1	1	1	162
683	89PSL 165	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	F	3	3	0	0	0	0	1	0	0	1	165
684	89PSL 171	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	2	U	3	3	0	0	0	0	1	1	0	0	171
685	89PSL 174	28	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	3	3	0	0	0	0	1	0	0	1	174
686	89PSL 178	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	F	3	3	0	0	0	0	1	1	2	1	178
687	89PSL 250	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	2	U	3	3	0	0	0	0	1	1	2	1	250
688	89PSL 265	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	F	3	3	0	0	0	0	1	1	2	1	265
689	89PSL 274	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	3	3	0	0	0	0	1	1	0	1	274
690	89PSL 316	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	F	3	3	0	0	0	0	1	1	0	1	316
691	89PSL 319	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	2	F	3	3	0	0	0	0	1	1	1	1	319
692	89PSL 365	28	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	3	3	0	0	0	0	0	0	0	2	365
693	89PSL 450	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	F	3	3	0	0	0	0	1	1	0	1	450
694	89PSL 454	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	3	3	0	0	0	0	1	1	3	2	454
695	89PSL 509	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	2	U	3	3	0	0	0	0	1	0	0	1	509
696	89PSL 525	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	2	F	3	3	0	0	0	0	1	0	2	1	525
697	89PSL 542	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	3	3	0	0	0	0	1	1	0	1	542
698	89PSL 563	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	3	3	0	0	0	0	0	1	2	1	563
699	89PSL 578	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	F	3	3	0	0	0	0	1	0	0	1	578
700	89PSL 590	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	U	3	3	0	0	0	NG	0	0	0	1	590
701	89PSL 667	30	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	2	F	3	3	0	0	0	0	1	0	1	2	667
702	89PSL 670	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	2	A	.	0	0	0	.	NG	0	0	2	1	670
703	89PSL 673	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	F	3	3	0	0	0	0	0	0	3	1	673
704	89PSL 683	29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1	F	3	3	0	0	0	0	0	0	0	1	683
Stats:	n =	32						Statistics:	n =	32	32		31	32	32	32	31	30	32	32	32	32	
	ave.	29							ave	1	1.3		3	2.9	0	0	0	0	.66	.56	.72	1.1	
	std.	.43							std	0	.43		0	.52	0	0	0	0	.47	.50	1.0	.35	
	SE =	.08							SE	0	.08		0	.09	0	0	0	0	.08	.09	.18	.06	
																			n =	18	12		
																			frequency =	.56	.38		

#	Proc. #	TL (mm)	S#	Jar number	Sample date	Stream #	Stream name	Location	OS	Atly	art	sex	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	SPF	Proc. #
705	89PSL 2	33	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	U	3	2	0	0	0	0	0	0	1	1	2
706	89PSL 94	32	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	U	3	2	0	0	0	0	0	0	1	0	94
707	89PSL 134	32	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	U	3	3	0	0	0	0	1	1	0	1	134
708	89PSL 147	33	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	U	3	2	0	0	0	0	1	1	1	1	147
709	89PSL 170	30	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	F	3	3	0	0	0	0	1	1	0	1	170
710	89PSL 172	30	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	F	3	3	0	0	0	0	1	0	0	1	172
711	89PSL 238	31	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	F	3	3	0	0	0	0	1	0	3	1	238
712	89PSL 278	31	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	U	3	3	0	0	0	0	2	0	0	1	278
713	89PSL 313	33	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	U	3	2	0	0	0	0	0	0	1	1	313
714	89PSL 324	32	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	U	3	3	0	0	0	0	1	1	0	1	324
715	89PSL 331	33	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	F	3	2	0	0	0	0	1	1	0	1	331
716	89PSL 346	31	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	F	3	3	0	0	0	0	1	1	0	1	346
717	89PSL 349	32	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	U	3	2	0	0	0	0	0	1	0	1	349
718	89PSL 367	30	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	U	3	3	0	0	0.	0	1	1	0	2	367
719	89PSL 393	32	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	F	3	2	0	0	0	0	0	1	0	1	393
720	89PSL 405	33	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	2	F	3	2	0	0	0	0	1	0	0	1	405
721	89PSL 463	29	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	F	3	3	0	0	0	0	1	1	0	1	463
722	89PSL 488	30	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	U	3	3	0	0	0	0	1	1	2	1	488
723	89PSL 503	32	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	U	3	3	0	0	0	0	1	0	0	1	503
724	89PSL 526	30	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	F	3	3	0	0	0	0	1	0	1	1	526
725	89PSL 527	31	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	F	3	3	0	0	0	0	1	0	0	1	527
726	89PSL 534	30	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	F	3	3	0	0	0	0	1	0	0	1	534
727	89PSL 552	32	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	F	3	3	0	0	0	0	1	1	0	1	552
728	89PSL 557	33	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	U	3	3	0	0	0	0	0	1	0	1	557
729	89PSL 572	30	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	U	3	3	0	0	0	0	1	1	0	1	572
730	89PSL 577	32	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	F	3	2	0	0	0	0	1	0	0	1	577
731	89PSL 586	30	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	U	3	3	0	0	0	0	1	1	1	1	586
732	89PSL 608	31	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	F	3	3	0	0	0	0	1	0	1	1	608
733	89PSL 637	33	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	U	3	2	0	0	0	0	1	1	0	1	637
734	89PSL 672	29	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	F	3	3	0	0	0	0	1	0	0	2	672
735	89PSL 689	32	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	U	3	2	0	0	0	0	1	1	2	1	689
736	89PSL 700	32	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS C		1	1	U	3	3	0	0	0	0	0	1	0	1	700
Stats:	n =	32						Statistics:	n =	32	32		32	32	32	32	32	32	32	32	32	32	
	ave.	31.							ave. =	1	1.0		3	2.7	0	0	0	0	.81	.56	.44	1.0	
	std.	1.2							std. =	0	.17		0	.47	0	0	0	0	.46	.50	.75	.30	
	SE =	.22							SE =	0	.03		0	.08	0	0	0	0	.08	.09	.13	.05	
																			n =	18	10		
																			frequency =	.56	.31		

	Frequency GIF			Frequency CT		
	C	LO	O	C	LO	O
	.53	.69	.50	.22	.22	.41
	.47	.34	.53	.72	.55	.53
	.22	.44	.44	.50	.28	.34
	.47		.47	.28		.34
	.37		.59	.52		.44
	.56		.41	.34		.41
	.28		.28	.63		.53
	.69		.69	.34		.34
	.56		.56	.38		.38
	.56		.56	.31		.31
mean	.47	.48	.46	.41	.40	.43

Mean TL (mm)	S#	Jar number	Sample date	Stream #	Stream name	Location	OS	Mean score: Atly	art	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	SPF
31.	1	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1.2	3	3	0	0	0	0	.81	.69	.53	.97
29	2	89-1500	14 APR 1989	485	NONE	WEST FINGER	C	1	1.3	3	2.9	0	0	0	0	.66	.56	.72	1.1
31.	3	89-1501	15 APR 1989	495	CHIMEVSKY CR	MCCLURE BAY	C	1	1.1	3	3.0	0	0	0	0	.81	.37	.81	1
30.	7	89-1505	16 APR 1989	630	BAINBRIDGE	WHALE BAY	C	1	1	3	2.9	0	0	0	0	.72	.44	.31	.97
24.	8	89-1506	17 APR 1989	632	CLAW	WHALE BAY	C	1	1.4	2.5	3	0	.09	0	0	.47	.22	.91	.47
32.	16	89-1514	20 APR 1989	695	NONE	PORT AUDREY	C	1.0	1	1.6	1.5	0	.09	0	0	0	.53	.31	.5
29.	19	89-1517	21 APR 1989	740	KELEZ	MONTAGUE IS	C	1	1.2	3	3	0	0	0	0	.84	.28	1.1	1.2
31.	20	89-1518	22 APR 1989	749	SHAD	MONTAGUE IS	C	1	1.0	3	2.8	0	0	0	0	.75	.56	.53	.97
31.	21	89-1519	23 APR 1989	828	COOKE	HICHINBROOKE IS	C	1	1.0	3	2.7	0	0	0	0	.81	.56	.44	1.0
31.	22	89-1520	23 APR 1989	861	BERNARD	HAWKINS IS	C	1.0	1.1	2.9	2.8	.03	0	0	0	.5	.47	.44	1.6
30.	23	89-1521	24 APR 1989	35	KOPPEN	SHEEP BAY	C	1.1	1.3	2.9	3	0	0	.03	0	.78	.47	.97	.03
31.	5	89-1503	16 APR 1989	604	ERB	EWAN	LO	1	1.0	2.9	2.8	0	0	0	0	.69	.47	.84	.91
32.	6	89-1504	16 APR 1989	621	TOTEMOFF	CHENEGA	LO	1	1.0	3.0	2.9	0	0	0	0	.69	.69	.31	.94
30.	10	89-1508	17 APR 1989	673	FALLS	LATOUCHE IS	LO	1.0	1.1	3	3.0	0	0	0	0	.52	.34	.93	.97
31.	11	89-1509	18 APR 1989	677	HAYDEN	LATOUCHE IS	LO	1	1.0	3.0	3.0	0	0	0	0	.56	.44	.44	1
31.	4	89-1502	15 APR 1989	506	LOOMIS	ESHAMY BAY	O	1	1.4	2.9	3.0	0	0	0	0	.72	.41	.66	.09
29.	9	89-1507	17 APR 1989	637	PT COUNTESS	WHALE BAY	O	1	1.1	2.8	2.9	0	0	0	0	.66	.47	.63	.97
30.	12	89-1510	18 APR 1989	678	NONE	SLEEPY BAY	O	1	1.0	3.0	3.0	0	.06	0	0	1.1	.5	.47	1
29.	13	89-1511	18 APR 1989	663	NONE	SHELTER BAY	O	1	1	2.9	3	0	.06	0	0	.84	.47	.72	.53
30.	14	89-1512	19 APR 1989	628	NONE	CHENEGA IS	O	1	1.1	2.9	3.0	.06	0	0	0	.72	.59	.66	.88
31.	15	89-1513	19 APR 1989	692	NONE	KNIGHT IS	O	1	1.1	3	3.0	0	0	0	0	.88	.44	.59	.78
32.	17	89-1515	21 APR 1989	682	NONE	SNUG HARBOR	O	1	1.1	2.9	2.9	0	0	0	0	.56	.28	.78	1
32.	18	89-1516	21 APR 1989	681	NONE	HOGAN BAY	O	1.0	1.1	2.1	1.9	.13	0	0	0	.72	.53	.84	.97

Length			Atly	art	GLY	YOLK	EA	MDN	IHN	VDGG	ECN	GIF	CT	SPF
30.	Overall mean	Clean	1.0	1.1	2.8	2.8	.00	.02	.00	0	.65	.47	.64	.89
2.3	Overall st. dev.		.02	.14	.42	.44	.01	.04	.01	0	.25	.14	.27	.42
31.	Overall mean	Lt. Oil	1.0	1.0	3.0	2.9	0	0	0	0	.61	.48	.63	.95
.56	Overall st. dev.		.02	.02	.03	.09	0	0	0	0	.09	.15	.30	.04
30.	Overall mean	Oiled	1.0	1.1	2.8	2.8	.02	.02	0	0	.78	.46	.67	.78
1.0	Overall st. dev.		.01	.12	.30	.38	.05	.03	0	0	.17	.09	.12	.32

CHAPTER 10 - Histopathology in pink salmon adults from Prince William Sound, Alaska, 1990.

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Methods

All tissues received by Dr. Hinton's laboratory were logged in and recorded in numerical order, as closely to the numbers designated by ADF&G as possible. All tissue vials we received had been labeled as to site of origin by ADF&G, but were not labeled by exposure history. A random number was assigned to each pink salmon in the "P" group (1 through 200, received from Sam Sharr; Table X-1) and "S" group (1 through 320, received from Henry Yuen; Table X-2). All tissues were processed routinely in paraffin and stained with hematoxylin and eosin. Slides were read in ascending numerical order based on the assigned random number (i.e., blind study); the "P" samples were read separately from the "S" group. Lesions were subjectively ranked using a 4 point scale: none (0), mild (1), moderate (2), or severe (3). Basic historical/site data and significant lesion scores are listed in Tables X-1 and X-2. After the nature and extent of lesions in each tissue were recorded, results were reassembled into groups by site of capture. Statistics were computed based on site of origin, and later (after results were reported to ADF&G and exposure history revealed) by exposure status of the sites.

Results

Histopathology

Lesion scores for each fish are listed in Tables X-1 and X-2; the final page of each table summarizes means of major lesions, separated by sex and exposure history of home streams. In the initial progress report, basic statistics for individual lesions in pink salmon adults were computed. Males were significantly different from females in the amount of hepatic glycogen depletion (t-test, $P<0.5$); therefore, site comparisons were made using "male" and "female" as additional treatments. With ANOVA, only 2 variables (glycogen depletion for males, and hepatic megalocytes for males and females), exhibited homogeneity of variance (and, hence, only these variables were appropriate for use with ANOVA) and are reported in the appropriate results section below. Overall statistical significance using principal components analysis is described on page X-5. Because we did not detect any oil related differences in lesion prevalence, these results will not be prepared for publication. The format of this chapter is retained in the original report format: primarily an outline and tabular presentation of findings. Basic findings in each organ are outlined below:

I. **Liver**
   A. Normal Histology: A few salmon had glycogen-laden livers without any inflammatory or degenerative lesions.

B. Megalocytes: Megalocytes were common, but only a few livers had large numbers. Affected hepatocytes had varying degrees of karyomegaly, and enlarged nuclei were usually round to oval, with prominent, sometimes multiple, nucleoli and vesicular chromatin. In a few fish with severe megalocytosis, there were also increased numbers of mitotic figures and clusters of bile preductular epithelial cells (presumptive stem cells).

When analyzed separately, site differences among mean megalocyte scores were weakly significant for males ($P = 0.066$) and for females ($P = 0.072$). However, when the sexes were combined in a 2-way ANOVA (with sex and site as the variables), scores by site were significantly different ($P = 0.005$), with no differences in scores due to sex or sex-site interaction ($P \geq 0.05$). Separating the means with Tukey's studentized range test demonstrated a significant difference between fish from Sleepy Bay (lower score) and West Finger Creek (higher score); no other site comparisons were significant at the $\alpha = 0.05$ level.

C. Sinusoidal fibrosis: Sinusoidal fibrosis, similar to that described in rockfish, was only observed in a few salmon. Other salmon had foci of fibrosis, but fibrosis seemed to be related to previous or ongoing necrosis of hepatocytes.

D. Necrosis
   1. Coagulation necrosis: Some salmon had randomly scattered, small foci of hepatocellular necrosis which were usually associated with infiltration with small numbers of lymphocytes, neutrophils, and varying degrees of fibrosis.

   2. Single cell necrosis: Single hepatocellular necrosis was common and was usually found in those livers which had glycogen depletion, but which did not have loss of hepatocyte volume. The hepatocytes in these livers had abundant, homogeneous, non-vacuolated, basophilic cytoplasm. Necrotic hepatocytes were usually rounded up and shrunken with pyknotic nuclei. Some livers had phagocytosis of dead hepatocytes by individual macrophages.

E. Inflammation
   1. Macrophage aggregates: not observed

   2. Lymphocytic aggregates: usually few in number and often associated with focal hepatocyte necrosis

   3. Neutrophilic aggregates: uncommon and usually associated with *Ichthyophonus hoferi* infection

F. Hepatocyte storage disorders
   1. Glycogen depletion: Glycogen depletion was common. In some fish, depleted hepatocytes had severe loss of cytoplasm and hepatocyte nuclei were tightly clustered together. In other fish, depletion of glycogen was not associated with

any loss of cytoplasm and affected hepatocytes were large with abundant basophilic cytoplasm. Differences among mean glycogen scores for various sites were highly significant for males ($P = 0.0001$).

2. Lipidosis (hepatic fatty change): Lipidosis was fairly common and varied from mild to severe. A few fish had discrete foci of lipidosis and some had large numbers of presumptive Ito cells which were packed with large lipid droplets.

3. Eosinophilic "protein" droplets: Some salmon had small to large amounts of round, refractile, eosinophilic, cytoplasmic droplets in hepatocytes. The material resembles protein that is occasionally seen within renal tubules. These protein droplets were primarily found in female fish and may represent vitellogenin or large heterolysosomes.

G. Bile duct hyperplasia: not observed

H. Parasitism
   1. *Ichthyophonus* sp.: A few were occasionally seen.

   2. Microsporidian sp.: Microsporidian xenomas were occasionally seen and may represent *Loma salmonae*.

   3. Myxosporean sp.: An unidentified myxosporean parasite was common in small numbers. The parasite had variably sized (50-200 µm diameter), spherical clusters of smaller multinucleate syncytia. The syncytia were 10-20 µm in diameter and contained from 5 to seven, small (1-3 µm), basophilic nuclei.

I. Many fish had congested sinusoids and in some, there was disruption of normal hepatic architecture with breakdown of sinusoidal walls and pooling of blood.

J. Preneoplastic Foci
   1. Eosinophilic foci: One fish (from Humpy Creek, a control site) had a large eosinophilic focus. The focus was discrete and composed of hepatocytes packed with large, refractile, eosinophilic vacuoles. The focus contained scattered dead hepatocytes.

   2. Clear cell foci: Two fish had clear cell foci which were composed of hepatocytes filled with large amounts of homogeneous pale white to amphophilic cytoplasm One of the fish was from Humpy Creek, a control site.

II. **Kidney**

A. Normal histology: Normal renal histology consisted of tubules, glomeruli, and interstitial hematopoietic tissue. Many sections also included the corpuscle of Stannius.

B. Renal tubular degeneration and necrosis: A few salmon had mild to moderate vacuolar degeneration and necrosis of renal tubular epithelial cells.

C. Glomerulonephritis: Membranous glomerulonephritis was common.

D. Inflammation: Inflammation was usually limited to small aggregates of lymphocytes.

E. Parasitism:
   1. Microsporidian sp.: Many salmon had microsporidian xenomas in glomeruli and occasionally in the renal interstitium. The xenomas probably represent *Loma salmonae*.

   2. Myxosporean sp.: A suspected myxosporean parasite was common and sometimes severe (extrasporogonic stage of *Sphaerospora* sp.?). These parasites were centered in renal tubular epithelium and were associated with marked tubular epithelial dysplasia (often with marked karyomegaly and nuclear distortion), degeneration, and necrosis. The parasites were similar to the myxosporeans in the liver and are probably the same species. In some kidneys, large numbers of parasites were also free within tubular lumina.

F. Protein: In some fish, small numbers of renal tubules were packed with refractile, eosinophilic, cytoplasmic, protein droplets.

III. **Spleen**
   A. Inflammation: Occasional lymphoid aggregates were seen.

   B. Periarteriolar sheath hyperplasia: none

   C. Parasitism: Some spleens had microsporidian xenomas similar to those in the liver and kidney.

IV. **Pancreas**: Pancreas was inadvertently submitted with liver or spleen in some fish. In some, there was severe single cell necrosis similar to that seen in the liver. A few fish also had marked ductular proliferation and vacuolation of exocrine cells.

V. **Nares**
   A. Inflammation
      1. Lymphocytic: The majority of nares had mild to moderate, diffuse, infiltration of the lamina propria with lymphocytes.

      2. Neutrophilic: Small numbers of neutrophils were often in the lamina propria.

      3. Eosinophilic granular leukocytes (EGLs): EGLs were a consistent finding in the perineural sheaths of large, unmyelinated nerves clustered at the base of the

sensory tufts. Infiltrates were severe in some fish and appeared in some to be associated with fibrosis of the nerve.

B. Hyperplasia
   1. Mucous cell hyperplasia: Some fish had mild to moderate mucous cell hyperplasia.

   2. Sensory epithelial hyperplasia: In a few fish, the stratified columnar epithelium appeared thickened.

Final comment on histopathologic lesions: Many of the lesions in the liver and pancreas were similar to those seen in both rockfish and adult Pacific herring, and were consistent with exposure to some hepatotoxic and pancreatotoxic agent. Of tissues submitted, the liver, pancreas (only a few were submitted, scores are not reported), and kidney had the most lesions and were the most useful. The nares proved to be less than adequate as a histological specimen for several reasons: 1) large amounts of gravel in the "S" group resulted in severe sectioning artifact; 2) additional artifact was encountered because the nares were not decalcified before processing; 3) normal histology varies depending on plane and depth of section [We step-sectioned completely through 2 nares and found significantly different morphology in different sections.]; and 4) the nares had few lesions. Because of the time involved in getting consistent sections and the lack of lesions, we think that resources could be better spent by eliminating the nares from histopathologic analysis under similar "damage assessment" type circumstances in the future.

Statistical analysis

For general details about the types of statistical analysis used, see part III, "Statistical Analysis" on page vii.

Statistical Consultant - Neil Willits, Senior Statistician, Division of Statistics, 380 Kerr Hall, University of California, Davis, 95616

Initial statistical analysis was by simple ANOVA of individual lesion scores (described above in the "Histopathology" section). Few site differences were identified with this type of analysis, and we were unable to speculate on exposure history of the various sites based on individual lesion scores. After exposure history of each site was revealed, lesions were subjected to principal components analysis to look for overall trends.

<u>1990 "P" Pink Salmon</u>

Due to missing values, the nares data was omitted from the analysis. Using scores from liver, kidney, and spleen, 190 of the 200 fish (95%) were used in the final principal components analysis. When differences in individual scale values were blocked by gender and compared using MANOVA, oiled vs. reference differences were not significant for the first, second, or third principal components but were significant for the fourth principal component. In the fourth

principal component, hepatic focal necrosis, single cell necrosis, and decreased hepatocellular volume contributed most to variability. Examination of mean lesion scores (Table X-1) reveals that these lesions were more severe in females than in males, but there was no clear trend towards increased lesion severity in fish from oiled sites.

Sex differences were significant for all but the fourth principal component. Individual scale values and mean lesion scores indicate that glycogen depletion and hepatocellular single cell necrosis were more severe in females than males. When the data were analyzed to include nested site effects, results were similar. Tests for overall effects were not significant for oiled vs. reference effects but were significant for sex effects (MANOVA). Although several pink salmon had hepatocellular megalocytosis, a lesion associated with toxicant exposure in other fish species, mean megalocytosis scores were greater in males and females from reference sites than in fish from oiled sites.

1990 "S" Pink Salmon Adults

Due to missing values, the nares data was omitted from the analysis. Using scores from liver, kidney, and spleen, 301 of the 320 fish (94%) were used in the final principal components analysis. When differences in individual scale values were blocked by gender, including nested site effects, and compared with MANOVA, oiled status differences were significant for the first, second, and third principal components but were not significant for the fourth principal component. However, when the "Type III MS for SITE(OS)" was used as an error term for oil status comparisons, none of the oil status differences were significant. Note that with this group of pink salmon, sites were classified in 3 ways: clean, lightly oiled, and oiled. By contrasting the differences between the 3 classes of exposure, in only the second principal component were lesions from "clean" salmon significantly different from "oiled" and "lightly oiled" fish, and "oiled" and "lightly oiled" salmon were not significantly different. In the second principal component, kidney scores for luminal debris and myxosporean parasites contributed most to variability. Mean lesion scores for the myxosporean parasites were greatest in males from clean sites, but female lesion scores were similar for all sites. Several pink salmon had hepatocellular megalocytosis, but mean scores were only slightly greater fish from oiled sites than in fish from clean sites.

Sex differences were significant for the first and second principal components. Individual scale values (first principal component) and mean lesion scores indicate that females had greater mean scores for glycogen depletion, decreased hepatocellular volume, hepatocellular single cell necrosis, as well as kidney and liver protein droplets.

When the data were analyzed without including nested site effects, overall results were similar. Tests for overall effects were not significant for oil status (MANOVA). However, the "oiled vs lightly oiled" differences were significant. Sex differences were highly significant.

Discussion

Recommendations for future sampling of pink salmon adults include:

1) sample liver, pancreas, kidney, and gill
2) submit 2 pieces of each organ
3) have a trained histopathologist on site for all necropsy and tissue sampling (as was done with the "P" group of salmon). This will ensure optimum specimen quality and lesion interpretation.

To conclude that lesions in pink salmon were related to oil exposure, sex, or sample site, similar findings would be expected in both studies ("P" and "S" groups). Indeed, site differences were obvious for several types of lesions, and sex differences were consistent for 3 hepatocellular changes: glycogen depletion, single cell necrosis, and decreased cytoplasmic volume. Also, kidney and liver protein droplets were increased in females in the S group of salmon, but only slightly increased in the P group. Both groups of pink salmon had lesions that have been associated with exposure to hydrocarbons or other toxicants: hepatic lipidosis (Khan and Kiceniuk 1984), hepatocellular glycogen depletion (Sabo et al. 1975, Hawkes 1977), decreased hepatocellular volume (Khan and Kiceniuk 1984), focal hepatocellular necrosis (Haensly et al. 1982, Solangi and Overstreet 1982), pancreatic acinar necrosis (DiMichele and Taylor 1978), single cell necrosis in the nares (Solangi and Overstreet 1982), and megalocytosis (Kent et al. 1988). However, in our study none of these lesions were clearly related to oil exposure status of the salmon's home stream. Possible explanations include: 1) lesions were not a result of oil exposure; or 2), because of common migration patterns of most pink salmon from Prince William Sound (Sam Sharr, personal communication), salmon from clean, lightly oiled, and oiled home streams might have been exposed to similar amounts of oil during the migratory growout phase of their life cycle. Note that oil exposure does not produce specific lesions, and lesions in fish might have resulted from secondary effects of oil (e.g., decreased or altered food supply). Resolving this problem would require comparison of histopathologic lesions in known-clean and oil-exposed pink salmon reared in a controlled setting.

Table X-1. Histopathology of "P" Pink Salmon Adult sampled from Prince William Sound in 1990.

Key to table symbols:
Alaska # = Sample number generated by ADF@G
Hinton processing # (proc. #) = Random number generated by Dr. Hinton's Laboratory
Sex = male (M) or female (F)
OS = oiled status; oiled (O), lightly oiled (LO), or control/clean (C)
Lesion scores = none (0), mild (1), moderate (2), severe (4), or tissue not present "."

LIVER:
- glycogen depletion (GLY)
- lipidosis (LIP)
- decreased hepatocyte volume (HV)
- hepatocellular protein droplets (PD)
- peliosis/congestion of sinusoids (PEL)
- single cell necrosis (SCN)
- focal necrosis (FN)
- hepatocellular karyomegaly (MEG)
- sinusoidal fibrosis (FIB)

SPLEEN (SPL): macrophage aggregates (MA)

KIDNEY:
- tubular epithelial vacuolar degeneration (VD)
- tubular epithelial protein droplets (PD)
- tubular luminal debris (LUM)
- tubular myxosporeans (MYX)
- glomerular microsporidians (GLM)

NARES:
- Eph'ic granulocytes in unmyelinated nerve sheathes (EGL)
- mucous cell hyperplasia in sensory epithelium (MUC)
- single cell necrosis in nasal sensory epithelium (SCN)

	Proc.		LIVER									KIDNEY					SPL	NARES			Stream			Alaska
#	#	Sex	GLY	LIP	HV	PD	PEL	SCN	FN	MEG	FIB	VD	PD	LUM	MYX	GLM	MA	EGL	MUC	SCN	#	Name	OS	#
1	P 4	F	2	0	2	0	1	1	0	1	0	3	0	0	0	0	0	1	1	1	678	Sleepy Bay	O	18
2	P 27	F	3	0	3	0	2	0	0	1	0	3	1	0	0	0	0	1	1	1	678	Sleepy Bay	O	9
3	P 46	F	3	0	3	0	2	0	0	0	0	3	0	1	0	0	0	3	2	0	678	Sleepy Bay	O	19
4	P 75	F	3	0	3	1	1	0	0	0	0	1	1	1	0	0	0	3	1	0	678	Sleepy Bay	O	12
5	P 77	F	3	0	0	3	2	0	0	1	0	2	0	0	0	1	0	2	1	0	678	Sleepy Bay	O	11
6	P 97	F	3	0	3	0	1	0	2	1	0	1	0	0	1	0	0	2	3	0	678	Sleepy Bay	O	3
7	P 101	F	3	0	2	0	1	0	0	0	0	3	0	0	0	0	0	.	.	.	678	Sleepy Bay	O	20
8	P 112	F	3	0	1	0	2	0	0	0	0	3	0	0	0	0	0	0	1	0	678	Sleepy Bay	O	14
9	P 194	F	3	0	1	1	2	0	1	2	0	3	0	1	0	1	0	2	1	0	678	Sleepy Bay	O	15
10	P 216	F	2	0	2	0	1	0	0	0	0	3	1	0	0	0	0	2	1	0	678	Sleepy Bay	O	16
11	P 258	F	3	0	3	0	1	0	0	0	0	2	1	1	0	2	0	2	3	0	678	Sleepy Bay	O	6
12	P 298	F	2	0	2	0	0	0	0	0	0	0	0	0	0	0	0	.	.	.	678	Sleepy Bay	O	17
13	P 324	F	3	0	3	0	1	0	0	0	0	0	0	1	0	0	0	1	0	0	678	Sleepy Bay	O	10
14	P 344	F	3	0	3	0	0	0	0	1	0	0	1	0	0	1	0	1	0	0	678	Sleepy Bay	O	7
15	P 360	F	3	0	3	0	0	0	0	1	0	0	0	0	0	0	0	1	0	0	678	Sleepy Bay	O	13
16	P 370	F	2	2	3	1	0	0	0	0	1	0	0	0	0	1	0	1	0	0	678	Sleepy Bay	O	4
17	P 404	F	3	0	2	0	0	0	0	0	0	0	1	0	0	0	0	1	0	0	678	Sleepy Bay	O	8
18	P 448	F	2	0	1	0	1	0	0	1	0	3	1	0	0	1	0	1	1	0	678	Sleepy Bay	O	1
19	P 449	F	3	0	2	0	2	0	0	0	0	2	1	1	0	0	0	3	1	0	678	Sleepy Bay	O	5
20	P 455	F	2	1	2	0	1	0	0	0	0	2	1	0	0	1	0	3	1	0	678	Sleepy Bay	O	2
21	P 9	M	.	.	.	.	.	.	.	.	.	3	1	1	0	0	0	2	1	0	678	Sleepy Bay	O	37
22	P 22	M	2	0	0	0	2	0	0	1	0	2	1	0	0	0	0	2	1	1	678	Sleepy Bay	O	30
23	P 52	M	1	1	1	0	1	0	0	1	0	2	0	0	0	0	0	2	1	2	678	Sleepy Bay	O	40
24	P 71	M	3	0	0	0	2	0	0	1	0	1	1	2	0	0	0	2	0	0	678	Sleepy Bay	O	25
25	P 117	M	1	1	0	0	2	0	0	0	0	1	1	1	0	1	0	1	1	0	678	Sleepy Bay	O	28

	Proc.		LIVER									KIDNEY					SPL	NARES			Stream			Alaska
#	#	Sex	GLY	LIP	HV	PD	PEL	SCN	FN	MEG	FIB	VD	PD	LUM	MYX	GLM	MA	EGL	MUC	SCN	#	Name	OS	#
26	P 179	M	1	0	1	0	2	0	0	0	0	2	1	1	0	0	0	1	1	1	678	Sleepy Bay	O	21
27	P 233	M	.	.	.	.	.	.	.	.	.	2	1	0	0	0	0	2	2	1	678	Sleepy Bay	O	22
28	P 257	M	1	0	0	0	2	0	0	0	0	2	0	1	0	0	0	3	0	0	678	Sleepy Bay	O	31
29	P 301	M	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	1	1	0	678	Sleepy Bay	O	26
30	P 321	M	3	2	1	0	0	1	0	0	0	0	1	1	0	0	0	0	0	0	678	Sleepy Bay	O	34
31	P 338	M	2	2	1	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	678	Sleepy Bay	O	27
32	P 345	M	1	0	0	0	0	1	0	1	0	0	0	0	0	0	0	1	0	0	678	Sleepy Bay	O	38
33	P 348	M	3	0	2	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	678	Sleepy Bay	O	23
34	P 367	M	1	0	1	0	0	0	0	0	0	1	1	1	0	0	0	1	0	0	678	Sleepy Bay	O	35
35	P 386	M	2	0	0	0	0	0	0	0	0	0	0	1	0	1	0	1	0	1	678	Sleepy Bay	O	29
36	P 443	M	2	0	0	0	0	0	0	1	0	0	1	0	0	0	0	1	0	0	678	Sleepy Bay	O	39
37	P 445	M	3	0	2	0	3	0	0	0	0	0	0	0	0	0	0	.	0	0	678	Sleepy Bay	O	33
38	P 447	M	2	0	1	0	2	0	0	1	0	3	1	1	0	0	0	1	0	1	678	Sleepy Bay	O	24
39	P 473	M	0	0	0	0	0	0	0	0	0	0	0	0	.	.	0	1	0	0	678	Sleepy Bay	O	36
40	P 487	M	3	0	1	0	0	0	0	1	0	0	0	0	0	0	0	1	0	1	678	Sleepy Bay	O	32
41	P 23	F	3	0	2	0	2	1	2	0	1	1	1	1	1	0	0	.	.	.	692	Herring Bay	O	17
42	P 53	F	3	0	2	1	2	1	1	1	0	2	1	1	1	0	0	1	1	0	692	Herring Bay	O	12
43	P 82	F	3	1	0	0	1	0	0	0	0	1	2	0	0	0	0	0	3	0	692	Herring Bay	O	1
44	P 99	F	3	0	0	0	1	3	2	0	0	0	2	0	1	1	0	2	0	1	692	Herring Bay	O	18
45	P 108	F	3	0	0	0	0	1	0	2	0	0	0	1	0	0	0	2	0	0	692	Herring Bay	O	13
46	P 131	F	3	1	0	0	0	1	0	1	0	1	1	2	2	0	0	3	1	0	692	Herring Bay	O	4
47	P 153	F	3	0	2	0	1	1	0	1	0	1	0	0	0	2	0	3	1	0	692	Herring Bay	O	2
48	P 188	F	3	1	1	0	1	2	1	0	0	1	2	1	0	0	0	2	0	1	692	Herring Bay	O	3
49	P 209	F	3	0	1	0	0	2	0	0	0	0	1	1	1	0	0	2	0	1	692	Herring Bay	O	7
50	P 210	F	3	1	2	0	1	3	2	1	1	1	1	0	0	0	0	2	1	1	692	Herring Bay	O	6
51	P 242	F	3	0	0	0	1	2	0	0	0	0	1	0	0	2	0	3	1	0	692	Herring Bay	O	10
52	P 290	F	3	0	3	0	2	0	0	0	0	0	0	1	2	0	0	2	0	0	692	Herring Bay	O	11
53	P 381	F	3	0	3	0	1	1	0	1	0	0	1	0	0	0	0	1	.	.	692	Herring Bay	O	14
54	P 398	F	3	0	2	0	0	1	0	0	0	0	0	0	0	0	0	1	0	1	692	Herring Bay	O	8
55	P 464	F	3	2	2	0	1	1	0	1	0	1	2	1	3	2	0	.	1	0	692	Herring Bay	O	5
56	P 488	F	3	0	0	0	0	1	0	1	0	0	0	0	0	1	0	1	0	1	692	Herring Bay	O	19
57	P 490	F	3	0	0	0	.	1	0	1	0	0	0	0	0	0	0	.	.	.	692	Herring Bay	O	9
58	P 491	F	3	0	1	0	0	1	0	1	0	0	1	0	0	1	0	.	.	.	692	Herring Bay	O	15
59	P 501	F	3	0	2	0	0	2	0	2	0	0	2	0	0	1	0	.	.	.	692	Herring Bay	O	16
60	P 37	M	1	0	0	0	2	0	0	1	0	1	0	3	2	0	0	1	2	0	692	Herring Bay	O	24
61	P 38	M	1	0	0	0	3	0	0	2	0	1	0	0	0	0	0	3	1	0	692	Herring Bay	O	30
62	P 42	M	1	0	0	0	2	1	1	1	0	1	0	1	1	0	0	1	0	0	692	Herring Bay	O	37
63	P 113	M	2	0	0	0	1	0	0	1	0	1	0	1	1	0	0	.	1	0	692	Herring Bay	O	27
64	P 130	M	2	0	0	0	0	1	0	1	0	1	0	0	0	0	0	2	.	.	692	Herring Bay	O	36
65	P 151	M	3	0	0	0	1	0	0	1	0	0	0	0	0	0	0	1	2	0	692	Herring Bay	O	33
66	P 166	M	2	0	1	0	1	0	1	0	0	0	1	3	3	0	0	1	1	0	692	Herring Bay	O	34
67	P 178	M	1	0	0	0	0	0	0	0	0	0	1	1	0	0	0	.	.	.	692	Herring Bay	O	32
68	P 193	M	2	0	0	0	0	1	2	1	0	1	0	1	0	0	0	1	2	0	692	Herring Bay	O	21
69	P 255	M	2	0	0	0	1	1	0	0	0	1	0	1	0	0	0	.	2	1	692	Herring Bay	O	28

	Proc.		LIVER									KIDNEY					SPL	NARES			Stream			Alaska
#	#	Sex	GLY	LIP	HV	PD	PEL	SCN	FN	MEG	FIB	VD	PD	LUM	MYX	GLM	MA	EGL	MUC	SCN	#	Name	OS	#
70	P 261	M	2	1	2	0	2	0	0	0	0	0	0	0	1	1	0	1	0	1	692	Herring Bay	O	35
71	P 346	M	3	0	1	0	0	1	0	1	0	0	0	0	0	0	0	1	.	.	692	Herring Bay	O	39
72	P 353	M	3	0	2	0	0	0	0	0	0	0	0	0	0	0	0	.	.	.	692	Herring Bay	O	20
73	P 371	M	2	0	1	0	1	0	0	2	0	0	0	0	0	1	0	.	.	.	692	Herring Bay	O	26
74	P 383	M	3	0	0	0	0	0	0	1	0	0	0	0	0	0	0	.	.	.	692	Herring Bay	O	31
75	P 410	M	3	0	1	0	0	1	1	1	0	0	0	0	0	1	0	.	.	.	692	Herring Bay	O	40
76	P 424	M	3	0	3	0	0	1	0	1	0	0	0	0	0	1	0	.	.	.	692	Herring Bay	O	29
77	P 430	M	3	3	2	0	1	0	0	0	1	0	0	1	0	0	0	0	1	0	692	Herring Bay	O	25
78	P 434	M	3	2	0	0	0	1	0	1	1	0	0	0	0	0	0	.	.	.	692	Herring Bay	O	22
79	P 452	M	3	0	0	0	0	0	1	0	0	1	0	0	0	0	0	3	1	1	692	Herring Bay	O	23
80	P 481	M	3	0	0	0	0	1	0	1	0	0	0	0	1	1	0	1	0	1	692	Herring Bay	O	38
81	P 26	F	3	0	1	0	1	1	0	1	0	0	0	0	0	0	0	3	1	0	506	Loomis Creek	O	14
82	P 32	F	3	0	0	0	1	0	0	1	0	0	0	0	0	1	0	3	1	0	506	Loomis Creek	O	20
83	P 62	F	3	3	0	0	1	1	0	1	0	1	1	1	1	0	0	.	.	.	506	Loomis Creek	O	15
84	P 68	F	3	0	3	0	1	1	0	0	0	1	1	1	1	0	0	.	.	.	506	Loomis Creek	O	10
85	P 143	F	3	1	2	0	1	1	0	1	0	0	0	1	1	0	0	1	0	0	506	Loomis Creek	O	12
86	P 147	F	3	0	3	0	2	0	0	0	0	1	2	0	0	0	0	1	1	0	506	Loomis Creek	O	19
87	P 185	F	3	0	0	0	2	1	0	1	0	2	2	1	1	0	0	.	.	.	506	Loomis Creek	O	6
88	P 186	F	3	0	1	0	2	0	0	0	0	1	1	1	3	0	0	1	0	1	506	Loomis Creek	O	1
89	P 195	F	3	1	3	0	1	1	0	0	0	1	3	1	0	1	0	.	.	.	506	Loomis Creek	O	17
90	P 238	F	3	2	1	0	0	0	0	1	0	0	1	0	0	0	0	.	.	.	506	Loomis Creek	O	4
91	P 283	F	2	0	1	0	1	0	0	0	0	0	0	0	1	0	0	.	.	.	506	Loomis Creek	O	16
92	P 302	F	3	0	3	0	0	1	0	0	0	0	1	1	0	0	0	0	0	1	506	Loomis Creek	O	18
93	P 340	F	3	1	1	0	0	1	0	1	0	0	1	0	1	0	0	.	.	.	506	Loomis Creek	O	7
94	P 355	F	3	1	2	0	3	3	1	0	0	0	1	0	1	1	0	0	0	0	506	Loomis Creek	O	13
95	P 405	F	3	1	3	0	0	3	0	1	0	1	3	0	2	0	0	1	1	0	506	Loomis Creek	O	2
96	P 409	F	3	1	0	0	0	0	0	1	0	0	0	0	0	0	0	.	.	.	506	Loomis Creek	O	8
97	P 411	F	3	1	3	0	1	1	0	1	0	1	1	1	1	1	0	.	.	.	506	Loomis Creek	O	11
98	P 433	F	3	0	3	1	0	0	0	0	1	0	3	1	0	0	0	1	0	1	506	Loomis Creek	O	9
99	P 67	M	0	0	0	0	2	0	0	0	0	1	0	2	1	0	0	.	1	0	506	Loomis Creek	O	33
100	P 83	M	3	0	0	0	3	0	0	0	0	1	0	1	1	0	0	.	.	.	506	Loomis Creek	O	35
101	P 94	M	3	1	0	0	1	0	1	1	0	0	1	1	0	0	0	.	0	2	506	Loomis Creek	O	3
102	P 126	M	3	0	0	0	0	1	0	1	0	0	1	0	0	1	0	0	.	.	506	Loomis Creek	O	5
103	P 150	M	3	0	0	0	1	1	3	0	0	0	0	0	0	1	0	1	0	0	506	Loomis Creek	O	37
104	P 177	M	3	1	0	0	2	0	0	0	0	0	0	0	0	0	0	.	1	1	506	Loomis Creek	O	31
105	P 217	M	3	0	0	0	2	0	0	0	0	1	0	2	2	0	0	.	.	.	506	Loomis Creek	O	24
106	P 222	M	2	1	0	0	0	0	0	0	0	0	0	0	0	0	0	.	.	.	506	Loomis Creek	O	22
107	P 226	M	2	1	0	0	0	0	0	1	0	0	0	2	1	0	0	.	0	0	506	Loomis Creek	O	25
108	P 270	M	2	3	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	2	506	Loomis Creek	O	29
109	P 282	M	3	0	3	0	0	0	0	0	0	0	1	1	3	0	0	.	.	.	506	Loomis Creek	O	34
110	P 289	M	2	0	2	0	1	0	0	0	0	0	0	0	1	0	0	.	.	.	506	Loomis Creek	O	32
111	P 293	M	0	0	0	0	1	0	0	2	0	0	0	0	0	1	0	0	1	0	506	Loomis Creek	O	38
112	P 332	M	3	0	2	0	1	0	0	0	0	0	0	0	0	1	0	0	0	0	506	Loomis Creek	O	39
113	P 380	M	3	2	2	0	0	1	0	1	1	0	0	0	2	0	0	1	0	1	506	Loomis Creek	O	30

	Proc.		LIVER									KIDNEY					SPL	NARES			Stream			Alaska
#	#	Sex	GLY	LIP	HV	PD	PEL	SCN	FN	MEG	FIB	VD	PD	LUM	MYX	GLM	MA	EGL	MUC	SCN	#	Name	OS	#
114	P 416	M	3	0	0	0	0	0	0	1	1	0	0	1	0	0	0	.	.	.	506	Loomis Creek	O	21
115	P 417	M	.	.	.	.	.	.	.	.	.	.	.	.	1	1	0	.	.	.	506	Loomis Creek	O	36
116	P 431	M	3	3	2	0	0	0	0	1	0	0	0	0	0	0	0	.	.	.	506	Loomis Creek	O	28
117	P 446	M	3	1	1	0	1	1	0	1	1	0	0	0	2	1	0	.	.	.	506	Loomis Creek	O	40
118	P 458	M	3	2	0	0	0	0	0	0	0	1	0	0	0	0	0	2	1	2	506	Loomis Creek	O	23
119	P 467	M	2	0	0	0	1	0	0	1	0	1	0	0	0	0	0	.	2	2	506	Loomis Creek	O	26
120	P 489	M	2	0	1	0	1	0	0	0	0	0	0	0	0	0	0	.	.	.	506	Loomis Creek	O	27
121	P 2	F	3	1	2	0	1	2	1	0	0	1	0	0	0	0	0	0	0	0	861	Bernard Creek	C	28
122	P 20	F	2	2	0	0	1	3	1	0	0	1	3	1	0	0	0	1	1	0	861	Bernard Creek	C	29
123	P 57	F	3	0	0	0	1	1	1	2	0	1	1	0	0	1	0	1	1	0	861	Bernard Creek	C	31
124	P 69	F	3	1	0	1	2	3	0	3	0	1	2	0	0	0	0	.	1	0	861	Bernard Creek	C	33
125	P 161	F	3	0	0	0	1	1	2	0	1	0	1	1	1	1	0	1	1	0	861	Bernard Creek	C	26
126	P 170	F	3	0	2	0	2	1	0	1	0	0	0	1	1	1	0	3	0	0	861	Bernard Creek	C	38
127	P 228	F	3	2	1	0	2	1	2	0	0	1	1	1	0	0	0	2	2	0	861	Bernard Creek	C	40
128	P 275	F	3	0	3	0	2	1	2	0	0	0	2	1	3	0	0	3	0	1	861	Bernard Creek	C	37
129	P 295	F	2	2	1	0	1	0	0	0	0	0	0	0	0	1	0	1	.	.	861	Bernard Creek	C	24
130	P 314	F	3	1	3	0	2	1	0	2	0	0	0	0	0	0	0	2	0	1	861	Bernard Creek	C	39
131	P 320	F	3	2	0	0	2	1	0	1	0	0	0	0	0	1	0	.	.	.	861	Bernard Creek	C	27
132	P 326	F	3	1	1	0	0	2	0	1	0	0	0	0	0	0	0	3	0	0	861	Bernard Creek	C	30
133	P 385	F	3	0	3	0	0	0	0	2	0	0	0	0	0	0	0	1	1	0	861	Bernard Creek	C	36
134	P 396	F	3	1	2	0	1	2	1	1	0	0	1	1	0	1	0	1	1	0	861	Bernard Creek	C	34
135	P 415	F	3	2	1	0	0	1	0	1	0	0	0	3	3	0	0	.	.	.	861	Bernard Creek	C	22
136	P 432	F	3	0	2	0	0	0	0	1	1	0	2	0	1	0	0	.	.	.	861	Bernard Creek	C	32
137	P 437	F	3	1	2	0	2	2	0	2	0	0	1	0	1	0	0	1	0	1	861	Bernard Creek	C	35
138	P 442	F	3	1	3	0	0	1	0	1	0	0	2	1	2	0	0	1	0	0	861	Bernard Creek	C	21
139	P 474	F	3	2	1	0	0	1	1	1	0	0	1	0	0	0	0	.	.	.	861	Bernard Creek	C	25
140	P 484	F	3	0	3	0	1	.	0	2	0	0	0	0	1	0	0	.	.	.	861	Bernard Creek	C	23
141	P 30	M	1	0	0	0	2	0	0	1	0	0	0	1	1	0	0	0	1	0	861	Bernard Creek	C	19
142	P 86	M	2	0	0	0	1	1	1	2	1	0	0	0	0	0	0	1	2	1	861	Bernard Creek	C	16
143	P 110	M	1	0	0	0	2	0	0	0	1	1	0	0	0	1	0	0	2	1	861	Bernard Creek	C	6
144	P 124	M	1	0	0	0	2	0	0	1	0	0	0	0	0	0	0	1	1	0	861	Bernard Creek	C	12
145	P 127	M	1	0	0	0	2	0	0	0	0	0	0	0	0	0	0	0	0	0	861	Bernard Creek	C	9
146	P 134	M	0	0	0	0	2	0	0	0	0	1	0	1	1	1	0	3	3	1	861	Bernard Creek	C	17
147	P 189	M	1	0	0	0	0	0	0	1	0	0	0	2	1	0	0	1	0	0	861	Bernard Creek	C	1
148	P 200	M	2	0	0	0	2	0	0	0	0	1	0	0	0	0	0	3	1	0	861	Bernard Creek	C	2
149	P 208	M	2	0	0	0	1	0	1	0	0	1	0	2	1	0	0	2	1	0	861	Bernard Creek	C	13
150	P 215	M	3	0	0	0	1	0	2	0	0	0	0	0	0	0	0	1	0	1	861	Bernard Creek	C	14
151	P 220	M	0	0	0	0	2	0	0	0	0	0	0	2	1	0	0	1	3	0	861	Bernard Creek	C	18
152	P 232	M	1	0	0	0	1	0	0	1	0	2	0	1	1	0	0	1	1	0	861	Bernard Creek	C	20
153	P 250	M	3	0	2	0	3	0	0	0	0	0	0	1	1	1	0	3	0	1	861	Bernard Creek	C	10
154	P 309	M	2	1	1	0	0	2	0	0	0	0	0	1	1	0	0	1	0	0	861	Bernard Creek	C	4
155	P 322	M	3	1	3	0	0	0	0	0	0	0	1	0	0	2	0	.	.	.	861	Bernard Creek	C	3
156	P 323	M	0	0	0	0	1	0	0	0	0	0	0	0	0	1	0	2	0	0	861	Bernard Creek	C	7
157	P 343	M	2	0	1	0	0	0	0	0	0	1	0	0	2	1	0	1	.	.	861	Bernard Creek	C	15

#	Proc. #	Sex	LIVER									KIDNEY					SPL	NARES			Stream			Alaska
			GLY	LIP	HV	PD	PEL	SCN	FN	MEG	FIB	VD	PD	LUM	MYX	GLM	MA	EGL	MUC	SCN	#	Name	OS	#
158	P 358	M	1	0	1	0	1	0	1	1	1	1	0	0	0	0	0	1	1	0	861	Bernard Creek	C	8
159	P 392	M	0	0	0	0	1	0	0	1	0	1	1	0	0	0	0	0	1	0	861	Bernard Creek	C	5
160	P 407	M	1	0	1	0	1	0	0	1	0	1	0	0	0	0	0	1	1	0	861	Bernard Creek	C	11
161	P 3	F	3	0	0	0	2	0	0	1	0	1	1	0	0	0	0	.	.	.	485	West Finger Creek	C	29
162	P 7	F	3	0	3	0	2	1	0	2	0	1	2	2	2	0	0	2	0	0	485	West Finger Creek	C	27
163	P 14	F	3	1	2	0	2	0	0	1	0	2	2	1	0	0	0	2	0	0	485	West Finger Creek	C	21
164	P 55	F	3	0	0	0	1	1	0	1	0	1	3	0	0	0	0	1	1	0	485	West Finger Creek	C	30
165	P 58	F	3	2	0	0	2	1	0	1	0	1	2	0	0	0	0	2	1	0	485	West Finger Creek	C	34
166	P 85	F	3	0	2	0	2	0	0	0	0	1	0	1	0	0	0	.	.	.	485	West Finger Creek	C	32
167	P 103	F	3	0	1	1	2	0	0	0	0	0	0	1	3	2	0	2	0	0	485	West Finger Creek	C	24
168	P 154	F	3	0	3	0	1	1	2	1	0	0	0	1	0	0	0	1	0	0	485	West Finger Creek	C	25
169	P 182	F	3	0	1	0	2	0	0	1	0	0	1	0	0	0	0	1	0	0	485	West Finger Creek	C	33
170	P 183	F	3	0	3	0	1	0	0	0	0	0	2	1	0	0	0	3	0	0	485	West Finger Creek	C	38
171	P 201	F	3	0	2	0	2	1	1	1	0	1	0	2	0	1	0	2	1	1	485	West Finger Creek	C	35
172	P 259	F	3	0	1	0	2	1	0	1	0	0	1	0	0	0	0	2	0	0	485	West Finger Creek	C	36
173	P 359	F	3	0	3	1	0	0	0	1	1	0	2	0	0	1	0	1	0	0	485	West Finger Creek	C	39
174	P 368	F	3	1	2	0	1	1	0	1	0	0	1	0	1	1	0	1	0	0	485	West Finger Creek	C	37
175	P 378	F	3	1	3	0	0	1	0	1	0	0	1	0	0	0	0	2	0	0	485	West Finger Creek	C	28
176	P 379	F	2	3	3	0	0	0	0	2	0	1	2	0	0	0	0	1	0	0	485	West Finger Creek	C	40
177	P 412	F	3	0	3	0	2	1	0	1	0	0	1	0	1	1	.	1	0	0	485	West Finger Creek	C	26
178	P 440	F	3	0	3	0	1	0	0	0	0	0	0	0	0	1	0	1	0	0	485	West Finger Creek	C	22
179	P 478	F	3	1	2	1	1	1	0	2	0	0	1	0	0	1	0	1	0	0	485	West Finger Creek	C	23
180	P 499	F	3	1	2	0	0	0	0	1	0	0	1	0	0	0	0	1	0	1	485	West Finger Creek	C	31
181	P 45	M	1	0	0	0	0	0	0	1	0	1	0	0	0	2	0	1	1	0	485	West Finger Creek	C	7
182	P 49	M	2	0	0	0	2	0	0	1	0	1	0	1	0	0	0	1	1	0	485	West Finger Creek	C	15
183	P 60	M	2	0	0	0	1	1	0	1	0	1	0	1	2	0	0	3	1	0	485	West Finger Creek	C	13
184	P 123	M	1	0	0	0	1	0	0	1	0	0	0	0	0	1	0	1	.	.	485	West Finger Creek	C	8
185	P 138	M	1	0	0	1	0	0	0	1	0	1	0	1	0	1	0	.	0	1	485	West Finger Creek	C	16
186	P 142	M	1	0	0	0	2	0	0	1	0	1	0	0	0	0	0	1	0	0	485	West Finger Creek	C	19
187	P 149	M	2	1	0	0	0	0	0	1	0	2	1	1	0	0	0	2	0	0	485	West Finger Creek	C	9
188	P 162	M	0	0	0	0	1	0	0	0	0	1	0	1	0	0	0	.	.	.	485	West Finger Creek	C	4
189	P 230	M	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0	.	0	0	485	West Finger Creek	C	11
190	P 248	M	0	0	0	0	0	0	0	1	0	1	0	1	0	0	0	1	1	1	485	West Finger Creek	C	5
191	P 264	M	1	0	1	0	2	0	0	1	0	0	0	0	0	1	0	1	1	0	485	West Finger Creek	C	18
192	P 278	M	1	0	1	0	2	2	0	1	0	0	0	1	0	0	0	1	0	0	485	West Finger Creek	C	10
193	P 279	M	0	0	0	0	2	1	0	0	0	0	0	0	0	0	0	1	1	0	485	West Finger Creek	C	17
194	P 294	M	3	2	0	0	0	1	0	0	0	0	0	0	1	0	0	.	.	.	485	West Finger Creek	C	20
195	P 327	M	0	0	0	0	0	0	0	1	0	0	1	1	0	0	0	.	.	.	485	West Finger Creek	C	2
196	P 333	M	2	2	2	0	0	0	0	1	0	0	1	0	0	0	0	.	.	.	485	West Finger Creek	C	6
197	P 389	M	3	1	1	0	1	1	0	1	0	0	0	0	0	0	0	1	0	0	485	West Finger Creek	C	12
198	P 400	M	1	0	1	0	1	0	0	2	0	0	1	0	1	0	.	.	.	.	485	West Finger Creek	C	14
199	P 470	M	1	0	0	0	2	0	0	1	0	.	.	.	.	.	.	2	0	0	485	West Finger Creek	C	3
200	P 483	M	1	0	1	0	2	0	0	0	0	0	.	0	0	0	0	0	0	0	485	West Finger Creek	C	1

MEAN LESION SCORES

		LIVER									KIDNEY					SPL	NARES			Stream		
	Sex	GLY	LIP	HV	PD	PEL	SCN	FN	MEG	FIB	VD	PD	LUM	MYX	GLM	MA	EGL	MUC	SCN	#	Name	OS
	F	2.7	.15	2.2	.3	1.1	.05	.15	.45	.05	1.7	.45	.3	.05	.4	0	1.7	1	.11	678	Sleepy Bay	O
	M	1.7	.33	.61	0	.89	.17	0	.39	0	.95	.55	.5	0	.11	0	1.3	.4	.4	678	Sleepy Bay	O
	F	3	.32	1.2	.05	.78	1.3	.42	.68	.11	.47	.95	.47	.58	.53	0	1.8	.64	.43	692	Herring Bay	O
	M	2.3	.29	.62	0	.71	.43	.29	.76	.10	.38	.10	.57	.43	.24	0	1.3	1.1	.33	692	Herring Bay	O
	F	2.9	.67	1.7	.06	.94	.83	.06	.56	.06	.5	1.2	.5	.72	.22	0	1.2	.44	.33	506	Loomis Creek	O
	M	2.4	.71	.67	0	.81	.19	.19	.48	.14	.24	.14	.48	.64	.27	0	.57	.55	.91	506	Loomis Creek	O
	F	2.9	.95	1.5	.05	1.1	1.3	.55	1.1	.1	.25	.85	.5	.65	.3	0	1.5	.57	.21	861	Bernard Creek	C
	M	1.4	.1	.45	0	1.3	.15	.25	.45	.15	.5	.1	.55	.5	.35	0	1.2	1	.28	861	Bernard Creek	C
	F	3.0	.5	2.0	.15	1.3	.5	.15	.95	.05	.45	1.2	.45	.35	.4	0	1.5	.17	.11	485	West Finger Creek	C
	M	1.2	.3	.35	.05	1	.3	0	.8	0	.47	.22	.42	.21	.26	0	1.2	.43	.14	485	West Finger Creek	C
oiled	F	2.9	.38	1.7	.14	.92	.73	.21	.56	.07	.89	.85	.42	.45	.38	0	1.6	.70	.29	Combined oiled sites		
ref.	F	2.9	.73	1.7	.1	1.2	.88	.35	1	.08	.35	1	.48	.5	.35	0	1.5	.37	.16	Combined reference sites		
oiled	M	2.1	.44	.63	0	.80	.26	.16	.54	.08	.52	.26	.52	.35	.21	0	1.1	.68	.55	Combined oiled sites		
ref.	M	1.3	.2	.4	.03	1.1	.23	.13	.63	.08	.49	.16	.49	.36	.31	0	1.2	.71	.21	Combined reference sites		

Table X-2. Histopathology of "S" Pink Salmon Adult sampled from Prince William Sound in 1990.

Key to table symbols:

Alaska # = Sample number generated by ADF@G
Hinton processing # = Random number generated by Dr. Hinton's Laboratory
Sex = male (M) or female (F)
OS = oiled status; oiled (O), lightly oiled (LO), or control/clean (C)

Lesion scores = none (0), mild (1), moderate (2), severe (4), or tissue not present "."

LIVER:
- glycogen depletion (GLY)
- lipidosis (LIP)
- decreased hepatocyte volume (HV)
- hepatocellular protein droplets (PD)
- peliosis/congestion of sinusoids (PEL)
- single cell necrosis (SCN)
- focal necrosis (FN)
- hepatocellular karyomegaly (MEG)
- sinusoidal fibrosis (FIB)

SPLEEN (SPL):
- macrophage aggregates (MA)

KIDNEY:
- tubular epithelial vacuolar degeneration (VD)
- tubular epithelial protein droplets (PD)
- tubular luminal debris (LUM)
- tubular myxosporeans (MYX)
- glomerular microsporidians (GLM)

NARES:
- Eph'ic granulocytes in unmyelinated nerve sheathes (EGL)
- mucous cell hyperplasia in sensory epithelium (MUC)
- single cell necrosis in nasal sensory epithelium (SCN)

	Proc.		LIVER									KIDNEY					SPL	NARES			Stream			Alaska
#	#	Sex	GLY	LIP	HV	PD	PEL	SCN	FN	MEG	FIB	VD	PD	LUM	MYX	GLM	MA	EGL	MUC	SCN	#	Name	OS	#
1	S 3	F	3	1	1	0	3	1	0	1	0	0	0	0	1	0	0	1	1	0	9	Port Dick	LO	423
2	S 10	F	3	0	1	0	2	1	0	0	0	0	1	0	0	0	0	1	2	0	9	Port Dick	LO	436
3	S 15	F	3	1	1	0	3	1	0	0	0	0	0	0	0	0	0	.	.	.	9	Port Dick	LO	438
4	S 72	F	3	0	0	1	1	1	0	1	0	1	2	1	2	0	0	1	0	1	9	Port Dick	LO	433
5	S 77	F	3	0	0	0	2	1	0	1	0	0	0	0	0	0	0	1	1	0	9	Port Dick	LO	424
6	S 93	F	3	0	1	0	2	1	0	1	0	1	1	1	0	0	0	1	1	1	9	Port Dick	LO	434
7	S 104	F	3	0	1	0	2	1	0	1	0	0	0	1	1	0	0	1	0	1	9	Port Dick	LO	425
8	S 108	F	3	1	2	1	2	1	0	2	0	0	0	1	0	0	0	2	0	1	9	Port Dick	LO	440
9	S 111	F	3	0	2	1	1	1	0	2	0	1	1	1	0	0	0	2	0	1	9	Port Dick	LO	427
10	S 115	F	3	1	2	0	1	1	0	0	0	0	0	1	1	0	0	2	0	1	9	Port Dick	LO	429
11	S 136	F	3	1	2	1	3	1	0	1	0	1	1	0	0	0	0	1	2	2	9	Port Dick	LO	435
12	S 176	F	3	0	0	0	2	2	0	1	0	.	.	.	.	.	0	2	1	0	9	Port Dick	LO	439
13	S 182	F	3	1	1	0	1	1	0	1	0	1	0	1	2	0	0	1	1	1	9	Port Dick	LO	426
14	S 186	F	3	0	0	0	1	1	2	1	0	0	1	3	2	0	0	1	1	1	9	Port Dick	LO	430
15	S 205	F	3	0	0	0	3	1	0	1	0	0	0	1	0	0	0	3	0	2	9	Port Dick	LO	421
16	S 207	F	3	0	2	0	2	1	1	1	0	1	1	0	0	1	0	1	1	1	9	Port Dick	LO	432
17	S 216	F	3	0	1	0	2	1	0	0	0	0	1	1	0	0	0	1	2	0	9	Port Dick	LO	422
18	S 258	F	3	0	0	0	2	1	0	0	0	.	.	.	.	.	0	1	2	2	9	Port Dick	LO	437

#	Proc. #	Sex	LIVER GLY	LIP	HV	PD	PEL	SCN	FN	MEG	FIB	KIDNEY VD	PD	LUM	MYX	GLM	SPL MA	NARES EGL	MUC	SCN	Stream #	Name	OS	Alaska #
19	S 279	F	3	0	0	0	1	1	0	1	0	0	1	1	0	0	0	0	3	2	9	Port Dick	LO	431
20	S 312	F	3	0	2	0	1	1	0	1	0	0	0	2	2	0	0	2	3	2	9	Port Dick	LO	428
21	S 7	M	2	1	0	0	2	0	0	1	0	0	0	0	0	0	0	0	1	0	9	Port Dick	LO	407
22	S 19	M	1	0	2	0	1	0	0	0	0	1	0	1	0	0	0	0	2	0	9	Port Dick	LO	418
23	S 22	M	3	0	2	0	2	2	0	3	0	0	0	0	0	0	0	0	1	0	9	Port Dick	LO	416
24	S 25	M	1	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	1	0	9	Port Dick	LO	409
25	S 29	M	1	0	0	0	2	0	0	0	0	2	0	0	2	0	0	0	1	0	9	Port Dick	LO	410
26	S 40	M	1	0	1	0	2	0	0	0	0	0	0	0	0	0	0	0	1	0	9	Port Dick	LO	411
27	S 51	M	2	0	1	0	1	0	0	1	0	0	0	0	2	0	0	1	1	0	9	Port Dick	LO	405
28	S 62	M	1	0	0	0	3	0	1	1	0	1	0	0	0	0	0	1	0	1	9	Port Dick	LO	417
29	S 154	M	0	0	1	0	1	0	0	1	0	0	0	1	1	0	0	2	1	1	9	Port Dick	LO	401
30	S 155	M	1	0	0	0	0	0	0	1	0	1	0	1	1	0	0	0	1	0	9	Port Dick	LO	414
31	S 181	M	0	0	0	0	1	0	0	0	0	1	0	1	0	0	0	0	1	1	9	Port Dick	LO	408
32	S 187	M	1	0	1	0	1	0	1	1	0	0	0	1	0	0	0	1	1	1	9	Port Dick	LO	412
33	S 199	M	1	0	0	0	3	0	0	1	0	0	0	2	2	0	0	2	1	0	9	Port Dick	LO	413
34	S 204	M	1	0	0	0	3	0	0	2	0	0	0	2	2	0	0	1	1	0	9	Port Dick	LO	402
35	S 212	M	2	0	0	0	3	0	0	1	0	1	0	0	0	1	0	2	2	1	9	Port Dick	LO	406
36	S 231	M	2	0	0	0	3	0	0	1	0	0	0	0	0	0	0	2	1	0	9	Port Dick	LO	403
37	S 288	M	3	0	0	0	3	1	0	1	0	1	0	1	2	0	0	1	1	1	9	Port Dick	LO	419
38	S 301	M	0	0	0	0	2	0	0	0	0	0	0	3	2	0	0	1	2	0	9	Port Dick	LO	404
39	S 302	M	3	0	0	0	2	0	0	0	0	2	0	0	0	0	0	1	1	0	9	Port Dick	LO	420
40	S 320	M	1	0	0	0	0	0	0	1	0	1	0	1	0	0	0	1	1	0	9	Port Dick	LO	415

#	Proc. #	Sex	LIVER									KIDNEY					SPL	NARES			Stream			Alaska
			GLY	LIP	HV	PD	PEL	SCN	FN	MEG	FIB	VD	PD	LUM	MYX	GLM	MA	EGL	MUC	SCN	#	Name	OS	#
41	S 1	F	3	0	3	0	1	1	0	0	0	0	0	0	0	0	0	1	2	2	11	South Nuka	LO	725
42	S 5	F	3	0	1	0	2	1	1	0	0	0	0	0	0	0	0	1	1	1	11	South Nuka	LO	726
43	S 6	F	3	0	2	3	0	1	0	0	1	0	1	1	2	0	0	1	1	0	11	South Nuka	LO	734
44	S 17	F	2	0	2	1	2	2	0	0	2	0	1	0	0	0	0	1	0	0	11	South Nuka	LO	732
45	S 39	F	3	0	0	2	1	1	0	1	0	0	1	0	0	0	0	1	0	0	11	South Nuka	LO	731
46	S 69	F	3	0	2	0	1	2	0	1	0	1	1	0	0	0	0	2	1	1	11	South Nuka	LO	739
47	S 82	F	3	0	1	0	0	2	2	0	0	0	2	1	0	0	0	2	2	0	11	South Nuka	LO	722
48	S 96	F	3	0	2	0	2	1	2	1	0	1	0	1	1	0	0	2	1	3	11	South Nuka	LO	728
49	S 112	F	3	0	2	1	1	1	0	1	0	3	1	1	0	0	0	1	0	1	11	South Nuka	LO	738
50	S 127	F	3	0	2	0	1	2	1	0	1	1	1	0	0	0	0	1	2	1	11	South Nuka	LO	740
51	S 132	F	3	0	2	1	0	1	0	0	0	3	1	0	0	0	0	3	1	0	11	South Nuka	LO	721
52	S 158	F	3	0	0	0	2	3	0	1	0	1	1	1	0	0	0	0	2	0	11	South Nuka	LO	724
53	S 169	F	3	0	1	0	1	3	1	0	0	0	2	0	2	0	0	1	0	0	11	South Nuka	LO	730
54	S 177	F	3	0	2	2	1	1	0	0	0	1	2	1	0	0	0	1	1	0	11	South Nuka	LO	733
55	S 193	F	3	0	2	0	2	1	0	2	0	0	1	0	0	0	0	0	3	0	11	South Nuka	LO	727
56	S 196	F	3	0	3	0	1	2	2	0	0	1	1	3	2	0	0	0	2	2	11	South Nuka	LO	729
57	S 247	F	3	0	1	0	1	2	0	1	0	1	0	0	0	0	0	2	0	3	11	South Nuka	LO	723
58	S 293	F	3	0	1	0	1	1	0	1	0	0	0	0	0	0	0	.	.	.	11	South Nuka	LO	735
59	S 297	F	3	0	0	0	0	0	0	1	0	0	0	1	0	0	0	3	1	0	11	South Nuka	LO	737
60	S 304	F	3	0	1	0	2	1	0	1	0	1	0	0	0	0	0	3	2	0	11	South Nuka	LO	736
61	S 13	M	2	2	0	0	2	1	0	0	0	1	1	2	3	0	0	0	2	1	11	South Nuka	LO	703
62	S 16	M	1	0	3	0	1	0	0	1	0	0	0	1	0	0	0	.	.	.	11	South Nuka	LO	718
63	S 24	M	0	0	0	0	0	0	0	1	0	0	0	0	1	0	0	1	0	0	11	South Nuka	LO	717
64	S 48	M	1	0	0	0	0	0	0	1	0	0	0	0	0	1	0	1	1	1	11	South Nuka	LO	708
65	S 63	M	1	0	1	0	1	0	0	1	0	1	0	0	0	0	0	0	0	1	11	South Nuka	LO	704
66	S 110	M	2	0	0	0	1	0	0	0	0	1	1	1	0	0	0	1	0	1	11	South Nuka	LO	719
67	S 117	M	2	0	0	0	2	1	1	0	0	0	0	0	1	0	0	1	0	1	11	South Nuka	LO	707
68	S 122	M	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0	2	1	0	11	South Nuka	LO	712
69	S 166	M	1	0	0	0	1	0	0	0	0	1	0	1	0	0	0	.	.	.	11	South Nuka	LO	701
70	S 224	M	1	0	0	0	1	0	0	1	0	0	0	1	1	0	0	0	2	0	11	South Nuka	LO	709
71	S 243	M	1	0	0	0	1	0	0	1	0	0	0	1	0	1	0	1	1	0	11	South Nuka	LO	705
72	S 246	M	1	0	0	0	2	1	0	0	0	2	0	0	0	0	0	1	2	0	11	South Nuka	LO	713
73	S 248	M	2	1	0	0	0	1	1	0	0	2	0	3	3	0	0	2	2	1	11	South Nuka	LO	702
74	S 251	M	2	0	0	0	1	1	1	0	0	2	0	1	0	0	0	1	1	1	11	South Nuka	LO	706
75	S 266	M	2	0	0	0	0	0	0	1	0	1	0	0	0	0	0	2	1	2	11	South Nuka	LO	714
76	S 268	M	0	0	0	0	0	0	0	0	0	1	0	2	1	0	0	3	2	1	11	South Nuka	LO	715
77	S 285	M	0	0	0	0	1	0	1	0	0	0	0	0	0	0	0	1	3	1	11	South Nuka	LO	711
78	S 296	M	0	0	0	0	1	1	0	1	0	1	0	1	1	1	0	3	3	0	11	South Nuka	LO	720
79	S 309	M	1	0	0	0	0	0	0	1	0	1	0	2	2	0	0	2	3	0	11	South Nuka	LO	716
80	S 325	M	0	1	0	0	0	0	0	0	0	0	1	1	0	0	0	0	1	0	11	South Nuka	LO	710

#	Proc. #	Sex	LIVER									KIDNEY					SPL	NARES			Stream			Alaska #
			GLY	LIP	HV	PD	PEL	SCN	FN	MEG	FIB	VD	PD	LUM	MYX	GLM	MA	EGL	MUC	SCN	#	Name	OS	
81	S 43	F	3	0	0	0	0	2	1	0	0	0	2	0	1	1	0	1	0	1	6	Humpy Creek	C	532
82	S 46	F	3	0	3	0	0	1	1	1	1	0	1	0	0	0	0	.	.	.	6	Humpy Creek	C	533
83	S 54	F	3	2	0	0	2	2	1	0	0	0	0	0	0	1	0	1	0	0	6	Humpy Creek	C	524
84	S 58	F	3	0	2	0	1	2	2	0	0	0	0	0	0	0	0	0	0	1	6	Humpy Creek	C	528
85	S 65	F	3	0	3	1	0	2	1	1	1	1	1	1	2	0	0	0	0	1	6	Humpy Creek	C	526
86	S 79	F	3	0	0	1	0	3	1	0	0	2	0	0	0	0	0	0	0	0	6	Humpy Creek	C	521
87	S 83	F	3	2	1	1	3	1	2	0	0	1	0	1	0	0	0	0	0	1	6	Humpy Creek	C	531
88	S 87	F	3	0	1	0	2	1	0	1	0	1	0	1	0	0	0	1	0	1	6	Humpy Creek	C	527
89	S 90	F	3	0	1	0	1	2	0	1	0	1	0	1	1	0	0	1	0	1	6	Humpy Creek	C	525
90	S 120	F	3	0	0	0	3	1	0	1	0	2	0	0	1	0	0	1	0	1	6	Humpy Creek	C	529
91	S 135	F	3	0	3	0	1	2	1	0	1	1	0	1	1	0	0	.	.	.	6	Humpy Creek	C	539
92	S 149	F	3	0	3	0	1	2	1	1	0	1	0	1	0	0	0	.	.	.	6	Humpy Creek	C	538
93	S 180	F	3	0	3	0	1	1	2	1	0	3	1	1	0	0	0	.	.	.	6	Humpy Creek	C	522
94	S 188	F	3	0	2	0	2	2	1	1	1	3	1	0	0	0	0	1	3	3	6	Humpy Creek	C	523
95	S 189	F	3	0	0	0	1	2	1	0	0	0	0	0	0	0	0	2	1	0	6	Humpy Creek	C	536
96	S 206	F	3	0	0	0	2	1	0	1	0	1	0	0	0	0	0	3	1	3	6	Humpy Creek	C	540
97	S 218	F	3	0	0	0	1	2	0	0	1	1	1	0	0	0	0	0	3	3	6	Humpy Creek	C	537
98	S 234	F	3	0	0	0	1	2	0	0	0	0	1	1	0	0	0	.	.	.	6	Humpy Creek	C	535
99	S 290	F	3	0	0	0	2	1	1	2	0	1	0	2	1	0	0	1	3	0	6	Humpy Creek	C	534
100	S 318	F	3	0	3	0	1	1	1	0	0	2	1	1	2	0	0	.	.	.	6	Humpy Creek	C	530
101	S 26	M	2	0	0	0	1	1	0	1	0	0	0	0	0	0	0	1	0	0	6	Humpy Creek	C	516
102	S 34	M	2	0	1	0	2	0	0	0	0	0	0	0	2	0	0	0	0	0	6	Humpy Creek	C	505
103	S 86	M	1	0	1	0	2	0	1	1	1	1	1	1	0	0	0	1	0	0	6	Humpy Creek	C	501
104	S 92	M	0	0	0	0	2	1	1	1	1	1	0	1	2	0	0	0	0	0	6	Humpy Creek	C	513
105	S 109	M	2	1	1	0	2	0	0	1	0	0	1	2	2	0	0	0	0	1	6	Humpy Creek	C	502
106	S 124	M	2	0	0	0	0	1	1	1	0	1	0	1	1	0	0	0	2	2	6	Humpy Creek	C	518
107	S 125	M	2	0	1	0	1	1	2	1	0	1	0	1	1	0	0	2	1	1	6	Humpy Creek	C	510
108	S 140	M	0	0	0	0	3	1	0	0	0	1	1	1	2	0	0	.	.	.	6	Humpy Creek	C	507
109	S 161	M	1	0	0	0	3	1	1	1	0	1	0	1	0	0	0	3	3	0	6	Humpy Creek	C	511
110	S 173	M	1	0	0	0	2	0	0	0	0	0	0	1	0	0	0	1	1	0	6	Humpy Creek	C	503
111	S 183	M	1	0	0	0	1	0	1	1	0	1	0	3	1	0	0	1	1	1	6	Humpy Creek	C	515
112	S 229	M	3	0	2	1	1	1	1	0	0	0	0	2	3	0	0	2	1	0	6	Humpy Creek	C	508
113	S 235	M	1	0	0	0	0	0	0	0	0	0	1	2	1	0	0	1	2	1	6	Humpy Creek	C	517
114	S 249	M	1	0	0	0	0	1	1	1	0	0	0	1	1	0	0	0	1	1	6	Humpy Creek	C	504
115	S 256	M	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	1	1	3	6	Humpy Creek	C	519
116	S 280	M	1	0	0	0	0	1	0	0	0	1	0	1	0	0	0	0	3	3	6	Humpy Creek	C	514
117	S 283	M	0	0	0	0	0	0	0	1	0	0	0	1	2	0	0	2	2	1	6	Humpy Creek	C	506
118	S 292	M	1	0	0	0	0	0	0	0	0	2	2	3	2	0	0	0	3	1	6	Humpy Creek	C	509
119	S 315	M	2	0	0	0	0	0	0	1	0	1	0	1	1	0	0	1	2	0	6	Humpy Creek	C	512
120	S 321	M	0	0	0	0	2	0	1	0	0	1	0	1	0	0	0	3	3	0	6	Humpy Creek	C	520

#	Proc. #	Sex	LIVER GLY	LIP	HV	PD	PEL	SCN	FN	MEG	FIB	KIDNEY VD	PD	LUM	MYX	GLM	SPL MA	NARES EGL	MUC	SCN	Stream #	Name	OS	Alaska #
121	S 28	F	3	0	1	0	3	1	0	0	0	0	1	0	0	0	0	.	.	.	7	Island Creek	LO	336
122	S 36	F	3	0	1	0	2	0	0	0	0	0	0	0	0	1	0	0	1	0	7	Island Creek	LO	332
123	S 49	F	3	0	0	0	2	1	0	1	0	0	1	0	0	0	0	1	1	1	7	Island Creek	LO	331
124	S 73	F	3	0	2	0	2	0	0	2	0	1	1	1	0	1	0	0	0	1	7	Island Creek	LO	338
125	S 84	F	3	0	0	1	2	1	0	1	0	1	1	0	0	0	0	1	2	1	7	Island Creek	LO	333
126	S 89	F	3	1	1	0	3	1	1	1	0	1	0	1	0	0	0	1	0	0	7	Island Creek	LO	322
127	S 95	F	3	0	1	0	2	1	0	1	0	0	1	0	0	0	0	1	0	1	7	Island Creek	LO	324
128	S 123	F	3	1	2	0	3	3	0	1	0	1	2	0	0	0	0	1	1	1	7	Island Creek	LO	326
129	S 145	F	3	0	3	0	1	0	0	1	0	1	2	1	0	0	0	1	1	1	7	Island Creek	LO	327
130	S 152	F	3	0	1	0	1	2	0	1	0	1	1	0	0	0	0	2	1	0	7	Island Creek	LO	329
131	S 153	F	3	0	3	0	1	1	0	0	0	0	0	1	1	1	0	0	1	0	7	Island Creek	LO	328
132	S 179	F	3	0	1	0	3	1	1	0	0	1	1	0	0	0	0	1	3	0	7	Island Creek	LO	339
133	S 185	F	3	0	3	0	2	1	0	0	0	0	1	2	0	0	2	2	2	2	7	Island Creek	LO	337
134	S 227	F	3	0	3	0	2	0	0	1	0	0	0	0	0	0	0	1	0	0	7	Island Creek	LO	334
135	S 237	F	3	1	0	0	2	1	0	1	0	0	2	0	0	0	0	0	2	2	7	Island Creek	LO	340
136	S 241	F	3	0	2	0	1	1	0	1	0	0	1	1	0	0	0	1	1	2	7	Island Creek	LO	325
137	S 253	F	3	0	1	0	2	1	1	1	0	0	2	0	0	0	0	2	1	2	7	Island Creek	LO	330
138	S 254	F	3	1	3	0	2	0	1	0	0	1	0	0	0	0	0	3	2	2	7	Island Creek	LO	335
139	S 273	F	2	2	3	2	2	0	0	1	0	2	1	1	0	0	0	2	1	0	7	Island Creek	LO	321
140	S 317	F	3	0	0	0	1	2	2	1	0	1	1	0	0	0	0	2	3	0	7	Island Creek	LO	323
141	S 8	M	1	0	0	0	2	0	0	1	0	0	0	0	1	0	0	0	1	0	7	Island Creek	LO	305
142	S 20	M	2	0	1	0	2	0	0	1	0	.	.	.	.	.	0	1	1	0	7	Island Creek	LO	320
143	S 33	M	0	0	1	0	0	0	0	1	0	0	0	0	0	0	0	1	1	0	7	Island Creek	LO	301
144	S 80	M	1	0	0	0	0	0	0	1	0	1	0	2	0	0	0	1	0	0	7	Island Creek	LO	308
145	S 105	M	2	1	1	0	3	0	0	0	0	1	0	1	2	0	0	.	.	.	7	Island Creek	LO	315
146	S 139	M	1	0	1	0	3	0	0	0	0	0	0	0	1	0	0	1	1	1	7	Island Creek	LO	310
147	S 148	M	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	1	1	0	7	Island Creek	LO	302
148	S 167	M	0	0	0	0	0	0	0	0	0	0	0	1	0	1	0	0	1	0	7	Island Creek	LO	319
149	S 198	M	0	0	0	0	3	0	0	1	0	1	0	1	1	0	0	1	1	0	7	Island Creek	LO	309
150	S 219	M	1	0	2	0	1	0	0	1	0	0	0	0	0	0	0	1	0	0	7	Island Creek	LO	306
151	S 226	M	2	0	1	0	3	0	0	1	0	0	0	0	0	0	0	1	0	0	7	Island Creek	LO	314
152	S 244	M	0	0	0	0	2	0	0	1	0	0	0	0	0	1	0	1	1	0	7	Island Creek	LO	316
153	S 257	M	2	0	0	0	2	0	0	1	0	0	0	1	0	1	2	1	1	2	7	Island Creek	LO	311
154	S 275	M	0	0	0	0	1	0	0	1	0	0	0	0	0	0	0	2	0	0	7	Island Creek	LO	312
155	S 286	M	1	0	1	0	1	0	0	1	0	0	0	1	0	0	0	0	1	0	7	Island Creek	LO	317
156	S 289	M	1	0	0	0	0	0	0	1	0	1	0	1	0	0	0	0	1	1	7	Island Creek	LO	304
157	S 291	M	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	7	Island Creek	LO	303
158	S 303	M	2	0	0	0	2	0	0	2	0	0	0	0	0	0	0	1	3	1	7	Island Creek	LO	307
159	S 307	M	0	0	0	0	0	0	0	1	0	0	0	1	0	0	0	2	1	0	7	Island Creek	LO	318
160	S 323	M	0	0	0	0	1	2	0	1	0	0	1	1	0	0	0	0	1	0	7	Island Creek	LO	313

	Proc.		LIVER									KIDNEY					SPL	NARES			Stream			Alaska
#	#	Sex	GLY	LIP	HV	PD	PEL	SCN	FN	MEG	FIB	VD	PD	LUM	MYX	GLM	MA	EGL	MUC	SCN	#	Name	OS	#
161	S 4	F	3	0	1	0	2	0	0	0	0	0	1	0	0	1	0	1	1	0	8	James Lagoon	LO	834
162	S 9	F	3	0	1	0	2	0	0	0	0	0	1	0	1	0	0	1	1	0	8	James Lagoon	LO	833
163	S 38	F	3	0	3	1	1	0	0	1	0	0	0	0	0	0	0	1	0	0	8	James Lagoon	LO	823
164	S 53	F	3	1	2	1	1	1	0	0	0	1	1	0	0	2	.	.	.	.	8	James Lagoon	LO	821
165	S 55	F	3	1	2	0	1	0	0	1	0	1	1	0	2	1	0	1	1	0	8	James Lagoon	LO	822
166	S 56	F	3	1	2	1	1	1	0	1	0	0	1	0	0	1	0	.	.	.	8	James Lagoon	LO	831
167	S 71	F	3	0	3	0	0	0	0	1	0	1	2	1	0	1	0	1	0	1	8	James Lagoon	LO	839
168	S 113	F	3	0	0	0	3	1	0	1	0	2	0	1	0	0	0	1	0	1	8	James Lagoon	LO	829
169	S 143	F	3	0	1	0	0	0	0	1	0	2	1	0	0	0	0	0	3	2	8	James Lagoon	LO	828
170	S 168	F	3	0	0	0	2	1	0	1	0	1	2	1	0	0	0	2	1	1	8	James Lagoon	LO	824
171	S 175	F	2	0	0	1	0	2	0	1	0	1	1	1	0	0	0	2	1	1	8	James Lagoon	LO	840
172	S 191	F	3	0	0	0	2	1	0	1	0	1	0	.	1	2	0	1	2	1	8	James Lagoon	LO	837
173	S 192	F	3	0	1	0	1	0	0	1	0	1	1	1	0	1	0	1	1	0	8	James Lagoon	LO	825
174	S 209	F	3	0	3	0	2	0	0	2	0	2	0	0	0	0	0	2	0	1	8	James Lagoon	LO	832
175	S 213	F	3	0	0	0	2	1	0	1	0	1	0	1	2	0	0	1	2	2	8	James Lagoon	LO	835
176	S 221	F	3	0	3	0	2	2	0	1	0	0	0	0	0	1	0	1	1	0	8	James Lagoon	LO	827
177	S 269	F	3	0	0	0	2	1	0	1	0	1	1	1	2	0	0	2	1	1	8	James Lagoon	LO	838
178	S 277	F	3	0	0	0	2	1	0	0	0	0	0	1	1	0	0	0	1	0	8	James Lagoon	LO	836
179	S 300	F	3	0	0	0	2	1	0	0	0	1	0	1	0	0	0	2	0	0	8	James Lagoon	LO	830
180	S 305	F	3	0	0	0	1	1	0	1	0	1	1	0	0	0	0	1	1	0	8	James Lagoon	LO	826
181	S 2	M	0	0	2	0	0	0	0	1	1	0	0	0	0	0	0	2	1	2	8	James Lagoon	LO	814
182	S 23	M	0	0	0	0	1	0	0	1	0	0	0	0	0	0	0	0	0	0	8	James Lagoon	LO	801
183	S 52	M	0	0	0	0	1	0	0	1	0	0	0	0	0	0	0	3	1	0	8	James Lagoon	LO	808
184	S 68	M	2	0	2	0	2	0	1	1	0	1	0	1	1	0	0	0	0	1	8	James Lagoon	LO	819
185	S 94	M	1	0	0	0	2	0	0	0	0	1	0	0	1	0	0	1	0	0	8	James Lagoon	LO	809
186	S 106	M	1	2	1	0	1	0	0	1	0	1	0	1	1	0	0	0	0	1	8	James Lagoon	LO	805
187	S 116	M	0	0	0	0	0	0	0	0	0	1	0	1	0	0	0	.	.	.	8	James Lagoon	LO	804
188	S 119	M	0	0	1	0	0	0	0	1	0	1	0	0	0	0	0	0	0	1	8	James Lagoon	LO	802
189	S 134	M	2	1	0	0	0	0	1	0	1	1	2	1	2	0	0	0	2	0	8	James Lagoon	LO	810
190	S 147	M	0	0	0	0	1	0	0	0	0	1	0	2	1	0	0	0	1	0	8	James Lagoon	LO	816
191	S 170	M	0	0	0	0	1	0	0	0	0	1	0	1	0	0	0	0	1	0	8	James Lagoon	LO	812
192	S 172	M	0	0	0	0	1	0	0	0	0	1	0	1	0	0	0	0	2	0	8	James Lagoon	LO	811
193	S 233	M	0	0	0	0	2	0	0	0	0	.	.	.	.	.	0	0	1	0	8	James Lagoon	LO	807
194	S 238	M	0	0	0	0	2	0	0	1	0	0	0	1	0	1	0	3	1	1	8	James Lagoon	LO	815
195	S 278	M	1	0	0	0	2	0	0	1	0	0	0	1	1	0	0	0	1	0	8	James Lagoon	LO	817
196	S 306	M	0	0	0	0	0	0	0	1	0	1	0	0	0	0	0	0	1	0	8	James Lagoon	LO	806
197	S 310	M	0	0	0	0	1	0	0	1	0	0	0	0	0	0	0	2	2	0	8	James Lagoon	LO	820
198	S 313	M	0	0	0	0	1	1	0	2	0	1	0	1	0	1	0	1	3	0	8	James Lagoon	LO	813
199	S 314	M	3	1	0	0	0	0	0	1	0	0	0	1	2	1	0	2	1	0	8	James Lagoon	LO	803
200	S 324	M	0	0	0	0	2	0	0	1	0	0	0	1	0	0	0	0	1	0	8	James Lagoon	LO	818

	Proc.			LIVER								KIDNEY					SPL	NARES			Stream			Alaska
#	#		Sex	GLY	LIP	HV	PD	PEL	SCN	FN	MEG	FIB	VD	PD	LUM	MYX	GLM	MA	EGL	MUC	SCN	#	Name	OS	#
201	S	11	F	3	1	3	1	1	1	0	1	0	0	0	0	2	0	0	1	1	1	10	Port Graham	C	637
202	S	30	F	3	0	1	1	1	0	0	0	1	2	0	2	3	0	0	1	0	0	10	Port Graham	C	627
203	S	74	F	3	1	2	1	0	1	2	1	3	1	1	1	1	0	0	1	0	1	10	Port Graham	C	633
204	S	100	F	3	1	3	1	1	0	0	0	0	2	2	1	0	0	0	2	1	1	10	Port Graham	C	630
205	S	146	F	3	1	1	1	1	1	0	1	0	2	0	1	0	0	0	0	1	1	10	Port Graham	C	626
206	S	157	F	3	1	2	0	0	1	0	2	0	3	1	0	0	0	0	2	2	2	10	Port Graham	C	639
207	S	184	F	3	0	2	3	1	1	2	1	2	2	1	1	0	0	0	2	1	1	10	Port Graham	C	622
208	S	190	F	3	0	3	2	3	0	1	1	0	2	2	0	0	0	0	2	1	1	10	Port Graham	C	623
209	S	200	F	2	1	3	0	0	0	1	0	0	2	0	2	0	0	0	2	3	2	10	Port Graham	C	631
210	S	202	F	2	2	1	1	2	0	0	0	0	1	1	0	0	0	0	2	1	2	10	Port Graham	C	632
211	S	208	F	3	0	0	0	1	1	0	1	0	1	1	0	0	0	0	1	2	2	10	Port Graham	C	636
212	S	217	F	3	0	3	2	1	1	0	1	0	2	3	0	0	0	0	3	2	0	10	Port Graham	C	635
213	S	222	F	3	0	2	2	3	1	0	1	0	0	1	0	0	0	0	0	2	0	10	Port Graham	C	628
214	S	232	F	3	2	0	1	0	2	0	1	0	1	1	2	0	1	0	.	.	.	10	Port Graham	C	640
215	S	259	F	3	1	3	0	1	1	0	1	0	2	1	0	0	0	0	1	3	1	10	Port Graham	C	638
216	S	267	F	.	.	.	.	.	.	.	.	.	2	0	1	0	2	0	2	2	3	10	Port Graham	C	629
217	S	284	F	1	1	0	1	1	0	0	2	0	0	1	1	0	1	0	2	2	1	10	Port Graham	C	621
218	S	298	F	3	0	2	1	0	0	0	1	0	2	0	1	1	0	0	2	1	2	10	Port Graham	C	624
219	S	311	F	3	1	2	1	1	0	0	1	0	0	0	0	0	0	0	2	1	1	10	Port Graham	C	634
220	S	322	F	3	1	2	2	1	1	2	1	0	1	1	1	0	1	0	2	2	0	10	Port Graham	C	625
221	S	32	M	2	2	1	0	1	0	0	1	0	0	0	0	0	0	0	1	1	0	10	Port Graham	C	602
222	S	35	M	3	1	1	0	3	0	0	0	0	0	2	0	0	0	0	1	2	0	10	Port Graham	C	609
223	S	41	M	3	2	0	0	1	0	0	1	0	0	3	2	2	0	0	.	.	.	10	Port Graham	C	613
224	S	50	M	2	3	0	0	0	0	0	1	0	1	2	1	2	0	0	.	.	.	10	Port Graham	C	610
225	S	76	M	2	1	1	0	2	1	0	0	0	0	2	0	0	0	0	1	0	1	10	Port Graham	C	614
226	S	98	M	2	1	1	0	1	0	0	1	1	0	0	1	0	0	0	2	0	1	10	Port Graham	C	601
227	S	138	M	3	0	1	0	3	0	1	0	0	1	1	1	0	1	0	1	2	2	10	Port Graham	C	616
228	S	163	M	2	1	0	0	1	1	1	1	0	0	1	0	0	0	0	1	1	1	10	Port Graham	C	606
229	S	164	M	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	1	1	10	Port Graham	C	612
230	S	214	M	2	1	0	0	2	0	0	1	0	2	0	1	0	0	0	.	.	.	10	Port Graham	C	607
231	S	223	M	3	2	0	0	1	0	0	0	0	0	2	0	0	0	1	1	1	0	10	Port Graham	C	611
232	S	230	M	2	0	0	0	3	1	0	1	0	0	2	1	0	0	0	1	2	0	10	Port Graham	C	603
233	S	240	M	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0	.	.	.	10	Port Graham	C	604
234	S	262	M	3	0	0	0	1	0	0	2	0	1	2	0	0	0	0	1	1	0	10	Port Graham	C	605
235	S	271	M	2	0	0	0	2	0	1	0	0	1	0	1	0	0	0	2	1	1	10	Port Graham	C	618
236	S	272	M	3	0	0	0	2	0	1	1	1	1	2	1	0	0	0	1	1	1	10	Port Graham	C	617
237	S	281	M	3	2	0	0	1	0	0	1	1	1	2	2	3	0	.	2	3	1	10	Port Graham	C	619
238	S	282	M	3	1	0	0	1	1	2	1	0	1	2	2	0	0	2	0	0	2	10	Port Graham	C	620
239	S	294	M	3	0	0	0	1	0	0	1	0	1	2	1	0	0	.	3	2	1	10	Port Graham	C	608
240	S	308	M	2	1	0	0	2	0	0	1	0	0	0	1	0	0	0	1	0	0	10	Port Graham	C	615

#	Proc. #	Sex	LIVER									KIDNEY					SPL	NARES			Stream			Alaska
			GLY	LIP	HV	PD	PEL	SCN	FN	MEG	FIB	VD	PD	LUM	MYX	GLM	MA	EGL	MUC	SCN	#	Name	OS	#
241	S 42	F	3	1	2	0	2	0	0	0	0	0	0	0	0	0	0	1	1	0	13	Windy Bay, Rt	O	234
242	S 60	F	3	0	3	2	1	0	0	1	0	1	1	0	0	0	0	2	1	1	13	Windy Bay, Rt	O	230
243	S 103	F	3	0	3	1	2	1	0	1	0	1	1	1	0	0	0	1	0	1	13	Windy Bay, Rt	O	233
244	S 128	F	3	0	2	2	2	0	0	2	0	1	0	1	2	0	0	2	1	0	13	Windy Bay, Rt	O	225
245	S 129	F	3	0	2	3	2	1	0	1	0	0	2	1	1	0	0	0	1	1	13	Windy Bay, Rt	O	222
246	S 131	F	3	1	2	3	0	0	0	1	0	1	1	1	1	0	0	.	.	.	13	Windy Bay, Rt	O	228
247	S 133	F	3	0	3	1	2	0	0	0	0	0	1	0	0	0	0	1	0	2	13	Windy Bay, Rt	O	221
248	S 137	F	2	3	3	0	0	0	0	1	0	1	0	0	2	0	0	3	2	0	13	Windy Bay, Rt	O	239
249	S 142	F	3	0	2	2	2	0	0	1	0	1	1	1	0	0	0	0	1	0	13	Windy Bay, Rt	O	223
250	S 150	F	3	1	2	0	1	1	1	0	0	1	1	2	1	0	0	3	1	3	13	Windy Bay, Rt	O	236
251	S 151	F	3	0	2	1	1	0	0	1	0	1	2	1	0	0	0	1	1	0	13	Windy Bay, Rt	O	226
252	S 201	F	3	0	3	0	0	0	0	2	0	.	.	.	.	.	0	1	1	0	13	Windy Bay, Rt	O	229
253	S 210	F	3	1	3	0	0	0	0	1	0	0	0	1	0	0	0	1	1	2	13	Windy Bay, Rt	O	240
254	S 220	F	3	0	2	0	2	0	0	0	0	0	0	0	0	1	0	2	1	0	13	Windy Bay, Rt	O	224
255	S 260	F	3	1	2	0	0	0	0	1	0	0	0	1	2	0	0	1	1	0	13	Windy Bay, Rt	O	232
256	S 261	F	3	1	3	0	1	0	0	1	0	1	0	0	0	0	0	1	1	0	13	Windy Bay, Rt	O	237
257	S 264	F	3	2	3	0	2	0	0	0	0	1	1	1	0	2	2	1	1	1	13	Windy Bay, Rt	O	235
258	S 270	F	3	0	1	0	1	0	0	0	0	1	1	1	0	0	0	1	1	0	13	Windy Bay, Rt	O	238
259	S 299	F	2	1	3	0	3	1	0	1	0	0	1	1	0	0	0	1	1	1	13	Windy Bay, Rt	O	231
260	S 327	F	3	1	3	1	0	1	0	1	1	0	1	0	0	0	2	1	0	0	13	Windy Bay, Rt	O	227
261	S 12	M	3	1	0	0	1	0	0	1	0	0	0	0	0	0	0	0	2	0	13	Windy Bay, Rt	O	215
262	S 57	M	3	1	1	0	2	0	0	0	0	1	1	0	0	0	0	.	.	.	13	Windy Bay, Rt	O	204
263	S 59	M	3	1	0	0	0	0	0	2	0	1	0	1	0	0	0	2	1	1	13	Windy Bay, Rt	O	202
264	S 66	M	3	1	0	0	2	1	0	1	0	2	1	0	0	0	0	0	1	1	13	Windy Bay, Rt	O	205
265	S 70	M	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	13	Windy Bay, Rt	O	209
266	S 85	M	1	0	1	0	3	0	0	1	0	1	0	1	0	0	0	3	0	0	13	Windy Bay, Rt	O	203
267	S 88	M	1	0	1	0	1	0	0	1	0	1	0	1	0	0	0	1	0	0	13	Windy Bay, Rt	O	213
268	S 91	M	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	13	Windy Bay, Rt	O	220
269	S 99	M	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	13	Windy Bay, Rt	O	211
270	S 121	M	3	1	1	0	1	1	0	1	0	1	1	1	2	0	0	2	1	1	13	Windy Bay, Rt	O	212
271	S 126	M	3	2	0	0	2	0	0	1	1	1	2	1	1	0	0	0	0	2	13	Windy Bay, Rt	O	208
272	S 160	M	3	2	1	0	2	1	0	1	0	1	1	1	0	0	0	1	1	0	13	Windy Bay, Rt	O	207
273	S 194	M	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	2	1	0	13	Windy Bay, Rt	O	217
274	S 203	M	.	.	.	.	.	.	.	.	.	0	0	1	1	0	0	1	1	2	13	Windy Bay, Rt	O	206
275	S 211	M	3	1	0	0	3	2	0	1	0	2	2	0	0	0	0	3	1	2	13	Windy Bay, Rt	O	214
276	S 228	M	2	2	0	0	2	1	0	1	0	0	1	0	0	0	0	0	1	0	13	Windy Bay, Rt	O	201
277	S 236	M	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	13	Windy Bay, Rt	O	210
278	S 252	M	2	3	0	0	3	0	0	0	0	0	0	1	0	0	0	3	1	1	13	Windy Bay, Rt	O	218
279	S 255	M	1	0	0	0	2	0	1	1	0	0	0	1	0	0	0	2	2	1	13	Windy Bay, Rt	O	216
280	S 287	M	3	3	0	0	2	0	0	1	0	1	0	1	0	0	0	0	1	1	13	Windy Bay, Rt	O	219

	Proc.			LIVER									KIDNEY					SPL	NARES			Stream			Alaska
#	#	Sex	GLY	LIP	HV	PD	PEL	SCN	FN	MEG	FIB	VD	PD	LUM	MYX	GLM	MA	EGL	MUC	SCN	#	Name	OS	#
281	S 37	F	3	0	2	0	1	1	2	0	0	0	1	0	0	0	0	0	1	0	12	Windy Bay, left	O	139
282	S 61	F	2	0	2	0	1	1	1	0	0	0	1	0	0	0	0	1	0	1	12	Windy Bay, left	O	123
283	S 64	F	3	0	0	0	2	1	1	2	0	1	1	0	0	0	0	0	1	1	12	Windy Bay, left	O	129
284	S 67	F	3	1	3	1	2	1	0	1	0	0	0	1	1	0	0	0	1	1	12	Windy Bay, left	O	125
285	S 75	F	3	0	0	0	1	2	0	1	0	1	2	0	0	0	0	2	0	1	12	Windy Bay, left	O	133
286	S 101	F	3	0	3	1	1	0	0	0	0	0	1	1	0	0	0	0	0	1	12	Windy Bay, left	O	135
287	S 102	F	3	1	3	2	1	1	0	1	0	1	2	1	1	0	0	1	0	1	12	Windy Bay, left	O	132
288	S 114	F	3	0	2	3	1	0	0	1	0	1	2	1	0	0	0	1	0	0	12	Windy Bay, left	O	140
289	S 130	F	3	1	2	1	3	0	0	1	0	0	1	2	2	0	0	1	0	2	12	Windy Bay, left	O	126
290	S 156	F	3	0	1	0	3	1	1	0	0	1	1	1	3	1	0	.	.	.	12	Windy Bay, left	O	130
291	S 162	F	3	0	3	2	2	1	1	1	0	0	1	0	0	0	0	0	1	2	12	Windy Bay, left	O	127
292	S 178	F	3	1	2	1	2	1	1	1	0	1	1	1	0	0	0	1	2	0	12	Windy Bay, left	O	128
293	S 195	F	3	0	3	0	2	1	1	1	0	0	0	1	2	0	0	1	2	2	12	Windy Bay, left	O	138
294	S 215	F	3	0	3	0	1	2	0	2	0	0	0	1	0	1	0	0	2	2	12	Windy Bay, left	O	136
295	S 245	F	3	0	2	0	1	2	1	0	0	0	2	1	0	1	.	2	1	2	12	Windy Bay, left	O	134
296	S 250	F	3	0	1	0	1	1	1	1	0	1	1	1	0	0	0	1	1	2	12	Windy Bay, left	O	131
297	S 263	F	3	0	1	0	1	2	1	1	0	1	0	0	0	0	0	0	2	2	12	Windy Bay, left	O	137
298	S 265	F	3	0	0	0	2	2	0	1	0	1	1	0	0	0	0	0	1	0	12	Windy Bay, left	O	124
299	S 274	F	3	0	2	0	2	1	1	1	0	0	1	1	0	0	0	1	1	1	12	Windy Bay, left	O	121
300	S 316	F	3	0	1	0	3	1	1	1	0	0	2	0	0	0	0	3	3	2	12	Windy Bay, left	O	122
301	S 14	M	2	3	1	0	0	0	0	1	0	1	2	1	0	0	0	1	1	0	12	Windy Bay, left	O	112
302	S 21	M	1	0	2	0	1	1	0	2	0	0	0	0	0	.	0	1	2	1	12	Windy Bay, left	O	104
303	S 31	M	2	0	1	0	1	0	0	0	0	1	1	0	0	0	0	1	1	0	12	Windy Bay, left	O	119
304	S 44	M	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	.	.	.	12	Windy Bay, left	O	113
305	S 45	M	.	.	.	.	.	.	.	.	.	.	.	.	.	.	0	.	.	.	12	Windy Bay, left	O	117
306	S 81	M	1	0	0	0	1	0	1	1	0	1	1	1	1	0	0	2	1	0	12	Windy Bay, left	O	115
307	S 97	M	1	0	1	0	1	0	1	1	0	0	0	0	0	0	0	0	0	1	12	Windy Bay, left	O	107
308	S 107	M	1	0	1	0	1	0	0	1	0	1	0	1	1	0	0	.	.	.	12	Windy Bay, left	O	108
309	S 118	M	2	0	1	0	1	0	0	1	0	0	0	2	1	0	0	0	1	0	12	Windy Bay, left	O	101
310	S 144	M	1	1	0	0	0	0	0	1	0	1	0	1	1	0	0	1	0	2	12	Windy Bay, left	O	109
311	S 159	M	0	0	1	0	0	0	0	0	0	1	0	1	0	0	0	2	2	0	12	Windy Bay, left	O	102
312	S 165	M	2	1	0	0	0	0	0	1	0	1	2	2	0	1	0	2	1	2	12	Windy Bay, left	O	120
313	S 171	M	2	1	0	0	1	0	0	0	0	2	0	1	0	0	0	2	1	1	12	Windy Bay, left	O	118
314	S 174	M	2	2	1	0	1	0	0	0	0	1	2	1	0	0	0	.	.	.	12	Windy Bay, left	O	116
315	S 197	M	1	0	0	0	2	0	1	1	0	1	0	1	0	0	0	1	3	2	12	Windy Bay, left	O	110
316	S 225	M	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0	12	Windy Bay, left	O	111
317	S 242	M	0	0	0	0	2	0	0	1	0	0	0	1	0	0	0	1	1	2	12	Windy Bay, left	O	105
318	S 295	M	2	2	0	1	0	0	0	1	0	0	1	1	0	0	0	1	2	1	12	Windy Bay, left	O	106
319	S 319	M	3	2	0	0	1	0	0	0	0	2	3	1	0	0	0	2	1	0	12	Windy Bay, left	O	114
320	S 326	M	1	0	0	0	0	0	0	1	0	0	0	2	0	0	0	.	.	.	12	Windy Bay, left	O	103

MEAN LESION SCORES

		LIVER									KIDNEY					SPL	NARES			Stream	
OS	Sex	GLY	LIP	HV	PD	PEL	SCN	FN	MEG	FIB	VD	PD	LUM	MYX	GLM	MA	EGL	MUC	SC	Name	OS
C	F	3	.2	1.3	.15	1.3	1.7	.85	.55	.25	1.1	.45	.55	.45	.1	0	.86	.79	1.1	Humpy Creek	C
C	M	1.2	.05	.3	.05	1.1	.45	.5	.6	.1	.6	.3	1.2	1.1	0	0	1	1.4	.79	Humpy Creek	C
C	F	2.8	.74	1.8	1.1	1	.63	.42	.89	.32	1.4	.85	.7	.35	.25	0	1.6	1.5	1.2	Port Graham	C
C	M	2.3	.9	.25	0	1.5	.2	.3	.7	.15	.5	1.3	.75	.35	.05	.17	1.2	1.1	.75	Port Graham	C
LO	F	3	.3	.95	.2	1.9	1.1	.15	.85	0	.33	.56	.83	.61	.06	0	1.3	1.1	1	Port Dick	LO
LO	M	1.4	.05	.4	0	1.8	.15	.1	.85	0	.55	0	.7	.7	.05	0	.8	1.1	.3	Port Dick	LO
LO	F	3.0	0	1.5	.5	1.1	1.5	.45	.55	.2	.7	.8	.5	.35	0	0	1.4	1.2	.74	South Nuka	LO
LO	M	1	.2	.2	0	.75	.3	.25	.5	0	.7	.15	.85	.65	.15	0	1.2	1.4	.61	South Nuka	LO
LO	F	3.0	.3	1.6	.15	2.0	.9	.3	.75	0	.55	.95	.4	.05	.15	.1	1.2	1.2	.84	Island Creek	LO
LO	M	.8	.05	.4	0	1.3	.1	0	.85	0	.21	.05	.53	.26	.16	.1	.79	.89	.26	Island Creek	LO
LO	F	3.0	.15	1.1	.2	1.5	.7	0	.8	0	.85	.7	.47	.45	.5	0	1.2	.94	.61	James Lagoon	LO
LO	M	.5	.2	.3	0	1	.05	.1	.7	.1	.58	.11	.68	.47	.16	0	.74	1	.32	James Lagoon	LO
O	F	2.9	.65	2.5	.8	1.2	.25	.05	.8	.05	.58	.74	.68	.47	.16	.2	1.3	.89	.63	Windy Bay, Rt	O
O	M	2.4	1.3	.36	0	1.9	.43	.07	.93	.07	.8	.6	.67	.27	0	0	1.3	.93	.8	Windy Bay, Rt	O
O	F	3.0	.2	1.8	.55	1.7	1.1	.65	.85	0	.45	1.1	.65	.45	.15	0	.79	1	1.2	Windy Bay, left	O
O	M	1.3	.5	.44	.06	.72	.06	.17	.67	0	.67	.56	.89	.22	.06	0	1.2	1.2	.86	Windy Bay, left	O
C	F	2.9	.47	1.5	.63	1.1	1.1	.64	.72	.28	1.2	.65	.63	.4	.18	0	1.2	1.1	1.2	Combined clean sites	
LO	F	3.0	.19	1.3	.26	1.6	1.0	.23	.74	.05	.61	.75	.55	.37	.18	.03	1.3	1.1	.80	Combined lightly oiled sites	
O	F	2.9	.43	2.1	.68	1.4	.68	.35	.83	.03	.51	.89	.67	.46	.15	.1	1.0	.95	.92	Combined oiled sites	
C	M	1.7	.48	.28	.03	1.3	.33	.4	.65	.13	.55	.78	.98	.7	.03	.08	1.1	1.2	.77	Combined clean sites	
LO	M	.91	.13	.33	0	1.2	.15	.11	.73	.03	.51	.08	.69	.52	.13	.03	.89	1.1	.37	Combined lightly oiled sites	
O	M	1.9	.89	.40	.03	1.3	.24	.12	.80	.04	.73	.58	.78	.24	.03	0	1.3	1.1	.83	Combined oiled sites	

CHAPTER 11 - General Literature Review of Lesions in Fish from Prince William Sound

Before the spill, little research on lesions in fish from actual oil spills had been published, but several laboratory studies on the effects of exposing fish to crude oil or refined hydrocarbons have been conducted. Some references directly describe morphologic lesions, whereas others concentrated on enzymatic, physiologic, or behavioral changes that might decrease the ability of exposed fish to survive in the environment. Histopathologic lesions associated with petroleum hydrocarbon exposure are described by organ for juvenile and adult fish (Table XI-1) and for larvae (Table XI-2). Tables with some of the same findings of earlier work were previously reviewed (Malins 1982). Although exposure to petroleum hydrocarbons produces several types of lesions, none of these lesions are specific for exposure to oil. General aspects of the effect of petroleum hydrocarbons on subtidal regions have recently been reviewed (Lee and Page 1997).

In our studies on the effects of the spill on fisheries in Prince William Sound, several lesions were found consistently in various fish species. For example, hepatocellular megalocytosis occurred in rockfish, pink salmon, Dolly Varden char, and in Pacific herring. The purpose of this section is to review lesions associated with petroleum hydrocarbons, with emphasis on lesions of significance in this report.

HEPATOCELLULAR LIPIDOSIS - Vacuolation of hepatocytes (fatty change) is a common response associated with exposure of fish to a variety of different agents (Meyers and Hendricks 1985). Studies in rat liver indicate a multitude of possible mechanisms to account for development of fatty liver (Lombardi 1966). In general, the condition is not due to excess uptake of lipid precursors, but a defect of exporting lipid from hepatocytes. This could signify one or more biochemical lesions. First, inhibition of protein synthesis: the apoprotein is not made in sufficient amount to bind with the lipid for transport from the cell. Second, energy depletion: lipid being transported from the cell normally moves through the endoplasmic reticulum (ER) and fuses with the Golgi apparatus. This fusion of ER and Golgi is thought to require energy, and if energy levels in the cell are deficient, lipid might accumulate within ER cisternae. Third, disaggregation of microtubules: once secretory vesicles containing lipoprotein substances have been formed, they must move from the Golgi apparatus to the plasma membrane. Microtubules guide movement of vesicles in cells. Thus, disaggregation of microtubules is another mechanism whereby fatty liver can arise (Bannasch et al. 1981). And fourth, shifts in substrate utilization (e.g., inhibition of metabolic pathways such as the ß-oxidation of fatty acids) might also lead to accumulation of excess lipid.

Mechanistic studies of fatty change in teleost livers have not been done, despite the relative common occurrence of fatty change in response to toxicant exposure (see Table XI-1). Hydrocarbon exposure often results in increased amounts of hepatocellular lipid (McCain et al. 1978, Eurell and Haensly 1981, Fletcher et al. 1982, Solangi and Overstreet 1982, Khan and Kiceniuk 1984). However, decreased amounts of hepatocellular lipid have been described in other studies following hydrocarbon exposure (Sabo et al. 1975, Haensly et al. 1982, Woodward et al. 1983). These changes must be differentiated from small lipid vacuoles, often multiple per cell, that are normal in hepatocytes of females producing vitellogenin for transfer to oocytes (van Boheman et al. 1981).

HEPATOCELLULAR MEGALOCYTOSIS - Hepatocellular megalocytosis is characterized by marked cellular and nuclear enlargement. With light microscopy, megalocytes are often 3 to 5 times larger that normal hepatocytes, and their enlarged nuclei frequently have eosinophilic inclusions; multinucleate megalocytes have been described. A condition involving megalocytosis, termed megalocytic hepatosis, is the most frequently encountered idiopathic lesion in the liver of English sole *Parophrys vetulus* from contaminant-laden sites within Puget Sound, Washington (Myers et al. 1990). These authors interpreted megalocytosis as a manifestation of chronic toxicity of sediment contaminants. Megalocytosis was seen in fish from chemically contaminated sites in the Kanawha River of West Virginia (Hinton and Lauren, unpublished observations) and in sea pen cultures of Atlantic salmon from Puget Sound (Kent et al. 1988, Kent et al. 1996). Megalocytosis has been produced in the laboratory in rainbow trout *Oncorhynchus mykiss* exposed to pyrrolizidine (produced by *Senecio* spp.) alkaloids (Hendricks et al. 1981) and medaka *Oryzias latipes* exposed to diethylnitrosamine (Hinton et al. 1988). Megalocytes are probably sublethally injured hepatocytes and are able to survive for months (Kent et al. 1988, Groff et al. 1992). Megalocytosis has not previously been described in fish associated with crude oil or other petroleum hydrocarbons.

MACROPHAGE AGGREGATES - Macrophage aggregates, most common in the liver, spleen, and kidney, have been used as indicators of contaminant exposure (Couillard and Hodson 1996) and more often as a generalized nonspecific response to several stressful stimuli (e.g., starvation, heat stress) in several studies (Wolke et al. 1985, Herraez and Zapata 1986, Blazer et al. 1987). Increases in macrophage aggregate area, density, or frequency in diseased fish collected from degraded environments support the use of macrophage aggregates as a biomarker (Wolke 1992). Tissue breakdown and age are the main factors contributing to formation of macrophage aggregates (Agius 1985, Brown and George 1985, Marty et al. 1998). Although tissue breakdown might occur with toxicant exposure, age-related levels of macrophage aggregates must be considered in any study of potential toxicant exposure; i.e., ideally, fish from reference and exposed sites should be of the same age. Little is known about the dynamics of macrophage aggregates in fish; e.g., how quickly they develop, and once developed, how long before they regress.

In winter flounder collected from 8 New England coastal and urban embayments, splenic area occupied by macrophage aggregates was correlated with chemical contamination of surface sediments (Gardner et al. 1989a). Because levels of polychlorinated biphenyls, polycyclic aromatic hydrocarbons, and trace metals measured in surface sediment at these sites correlated and co-varied in the same way, benzo(a)pyrene (BaP) was used as an index of exposure against which macrophage aggregate area was compared (Gardner et al. 1989b). Macrophage aggregate area was not age dependent, and the frequency of macrophage aggregates and their total area increased with increasing levels of BaP (Gardner et al. 1989a). Mean macrophage aggregate percent area in spleens of flounder from offshore locations (i.e., Martha's Vineyard, Massachusetts, Gorges Bank) was 0.4, and corresponding sediment BaP levels were less than 0.001 µg/g. By comparison, mean macrophage aggregate percent area in flounder from polluted urban estuaries was greater than 12% and BaP levels in surface sediment approached 9 µg/g. In addition, area of splenic macrophage aggregates was greater in fish with hepatic neoplasms. Flounder from Quincy Bay (Boston Harbor, Massachusetts) without hepatic neoplasms had an

average macrophage aggregate area of 8%, whereas those with neoplasms approached 50%. Linkage of increased splenic macrophage aggregate area to contaminated sediment was demonstrated in the same species using laboratory exposures to contaminated sediment (Gardner and Pruell 1987).

Despite the potential of macrophage aggregates as biomarkers of exposure, comparatively few fish studies on effects of petroleum hydrocarbons have described macrophage aggregates. Haensly et al. (1982) found increased numbers of macrophage aggregates in the liver of plaice *Pleuronectes platessa* more than one year after the 1978 *Amoco Cadiz* Oil Spill, but macrophage aggregate numbers were not significantly increased in plaice sampled 2 years after the spill. In 3 species of fish sampled from an oiled site in Prince William Sound, Alaska, in 1990, the % area of macrophage aggregates in the spleen was significantly greater than in spleens from size-matched fish sampled from a reference site near Seward (Khan and Nag 1993). Laboratory studies have found increased numbers of macrophage aggregates in the kidney and spleen following crude oil exposure in 2 marine species (Khan and Kiceniuk 1984, Khan 1991). By comparison, laboratory studies with flatfish have found decreased numbers of macrophage aggregates after long-term exposure to crude oil in sediments (Payne and Fancey 1989, Moles and Norcross 1998).

NECROSIS - Necrosis is defined as "the sum of the morphologic changes that follow cell death in a living tissue or organ" (Cotran et al. 1989). In the most common type, coagulative necrosis, the nucleus is lost but basic cellular shape is maintained. Histologically, necrotic cells have pyknotic, karyorrhectic, or karyolytic nuclei, and hypereosinophilic coagulated cytoplasm. This type of necrosis often results from tissue ischemia (i.e., inadequate oxygenation of tissues) or cellular ischemia (e.g., blockage of mitochondrial oxidative phosphorylation, as occurs with some toxicants). Because necrosis involves tissue breakdown, necrotic cells in fish are thought to be scavenged, in part, by macrophage aggregates (Wolke 1992).

Studies on fish exposed to petroleum hydrocarbons have revealed necrosis of various tissues: gill epithelial cells (Woodward et al. 1983); liver/hepatocytes (Haensly et al. 1982, Solangi and Overstreet 1982); olfactory organ or nares (Solangi and Overstreet 1982, Latendresse and Fisher 1983); ovary/oocytes (Lopez et al. 1981) from (Malins 1982); pancreas (Solangi and Overstreet 1982); spleen (Khan and Kiceniuk 1984); and the tail (Haensly et al. 1982). In addition, necrosis in the forebrain and neuronal layer of the retina was described in embryonic Surf smelt *Hypomesus pretiosus* exposed to a seawater-accommodated fraction of crude oil during development (Hawkes and Stehr 1982). Solangi and Overstreet (1982) concluded that necrosis of pancreatic acinar cells was the most specific oil-related lesion. In Pacific herring sampled in 1989, hepatic necrosis occurred in fish from oiled sites only, but this was later attributed to expression of viral hemorrhagic septicemia virus in affected fish (Carls et al. 1998).

Table XI-1. Histopathological or ultrastructural lesions in juvenile or adult fish exposed to crude oil or petroleum components.

Organ	Significant Lesions	Species	Type of petroleum. mode of exposure	Concentration (exposure period)	Reference
eye	increased lens diameter	Rainbow trout *Oncorhynchus mykiss*	Prudhoe Bay crude oil, feeding	17 mg crude oil/kg fish weight/d (13 months)	(Hawkes 1977)
eye	increased lens diameter	Cunner *Tautogolabrus adspersus*	Venezuelan crude oil as a continuous flow surface slick	actual concentration was not measured (6 months)	(Payne et al. 1978)
eye	lens diameter was not significantly different	Cunner	Venezuelan crude oil as a continuous flow surface slick	concentrations were not determined; surface oil was replaced with fresh oil every week (14 d)	(Kiceniuk et al. 1980)
eye	cataract formation; SEM: lateral projections of lens fiber cells were absent or grossly misshapen	Rainbow trout	Prudhoe Bay crude oil, feeding	1 g oil/kg feed (3 years)	(Hawkes 1980)
fin	see skin				
gastrointestinal tract	prevalence and intensity of infections with the digenetic trematode *Steringophorus furciger* were lower in oil treated fish	Atlantic cod *Gadus morhua*	Venezuelan and Hibernia crude oil extracts	I didn't write down the concentrations (81-140 d)	(Khan and Kiceniuk 1983)
gastrointestinal tract	prevalence and intensity of infections with the acanthocephalan *Echinorhynchus gadi* were lower in oil treated fish (effect was most pronounced in fish exposed to WSF)	winter flounder *Pseudopleuronectes americanus*	Venezuelan crude oil, water soluble fraction or 1-yr-old contaminated sediment	I didn't write down the concentrations; WSF (34 d) or sediment (160 d)	(Khan and Kiceniuk 1983)
gastrointestinal tract	hydropic degeneration of gastric gland epithelial cells and lower frequency of gastric parasites; significant in all samples	Plaice *Pleuronectes platessa*	*Amoco Cadiz* Oil Spill (March 16, 1978); crude oil, natural exposure	unknown concentration (fish sampled in Dec 78, Apr 79, Aug 79, Feb 80, & Jun 80)	(Haensly et al. 1982)

Organ	Significant Lesions	Species	Type of petroleum. mode of exposure	Concentration (exposure period)	Reference
gastrointestinal tract	chlorinated biphenyls, but not petroleum hydrocarbons, caused exfoliation of intestinal epithelial cells; ultrastructurally, epithelial cells in both groups had intracellular vacuoles of flocculent, finely granular material, and combined-group vacuoles had variable electron density; different lesions based on sampling day were not described	Chinook salmon *Oncorhynchus tshawytscha*	chlorinated biphenyls and/or a mixture of 8 petroleum hydrocarbons; dietary exposure	5 ppm in feed (28 d exposure, with 21 d depuration on clean diet); the mixture group received 10 ppm in feed (same exposure times); tissues sampled at 14, 28, and 49 d	(Hawkes et al. 1980)
gastrointestinal tract	decreased prevalence of digenetic trematodes	yellowfin sole *Pleuronectes asper* rock sole *Pleuronectes bilineatus*	Alaska North slope crude oil in sediments (laboratory exposure)	90-d exposure to sediments laden with 0, 1600-1800, and 4300-4700 µg oil/g sand or mud in unfiltered seawater	(Moles and Norcross 1998)
fin	(see skin)				
gill	loss of epithelial and mucous cells, and considerable reduction in the number of acidophilic (chloride) cells	flounder *Ancyclopsetta quadrocellata*	low-boiling petroleum fractions; natural exposure	concentration and time since spill not known (source of spill unknown); only one control fish was examined for comparison	(Blanton and Robinson 1973)
gill	changes were similar to the flounder (above), but some mucous cells remained and fewer chloride cells were missing from the filaments	*Micropogon undulatus*	low-boiling petroleum fractions; natural exposure	concentration and time since spill not known (source of spill unknown); only one control fish was examined for comparison	(Blanton and Robinson 1973)
gill	changes identical to the flounder (above); the single specimen from the control area had no lesions	sole *Etropus crossotus*	low-boiling petroleum fractions; natural exposure	concentration and time since spill not known (source of spill unknown); only one control fish was examined for comparison	(Blanton and Robinson 1973)
gill	higher prevalence of parasitsm (11 vs. 4%) and epithelial hypertrophy (20 vs. 0%) than gills from reference areas	flathead sole *Hippoglossoides elassodon*	*Exxon Valdez* Oil Spill (March 24, 1989); crude oil, natural exposure)	unknown concentration (fish sampled in 1989)	(Armstrong et al. 1995)

Organ	Significant Lesions	Species	Type of petroleum. mode of exposure	Concentration (exposure period)	Reference
gill	Bunker C oil caused curling of lamellar tips, cell rupture, and increased necrotic debris. The oil dispersant, with or without bunker C oil, caused extensive deterioration of the gill structure, with lifting and rupture of the lamellar epithelium and blood vessels. Figures show lamellar epithelial hypertrophy and hyperplasia (i.e., lesions after exposure to the oil dispersant are more severe than lesions from bunker C oil alone).	17-cm-long rainbow trout	bunker C oil alone and in combination with an oil dispersant (Oilsperse 43); dissolved in water	200 mg/L bunker C oil alone (96h), or 200 mg/L Oilsperse alone (96h), or 200 mg/L bunker C oil combined with 200 mg/L Oilsperse (96h)	(McKeown and March 1978)
gill	branchial hyperplasia; pseudobranch: acidophilic cells were swollen and vacuolated	Fathead minnow *Pimephales promelas*	JP-4 aviation fuel, WSF as a static exposure	25 and 50% WSF (= 5 and 10 ppm); sample at 6, 12, 48, and 72 h	(Latendresse and Fisher 1983)
gill	lamellar hyperplasia, excess mucus secretion, increased numbers of *Trichodina*	Longhorn sculpin *Myxocephalus octodecemspinosus*	Hibernia crude oil in sediment	1 L crude oil/45 kg washed sand (3-6 months); total hydrocarbons were 2-3 mg/g	(Khan 1991)
gill	epithelial cell hyperplasia and fusion of gill lamellae (decreased markedly after a 17-d depuration period), separation of respiratory epithelium from underlying tissues; gill lesions were milder in *T. maculatus*	Tidewater silverside *Menidia beryllina* and the Hogchoker *Trinectes maculatus*	Louisiana whole crude oil (WCO) poured directly into the aquaria, and its WSF	5 and 100 mg WCO/L; 5 and 50% WSF; (*M. beryllina*, 21-30 d) (*T. maculatus*, 38-60 d)	(Solangi and Overstreet 1982)
gill	oil emulsions: epithelial cell separation, chloride cell abnormalities, fusion of secondary lamellae WSF and IP injection: no lesions	Rainbow trout	paraffin oil and 2 crude oils, as particulate oil emulsions, WSF, or IP injection	emulsion: 200 µL oil/L water (7 d) WSF: 35-45 µL/L (7 d) IP injection: 100 µL/kg fish/d (7 d)	(Engelhardt et al. 1981)
gill	increased numbers of mucous-producing epithelial cells, capillary dilation, lamellar hyperplasia, and fusion of adjacent filaments	Atlantic cod	Venezuelan or Hibernia crude oil, WSF in a flow-through seawater system	50-300 ppb (12-13 weeks)	(Khan and Kiceniuk 1984)
gill	sloughing of surface epithelial cells and discharge of mucous glands; numbers of *Gyrodactylus* were similar in control and exposed fish	Coho salmon *Oncorhynchus kisutch* and Starry Founder *Platichthys stellatus*	WSF Prudhoe Bay crude oil in a seawater flow-through system	100 ± 90 ppb (5 d)	(Roubal et al. 1977), from (Hawkes 1977)
gill	excess mucus production, fusion of gill filaments	Goldfish *Carassius auratus*	outboard motor exhaust (OME) or toluene, dissolved in water for a continuous flow bioassay	200, 152, and 82 ppm leaded OME, or 10 and 5 ppm toluene (up to 30 d)	(Brenniman et al. 1979)

Organ	Significant Lesions	Species	Type of petroleum. mode of exposure	Concentration (exposure period)	Reference
gill	hyperplasia and some fusion of lamellae, chloride cell hyperplasia, and a few necrotic epithelial cells	Cutthroat trout *Oncorhynchus clarki*	refined oil, dissolved in water	0 - 183 µg/L total oil (90 d)	(Woodward et al. 1983)
gill	mucous cell hyperplasia over gill rakers (500 ppm); after 24 h at 1500 ppm (lethal dose), number and size of mucous cells were decreased	*Colisa fasciatus* (freshwater fish)	Assam crude oil in aqueous solution	200, 500, & 700 ppm (up to 360 h) 1000 or 1500 ppm (2-24 h)	(Prasad 1988)
gill	hyperplasia & hypertrophy of gill lamellar mucous cells (differences were gone by the Jun 80 sample, 26 months after the spill)	Plaice	*Amoco Cadiz* Oil Spill (March 16, 1978); crude oil, natural exposure	unknown concentration (fish sampled in Dec 78, Apr 79, Aug 79, Feb 80, & Jun 80)	(Haensly et al. 1982)
gill	Increased numbers (100 fold increase) of *Trichodina*, severe hyperplasia of lamella, excess mucous secretion	Atlantic cod	crude oil/ seawater soluble fraction	50-100 µg/L (12 wk, with 14-wk depuration)	(Khan 1990)
gill	Increased numbers (17 fold increase) of *Trichodina*, severe hyperplasia of lamella, excess mucous secretion	Longhorn sculpin	crude oil/ in sediment	2200 µg/g (12 wk, with 20-wk depuration)	(Khan 1990)
gill	Increased prevalence and frequency of *Trichodina* infection	Intertidal sculpin *Oligocottus maculosus*	Prudhoe Bay crude oil/ EVOS[1] exposure	concentration not reported (collected 8-20-89 from Wildcat Cove, Pye Islands, exposed; or Seward, clean)	(Khan 1990)
gill	Before depuration - dose dependent lamellar epithelial hyperplasia, goblet cell hyperplasia, lamellar fusion, lamellar capillary dilation; after depuration- increased numbers of the parasitic monogeneid *Gyrodactylus*	Atlantic cod	Venezuelan crude oil/ seawater soluble fraction	30, 50, and 500 ppb (10-14 wks, with 20-wk depuration)	(Khan and Kiceniuk 1988)
gill	hyperplasia of chloride cells and mucous cells; hyperemia and lysis of respiratory platelets	eels (species not given)	*Amoco Cadiz* Oil Spill (March 16, 1978); crude oil, natural exposure	unknown concentration	(Lopez et al. 1981) from (Malins 1982)

EVOS = *Exxon Valdez* Oil Spill (March 24, 1989)

Organ	Significant Lesions	Species	Type of petroleum. mode of exposure	Concentration (exposure period)	Reference
gill	epithelial hyperplasia of filaments and lamellae, with fusion of lamellar tips; increased prevalence of *Trichodina borealis*	yellowfin sole rock sole	Alaska North slope crude oil in sediments (laboratory exposure)	90-d exposure to sediments laden with 0, 1600-1800, and 4300-4700 µg oil/g sand or mud in unfiltered seawater	(Moles and Norcross 1998)
gill	lamellar hyperplasia >300 ppn lamellar mucous hyperplasia at highest dose	winter flounder, *Pleuronectes americanus*	Grand Banks crude oil-contaminated sediments; exposed in winter	8-wk exposure to 0, 100, 300, 600, 1000, and 2200 µg/g total hydrocarbon concentration	(Khan 1995)
gonad	(see ovary or testis)				
intestine	see gastrointestinal tract				
kidney	increased numbers of melanomacrophage centers	Atlantic cod	Venezuelan or Hibernia crude oil, WSF in a flow-through seawater system	50-300 ppb (12-13 weeks)	(Khan and Kiceniuk 1984)
kidney	tubular vacuolization	Goldfish	outboard motor exhaust or toluene, dissolved in water for a continuous flow bioassay	200, 152, and 82 ppm leaded OME, or 10 and 5 ppm toluene (up to 30 d)	(Brenniman et al. 1979)
lens	(see eye)				
liver	decreased hepatocyte size	Atlantic cod	crude oil	long term (article not read)	(Khan et al. 1981), from (Fletcher et al. 1979)
liver	decreased hepatocellular glycogen, but increased G-6-PDH, lipid, and cholesterol; changes most severe in hepatocytes nearest afferent hepatic blood vessels	saltwater fish *Micropogon undulatus*	Southern Louisiana crude oil, WSF	5-10% WSF (1, 3, 7, 14, and 21 d)	(Eurell and Haensly 1981)
liver	*M. beryllina*: extensive hepatocellular lipid vacuolization, with slight necrosis of acinar and hepatic cells; *T. maculatus*: no histologic lesions	Tidewater silverside and the Hogchoker	Louisiana whole crude oil (WCO) poured directly into the aquaria, and its WSF	5 and 100 mg WCO/L; 5 and 50% WSF; (*M. beryllina*, 21-30 d) (*T. maculatus*, 38-60 d)	(Solangi and Overstreet 1982)
liver	hepatocytes with RER hyperplasia, decreased glycogen and lipid stores, and increased numbers of free ribosomes	Killifish	natural exposure in oil-contaminated water	unknown concentration, lifetime exposure	(Sabo et al. 1975)

Organ	Significant Lesions	Species	Type of petroleum. mode of exposure	Concentration (exposure period)	Reference
liver	formation of microvesicles (lipid?) in hepatocytes, decreased hepatocyte volume	Atlantic cod	Venezuelan or Hibernia crude oil, WSF in a flow-through seawater system	50-300 ppb (12-13 weeks)	(Khan and Kiceniuk 1984)
liver	histopathology and TEM: increased perisinusoidal collagen (sinusoidal fibrosis)	rainbow trout	Prudhoe Bay crude oil, feeding	17 mg crude oil/kg fish/d (13 months)	(Hawkes 1977)
liver	hepatocellular glycogen depletion, proliferation of ER and cochlear ribosomes; decreased weight gain after 75 d	rainbow trout	Prudhoe Bay crude oil, feeding	11 mg crude oil/fish/d (14 or 75 d)	(Hawkes 1977)
liver	histopathology: severe hepatocellular lipid vacuolization; oil-exposed fish ate less and failed to gain weight (no histologic lesions in spleen, kidney, intestine, fin, gills, and skin); TEM: SER hyperplasia and excess intracellular lipid	English sole *Parophrys vetulus*	Alaskan North Slope crude oil, via contaminated sediments	initial 700 µg oil/g dry sediment had decreased to 400 µg/g after 1 month exposure; 4 months total exposure	(McCain et al. 1978)
liver	no histopathology; biochemistry: increased liver weight due to increased concentrations of lipid and phospholipid; concentrations of DNA, protein, and sodium were decreased (evidence for hypertrophy rather than hyperplasia)	winter flounder, males only	Venezuelan crude oil, via contaminated sediments	approximately 3,000 µg oil\g dry sediment (4-5 months)	(Fletcher et al. 1982)
liver	nuclear pleomorphism prevalence greater in oiled than in unoiled sites in 1989, 1990, but not 1991	flathead sole	*Exxon Valdez* Oil Spill (March 24, 1989); crude oil, natural exposure)	unknown concentration (fish sampled in 1989)	(Armstrong et al. 1995)
liver	gross - livers were small and pale; histopathology - hepatocytes had decreased cytoplasm to nucleus ratio	Goldfish	outboard motor exhaust or toluene, dissolved in water for a continuous flow bioassay	200, 152, and 82 ppm leaded OME, or 10 and 5 ppm toluene (up to 30 d)	(Brenniman et al. 1979)
liver	decreased hepatocellular vacuolization (due to decreased glycogen and/or lipid)	Cutthroat trout	refined oil, dissolved in water	0 - 183 µg/L total oil (90 d)	(Woodward et al. 1983)

Organ	Significant Lesions	Species	Type of petroleum. mode of exposure	Concentration (exposure period)	Reference
liver	in exposed fish, decreased hepatocellular lipid (and/or glycogen ?) vacuolation was associated with increased concentration of macrophage centers; all differences were gone by the Feb & Jun 80 samples; increased frequency of hepatocellular necrosis in Dec 78 samples only	Plaice	*Amoco Cadiz* Oil Spill (March 16, 1978); crude oil, natural exposure	unknown concentration (fish sampled in Dec 78, Apr 79, Aug 79, Feb 80, & Jun 80)	(Haensly et al. 1982)
liver	decreased numbers of pigmented macrophage aggregates; in Pacific halibut only, lipidosis and coagulative hepatic necrosis (only 2 halibut were examined)	yellowfin sole rock sole Pacific halibut *Hippoglossus stenolepis*	Alaska North slope crude oil in sediments (laboratory exposure)	90-d exposure to sediments laden with 0, 1600-1800, and 4300-4700 µg oil/g sand or mud in unfiltered seawater	(Moles and Norcross 1998)
liver	exposure resulted in decreased numbers of macrophage aggregates (> 50 µg/g); liver hypertophy "followed a similar pattern"	winter flounder (males only)	Venezuelan crude oil in sediments (laboratory exposure)	4-month exposure to 0 to 500,000 µg oil/g sediment	(Payne and Fancey 1989)
liver	bile duct hyperplasia; depletion of hepatocellular glycogen and lipid	winter flounder	Grand Banks crude oil-contaminated sediments; exposed in winter	24-wk exposure to 2200 µg/g total hydrocarbons	(Khan 1995)
liver	1989 - hepatic necrosis (coagulative necrosis and single cell necrosis); no differences related to oil exposure after 1989	Pacific herring	*Exxon Valdez* Oil Spill (March 24, 1989); crude oil, natural exposure)	unknown concentration (fish sampled in Apr 1989, Oct 1990, Apr 1991, and Oct 1991)	(Marty et al. 1999)
liver	decreased numbers of periportal and perivascular leukocytes (evidence of immune suppression); hepatic necrosis was attributed to natural expression of viral hemorrhagic septicemia virus in oil-exposed fish	Pacific herring in spawning condition	weathered Alaska North Slope crude oil (laboratory exposure)	16-18 day exposure; initial total PAH concentrations of 0.03 (control) to 58.3 ppb	(Carls et al. 1998)
muscle	dystrophy of white muscle fibers	Atlantic cod	Venezuelan crude oil, WSF in a flow-through seawater system	50-300 ppb (12-13 weeks)	(Khan and Kiceniuk 1984)
nares	(see olfactory organ)				
olfactory organ	degeneration and necrosis of segments of the mucosa covering the olfactory rosette	Fathead minnow	JP-4 aviation fuel, WSF as a static exposure	25 and 50% WSF (= 5 and 10 ppm); sample at 6, 12, 48, and 72 h	(Latendresse and Fisher 1983)

Organ	Significant Lesions	Species	Type of petroleum. mode of exposure	Concentration (exposure period)	Reference
olfactory organ	hyperplasia of sustentacular cells of olfactory lamellae, necrosis of both neurosensory and sustentacular epithelium	Tidewater silverside and the Hogchoker	Louisiana whole crude oil (WCO) poured directly into the aquaria, and its WSF	5 and 100 mg WCO/L; 5 and 50% WSF; (*M. beryllina*, 21-30 d) (*T. maculatus*, 38-60 d)	(Solangi and Overstreet 1982)
ovary	increased ovary-somatic index; decreased reproductive success	Longhorn sculpin	Hibernia crude oil in sediment	1 L crude oil/45 kg washed sand (3-6 months); total hydrocarbons were 2-3 mg/g	(Khan 1991)
ovary	basophilic intracytoplasmic inclusions in oocytes; necrosis of follicles was enhanced after 8 months, resulting in complete degeneration of oocytes	eels (species not given)	*Amoco Cadiz* Oil Spill (March 16, 1978); crude oil, natural exposure	unknown concentration	(Lopez et al. 1981) from (Malins 1982)
ovary	mature follicles were either decreased in number or absent in fish from exposed sites (control fish had mature follicles); Jun 1980 samples from exposed site had increased frequency of atretic follicles	Plaice	*Amoco Cadiz* Oil Spill (March 16, 1978); crude oil, natural exposure	unknown concentration (fish sampled in Dec 78, Aug 79, Feb 80, and Jun 80)	(Stott et al. 1983)
pancreas	atrophy and necrosis of intrahepatic pancreatic nodules (considered the most oil-specific change)	Tidewater silverside and the Hogchoker	Louisiana whole crude oil (WCO) poured directly into the aquaria, and its WSF	5 and 100 mg WCO/L; 5 and 50% WSF; (*M. beryllina*, 21-30 d) (*T. maculatus*, 38-60 d)	(Solangi and Overstreet 1982)
peritoneal cavity	ascites with <7 d of exposure, tenacious exudate at 15-30 d exposure	Goldfish	outboard motor exhaust or toluene, dissolved in water for a continuous flow bioassay	200, 152, and 82 ppm leaded OME, or 10 and 5 ppm toluene (up to 30 d)	(Brenniman et al. 1979)
skin	inhibition of mucigenesis; inhibition of cellular proliferation and elongation; significantly decreased epidermal thickness and goblet cell frequency after 28 d exposure	Atlantic salmon *Salmo salar* sexually mature males	Venezuelan crude oil, WSF, continuous flow	0.9 ppm (14 and 28 d)	(Burton et al. 1985)
skin	after surgical removal of half of the caudal fin, regeneration was decreased in exposed fish	Gulf Coast Killifish *Fundulus grandis*	fuel oil or dispersant (BP1100X), intubated per os	0.0125 mL fuel oil or dispersant/g fish (28 d)	(Fingerman 1980)
skin	erosion of caudal fin; hemorrhage in underlying muscle, with accumulation of syncytial cells (macrophage giant cells)	Cutthroat trout	refined oil, dissolved in water	0 - 183 µg/L total oil (90 d)	(Woodward et al. 1983)

Organ	Significant Lesions	Species	Type of petroleum. mode of exposure	Concentration (exposure period)	Reference
skin	frequency of fish with fin and tail necrosis was increased in all samples from Apr 79 - Jun 80	Plaice	*Amoco Cadiz* Oil Spill (March 16, 1978); crude oil, natural exposure	unknown concentration (fish sampled in Dec 78, Apr 79, Aug 79, Feb 80, & Jun 80)	(Haensly et al. 1982)
skin	no gross lesions (e.g., no fin erosions); in static exposures, oiled fish had decreased survival, and epidermal thickness, cell dissociation, and goblet cell concentration were decreased; WSF in running water produced only the decreased epidermal dissociation	winter flounder	Venezuelan crude oil (WSF in running water, or static surface exposure)	concentration not stated (20-30 days)	(Burton et al. 1984)
skin	fin erosion (most severe in Pacific halibut)	yellowfin sole rock sole Pacific halibut	Alaska North slope crude oil in sediments (laboratory exposure)	90-d exposure to sediments laden with 0, 1600-1800, and 4300-4700 µg oil/g sand or mud in unfiltered seawater	(Moles and Norcross 1998)
spleen	increased numbers of macrophage aggregates	longhorn sculpin	Hibernia crude oil in sediment	1 L crude oil/45 kg washed sand (3-6 months); total hydrocarbons were 2-3 mg/g	(Khan 1991)
spleen	hemosiderin increased as a % of total sectional area scanned	longhorn sculpin	oil in sediment	fish sampled after 3, 6, and 12 months exposure (hydrocarbon concentration not stated)	(Khan and Nag 1993)
spleen	increased numbers of melanomacrophage centers; foci of necrosis (Hibernia crude only)	Atlantic cod	Venezuelan or Hibernia crude oil, WSF in a flow-through seawater system	50-300 ppb (12-13 weeks)	(Khan and Kiceniuk 1984)
spleen	hemosiderin increased as a % of total sectional area scanned (about 12% exposed vs. 1% reference, all 3 species)	yellowfin sole *Limanda aspersa*, quillback rockfish *Sebastes maliger*, and kelp greenling *Hexagrammos decagrammus*	*Exxon Valdez* Oil Spill (March 24, 1989); crude oil, natural exposure)	unknown concentration (fish sampled in July 1990)	(Khan and Nag 1993)
spleen	thrombosis was attributed to natural expression of viral hemorrhagic septicemia virus (occurred only in oil-exposed fish)	Pacific herring in spawning condition	weathered Alaska North Slope crude oil (laboratory exposure)	16-18 day exposure; initial total PAH concentrations of 0.03 (control) to 58.3 ppb	(Carls et al. 1998)
stomach	see gastrointestinal tract				

Organ	Significant Lesions	Species	Type of petroleum. mode of exposure	Concentration (exposure period)	Reference
testis	decreased testis-somatic index	Cunner	Venezuelan crude oil as a continuous flow surface slick	actual concentration was not measured (6 months)	(Payne et al. 1978)
testis	delayed spermatogenesis, intratubular multinucleate cells	Atlantic cod	Venezuelan or Hibernia crude oil, WSF in a flow-through seawater system	50-300 ppb (12-13 weeks)	(Khan and Kiceniuk 1984)

Table XI-2. Histopathological or ultrastructural lesions arising from exposing fish embryos or larvae to crude oil or petroleum components.

Organ	Significant lesions in Exposed Embryos/Larvae	Species	Type of petroleum or petroleum component/ mode of exposure	Concentration (exposure period)	Reference
brain	inter- and intracellular spaces were irregular and not membrane bound; eyes had no lesions	Pacific herring *Clupea pallasi*	Prudhoe Bay crude oil, WSF	0.68 mg/L (exposure began in late neurula or early tail bud stages and continued for 4, 8, 12, 24, 48 (TEM group), or 144 h, followed by transfer to clean water for hatch)	(Cameron and Smith 1980)
brain and eye	necrotic neurons in forebrain and neuronal layer of the retina; 27-d-old embryos had severely damaged ellipsoid and myoid regions of the receptor cell inner segments of retina (some with cytoplasmic vacuolation)	surf smelt *Hypomesus pretiosus*	seawater-accommodated fraction of Cook Inlet crude oil	54 or 113 ppb for 3 h/d beginning 6 d postfertilization; sample at 21- and 27-d postfertilization (just before hatch)	(Hawkes and Stehr 1982)
heart, et al.	lethargic cardiac and body movements, hypopigmentation, decreased hatch ≥ 1 ppt	killifish	drilling fluids; continuous static aqueous exposure, with daily renewal	10 ppt, 1 ppt, 100 ppm, 10 ppm, 1 ppm based on dilution of original drilling fluid (begin 1 min after fertilization, continue to hatch)	(Crawford and Gates 1981)
muscle, striated	mitochondria were 57% more numerous and 13% larger on their long axis; many mitochondria had disrupted internal membranes and cristae that created spaces of varying sizes within their frameworks	Pacific herring	Prudhoe Bay crude oil, WSF	0.68 mg/L (exposure began in late neurula or early tail bud stages and continued for 4, 8, 12, 24, 48 (TEM group), or 144 h, followed by transfer to clean water for hatch)	(Cameron and Smith 1980)
musculoskeletal, et al.	144 h exposure: all embryos died; 48 h exposure: bent spine, pericardial edema ("enlarged pericardial cavity"), retarded growth SEM: erosion of pectoral fins, failure of the jaw to fully differentiate, absence of the maxillary bone, and absence of branchiostegal membranes	Pacific herring	Prudhoe Bay crude oil, WSF	0.68 µg hydrocarbon/g H_2O (exposure began in late neurula or early tail bud stages and continued for 4, 8, 12, 24, 48 (SEM group), or 144 h, with renewal every 48 h, followed by transfer to clean water for hatch)	(Smith and Cameron 1979)
olfactory organ	SEM: degeneration of chemosensory cilia and loss of microridges that circumscribe the perimeter of the epithelial cells surrounding the olfactory organ	sand sole *Psettichthys melanostictus*	Prudhoe Bay crude oil WSF	approximately 800 ppb for 8 days	(Hawkes 1980)
peritoneal cavity	ascites (plus, decreased gastrointestinal food and decreased growth)	Pacific herring	*Exxon Valdez* Oil Spill (March 24, 1989); crude oil, natural exposure)	unknown concentration, PAH probably less than 1 ppb (fish sampled in May and Jun 1989)	(Marty et al. 1997b)

Organ	Significant lesions in Exposed Embryos/Larvae	Species	Type of petroleum or petroleum component/ mode of exposure	Concentration (exposure period)	Reference
peritoneal cavity	ascites and decreased growth	Pacific herring	Prudhoe Bay crude oil, oil-water dispersion	exposure from fertilization to hatch; 0.0 to 2.41 mg/L	(Marty et al. 1997b)
peritoneal cavity	ascites (and expression of cytochrome P450, premature emergence/swim-up)	pink salmon *Oncorhynchus gorbuscha*	weathered Prudhoe Bay crude oil	continuous exposure from fertilization to emergence/swim-up (significant effects as low as 4.4 µg PAH/L)	(Marty et al. 1997c)

CHAPTER 12 - Editorial Perspectives and Recommendations for Future Oil Spills

by Gary D. Marty and David E. Hinton

Histopathologic examination of the effects of the *Exxon Valdez* oil spill on fishes included 18 data sets, over 7,500 fish tissues, 5 progress reports, and this final summary report. The volume of the project makes in the largest study of its nature yet performed in relation to a single environmental episode.

While work on this contract was being conducted, major spills occurred in Kuwait (associated with the Gulf War), Spain, and off the coast of Scotland. Other major spills will occur. We can learn a lot about the process of response and damage assessment from our experiences with the *Exxon Valdez* oil spill, and the following perspectives from a histopathologic standpoint are offered.

We commend the Trustee Council for supporting fish histopathology as part of the damage assessment plan. Further, this study would not have been possible without the effort by biologists from the Alaska Department of Fish and Game (ADF&G) and the National Marine Fisheries Service (NMFS) who were committed to producing the most information possible from finite resources. Site selection and basic fisheries data such as fish age, weight, and length were critical in interpreting eventual histopathologic results.

Despite blind coding of samples, results of histopathologic analysis identified exposure history (i.e., oiled vs. reference) for every sample site involving Pacific herring adults (samples from 1989 and 1990), Dolly Varden char (samples from spring of 1990), and rockfish (samples from 1991). Exposure histories were not identified by histopathologic analysis for pink salmon larvae (1990 and 1991) or adults (1990), or for herring larvae (1989 and 1990); additional histopathologic analysis on these fish groups was not recommended and was not done. Additional histopathologic analysis on herring in spring 1991 and Dolly Varden Char in Fall of 1990 yielded results that could not clearly be attributed to oil; additional studies of the same nature were not recommended, although 50 herring were sampled in Fall of 1991 (no oil-related differences). Hence, for nearly every species group, we are confident that histopathologic analysis was extended significantly long after the spill to determine longterm effects of the spill.

Despite many positive comments, certain aspects of the study, from the standpoint of histopathologic analysis, need attention to improve upon results obtained herein. Many of the deficiencies of this study can be attributed to lack of organization and planning. Given the relatively common occurrence of major oil spills worldwide, response and damage assessment of fish resources would be improved by development of procedures **before** the next spill. It is likely that use of a trained team (e.g., biologist, chemist, and histopathologist) would pay for itself many times over when damage can be unequivocally established. What follows are recommendations and descriptive anecdotes illustrating the need for a prepared team.

In the weeks immediately after the spill, about 20 dead rockfish were brought to collection centers in Valdez and Cordova by commercial fishermen and concerned citizens. Of the 5 fish suitable for necropsy, all had oil metabolites in their bile (Hoffman et al., *Exxon Valdez* Oil Spill Symposium

Abstracts, 1993). Later, 11 of 30 live rockfish collected from contaminated sites had biochemical evidence of oil exposure, but none of the 13 rockfish from reference sites had evidence of hydrocarbon exposure. Because exposure does not in itself indicate injury, demonstration of injury would have required gross and histologic examination of tissues by a trained pathologist. At least 2 pathology-trained veterinarians from the University of California, Davis (Dr. Joseph Groff and Dr. Bruce Rideout), were in Valdez immediately after the spill, conducting necropsies on affected sea otters. They were aware that dead rockfish had been submitted. But despite Dr. Groff's extensive experience in fish pathology, he was told outright not to examine rockfish. Later, other veterinary pathologists examined more sea otters (Lipscomb et al. 1993), but not rockfish.

At least 2 major errors in judgment were made in 1989. First, it was assumed that ADF&G fish pathologists would coordinate sampling, but this never happened. In retrospect, they may not have been able to handle the volume of this project with available staff. A future response plan is needed that includes responsibilities for fish histopathology and funding to ensure that adequate expertise is available when needed. Second, it was assumed that fisheries biologists could do an adequate necropsy on sampled fish. This proved wrong. Certainly, with training, fisheries biologists could do an adequate job of collecting tissues and recording gross alterations. However, because of the need for rapid response to the spill, there was no time to learn new skills. Trained pathologists examined sea otters but not fish in 1989. As a result of lack of specific training or compliance with short-course methods (provided by D.E. Hinton in 1990), several groups of tissues were sampled poorly. The biggest problems were with dead (autolyzed) herring larvae in 1989, pink salmon tissues filled with fine beach sand in 1990, and herring tissues that had been removed from fixative for several hours and became desiccated. Although these tissues were mostly salvageable, precision in interpretation was lost.

The greatest disappointment in working on this project was the lack of adult fish to examine from 1989. A common argument against the significance of our findings (e.g., fish from oiled sites often had increased incidence or severity of lesions such as macrophage aggregates and hepatic lipidosis) is that "the lesions are not specific." Although this statement is true, it must be understood that no single lesion or suite of lesions are currently specific for oil exposure. Fortuitously, Adam Moles, NMFS, collected 40 adult herring in April 1989: 10 each from 2 oiled sites and 2 reference sites. The types of acute lesions in herring from oiled sites–particularly hemorrhage and multifocal, coagulative, hepatic necrosis–were the same as described in sea otters affected by EVOS (Lipscomb et al. 1993). Although these acute lesions also are nonspecific, possible causes such as reproductive status, age, or inanition can almost certainly be ruled out as causes. Unexpectedly, recent study has attributed hepatic necrosis in Pacific herring to expression of viral hemorrhagic septicemia virus (Carls et al. 1998). In short, acute lesions found immediately after the spill are more readily associated with oil exposure than are the chronic lesions used in most of our analyses. It seems likely that fish species other than Pacific herring had acute lesions, but they were not examined.

Rockfish are the only group of species for which funding for additional study was rescinded despite statistically and biologically significant lesion differences between rockfish from oiled and reference sites. Unlike the Dolly Varden char samples from fall 1990, where statistically significant oiled vs. reference differences did not seem biologically relevant, the lesions

contributing most to variability in 1991 rockfish samples–macrophage aggregates in the spleen and kidney–have clearly been associated with toxicant exposure and stress in many studies. In addition, yelloweye rockfish had several lesions (e.g., hepatic lipidosis, renal tubular epithelial vacuolation, and renal tubular necrosis) that occurred only in fish captured from oiled sites.

Why were these lesions more severe and in higher prevalence in rockfish from oiled sites in 1991, more than 2 years after the spill? Several explanations are possible: 1) ongoing low-grade hydrocarbon exposure; 2) oil-related environmental alterations such as decreased or altered food supply; 3) residual effects (i.e., "scars") of acute exposure in 1989; and/or 4) an aberration of site selection independent of oil exposure (Do different populations of yelloweye rockfish normally have abundant lipid in their livers?). Hydrocarbon samples from rockfish were negative in 1990: evidence against continued exposure to *Exxon Valdez* oil. No literature is available on the dynamics and duration of lesions in rockfish such as macrophage aggregates or hepatic fibrosis. Therefore, we were unable to determine whether the lesions resulted from ongoing environmental changes or were residual effects of previous exposure. Hepatic lipidosis seems more likely to be related to ongoing damage rather that represent residual effects of previous exposure. Determination of the significance of site selection would require additional sampling. In conclusion, with no evidence for alternative explanations for the observed lesion differences, we must conclude what the study was designed to show–that differences were more likely than not related to the spill. Clearly, the only way to answer many of these questions is to do additional sampling, and to expand the number of control sites. We recommended sampling in 1992, but funding was not approved. Funding was approved for study in 1993, but was later rescinded.

The decision not to support the additional studies was apparently made because "population" or "significant" effects were not demonstrated. The studies were generally designed to detect site differences; therefore, attempting to expand the results to speculate on population level effects was, by design, destined for failure. In retrospect, it is important to understand the types of information likely to be gained from histopathologic analysis of fish tissues after an oil spill:

4) Expected lesion prevalence in oiled fish - It is not reasonable to expect a lesion frequency of 100% in fish from oiled areas, compared with 0% in fish from reference areas. Even among sea otters visually classified as "severely oiled" at the rehabilitation centers in 1989, necropsy examination revealed that the lesion with the highest incidence–interstitial pulmonary emphysema–occurred in only 67% of affected individuals (Lipscomb et al. 1993). For fish samples, hepatic lipidosis in yelloweye rockfish sampled in 1991 had the clearest differences (oiled sites, 18 of 30, 60% vs. reference sites, 0 of 17). More commonly, though, fish lesions such as macrophage aggregates occurred at increased frequency or severity in fish from oiled sites than in fish from reference sites.

2) Expected lesion prevalence in reference fish- It is unlikely that fish from oiled sites would contain several types of lesions, but fish from reference sites would have no lesions. Some of the literature reports of histopathologic examination of fish after oil spills have reported an absence of lesions. Either these histopathologists were incompetent, or what they really meant was that they didn't find any lesions they could clearly attribute to hydrocarbon exposure. All wild fish populations have lesions, and these lesions can may be exacerbated by hydrocarbon exposure. Examples include macrophage aggregates which are normal in

every teleost species (Wolke 1992), lesions associated with active bacterial and viral infections, and lesions associated with parasites, some of which have been shown to increase after hydrocarbon exposure (Khan 1990). Specifically, some Pacific herring in Prince William Sound normally carry viral hemorrhagic septicemia virus, but the virus causes severe disease only when fish are stressed by something like spawning or an oil spill (Meyers et al. 1994, Marty et al. 1998). Many laboratory studies have described the effects of crude oil on fish tissues, but these studies cannot mimic the many interactions and secondary effects of an oil spill in the natural environment. Each species and oil spill will interact differently, but by careful site selection and follow-up study, significant lesions can be identified.

3) Importance of long-term study - Certain lesions were in high frequency only in fish from oiled sites, and other lesions were more severe in fish from oiled sites. This suggests that fish inhabiting oiled sites paid a greater price for their location. The definitive way to determine whether certain lesions were due or related to oil is to follow their resolution and repair with time after the spill. Those lesion frequencies which diminish would indicate strong episode-related causality.

4) Oil-related lesions are not specific - It is unlikely that fish from oiled sites would have specific lesions that would prove that they were exposed to crude oil. Although many studies have linked similar lesions with exposure to petroleum hydrocarbons, none of the resultant lesions were specific for oil exposure. Acute lesions are more likely linked to crude oil exposure than are chronic lesions such as increased macrophage aggregates, hence the need for sampling immediately after a spill.

It must also be remembered that the fish histopathologists were asked to identify differences that could not have been more than subtle. The pathologists who examined the sea otters knew they were dealing with animals that were known to have been exposed to oil, and many of the otters died as a result of that exposure or subsequent handling. By comparison, no fish known to have died as a result of oil exposure were examined histopathologically. What might the lesion incidence have been if only sick fish had been examined? With rockfish, we were asked to look for significant lesions differences in fish that had survived and were collected more than 2 years after the spill. Their survival is evidence that only subtle lesions could be expected.

Recommendations for Future Studies

Among all larvae and adult groups examined, adult yelloweye rockfish seemed to be the best indicator species for documenting oil-associated damage. Among lesions, macrophage aggregates were the best evidence for chronic exposure. In fact, the persistent nature of macrophage aggregates, and the propensity of rockfish to stay in the same rock bed for many years, makes these fish potentially superior to any avian or mammalian species for determining chronic effects of the spill.

It is important to point out deficiencies in past procedures; however, criticism is useful only if can be used constructively to improve response and damage assessment for the next major oil spill. We offer these suggestions regarding fish histopathology:

1) **A trained fish pathologist will be under contract to be available as needed for response and damage assessment work.** This "contract" would ideally be worked into the job description of a pathologist with ADF&G or NMFS, although an outside pathologist/group could be used. Part of the assigned pathologist's duties would be to submit a biannual literature review on recent information on the histopathologic response of fish to crude oil and petroleum hydrocarbons in general.

2) **A trained fish pathologist/technician will be on site for all necropsies** and tissue collection. In the rush to collect samples in the critical period immediately after a spill, there is no time to learn new skills. Field biologists have enough to do coordinating logistics of sampling (boats, planes, sample sites, etc.), and concerns of about proper necropsy techniques are better handled by a trained pathologist. Further, observations of a trained pathologist are usually mor acceptable for litigation purposes. This protocol is far different than what happened after the spill, where the fish histopathology contract did not get signed until February, 1991. The utility of having the pathologist on site, working closely with fishery biologists, has been realized in the ongoing long term study of disease in the Pacific herring population of Prince William Sound (e.g., Marty et al. 1998).

3) **Number of sample sites** - Sample a minimum of 2 oiled sites and 2 reference sites. Because all oil-related lesions are nonspecific, the only way lesions are associated with oil exposure is by increased incidence and/or severity in fish from oiled sites compared with fish from reference sites. Hence, examination only of fish from an oiled site provides almost no useful information. If only one reference and one oiled site are chosen, we are less able to determine if differences in significant lesions are a result of variables other than oil exposure. On the other hand, choosing too many sample sites, particularly if contamination of the "oiled" sites is not firmly established, might result in dilution of significant lesions and failure to demonstrate significant effects when, in fact, oil-related damage occurred.

4) **Sample size** - Given the complexity of living systems and their interaction with a dynamic environment, sample a minimum of 25 fish from each site. If major differences in sex are anticipated (e.g., pink salmon nearing spawning condition), then sample 25 fish of each sex from each site. If large variations in age are expected, as with rockfish, a sample size of 40 is more appropriate (important biomarkers such as macrophage aggregates normally increase with age). For demonstration of population-level effects samples size should be greater than 200 and probably should approach 300. For laboratory exposures of a homogeneous group of fish (e.g., same-age larvae) a sample size of 12 is adequate. For all laboratory exposures, an unexposed control group must be sampled each time an exposure group is sampled; these controls will account for any vagaries in culture conditions.

5) **Basic Fisheries Data** - Age, weight, and length are critical variables for interpreting histopathologic findings. Age, to properly interpret the significance of macrophage aggregates (Marty et al. 1999). Weight and length, along with a calculated condition factor, as evidence for sublethal damage. More **advanced fisheries data** such as incremental growth derived from analysis of otoliths, might provide important supporting evidence of oil-related damage.

6) **Species** - During the acute phase of a spill (e.g., the first 2 months), representatives of any species known to have been impacted by the spill should be examined. If freshly dead or sick fish are submitted, they should be subjected to complete necropsy examination. Although lesions resulting from crude oil exposure are nonspecific, acute lesions such as liver necrosis provide better evidence for toxicant exposure than do chronic lesions such as macrophage aggregates. Overall, quillback rockfish and yelloweye rockfish seemed to be the best species in this study for determining long-term effects of EVOS.

7) **Tissues** - All samples should include liver, kidney, gill, and spleen. During the acute phase of study, intestine, heart, exocrine pancreas, skin, muscle, and brain should also be sampled. Because most of the expense of histopathologic analysis goes to capturing the fish, the incremental cost of a more complete examination of each fish is minimal. Because viral hemorrhagic septicemia is an important cofactor in Pacific herring exposed to oil, and other unidentified viruses may be important in other fish species, viral and bacterial analyses of each sampled fish is also important.

8) **Fixative** - Although we have experience with several types of fixatives, some of which are superior for controlled laboratory studies, **10% Neutral Buffered Formalin** is the best choice for field samples. The contracted pathologist will provide the proper formula for mixing the formalin. The necessary ingredients for preparing the fixative should be stored at each response center and by the pathologist.

9) **Specimen Jars** - Glass jars are heavy and might break during shipment. We have had good success with 250-mL (= 8 oz.) Nalgene® jars.

10) **Background Data** - One argument against lesion significance in fish in this study is that background information on lesion incidence is lacking for most species. Because the cost for obtaining the samples far exceeds the cost of histopathologic analysis, I recommend that whenever sentinel species (e.g., rockfish, herring, pink salmon, and Dolly Varden char) are collected for any reason, tissues be fixed in 10% neutral buffered formalin. These tissues can either be examined immediately, or they can be trimmed, embedded in paraffin, and archived for years until the next spill warrants additional study.

In addition, selected species of aquatic organisms should be exposed to fingerprinted oil from each of the crude oil sources being transported in state waters. Fate of that oil in stomach contents and target organs, compared with resultant histopathologic lesions should be established. Information so gained would form the benchmark for future spill assessments. A bank of tissues collected over many years would provide a solid base of background information on the nature of important lesions in fish, thereby increasing the precision by which significant lesions can be identified.

Concluding Remarks

This report confers results of the single most comprehensive study of the effects of a crude oil spill on fish. Related studies on effects of the spill on Pacific herring and pink salmon will continue to add to our knowledge. Although fish populations were not devastated by EVOS, they clearly were damaged. Many of the weaknesses in response to EVOS and damage assessment after EVOS can be attributed to lack of planning. Given the knowledge we have gained from this spill, development of a Response Plan for future spills is strongly encouraged. A well-designed plan will include contingencies for histopathologic examination of potentially impacted fish species.

Project Acknowledgments

We thank the many ADFG and NMFS personnel that sampled and shipped tissues for these studies. We thank Kelly Henninger and Wendy Widmann for histotechnology. Diego Fridmann, David Ostrach, and Thomas Strella provided technical assistance.

Literature Cited and Annotated Bibliography of Fish Histopathology Related to Oil Spills

by Gary D. Marty

This reference list includes all literature cited in this report. In addition, it contains literature not cited, but relevant to the study of oil effects on living systems. Most of the articles that were read but not cited are annotated. Those not read are so marked. References that could not be located are marked "not found."

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Barry, M., and P. P. Yevich. 1975. The ecological, chemical and histopathological evaluation of an oil spill site. Part III. Histopathological studies. Marine Pollution Bulletin 6:171-173.

Number 2 fuel oil mixed with JP5 jet fuel was spilled near Searsport, Maine, in March 1971. In 1974, soft-shelled clams *Mya arenaria* had a high incidence of gonadal tumors. The area of highest impact correlated with the highest percent of tumors. Tumor incidence was highest in 1971 [i.e., relation of tumors to oil, independent of other pollutants, was not established].

Beck, L. S., D. I. Hepler, and K. L. Hansen. 1984. The acute toxicology of selected petroleum hydrocarbons. Pages 1-16 *in* M. A. Mehlman, Editor Applied Toxicology of Petroleum Hydrocarbons, Advances in Modern Environmental Toxicology. Volume VI. Princeton Scientific Publishing, Princeton, New Jersey.

No histopathology. Mammalian study. Determined the LD50 of 19 selected petroleum hydrocarbons (no crude oil) in rabbits, Guinea pigs, and rats. Number 6 heavy fuel oild was most toxic (LD50 = 4.7 g/kg). Oral exposure caused diarrhea. Gross impressions included hemorrhagic enteritis, gastrointestinal tympany, and pneumonia with abscessation.

Blanton, W. G., and M. C. Robinson. 1973. Some acute effects of low-boiling petroleum fractions on the cellular structures of fish gills under field conditions. Center for Wetland Resources. Louisiana State Univ., Baton Rouge, LA. Pub. No. LSU-SG-73-01.

Blazer, V. S., R. E. Wolke, J. Brown, and C. A. Powell. 1987. Piscine macrophage aggregate parameters as health monitors: effect of age, sex, relative weight, season and site quality in largemouth bass (*Micropterus salmoides*). Aquatic Toxicology 10:199-215.

Brannon, E. L., L. L. Moulton, L. G. Gilbertson, A. W. Maki, and J. R. Skalski. 1995. An assessment of oil-spill effects on pink salmon populations following the *Exxon Valdez* oil spill--part 1: early life history. Pages 548-584 *in* P. G. Wells, J. N. Butler, and J. S. Hughes, Editors. *Exxon Valdez* oil spill: fate and effects in Alaskan waters, ASTM STP 1219. Special Technical Publication 1219. American Society for Testing and Materials, Philadelphia.

Brenniman, G. R., M. R. Anver, R. Hartung, and S. H. Rosenberg. 1979. Effects of outboard motor exhaust emissions on goldfish (*Carassius auratus*). Journal of Environmental Pathology and Toxicology 2:1267-1281.

Britvic, S., D. Lucic, and B. Kurelec. 1993. Bile fluorescence and some early biological effects in fish as indicators of pollution of xenobiotics. Environmental Toxicology and Chemistry 12:765-773.

Brown, C. L., and C. J. George. 1985. Age-dependent accumulation of macrophage aggregates in the yellow perch, *Perca flavescens* (Mitchill). Journal of Fish Diseases 8:135-138.

Brown, E. D., T. T. Baker, J. E. Hose, R. M. Kocan, G. D. Marty, M. D. McGurk, B. L. Norcross, and J. Short. 1996. Injury to the early life history stages of Pacific herring in Prince William Sound after the *Exxon Valdez* Oil Spill. American Fisheries Society Symposium 18:448-462.

Bue, B. G., S. Sharr, S. D. Moffitt, and A. K. Craig. 1996. Effects of the *Exxon Valdez* oil spill on pink salmon embryos and preemergent fry. American Fisheries Society Symposium 18:619-627.

Burton, D., M. P. Burton, and D. R. Idler. 1984. Epidermal condition in post-spawned winter flounder, *Pseudopleuronectes americanus* (Walbaum), maintained in the laboratory and after exposure to crude petroleum. Journal of Fish Biology 25:593-606.

Burton, D., M. P. Burton, B. Truscott, and D. R. Idler. 1985. Epidermal cellular proliferation and differentiation in sexually mature male *Salmo salar* with androgen levels depressed by oil. Proceedings of the Royal Society of London (Biology) 225:121-128.

Burton, D., and G. L. Fletcher. 1983. Seasonal changes in the epidermis of the winter flounder (*Pseudopleuronectes americanus*). Journal of the Marine Biological Association of the United Kingdom 63:273-287. **Not Read**

Cada, G. F., and M. Kenna. 1985. Effectiveness of hydrotreatment in reducing the toxicity of a coal liquefaction product to juvenile channel catfish. Bulletin of Environmental Contamination and Toxicology 34:746-753.

No histopathology. Coal-derived oils contain many toxic products (e.g., phenols and anilines) that are readily soluble in water; hence, they are 2x more toxic/potent than petroleum oils. Calculated a 96h LC50 in juvenile channel catfish.

Caldwell, C. A. 1997. Aromatic hydrocarbon pathology in fish following a large spill into the Nemadji River, Wisconsin, USA. Bulletin of Environmental Contamination and Toxicology 58:574-581.

Cameron, J. A., and R. L. Smith. 1980. Ultrastructural effects of crude oil on early life stages of Pacific herring. Transactions of the American Fisheries Society 109:224-228.

Carls, M. G. 1987. Effects of dietary and water-borne oil exposure on larval Pacific herring (*Clupea harengus pallasi*). Marine Environmental Research 22:253-270.

No histopathology. Hering larvae were exposed directly to WSF or indirectly via oil-comtaminated prey (OCP) for 28d. WSF decreased feeding and growth, OCP decreased survival but survivors were normal. Concluded that prey were not a major source of hydrocarbon toxicity to herring.

Carls, M. G., L. Holland, M. Larsen, J. L. Lum, D. G. Mortensen, S. Y. Wang, and A. C. Wertheimer. 1996a. Growth, feeding, and survival of pink salmon fry exposed to food contaminated with crude oil. American Fisheries Society Symposium 18:608-618.

Carls, M. G., A. C. Wertheimer, J. W. Short, R. M. Smolowitz, and J. J. Stegeman. 1996b. Contamination of juvenile pink and chum salmon by hydrocarbons in Prince William Sound after the *Exxon Valdez* oil spill. American Fisheries Society Symposium 18:593-607.

Carls, M. G., G. D. Marty, T. R. Meyers, R. E. Thomas, and S. D. Rice. 1998. Expression of viral hemorrhagic septicemia virus in pre-spawning Pacific herring (*Clupea pallasi*) exposed to weathered crude oil. Canadian Journal of Fisheries and Aquatic Sciences 55:1-10.

Celewycz, A. G., and A. C. Wertheimer. 1996. Prey availability to juvenile salmon after the *Exxon Valdez* oil spill. American Fisheries Society Symposium 18:564-577.

Collier, T. K., and U. Varanasi. 1991. Hepatic activities of xenobiotic metabolizing enzymes and biliary levels of xenobiotics in English sole (*Parophrys vetulus*) exposed to environmental contaminants. Archives of Environmental Contamination and Toxicology 20:462-473.

Cotran, R. S., V. Kumar, and S. L. Robbins. 1989. Pathologic Basis of Disease. (4th edition), W.B. Saunders, Philadelphia.

Couch, J. A. 1975. Histopathological effects of pesticides and related chemicals on the livers of fishes. Pages 559-584 *in* W. E. Ribelin, and G. Migaki, Editors. Pathology of Fishes. University of Wisconsin Press, Madison, Wisconsin. **Not Read**

Couillard, C. M., and P. V. Hodson. 1996. Pigmented macrophage aggregates: a toxic response in fish exposed to bleached-kraft mill effluent? Environmental Toxicology and Chemistry 15:1844-1854.

Cox, G. V., A. Barnett, J. R. Gould, K. G. Hay, J. Hirota, C. D. McAuliffe, and A. D. Michael. 1979. Oil spill studies: strategies and techniques. Journal of Environmental Pathology and Toxicology 3:1-148.

No histopathology; review article; oil spill study methods. Recommendations for statistical analysis include multivariate analysis and principal components analysis (PCA). PCA "determines which of the independent variables (X) are most important in the prediction of the dependent variable (Y) in the sense that they account for most of the variability found in Y."

If "the spilled oil is difficult to quantify among other hydrocarbon sources, then the contribution of the spilled oil to possible adverse affects is low." Benzene and toluene comprise 70-85% of the total dissolved aromatics. All natural waters tested have hydrocarbon using bacteria, yeasts, and fungi, and numbers are especially high in areas of chronic spills and natural seeps. Recommends use of sealed vials for invertebrate bioassays. For fish, "obvious and measurable mortalities of large mobile species are extremely rare..."

Crawford, R. B., and J. D. Gates. 1981. Effects of a drilling fluid on the development of a teleost and an echinoderm. Bulletin of Environmental Contamination and Toxicology 26:207-212.

No histopathology. Studied *Fundulus heteroclitus* (begin 1 min after fertilization, continuous exposure through embryonic development) and the sand dollar *Echinarachnius parma*. *F. heteroclitus* was unaffected early, but developments was slowed by d 7 (hypopigmentation, lethargic cardiac and body movements); percent hatch was decreased at 1 ppt. Those that hatched appeared normal.

DiMichele, L., and M. H. Taylor. 1978. Histopathological and physiological responses of *Fundulus heteroclitus* to naphthalene exposure. Journal of the Fisheries Research Board of Canada 35:1060-1066.

Dushkina, L. A. 1973. Influence of salinity on eggs, sperm and larvae of low-vertebral herring reproducing in the coastal waters of the Soviet Union. Marine Biology 19:210-223.

Ehrenberg, A. S. C. 1975. Data reduction; analysing and interpreting statistical data. Wiley, New York.

Engelhardt, F. R., M. P. Wong, and M. E. Duey. 1981. Hydromineral balance and gill morphology in rainbow trout, *Salmo gairdneri*, acclimated to fresh and sea water, as affected by petroleum exposure. Aquatic Toxicology 1:175-186.

Ernst, V. V., J. M. Neff, and J. W. Anderson. 1977. The effects of the water-soluble fractions of No. 2 fuel oil on the early development of the estuarine fish, *Fundulus grandis* Baird and Girard. Environmental Pollution 14:25-35. **Not Read**

Eurell, J. A. C., and W. E. Haensly. 1981. The effects of exposure to water soluble fractions of crude oil on selected histochemical parameters of the liver of the Atlantic croaker, *Micropogon undulatus* L. Journal of Fish Diseases 4:187-194.

Fair, P. H., and A. R. Fortner. 1987. Effect of ingested benzo[a]pyrene and cadmium on tissue accumulation, hydroxylase activity, and intestinal morphology of the black sea bass *Centropristis striata*. Environmental Research 42:185-195.

Fingerman, S. W. 1980. Differences in the effects of fuel oil, an oil dispersant, and three polychlorinated biphenyls on fin regeneration in the Gulf Coast killifish, *Fundulus grandis*. Bulletin of Environmental Contamination and Toxicology 25:234-240.

Fletcher, G. L., J. W. Kiceniuk, M. J. King, and J. F. Payne. 1979. Reduction of blood plasma copper concentrations in a marine fish following a six month exposure to crude oil. Bulletin of Environmental Contamination and Toxicology 22:548-551.

No histopathology; blood/serum. Continuously exposed cunner *Tautogolabrus adspersus* to a surface slick of Venezuelan crude oil for 6 months. Plasma copper and chloride were decreased in oil-exposed fish.

Fletcher, G. L., M. J. King, J. W. Kiceniuk, and R. F. Addison. 1982. Liver hypertrophy in winter flounder following exposure to experimentally oiled sediments. Comparative Biochemistry and Physiology, Part C 73:457-462.

No histopathology; Venezuelan crude oil; sediment exposure; 4-5 months duration. Oil exposure resulted in significant increases in liver weight; affected livers had decreased concentrations of DNA, protein, sodium, and zinc, with increased concentrations of lipid and phospholipid. They concluded that increased phospholipid indicated ER hyperplasia rather than fatty change. Khan et al. (1981) had morphologic evidence of decreased hepatocyte size in oil treated group.

Gardner, G. R. 1975. Chemically induced lesions in estuarine or marine teleosts. Pages 657-694 *in* W. C. Ribelin, and G. Migaki, Editors. The Pathology of Fishes. The University of Wisconsin Press, Madison, Wisconsin.

Gardner, G. R., P. P. Yevich, and P. F. Rogerson. 1975. Morphological anomalies in adult oysters, scallop, and Atlantic silversides exposed to waste motor oil. Pages 473-477 *in* Proceedings of the 1975 Conference on Prevention and Control of Oil Pollution.

Gardner, G. R., P. P. Yevich, A. R. Malcolm, R. J. Pruell, P. F. Rogerson, J. Heltshe, T. C. Lee, and A. Senecal. 1987. Carcinogenic effects of Black Rock Harbor sediment on molluscs

and fish. Final Report to the National Cancer Institute NCI/EPA Collaborative Program on Environmental Cancer.

Gardner, G. R., and R. J. Pruell. 1987. Quincy Bay Study, Boston Harbor: A histopathological and chemical assessment of winter flounder, lobster and soft-shelled clam indigenous to Quincy Bay, Boston Harbor and an in situ evaluation of oysters including sediment (surface and cores) chemistry U.S. EPA Report, Region I, Boston, MA.

Gardner, G. R., S. J. Benyi, J. F. Heltshe, and J. Rosen. 1989a. Pigment localization in lymphoid organs of the winter flounder (*Pseudopleuronectes americanus*) in relation to contaminated sediment. Society of Environmental Toxicology and Chemistry. Proceedings of 10th Annual Meeting, Toronto, November 1989

Gardner, G. R., R. J. Pruell, and L. C. Folmar. 1989b. A comparison of both neoplastic and non-neoplastic disorders in winter flounder (*Pseudopleuronectes americanus*) from eight areas in New England. Marine Environmental Research 28:393-397.

George, S. G., J. Wright, and J. Conroy. 1995. Temporal studies of the impact of the Braer oilspill on inshore feral fish from Shetland, Scotland. Archives of Environmental Contamination and Toxicology 29:530-534.

Grahl-Nielsen, O., T. Neppelberg, K. H. Palmork, K. Westrheim, and S. Wilhelmsen. 1976. The Drupa oil spill, investigation concerning oil, water and fish. International Council for Exploration of the Sea [ICES] C.M. 1976/E:34:1-18.

Greve, P. A. 1971. Chemical wastes in the sea: new forms of marine pollution. Science 173:1021-1022. **Not Read**

Grizzle, J. M. 1986. Lesions in fishes captured near drilling platforms in the Gulf of Mexico. Marine Environmental Research 18:267-276.

Actual type of exposure was unknown, but fish near oil drilling platforms had hepatomegaly, gill lesions (branchial edema, telangiectasis, hyperplasia, and necrosis), and hepatic fatty change.

Groff, J. M., D. E. Hinton, T. S. McDowell, and R. P. Hedrick. 1992. Progression and resolution of megalocytic hepatopathy with exocrine pancreatic metaplasia in a population of cultured juvenile striped bass *Morone saxatilis*. Diseases of Aquatic Organisms 13:189-202.

Gruger, E. H., Jr., M. M. Wekell, P. T. Numoto, and D. R. Craddock. 1977. Induction of hepatic aryl hydrocarbon hydroxylase in salmon exposed to petroleum dissolved in seawater and to petroleum and polychlorinated biphenyls, separate and together, in food. Bulletin of Environmental Contamination and Toxicology 17:512-520. **Not Read**

Guiney, P. D., R. M. Smolowitz, R. M. Peterson, and J. J. Stegeman. 1997. Correlation of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin induction of cytochrome P4501A in vascular endothelium with toxicity in early Life stages of lake trout. Toxicology and Applied Pharmacology 143:256-273.

Haensly, W. E., J. M. Neff, J. R. Sharp, A. C. Morris, M. F. Bedgood, and P. D. Boem. 1982. Histopathology of *Pleuronectes platessa* L. from Aber Wrac'h and Aber Benoit, Brittany, France: long-term effects of the Amoco Cadiz crude oil spill. Journal of Fish Diseases 5:365-391.

Haux, C., A. Larsson, U. Lidman, L. Foerlin, T. Hansson, and M.-J. Johansson-Sjoebeck. 1982. Sublethal physiological effects of chlorinated paraffins on the flounder, *Platichthys flesus* L. Ecotoxicology and Environmental Safety 6:49-59. **Not Read**

Hawkes, J. W. 1977. The effects of petroleum hydrocarbon exposure on the structure of fish tissues. Pages 115-128 *in* D. A. Wolfe, Editor Fate and Effects of Petroleum Hydrocarbons in Marine Organisms and Ecosystems, Proceedings. Pergamon Press, New York.

Hawkes, J. W. 1980. The effects of xenobiotics on fish tissues: morphological studies. Federation Proceedings 39:3230-3236.

Hawkes, J. W., E. H. Gruger, Jr., and O. P. Olson. 1980. Effects of petroleum hydrocarbons and chlorinated biphenyls on the morphology of the intestines of chinook salmon (*Oncorhynchus tshawytscha*). Environmental Research 23:149-161.

Hawkes, J. W., and C. M. Stehr. 1982. Cytopathology of the brain and retina of embryonic surf smelt (*Hypomesus pretiosus*) exposed to crude oil. Environmental Research 27:164-178.

Hay, D. E. 1982. Fixation shrinkage of herring larvae: effects of salinity, formalin concentration, and other factors. Canadian Journal of Fisheries and Aquatic Sciences 39:1138-1143.

No histopathology. Young herring larvae have greater water content that older larvae. In 2-5% formalin, shrinkage (due to osmotic water loss) increased from less than 2% at low salinities to about 10% shrinkage in seawater formalin. Buffering agents and starvation had no effect on shrinkage. Shrinkage increased with handling, time in fixative, and time after death before fixation.

Hedtke, S. F., and F. A. Puglisi. 1980. Effects of waste oil on the suvival and reproduction of the American flagfish *Jordanella floridae*. Canadian Journal of Fisheries and Aquatic Sciences 37:757-764.

Hedtke, S. F., and F. A. Puglisi. 1982. Short-term toxicity of five oils to four freshwater species. Archives of Environmental Contamination and Toxicology 11:425-430.

No histopathology; LC50s determined for: waste oil (American flagfish *Jordanella floridae*); #1 fuel oil (fathead minnow *Pimephales promelas*); #2 fuel oil (wood frog larvae *Rana sylvatica*); and mixed blend and Lloydminister crude oil (spotted salamander *Ambystoma maculatum*)

Hendricks, J. D., R. O. Sinnhuber, M. C. Henderson, and D. R. Buhler. 1981. Liver and kidney pathology in rainbow trout (*Salmo gairdneri)* exposed to dietary pyrrolizidine (Senecio) alkaloids. Experimental Molecular Pathology 35:170-183.

Herraez, M. P., and A. G. Zapata. 1986. Structure and function of the melano-macrophage centres of the goldfish *Carassius auratus*. Veterinary Immunology Immunopathology 12:117-126.

Hinton, D. E., J. A. Couch, S. J. Teh, and L. A. Courtney. 1988. Cytological changes during progression of neoplasia in selected fish species. Aquatic Toxicology 11:77-112.

Hinton, D. E., P. C. Baumann, G. R. Gardner, W. E. Hawkins, J. D. Hendricks, R. A. Murchelano, and M. S. Okihiro. 1992. Histopathological biomarkers. Pages 155-209 *in* R. J. Huggett, R. A. Kimerle, P. M. Mehrle, and H. L. Bergman, Editors. Biomarkers: Biochemical, Physiological, and Histological Markers of Anthropogenic Stress. Lewis Publishers, Boca Raton.

Review article, biomarkers of toxicity.

Hinton, D. E., J. A. Couch, S. J. Teh, and L. A. Courtney. 1988. Cytological changes during progression of neoplasia in selected fish species. Aquatic Toxicology 11:77-112.

Hinton, D. E., and D. J. Laurén. 1990. Liver structural alterations accompanying chronic toxicity in fishes: potential biomarkers of exposure. Pages 17-57 *in* J. F. McCarthy, and L. R. Shugart, Editors. Biomarkers of Environmental Contamination. Lewis Publishers, Boca Raton, Florida.

Review article, biomarkers of exposure in the liver.

Hodgins, H. O., W. D. Gronlund, J. L. Mighill, J. W. Hawkes, and P. A. Robisch. 1977a. Effects of crude oil on trout reproduction. Pages 143-150 *in* D. A. Wolfe, Editor Fate and Effects of Petroleum Hydrocarbons in Marine Ecosystems and Organisms. Pergamon Press, Toronto. **Not Read**

Hodgins, H. O., B. B. McCain, and J. W. Hawkes. 1977b. Marine fish and invertebrate diseases, host disease resistance, and pathological effects of petroleum. Pages 95-173 *in* D. C. Malins, Editor Effects of Petroleum on Arctic and Subarctic Marine Environments and Organisms. Volume 2. Academic Press, New York.

Review article. Petroleum, petroleum products, and petroleum-associated metals are implicated in neoplasia (several types of studies cited), but no clear relationship.

Some components of crude oil (e.g., benzene) are oxidized to phenol in the liver (Brocksen and Bailey 1973). Phenol caused epithelial cell hyperplasia (Waluga 1966). Coal tar and crude oil caused alterations in pigment inclusions in the liver (Vishnevetskii 1961). Phenol caused increased lipofuscin and hemosiderin, but no change in bilirubin (Waluga 1966).

Petroleum "may have considerable impact on diseases of marine animals... potential for suppressing immune responses and disease resistance."

Hollister, T. A., G. S. Ward, and P. R. Parrish. 1980. Acute toxicity of a #6 fuel oil to marine organisms. Bulletin of Environmental Contamination and Toxicology 24:656-661. **Not Read**

Hose, J. E. 1998. Field applications of the piscine anaphase aberration test: lessons from the Exxon Valdez oil spill. Mutation Research 399:167-178.

Hose, J. E., M. D. McGurk, G. D. Marty, D. E. Hinton, E. D. Brown, and T. T. Baker. 1996. Sublethal effects of the *Exxon Valdez* oil spill on herring embryos and larvae: morphological, cytogenetic, and histopathological assessments, 1989-1991. Canadian Journal of Fisheries and Aquatic Sciences 53:2355-2365.

Hyde, D. M., T. E. King, Jr., T. McDermott, J. A. Waldron, Jr., T. V. Colby, W. M. Thurlbeck, A. Flint, L. Ackerson, and R. M. Cherniack. 1992a. Idiopathic pulmonary fibrosis. Quantitative assessment of lung pathology: comparison of a semiquantitative and a morphometric histopathologic scoring system. The American Review of Respiratory Disease 146:1042-1047.

Jackivicz, T. J., and L. N. Kuzminski. 1973. The effects of the interaction of outboard motors with the aquatic environment--a review. Environmental Research 6:436-454. **Not Read**

Jimenez, B. D., and J. J. Stegeman. 1990. Detoxication enzymes as indicators of environmental stress on fish. American Fisheries Society Symposium 8:67-79.

Johnson, P. A., S. D. Rice, M. M. Babcock, and (compilers). 1992. Impacts of oil pollution and Prince William Sound studies: Bibliography of 1960-1991 publications and reports, Auke Bay Laboratory. U.S. Department of Commerce. NOAA Tech. Memo. NMFS-AFSC-3.

Johnson, R. A., and D. W. Wichern. 1992. Applied multivariate statistical analysis. (3rd ed.), Prentice Hall, Englewood Cliffs, New Jersey.

Keizer, P. D., T. P. Ahern, J. Dale, and J. H. Vandermeulen. 1978. Residues of bunker C oil in Chedabucto Bay, Nova Scotia, 6 years after the Arrow spill. Journal of the Fisheries Research Board of Canada 35:528-535.

No histopathology; persistent oil. The Arrow dumped about 70,000 barrels of Bunker C fuel oil in February 1970. Concentrations of total oil in the water column were as high as 100 ppb in May 1970, but had dropped to a background level of <2 ppb by

April 1971. After 6 years (in 1976), oil still remained in the intertidal segments, sometimes heavy in a "pavement-like" consistency. In 1976, concentrations of hydrocarbons reaching the water column were far below levels considered toxic to benthic organisms.

Kent, M. L., M. S. Myers, D. E. Hinton, W. D. Eaton, and R. A. Elston. 1988. Suspected toxicopathic hepatic necrosis and megalocytosis in pen-reared Atlantic Salmon *Salmo salar* in Puget Sound, Washington, USA. Diseases of Aquatic Organisms 49:91-100.

Kent, M. L., S. C. Dawe, S. St. Hilaire, and R. J. Andersen. 1996. Effects of feeding rate, seawater entry, and exposure to natural biota on the severity of net-pen liver disease among pen-reared Atlantic salmon. Progressive Fish-Culturist 58:43-46.

Khan, R. A. 1987a. Crude oil and parasites of fish. Parasitology Today 3:99-100.

Khan, R. A. 1987b. Effects of chronic exposure to petroleum hydrocarbons on two species of marine fish infected with a hemoprotozoan, *Trypanosoma murmanensis*. Canadian Journal of Zoology 65:2703-2709.

Khan, R. A. 1990. Parasitism in marine fish after chronic exposure to petroleum hydrocarbons in the laboratory and to the Exxon Valdez oil spill. Bulletin of Environmental Contamination and Toxicology 44:759-763.

Khan, R. A. 1991a. Effect of oil-contaminated sediment on the longhorn sculpin (*Myoxocephalus octodecemspinosus*) following chronic exposure. Bulletin of Environmental Contamination and Toxicology 47:63-69.

Khan, R. A. 1991b. Influence of concurrent exposure to crude oil and infection with *Trypanosoma murmanensis* (Protozoa: Mastigophora) on mortality in winter founder, *Pseudopleuronectes americanus*. Canadian Journal of Zoology 69:876-880.

Khan, R. A. 1995. Histopathology in winter flounder, *Pleuronectes americanus*, following chronic exposure to crude oil. Bulletin of Environmental Contamination and Toxicology 54:297-301.

Khan, R. A., and J. Kiceniuk. 1983. Effects of crude oils on the gastrointestinal parasites of two species of marine fish. Journal of Wildlife Diseases 19:253-258.

Khan, R. A., and J. Kiceniuk. 1984. Histopathological effects of crude oil on Atlantic cod following chronic exposure. Canadian Journal of Zoology 62:2038-2043.

Khan, R. A., and J. W. Kiceniuk. 1988. Effect of petroleum aromatic hydrocarbons on monogeneids parasitizing Atlantic cod, *Gadus morhua* L. Bulletin of Environmental Contamination and Toxicology 41:94-100.

Khan, R. A., J. W. Kiceniuk, M. Dawe, and U. Williams. 1981. Long term effects of crude oil on Atlantic cod. International Council for Exploration of the Sea [ICES] C.M. 1981/E:40. **Not read, Not found**

Khan, R. A., and K. Nag. 1993. Estimation of hemosiderosis in seabirds and fish exposed to petroleum. Bulletin of Environmental Contamination and Toxicology 50:125-131.

Kiceniuk, J. W., G. L. Fletcher, and R. Misra. 1980. Physiological and morphological changes in a cold torpid marine fish upon acute exposure to petroleum. Bulletin of Environmental Contamination and Toxicology 24:313-319.

Kiceniuk, J. W., and R. A. Khan. 1987. Effect of petroleum hydrocarbons on Atlantic cod, *Gadus morhua*, following chronic exposure. Canadian Journal of Zoology 65:490-494.

Kiceniuk, J. W., R. A. Khan, M. Dawe, and U. Williams. 1982. Examination of interaction of trypanosome infection and crude oil exposure on hematology of the longhorn sculpin (*Myoxocephalus octodecemspinosus*). Bulletin of Environmental Contamination and Toxicology 28:435-438. **Not Read**

Kocan, R. M., J. E. Hose, E. D. Brown, and T. T. Baker. 1996. Pacific herring (*Clupea pallasi*) embryo sensitivity to Prudhoe Bay petroleum hydrocarbons: laboratory evaluation and in situ exposure at oiled and unoiled sites in Prince William Sound. Canadian Journal of Fisheries and Aquatic Sciences 53:2366-2375.

Kocan, R. M., G. D. Marty, M. S. Okihiro, E. D. Brown, and T. T. Baker. 1996. Reproductive success and histopathology of individual Prince William Sound herring 3 years after the *Exxon Valdez* oil spill. Canadian Journal of Fisheries and Aquatic Sciences 53:2388-2393.

Kuhnhold, W. W. 1978. Effects of the water soluble fraction of a Venezuelan heavy fuel oil (No. 6) on cod eggs and larvae. Pages 126-130 *in* In the Wake of the Argo Merchant. Center for Ocean Management Studies, University of Rhode Island.

No histopathology; cod (*Gadus morhua*). Eggs (0.5-, 3-, and 7-d-old for 15.5-, 13-, and 9-d exposures) and 8-d-old larvae were exposed to the WSF of Bunker C oil in static tests and open jars. The EC50 for viable hatch was about 25 ppm (0.5-d-old), 35 ppm (3-d-old), and 175 ppb (7-d-old); the EC50 for total hatch was about 5 times this amount. Embryonic heartrate was unaffected if <100ppb, but heart development was decreased at concentrations >100ppb. In larvae, the apical part of the primordial fin fold was abnormally developed at higher concentrations.

Latendresse, J. R., II, and J. W. Fisher. 1983. Histopathologic effects of JP-4 aviation fuel on fathead minnows (*Pimephales promelas*). Bulletin of Environmental Contamination and Toxicology 30:536-543.

Lebsack, M. E., A. D. Anderson, K. F. Nelson, and D. S. Farrier. 1980. Sublethal effects of an in situ oil shale retort water on rainbow trout. Toxicology and Applied Pharmacology 54:462-468.

No histopathology; serum/blood chemistry. A 96-h exposure to 0.3% Omega-9 oil shale retort water (=70% of LC50) decreased PCV, hemoglobin concentration, plasma alkaline phosphatase and protein, and caused a three-fold increase in plasma ammonia levels.

Lee, R. F., and D. S. Page. 1997. Petroleum hydrocarbons and their effects in subtidal regions after major oil spills. Marine Pollution Bulletin 34:928-940.

Leighton, F. A. 1986. Clinical, gross, and histologic findings in herring gulls and Atlantic puffins that ingested Prudhoe Bay crude oil. Veterinary Pathology 23:254-263.

Bird study; feeding experiment. Orally dosed 0-20 mL Prudhoe Bay crude oil/kg body wt/d to herring gull and Atlantic puffin nestlings for 5-7 consecutive days. Clinical signs and lesions occurred only in birds given □10 mL oil/kg bw/d. The primary target of oil toxicity was the peripheral RBC: a Heinz-body hemolytic anemia. Some lesions were those secondary to hemolytic disease: phagocytosis of degenerative RBCs in liver and spleen, hemoglobin resorption droplets in renal proximal tubules, and erythroid hyperplasia in bone marrow. Other lesions were nonspecific reactions to stress: lymphocyte depletion in primary lymphoid tissues, increased heterophil:lymphocyte ratio in peripheral blood, lipid depletion and necrosis in adrenal steroidogenic cells, etc.. Liver lesions included multifocal hepatic necrosis and necrosis of individual hepatocytes. Cites 6 other bird studies that describe lesions after oil exposure (e.g., enteritis with and without necrosis, and hepatic lipidosis).

Linden, O. 1976. The influence of crude oil and mixtures of crude oil/dispersants on the ontogenic development of the Baltic herring, *Clupea harengus membras* L. Ambio 5:136-140.

Linden, O. 1978. Biological effects of oil on early development of Baltic herring, *Clupea harengus membras*. Marine Biology 45:273-283.

Lipscomb, T. P., R. K. Harris, R. B. Moeller, J. M. Pletcher, R. J. Haebler, and B. E. Ballachey. 1993. Histopathologic lesions in sea otters exposed to crude oil. Veterinary Pathology 30:1-11.

Lombardi, B. 1966. Considerations on the pathogenesis of fatty liver. Laboratory Investigation 15:1-20.

Longwell, A. C. 1978. Field and laboratory measurements of stress responses at the chromosome and cell levels in planktonic fish eggs and the oil problem. Pages 116-125 *In*

In the Wake of the Argo Merchant. Center for Ocean Management Studies, Universtiy of Rhode Island. **Not Read**

Lönning, S. 1977. The effects of crude Ekofisk oil and oil products on marine fish larvae. Astarte 10:37-47.

No histopathology; crude Ekofisk oil and some of its fractions, benzene and xylene (50-100 ppm, 1-h pulse to continuous exposure); marine fish larvae (*Gadus morhua*, *Pleuronectes platessa*, *Platichthys flesus*). During organogenesis, larvae had poor differentiation of the head region, protruding eye lenses, abnormally bent notochord, various levels of inhibition of hatching, and breakdown of yolk. High boiling point samples gave similar results, whereas the low boiling point samples caused a high incidence of rapid cytolysis and often delay and irregularities in cleavage and development. Benzene and xylene lesions were distinct from oil lesions.

Lopez, E., J. Leloup-Hatey, A. Hardy, F. Lallier, E. Martelly, J. Oudot, J. Peignoux-Deville, and Y. A. Fontaine. 1981. Modifications histopathologiques et stress chez des anguilles soumises a une exposition prolongée aux hydrocarbures. Pages 645-653 *in* AMOCO CADIZ, Consequences d'une pollution accidentelle par les hydrocarbures, Actes du Colloque International Centre Oceanologique de Bretagne Brest (FRANCE) 19-22 Novembre, 1979. **Not read**

Maki, A. W., E. J. Brannon, L. G. Gilbertson, L. L. Moulton, and J. R. Skalski. 1995. An assessment of oil-spill effect on pink salmon populations following the *Exxon Valdez* oil spill--part 2: adults and escapement. Pages 585-625 *in* P. G. Wells, J. N. Butler, and J. S. Hughes, Editors. *Exxon Valdez* oil spill: fate and effects in Alaskan waters, ASTM STP 1219. Special Technical Publication 1219. American Society for Testing and Materials, Philadelphia.

Malins, D. C. 1982. Alterations in the cellular and subcellular structure of marine teleosts and invertebrates exposed to petroleum in the laboratory and field: A critical review. Canadian Journal of Fisheries and Aquatic Sciences 39:877-889.

Marty, G. D., E. F. Freiberg, T. R. Meyers, J. Wilcock, T. B. Farver, and D. E. Hinton. 1998. Viral hemorrhagic septicemia virus, *Ichthyophonus hoferi*, and other causes of morbidity in Pacific herring *Clupea pallasi* spawning in Prince William Sound, Alaska, USA. Diseases of Aquatic Organisms 32:15-40.

Marty, G. D., R. A. Heintz, and D. E. Hinton. 1997a. Histology and teratology of pink salmon larvae near the time of emergence from gravel substrate in the laboratory. Canadian Journal of Zoology 75:978-988.

Marty, G. D., A. Hoffmann, M. S. Okihiro, K. Hepler, and D. Hanes. In review. Histopathology and bile hydrocarbon analysis of demersal rockfish in Prince William Sound, Alaska, after the *Exxon Valdez* Oil Spill. Canadian Journal of Fisheries and Aquatic Sciences

Marty, G. D., J. E. Hose, M. D. McGurk, E. D. Brown, and D. E. Hinton. 1997b. Histopathology and cytogenetic evaluation of Pacific herring larvae exposed to petroleum hydrocarbons in the laboratory or in Prince William Sound, Alaska, after the *Exxon Valdez* oil spill. Canadian Journal of Fisheries and Aquatic Sciences 54:1846-1857.

Marty, G. D., M. S. Okihiro, E. D. Brown, D. Hanes, and D. E. Hinton. 1999. Histopathology of adult Pacific herring in Prince William Sound, Alaska, after the *Exxon Valdez* oil spill. Canadian Journal of Fisheries and Aquatic Sciences 56:419-426.

Marty, G. D., J. W. Short, D. M. Dambach, N. H. Willits, R. A. Heintz, S. D. Rice, J. J. Stegeman, and D. E. Hinton. 1997c. Ascites, premature emergence, increased gonadal cell apoptosis, and cytochrome-P4501A induction in pink salmon larvae continuously exposed to oil-contaminated gravel during development. Canadian Journal of Zoology 75:989-1007.

Mayo, D. W., D. S. Page, J. Cooley, E. Sorenson, F. Bradley, E. S. Gilfillan, and S. A. Hanson. 1978. Weathering characteristics of petroleum hydrocarbons deposited on fine clay marine sediments, Searsport, Maine. Journal of the Fisheries Research Board of Canada 35:552-562.

No histopathology; persistent oil. A U.S. Air Force pipeline ruptured and released JP-4-jet fuel and No. 2 heating oil into a cove near Searsport, Maine on March 16, 1971. In 1976 (5 years after the spill), several sites seemed to have little or no decline in gross hydrocarbon concentration, and essentially no weathering of the aliphatic portions of petroleum residues. Chromatograph spikes were similar in 1971 and 1976 from sediment samples. Clam *Mya arenaria* repopulation of the cove occurred only after sediment hydrocarbon concentrations dropped to <49 ppm.

Mazmanidi, N. D., and T. R. Bazhashvili. 1975. Effects of dissolved petroleum products on the embryonic development of the Black Sea flounder. Hydrobiological Journal 11:39-43.

Continuously exposed eggs at gastrulation, organogenesis, motile-embryo stage, and 24-h and 72-h larvae; levels of dissolved petroleum products were 0 to 2.5 mg/L. At levels above 0.025 mg/L, heartrate was depressed; anomalies included crooked spine and sluggish activity. The early stage was most sensitive. Hatching was delayed at higher concentrations.

McCain, B. B., H. O. Hodgins, W. D. Gronlund, J. W. Hawkes, D. W. Brown, M. S. Myers, and J. H. Vandermeulen. 1978. Bioavailability of crude oil from experimentally oiled sediments to English sole (*Parophrys vetulus*), and pathological consequences. Journal of Fisheries Research Board of Canada 35:657-664.

McGurk, M. D. 1984. Effects of delayed feeding and temperature on the age of irreversible starvation and on the rates of growth and mortality of Pacific herring larvae. Marine Biology 84:13-26.

No histopathology. The time from exhaustion of yolk to age of irreversible starvation for herring larvae was 8.5, 7.0, and 6.0 d at 6, 8, and 10° C, respectively. Lists 25 references for data similar to this paper; does not include salmon or char.

McGurk, M. D., and E. D. Brown. 1996. Egg-larval mortality of Pacific herring in Prince William Sound, Alaska, after the *Exxon Valdez* oil spill. Canadian Journal of Fisheries and Aquatic Sciences 53:2343-2354.

McKeown, B. A., and G. L. March. 1978. The acute effect of bunker C oil and an oil dispersant on: 1 serum glucose, serum sodium and gill morphology in both freshwater and seawater acclimated rainbow trout (*Salmo gairdneri*). Water Research 12:157-163.

In addition to histopathology, Bunker C oil and the oil dispersant decreased serum glucose levels. Freshwater-acclimated rainbow trout had a significant decrease in serum sodium, whereas seawater-acclimated RT had a marked increase in sodium levels. Osmoregulatory abnormalities were due to direct interference with the energy activated sodium transport systems of the gills.

Metcalfe, C. D., and R. A. Sonstegard. 1985. Oil refinery effluents: evidence of cocarcinogenic activity in the trout embryo microinjection assay. Journal of the National Cancer Institute 75:1091-1097.

Includes histopathology. Oil refinery effluents soxhlet and XAD-2 were microinjected into eyed rainbow trout (*Oncorhynchus mykiss*) embryos. Alone, the two effluents increased the incidence of spinal abnormalities but not hepatic neoplasms (fish were sampled one or two years after injection). Incidence of hepatic tumors increased when the effluents were coinjected with aflatoxin B1 (more tumors than with AFB1 alone) but not with coinjection of MNNG.

Meyers, T. R., and J. D. Hendricks. 1985. Histopathology. Pages 283-331 *in* G. M. Rand, and S. R. Petrocelli, Editors. Fundamentals of Aquatic Toxicology. Hemisphere Publishing, Washington D.C.

Meyers, T. R., S. Short, K. Lipson, W. N. Batts, J. R. Winton, J. Wilcock, and E. Brown. 1994. Association of viral hemorrhagic septicemia virus with epizootic hemorrhages of the skin in Pacific herring *Clupea harengus pallasi* from Prince William Sound and Kodiak Island, Alaska, USA. Diseases of Aquatic Organisms 19:27-37.

Middaugh, D., P,, P. J. Chapman, and M. E. Shelton. 1996. Responses of embryonic and larval Inland Silversides to a water-soluble fraction formed during bidegredation of artificially weathered Alaska North Slope crude oil. Archives of Environmental Contamination and Toxicology 31:410-419.

Miller, D. S., D. B. Peakall, and W. B. Kinter. 1978. Ingestion of crude oil: Sublethal effects in herring gull chicks. Science 199:315-317.

Bird study; feeding experiment in herring gull chicks. Gave a single oral dose of crude oil (0.3 mL oil/kg bw). Results in exposed birds: 1) cessation of growth despite normal feeding; 2) osmoregulatory impairment; 3) hypertrophy of hepatic, adrenal, and nasal gland tissue; 4) induction of cytochrome P-450 enzymes; and 5) histologic lesions described as "proliferative edema with considerable cytoplasmic disruption" (no evidence of enteritis).

Miller, M. R., D. E. Hinton, J. J. Blair, and J. J. Stegeman. 1988. Immunohistochemical localization of cytochrome P-450E in liver, gill and heart of scup (*Stenotomus chrysops*) and rainbow trout (*Salmo gairdneri*). Marine Environmental Research 24:37-39.

Minchew, C. D., and J. D. Yarborough. 1977. The occurence of fin rot in mullet (*Mugil cephalus*) associated with crude oil contamination of an estuarine pond-ecosystem. Journal of Fish Biology 10:319-323. **Not Read**

Mitrovic, U. V., U. M. Brown, D. G. Shurben, and M. H. Berryman. 1968. Some pathological effects of sub-acute and acute poisoning of rainbow trout by phenol in hard water. Water Research 2:249-254.

Limited histopathology; phenol (references older papers); rainbow trout (*Oncorhynchus mykiss*); gross hemorrhage, gill, skin, liver congestion. Rainbow trout were exposed to levels near the 48-h LC50; no control fish were examined. Fish killed within a few hours of exposure had inflammation and necrosis of the pharynx and gills, bloody ascites, and splenomegaly. In the gills, initial inflammation was followed by stripping of the epithelium from secondary lamellae and from the filaments. Fish surviving for 7 d had lesions in the skin, liver, kidney, spleen, small intestine, and ovary.

Moles, A., M. M. Babcock, and S. D. Rice. 1987. Effect of oil exposure on pink salmon, *Oncorhynchus gorbuscha*, alevins in a simulated intertidal environment. Marine Environmental Research 21:49-58.

Moles, A., and B. L. Norcross. 1998. Effects of oil-laden sediments on growth and health of juvenile flatfishes. Canadian Journal of Fisheries and Aquatic Sciences 55:605-610.

Moles, A. D., S. D. Rice, and M. S. Okihiro. 1993. Herring parasite and tissue alterations following the *Exxon Valdez* oil spill. Pages 325-328 *in* 1993 International Oil Spill Conference (Prevention, Preparedness, Response). United States Coast Guard, American Petroleum Institute, and U.S. Environmental Protection Agency.

Morris, R. W. 1989. Testing statistical hypotheses about rat liver foci. Toxicologic Pathology 17:569-578.

Myers, M. S., L. D. Rhodes, and B. B. McCain. 1987. Pathologic anatomy and patterns of occurrence of hepatic neoplasms, putative preneoplastic lesions, and other idiopathic

hepatic conditions in English sole (*Parophrys vetulus*) from Puget Sound, Washington. Journal of the National Cancer Institute 78:333-363.

Myers, M. S., J. T. Landahl, M. M. Krahn, L. L. Johnson, and B. B. McCain. 1990. Overview of studies on liver carcinogenesis in English sole from Puget Sound; Evidence for a xenobiotic chemical etiology: pathology and epizootiology. The Science of the Total Environment 94:33-50.

Nava, M. E., and F. R. Engelhardt. 1982. Induction of mixed function oxidases by petroleum in the American eel, Anguilla rostrata. Archives of Environmental Contamination and Toxicology 11:141-5. **Not Read**

Neff, J. M., and W. A. Stubblefield. 1995. Chemical and toxicological evaluation of water quality following the *Exxon Valdez* oil spill. Pages 141-177 *in* P. G. Wells, J. N. Butler, and J. S. Hughes, Editors. *Exxon Valdez* oil spill: fate and effects in Alaskan waters, ASTM STP 1219. Special Technical Publication 1219. American Society for Testing and Materials, Philadelphia.

Nikunen, E. 1985. Toxic impact of effluents from petrochemical industry. Ecotoxicology and Environmental Safety 9:84-91. **Not Read**

Nuwayhid, M. A., P. S. Davies, and H. Y. Eldes. 1980. Changes in the ultrastructure of the gill epithelium of *Patella vulgata* after exposure to North Sea crude oil and dispersants. 60:439-448.

Limpet *Patella vulgata* were exposed to 25 and 100% WSF of North Sea crude oil and 1.0, 2.5, and 10.0 mL/L of two dispersants (BP1100X and BP1100WD). TEM revealed a great increase in the numbers of lysosomes, vacuolation of mitochondria, and extrusion of cytoplasm and damaged organelles through the apical surface.

Nystrom, R. R., and G. Post. 1982. Chronic effects of ammonia-stripped oil shale retort water on fishes, birds, and mammals. Bulletin of Environmental Contamination and Toxicology 28:271-276.

No histologic lesions. Any oil component in the retort water is of little concern.

Onwumere, B. G., and A. A. Oladimeji. 1990. Accumulation of metals and histopathology in *Oreochromis niloticus* exposed to treated NNPC Kaduna (Nigeria) petroleum refinery effluent. Ecotoxicology and Environmental Safety 19:123-134.

Refinery effluents ≠ crude oil

Owens, E. H. 1978. Mechanical dispersal of oil stranded in the littoral zone. Journal of the Fisheries Research Board of Canada 35:563-572.

No histopathology; persistent oil; review article. The residence time or persistence of stranded oils increases as mechanical energy levels at the shoreline decrease. Ice tends to decrease energy in the littoral zone.

Paine, M. D., W. C. Leggett, J. K. McRuer, and K. T. Frank. 1991. Effects of incubation in oiled sediment on emergence of capelin (*Mallotus villosus*) larvae. Canadian Journal of Fisheries and Aquatic Sciences 48:2228-2239.

Paine, M. D., W. C. Leggett, J. K. McRuer, and K. T. Frank. 1992. Effects of Hibernia crude oil on capelin (*Mallotus villosus*) embryos and larvae. Marine Environmental Research 33:159-187.

Park, S. S., H. Miller, A. V. Klotz, P. J. Kloepper-Sams, J. J. Stegeman, and H. V. Gelboin. 1986. Monoclonal antibodies to liver cytochrome P450 E of the marine fish scup. Archives of Biochemistry and Biophysics 249:339-350.

Payne, J. F., and L. F. Fancey. 1989. Effect of polycyclic aromatic hydrocarbons on immune responses in fish: change in melanomacrophae centers in flounder (*Pseudopleuronectes americanus*) exposed to hydrocarbon-contaminated sediments. Marine Environmental Research 28:431-435. **Not Read**

Payne, J. F., L. L. Fancey, J. Hellou, M. J. King, and G. L. Fletcher. 1995. Aliphatic hydrocarbons in sediments: A chronic toxicity study with winter flounder (*Pleuronectes americanus*) exposed to oil well drill cuttings. Canadian Journal of Fisheries and Aquatic Sciences 52:2724-2735.

Exposed male winter flounder in winter (no feeding) to realistic concentrations of drill cuttings enriched in aliphatic hydrocarbons; initial hydrocarbon levels were as high as 1500 ppm; 80-day exposure; no significant changes in body condition indices, muscle and liver energy reserves, mixed function oxidases (MFOs), blood parameters (PCV, Chloride, total protein), or liver and gill histopathology

Payne, J. F., J. W. Kiceniuk, W. R. Squires, and G. L. Fletcher. 1978a. Pathological changes in a marine fish after a 6-month exposure to petroleum. Journal of the Fisheries Research Board of Canada 35:665-667.

After 6-month exposure to continuous flow petroleum, no lesions were found in cunner *Tautogolabrus adspersus* liver, kidney, heart, spleen, gonad, gill, muscle, or gut tissues. Packed cell volume (PCV) was unchanged. The only changes in oiled fish were a decreased testes:somatic index and an increased lens diameter.

Payne, J. F., I. Martins, and A. Rahimtula. 1978b. Crankcase oils: are they a major mutagenic burden in the aquatic environment? Science 200:329-330.

No histopathology. Fractions from various crude and refined oils were not mutagenic. Fractions from used crankcase oils enriched in PAHs induced revertant

colonies in *Salmonella typhimurium* TA98 when activated by rat or trout liver extracts (reversions was not due to BaP or benzanthracene in the mixture).

Pearson, W. H., E. Moksness, and J. R. Skalski. 1995. A field and laboratory assessment of oil-spill effects on survival and reproduction of Pacific herring following the *Exxon Valdez* oil spill. Pages 626-661 *in* P. G. Wells, J. N. Butler, and J. S. Hughes, Editors. *Exxon Valdez* oil spill: fate and effects in Alaskan waters, ASTM STP 1219. Special Technical Publication 1219. American Society for Testing and Materials, Philadelphia.

Prasad, M. S. 1988. Sensitivity of branchial mucous to crude oil toxicity in a freshwater fish, *Colisa fasciatus*. Bulletin of Environmental Contamination and Toxicology 41:754-758.

Prasad, M. S. 1989. Effects of crude oil on the air-breathing organs of the striped gourami, *Colisa fasciatus*: a SEM study. Ecotoxicology and Environmental Safety 18:211-218. **Not Read**

Prasad, M. S. 1991. SEM study on the effects of crude oil on the gills and air breathing organs of climbing perch, *Anabas testudineus*. Bulletin of Environmental Contamination and Toxicology 47:882-889.

Ramusino, M. C., P. Dellavedova, and D. Zanzottera. 1984. Effects of crude Dubai oil on *Salmo gairdneri* Rich. and *Carassius auratus* L. Bulletin of Environmental Contamination and Toxicology 32:368-376.

No histopathology. 48 h LC50, static tests, WSF crude oil, rainbow trout (larvae a few days posthatch, and 5-cm-long fingerlings), and goldfish. Fish rolled on their sides and turned upside down. RT fry had decreased resorption of yolk sac and darker pigmentation. WSF was more toxic than oil poured directly on water.

Reimschuessel, R., R. O. Bennett, and M. M. Lipsky. 1992. A classification system for histologic lesions. Journal of Aquatic Animal Health 4:135-143.

This histopathologic classification system organizes lesions by 4 features: location (system, tissue), change (lesion), extent (focal, multifocal, diffuse) and severity (mild to severe), and coded data.

Rice, S. D., M. M. Babcock, C. C. Brodersen, M. G. Carls, J. A. Gharrett, S. Korn, A. Moles, and J. W. Short. 1987a. Lethal and sublethal effects of the water-soluble fraction of Cook Inlet crude oil on Pacific herring (*Clupea harengus pallasi*) reproduction. U.S Department of Commerce, NOAA Technical Memorandum NMFS F/NWC-111.

No histopathology; 2- and 12-d LC50 for herring adults (2.3 ppm aromatics), larvae (2.3-2.8 ppm), and eggs (1.5 ppm at 12 d, 5.3 ppm at 2 d); feeding yolk-sac larvae was most sensitive (21d LC50 = 0.36 ppm). Concluded that if adult herring survived oil exposure, then their eggs hatched. Larval growth was not affected by a diet of oil-contaminated prey

Rice, S. D., M. M. Babcock, C. C. Brodersen, J. A. Gharrett, and S. Korn. 1987b. Uptake and depuration of aromatic hydrocarbons by reproductively ripe Pacific herring and the subsequent effect of residues on egg hatching and survival. Pages 139-154 *in* W. B. Vernberg, A. Calabrese, F. P. Thurberg, and F. J. Vernberg, Editors. Pollution Physiology of Estuarine Organisms. Belle W. Baruch Libr. Mar. Sci. 17, University of South Carolina Press, Columbia.

No histopathology. In adult herring, depuration took 14 d until hydrocarbon levels in exposed fish were equivalent to controls. Cites papers on uptake-depuration in pink salmon fry and benzene effects of herring spawning.

Rice, S. D., R. E. Thomas, and J. W. Short. 1977. Effect of petroleum hydrocarbons on breathing and coughing rates and hydrocarbon uptake-depuration in pink salmon fry. Pages 259-277 *in* F. J. Vernberg, A. Calabrese, F. P. Thurberg, and W. B. Vernberg, Editors. Physiological Responses of Marine Biota to Pollutants. Academic Press, New York. **Not Read**

Roubal, W. T., D. H. Bovee, T. K. Collier, and S. I. Stranahan. 1977. Flow-through system for chronic exposure of aquatic organisms to seawater-soluble hydrocarbons from crude oil: construction and applications. Pages 1977 Oil Spill Conference (Prevention, Behavior, Control, Clean-Up). **Not read**

Roubal, W. T., S. I. Stranahan, and D. C. Malins. 1978. The accumulation of low molecular weight aromatic hydrocarbons of crude oil by coho salmon (*Oncorhynchus kisutch*) and starry flounder (*Platichthys stellatus*). Archives of Environmental Contamination and Toxicology 7:237-244.

No histopathology; oil accumulation/depuration in tissue. They exposed coho salmon and starry founder to 0.9±0.1 ppm Prudhoe Bay crude oil. Alkylated hydrocarbons accumulated in tissues more than unsubstituted derivatives. Muscle of starry founder had 17 ppm C4- and C5-substituted benzenes (bioconcentration factor of 1,700) but coho salmon muscle had only 1.5 ppm. Hydrocarbon levels were undetectable 1 wk after transfer to clean water.

Bioconcentration of various components of crude oil are different, making "fingerprinting" of HC source from fish tissues difficult.

Russell, L. C., and M. Fingerman. 1984. Exposure to the water soluble fraction of crude oil or to naphthalenes alters breathing rates in Gulf killifish, *Fundulus grandis*. Bulletin of Environmental Contamination and Toxicology 32:363-367. **Not Read**

Sabo, D. J., and J. J. Stegeman. 1977. Some metabolic effects of petroleum hydrocarbons in marine fish. Pages *in* A. Calabrese, and J. F. Vernberg, Editors. Pollution and Physiology of Marine Organisms II. Academic Press, New York. **Not read; Not found**

Sabo, D. J., J. J. Stegeman, and L. S. Gottlieb. 1975. Petroleum hydrocarbon pollution and hepatic lipogenesis in the marine fish *Fundulus heteroclitus*. Federation Proceedings 34:810.

Sawyer, T. K. 1978. Microscopic observations on vertebrates and invertebrates collected near the Argo Merchant oil spill. Pages 93-95 *in* In the wake of the Argo Merchant. Center for Ocean Management Studies, Universtiy of Rhode Island.

Histopathology was done on winter flounder (*Pleuronectes americanus*), yellowtail flounder (*Limanda ferruginea*), *Ammodytes* sp. larvae, mollusks, crustaceans, sea urchins, and starfish collected near the Argo Merchant oil spill. No lesions were attributed solely to exposure to petroleum. Adult fish had edematous gills, detached epithelium, and hyperplasia of the olfactory epithelium; larval fish had ocular lesions and malformations or lack of pigmentation of the eye. Authors did not state the incidence of lesions in fish or larvae from control vs. exposed sites. Mollusks had no lesions.

Scheier, A., and D. Gominger. 1976. A preliminary study of the toxic effects of irradiated vs. non-irradiated water soluble fractions of No. 2 fuel oil. Bulletin of Environmental Contamination and Toxicology 16:595-603. **Not Read**

Schwartz, J. P. 1985. Effect of oil-contaminated prey on the feeding and growth rate of pink salmon fry (*Oncorhynchus gorbuscha*). Pages 459-476 *in* F. J. Vernberg, F. P. Thurberg, A. Calabrese, and W. Vernberg, Editors. Marine pollution and physiology: recent advances. Univ. South Carolina Press, Columbia, South Carolina.

No histopathology. Fry were fed 0.6, 3.2, and 6.5 ppm oil-contaminated prey (OCP) for 10, 23, 36, or 50 d. After 10 d, fry had decreased growth during exposure and for 4 d after exposure. After 50-d, the higher levels of exposure resulted in decreased weight gain. Fry were able to feed and grow at OCP concentrations that were 5 to 10 times the 96-h LC50 of crude oil WSF in seawater. Cites similar feeding studies.

Sherman, K., and D. Busch. 1978. The Argo Merchant oil spill and the fisheries. Pages 149-165 *in* In the Wake of the Argo Merchant. Center for Ocean Management Studies, University of Rhode Island.

No histopathology. The impact of oil spilled from the Argo Merchant on fish stocks "has not been catastrophic." A more significant problem concerns "the chronic background levels of petroleum hydrocarbons present in the surface waters inhabited by fish eggs and larvae." The tanker Argo Merchant ran aground on Nantucket Shoals 15 December 1976; by 8 February 1977, approximately 7.7 million gallons of No. 6 fuel oil had been released into the waters of the Continental Shelf. In the immediate vicinity of the wreck, concentrations of petroleum hydrocarbons up to 250 ppb were detected. The only damage reported in fish was limited to the observation of oil in the stomach of two codfish shortly after the spill. Overall, <5% of fish sampled had clear evidence of Argo contamination.

Short, J. W., and P. M. Harris. 1996a. Chemical sampling and analysis of petroleum hydrocarbons in near-surface seawater of Prince William Sound after the *Exxon Valdez* oil spill. American Fisheries Society Symposium 18:17-28.

Short, J. W., and P. M. Harris. 1996b. Petroleum hydrocarbons in caged mussels deployed in Prince William Sound after the *Exxon Valdez* oil spill. American Fisheries Society Symposium 18:29-39.

Short, J. W., and R. A. Heintz. 1997. Identification of *Exxon Valdez* oil in sediments and tissues from Prince William Sound and the Northwestern Gulf of Alaska based on a PAH weathering model. Environmental Science and Technology 31:2375-2384.

Sindermann, C. J. 1979. Pollution-associated diseases and abnormalities of fish and shellfish: A review. Fishery Bulletin 76:717-749. **Not Read**

Slade, G. J. 1982. Effect of Ixtoc I crude oil and Corexit 9527 dispersant on spot (*Leiostomus xanthurus*) egg mortality. Bulletin of Environmental Contamination and Toxicology 29:525-530. **Not Read**

Smith, R. L., and J. A. Cameron. 1979. Effect of water soluble fraction of Prudhoe Bay crude oil on embryonic development of Pacific herring. Transactions of the American Fisheries Society 108:70-75.

Smolowitz, R. M., M. E. Hahn, and J. J. Stegeman. 1991. Immunohistochemical localization of cytochrome P-4501A1 induced by 3,3',4,4'-tetrachlorobiphenyl and by 2,3,7,8-tetrachlorodibenzo furan in liver and extrahepatic tissues of the teleost *Stenotomus chrysops* (scup). Drug Metabolism and Disposition 19:113-123.

Solangi, M. A., and R. M. Overstreet. 1982. Histopathological changes in two estuarine fishes, *Menidia beryllina* (Cope) and *Trinectes maculatus* (Bloch and Schneider), exposed to crude oil and its water soluble fractions. Journal of Fish Diseases 5:13-35.

Spies, R. B., J. J. Stegeman, D. E. Hinton, B. Woodin, R. Smolowitz, M. Okihiro, and D. Shea. 1996. Biomarkers of hydrocarbon exposure and sublethal effects in embiotocid fishes from a natural petroleum seep in the Santa Barbara Channel. Aquatic Toxicology 34:195-219.

Stegeman, J. J., and M. E. Hahn. 1995. Biochemical and molecular biology of monooxygenases: current perspectives on forms, functions, and regulation of cytochrome P450 in aquatic species. Pages 87-206 *in* D. C. Malins, and G. K. Ostrander, editors Aquatic toxicology: molecular, biochemical, and cellular perspectives. Lewis Publishers, Boca Raton, Florida.

Stott, G. G., W. E. Haensly, J. M. Neff, and J. R. Sharp. 1983. Histopathologic survey of ovaries of plaice, *Pleuronectes platessa* L., from Aber Wrac'h and Aber Benoit, Brittany, France: long-term effects of the Amoco Cadiz crude oil spill. Journal of Fish Diseases 6:429-437.

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