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Investigation of Persistent Seabird Mortalities along the Oregon Coast

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Abstract

From 1978 until 1997, Oregon experienced large annual die-offs of common murres (*Uria aalge*) from July to October. The mortality was predominantly among juveniles, but adults were impacted. Juvenile common murre require extensive parental care for several months, both at the colony site and at sea. At this stage they are vulnerable to natural and anthropogenic disturbances as well as changes in nearshore marine productivity. Given the vulnerability of birds at this age, exposure to contaminants may increase seabird mortality. However, aside from large oil spills, there was little information as to the potential contribution of contaminants to the annual die-offs. In addition to annual die-offs, Oregon common murres experienced some of their worst reproductive seasons on record during that time. Poor reproduction coupled with high mortality could have serious impacts on the stability of common murre breeding populations in Oregon and elsewhere.

The primary objectives of this study were to determine concentrations of contaminants in common murres during the annual die-off, assess the importance of contaminants as causative agents in the die-off, and identify potential sources and pathways of these contaminants.

Concentrations of inorganic and organic compounds in common murres were not life threatening. However, murres are accumulating some inorganic and organic contaminants to low concentrations that may have sublethal effects. In addition, blood analyses and necropsy results were not suggestive of contaminant exposure or significant disease, but they provided further evidence that starvation from limited food resources is likely a significant contributor to die-off events.

The tissue residue analyses suggest that common murres in Oregon are likely exposed primarily to non-point source pollutants (e.g., those from land runoff or atmospheric deposition), which are not readily controlled through direct management actions. Consequently, the only management recommendation from this study is to continue ongoing efforts in Oregon to decrease stress on common murres from disturbance (boats, aircraft, and people) which increase the potential for breeding colony abandonment and cause general stress that may exacerbate the effects of limited food resources, low levels of environmental contaminants, minor disease events, or any combination of these factors.

Keywords

Common murre, *Uria aalge*, die-offs, DEC ID 199510002, FFS 1N23, Oregon Congressional Districts 1, 4 and 5, contaminants, DDT, PCB, heavy metals

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List of Acronyms/Abbreviations

ALT	Alanine amino transferase
AST	Aspartate amino transferase
BC	Buffy coat
BHC	1,2,3,4,5,6-Hexachlorocyclohexane
Ca	Calcium
CK	Creatine kinase
Cl	Chloride
DBili	Direct bilirubin
DDD	Dichlorodiphenyldichloroethane
DDE	Dichlorodiphenyldichloroethylene
DDT	Dichlorodiphenyltrichloroethane
ELISA	Enzyme-linked immunosorbent assay
ENSO	El Niño Southern Oscillation
GERG	Geochemical and Environmental Research Group
GGT	Gamma glutamyltransferase
GLC	Gas-liquid chromatography
HCB	Hexachlorobenzene
HCO_3	Bicarbonate
ICP	Inductively coupled plasma
K	Potassium
mg/kg	Milligram/kilogram
Na	Sodium
ND	Non detect
NOAA	National Oceanic and Atmospheric Administration
NWHC	National Wildlife Health Center
P	Phosphorus
PACF	Patuxent Analytical Control Facility
PCB	Polychlorinated biphenyl
PCV	Packed cell volume
ppb	Part per billion
ppm	Part per million
ppt	Part per trillion
QA	Quality control
QC	Quality assurance
SOP	Standard operating procedure
SRM	Spike recovery matrix
TBili	Total bilirubin
TS	Total solids
TP	Total protein
UA	Uric acid
USGS	U.S. Geological Survey

INTRODUCTION

Seabirds are subject to a variety of human-caused perturbations including oil spills, gillnet mortality, overfishing of seabird prey stocks, diseases, and pollution (Newman et al. 2006, Carter et al. 2001, Ainley et al. 2002). These perturbations have resulted in seabird population declines at many locations in the U.S. and around the world. From 1989 to 1994, large die-offs of seabirds along the Pacific coast were reported by the U.S. Geological Survey's (USGS) National Wildlife Health Center (NWHC) located in Madison, Wisconsin. Although many of these die-offs were associated with oil spills, other seabird mortalities appear to be the result of starvation, infectious diseases, and other anthropogenic causes (Newman et al. 2007). The role of contaminants in these starvation die-offs has not been assessed.

Seabirds along the Pacific coast have great potential to be exposed to contaminants. Oil spills regularly occur and cause significant acute mortality (Carter et al. 2001). Whereas acute toxicity resulting from oil is usually evident, chronic and long-term effects to seabird populations and their habitats from contaminants are not well known. Sublethal effects can be delayed and result in short-, medium-, and long-term increases in vulnerability to disease, predation, and starvation resulting from effects on the immune system, stress response, energetics, and neurobehavioral function (Fox 2000). In addition, some contaminants can damage or alter genetic material, which may have serious implications for populations and gene pools (Hebert and Luiker 1996, Theodorakis 2001).

Common murres (*Uria aalge*) are members of the Alcidae family, a group of wing-propelled diving birds with high adult survivorship, delayed maturity, and low clutch size of a single egg (Manuwal and Carter 2001). Murres feed in various marine habitats, from estuarine areas near shore to offshore areas (Manuwal and Carter 2001), foraging from 22 to 124 miles from the colony (Briggs et al. 1987, Cairns et al. 1990) and diving to depths of over 590 feet (Piatt and Nettleship 1985). Prey varies with season and location, with fish being the primary food source during the breeding season and euphausiids (pelagic, shrimp-like crustaceans) and squid being the main dietary items during winter and pre-breeding periods (Manuwal et al. 2001). Common murres nest in very close proximity to one another in colonies on ledges of coastal cliffs and on sides and tops of rock stacks and other offshore islands (Csuti et al. 1997). Egg laying typically occurs from early May to early June on rocky substrate with both sexes incubating the egg for about 28 to 35 days (Boekelheide et al. 1990, Browning and English 1972, Scott 1973). Young leave for the sea approximately 3 weeks after hatching (Csuti et al. 1997) where further development takes place while the young is accompanied by the male parent (Ainley et al. 2002); parental care at sea lasts at least 1 to 2 months (Varoujean et al. 1979). The young can fly at approximately 6 weeks of age (Csuti et al. 1997).

From 1978 until 1997, Oregon experienced large annual die-offs of common murres from July to October. The mortality was predominantly among juveniles, but adults were impacted. Common murre mortalities along a single beach ranged from 135 to 1,273 individuals per year (Bayer et al. 1991). Juvenile common murre require extensive parental care for several months, both at the colony site and at sea (Welty 1975; Roy Lowe, Project Leader, U.S. Fish and Wildlife Service, Oregon Coast National Wildlife Refuge Complex, Newport, Oregon, pers. comm., 1997). At this stage they are vulnerable to natural (e.g., coastal storms, separation from the

parent) and anthropogenic (e.g., harassment, pollution) disturbances as well as changes in nearshore marine productivity. Given the vulnerability of birds at this age, exposure to contaminants may increase seabird mortality. However, aside from large oil spills (Carter 2003), there was little information as to the potential contribution of contaminants to the annual die-offs. Due to the high incidence of common murre mortality and the public interest in the die-offs, it is important to determine the causes of these mortalities.

In addition to the annual die-offs, Oregon common murres experienced some of their worst reproductive seasons on record during the 1990s. Lowe (1993) reported low common murre colony attendance but high chick production during the 1992 El Niño Southern Oscillation (ENSO) event. However, heavy chick mortality in their first month at sea (July) may have eliminated most of the 1992 year class. Other seabird species were also impacted by the 1992 ENSO event. Very high mortality of adult and subadult Cassin's auklets was recorded in Oregon and Washington during this time. Pelagic cormorants also abandoned colonies and no young were produced in 1992. These summer mortalities were followed by numerous seabird mortalities along Oregon beaches from November 1992 through February 1993 (Lowe 1993). On a 7.4-km beach transect, 511 dead Cassin's auklets were recovered from November to February (1,403% above a 14-year average) and 91 dead common murres were recovered from December to February (310% above a 14-year average) (Lowe 1993).

Widespread abandonment of major and minor common murre colonies also was observed along the entire Oregon coastline in May 1993 (Lowe 1993). A large number of eggs were laid at many colonies but nests were abandoned in mid- to late incubation. No chicks were produced at Three Arch Rocks where an estimated 220,000 common murres nest; the largest colony south of the Semidi Islands, Alaska. Additionally, during small boat transects, no common murre chicks were seen on the water where normally thousands would have been present. Chick production in the 1994 breeding season also was below average. Poor reproduction coupled with high mortality could have serious impacts on the stability of common murre breeding populations in Oregon and elsewhere.

In 1995, another large die-off occurred involving common murres and other seabirds including Cassin's auklets, surf scoters, and brown pelicans (R. Lowe, pers. comm., 1997). The murres in this die-off were primarily juveniles (77%). Again in 1996, record numbers of dead murres were found along Oregon beaches, almost entirely adults (99%), with large murre colonies being completely abandoned (R. Lowe, pers. comm., 1996). An estimated 10,000 murres died in spring/summer 1996 (Meehan 1996), the fifth year in a row that Oregon murres experienced a difficult year and also the fifth year of no measurable cold-water upwelling. These upwellings deliver nutrient-rich waters from deep depths up to the photic zone, promoting significant plankton growth, which ultimately benefits the forage fish murres depend upon. A similar situation also occurred in 1997 where birds also abandoned their nests, although this year was apparently not as severe (Meehan 1997).

Oregon common murre mortalities mirror findings recorded elsewhere along the Pacific coast (Piatt and van Pelt 1993). In the spring of 1993, more than 3,100 dead common murres were recovered in Alaska with unconfirmed observations of "hundreds to thousands" dead at sea. The predicted total mortality was in the range of 10,000 to 100,000. Necropsies revealed that most

common murres were extremely emaciated; some had pulmonary edema and unusual focal hemorrhaging in the intestines. A large proportion appeared to be sub-adults. Explanations for the mortalities included widespread starvation, anomalous warm sea surface temperatures, and biotoxins (Barclay 1993, Piatt and van Velt 1993). However, a few recovered birds were of normal weight and some of the pathological results remain unexplained. Dead common murres were also found in numbers three to four times higher than usual for winter beach surveys on Vancouver Island, British Columbia, and moribund common murres were recovered in California.

Further concern regarding common murre populations is warranted by the dramatic decline in the common murre breeding population along the Washington coast. The Washington common murre population was reduced 89% from 28,940 individuals in 1982 to 3,190 individuals in 1983, a decline attributed to the 1982 ENSO event (Wilson 1991). While populations in Oregon and California recovered from this event, Washington populations only partially recovered and now fluctuate between 10,000 and 13,000 individuals (R. Lowe, pers. comm., 2011). It is unknown why the population has not recovered, but the potential role of contaminants in the common murre's recovery has not been investigated. Since many common murres that breed in Oregon spend the winter in Washington waters, an examination of common murre mortalities along the Oregon coast may provide insight into the failure of Washington common murre populations to recover to former levels.

Common murres may also serve as an indicator of potential threats to marbled murrelets (*Brachyramphus marmoratus*). In 1992, the marbled murrelet was listed as threatened under the Federal Endangered Species Act of 1973, as amended, in Washington, Oregon, and California. In the Pacific Northwest, marbled murrelets spend the majority of their life cycle in the nearshore marine waters and fly inland to nest in older forests (U.S. Fish and Wildlife Service 1997). Foraging areas and prey composition are similar for common murres and marbled murrelets, although common murres generally tend to feed further offshore (Nelson 1997, Ainley et al. 2002). Virtually no information is available on the effects of contaminants, other than oil, on marbled murrelets, but poor food resources in central California may play some role in the declining populations, in addition to reduced nesting habitat (Peery et al. 2004, 2006). Evaluation of contaminant residues in common murres will provide insight into potential marbled murrelet contaminant concerns.

Study Objectives

The specific objectives of this study are:

1. Determine concentrations of contaminants in common murres during the annual die-off, assess the importance of contaminants as causative agents in the die-off, and identify potential sources and pathways of these contaminants.

2. Relate common murre contaminant concentrations in Oregon to similar data from other populations on the Pacific coast.

3. Interpret the significance of contaminant concentrations to common murre and other seabird species, most notably marbled murrelet populations.

4. Use information on contaminant concentrations to develop appropriate management options to reduce annual mortality.

METHODS

Murre Collection

Common murres were collected in 1995, 1996, and 1997, coinciding with summer die-offs. In 1995, a total of 40 common murres were collected near Newport (Figure 1) including 10 adult live birds, 10 juvenile live birds, and 20 freshly dead carcasses, primarily juveniles. The 20 live birds, collected on the ocean using a shotgun with steel shot on July 12, 1995, were considered reference birds. Of these live birds, 10 (five adults and five juveniles) were submitted to the NWHC for necropsy, histopathology, and tissue collection for contaminant analysis. The remaining 10 murres were necropsied by Dr. Scott Newman (Wildlife Health Center, School of Veterinary Medicine, University of California, Davis) on site and tissues collected for histopathology and future contaminant analysis. Moribund murres or carcasses were collected on July 12, 1995, from the ocean (one dead juvenile recovered while collecting live murres) or from July 14 through September 20, 1995, through beach surveys. These 20 moribund or dead birds were submitted to the NWHC for necropsy, histopathology, and tissue collection for contaminant analysis. Detailed sample information is provided in Appendix A.

Eight dead adult murres were collected on June 27 and 29, 1996, during beach surveys in the vicinity of Newport and Bandon (Figure 1). Specimens were not necropsied but tissues were extracted for chemical analysis. Results from these dead murres collected near Newport were compared to reference birds from 1995.

On June 30, 1997, four adult murres (two males, two females) were collected north of Gearhart, (Figure 1) during another die-off event. The die-off involved adult murres and followed a period of stormy weather and higher than normal sea surface temperatures. Three of the murres were collected dead, while one was collected alive and euthanized; the birds were sent to the NWHC for necropsy.

Blood

Blood samples were obtained in 1995 via heart puncture from reference birds immediately following collection on the ocean; sufficient samples were obtained from nine juveniles and 10 adults. Where possible, blood samples were collected from murres prior to euthanizing any individuals for necropsy.

Blood samples were placed either in heparinized plasma tubes or EDTA tubes. Tubes were immediately wrapped in towels and then placed on ice inside coolers within 5 minutes after collection. Whole blood was used to obtain packed cell volume (PCV), buffy coat (BC), and total solids (TS). Plasma was obtained by centrifuging plasma tubes for 10 to 15 minutes to

separate blood cells from plasma. The plasma was removed and placed in cryovials and frozen. The blood samples were analyzed at the School of Veterinary Medicine, University of California, Davis, by Dr. Scott Newman under contract with the Oregon Fish and Wildlife Office. Activity levels and concentrations of the following parameters were measured: alkaline phosphatase, alanine amino transferase (ALT), aspartate amino transferase (AST), creatine kinase (CK), gamma glutamyltransferase (GGT), albumin, globulin, total protein (TP), total bilirubin (TBili), direct bilirubin (DBili), creatinine, cholesterol, glucose, calcium (Ca), phosphorus (P), bicarbonate (HCO_3), chloride (Cl), potassium (K), sodium (Na), albumin:globulin ratio, and uric acid (UA).

Necropsy

Twenty dead or moribund murres obtained via beach surveys in 1995 were necropsied by the NWHC to document the basic health, body condition, and obvious pathological conditions that could be associated with disease or contaminants. This examination included histological studies to further identify pathological conditions possibly related to contaminant exposure. Ten reference carcasses collected in 1995 also were necropsied by the NWHC. The remaining 10 references murres collected from coastal habitats in 1995 were necropsied by Dr. Scott Newman who examined various tissues using similar gross pathology and histopathology techniques as the NWHC. Four carcasses collected in 1997 were necropsied at the NWHC.

The necropsies performed at the NWHC included gross external and internal examination followed by sample extraction for disease testing and histopathological examination. Bacterial isolation attempts from liver, intestine, and lung were conducted by inoculating tissues onto 5% sheep red blood agar and eosin-methylene blue plates (Difco Laboratories, Detroit, Michigan). Plates were incubated at 37 ˚C for 72 hours, and bacterial isolates were characterized by the API-20E system (Analytab Products, Plainview, New York). Heart blood was tested for botulism toxin by the use of enzyme-linked immunosorbent assay (ELISA). Cell cultures and embryonating eggs were used for virus isolation attempts from liver, spleen, intestine, and lung. Intestinal contents and mucosa were examined for the presence of helminthes parasites. Flotations and direct smears were prepared to evaluate samples for the presence of coccidian and helminth ova. Tissues for histopathology were fixed in 10% neutral buffered formalin, embedded in paraffin, sectioned to five microns, and stained with hematoxylin and eosin. A consistent set of tissues for histopathology was collected from each bird: brain, skeletal muscle, heart, esophagus, proventriculus, gizzard, small intestine, large intestine, bursa, cloaca, trachea, lung, thyroid, liver, spleen, kidney, and gonad.

Various tissues were also saved during necropsy for contaminant analyses. Individual liver and kidney tissue samples were double-wrapped in plastic bags. Individual brain tissue samples were double-wrapped in aluminum foil and placed in plastic bags. Samples were frozen until analyzed for contaminants.

Transport

Procedures for transport and shipment of samples followed QA/QC guidelines documented in Rope and Breckenridge (1993), Oregon Fish and Wildlife Office SOPs, and the Field Guide to Wildlife Diseases (Franson 1987). Carcasses were placed in coolers with chemical ice (e.g., Blue Ice) and shipped via overnight express service to the NWHC. Blood samples were shipped

on dry ice to the University of California, Davis. Tissue samples were shipped frozen to the Oregon Fish and Wildlife Office from the NWHC so that these samples could be sent on to the contract laboratories as directed by the U.S. Fish and Wildlife Service's Patuxent Analytical Control Facility (PACF) in Laurel, Maryland (now called the Analytical Control Facility and located in Shepherdstown, West Virginia).

Residue Analysis

Liver tissue was analyzed for trace elements (inorganic compounds), brain tissue for organic compounds, including total PCBs, and kidney tissue for cadmium. Tissue samples from 48 murres were analyzed for organic and inorganic compounds at two analytical laboratories: PACF and Geochemical and Environmental Research Group (GERG) in College Station, Texas. Percent lipid and moisture for these samples are provided in Appendix B.

Inorganics

All elements except for arsenic, selenium, and mercury were analyzed by inductively coupled plasma (ICP) according to Haseltine et al. (1981). Briefly, tissue was dried and ashed prior to dissolving in nitric and hydrochloric acids and adding a scandium internal standard and diluting. Determination was performed using a Perkin Elmer Plasma II sequential ICP emission spectrometer.

Tissue samples were analyzed for selenium and arsenic according to Krynitsky (1987). Determination was by stabilized temperature platform graphite furnace atomic absorption spectroscopy using Zeeman effect background correction. The nominal detection limit was 0.1 ppm wet weight.

At PACF, samples were prepared for mercury analysis by digesting 1 g aliquots of tissue under reflux in sulfuric and nitric acids as described by Monk (1961). The determination was performed by cold vapor atomic absorption spectrophotometry using a Spectro Products mercury analyzer equipped with a Varian VGA-76 vapor generation accessory. The nominal lower limit of detection is 0.05 ppm wet weight. At GERG, mercury analysis followed U.S. Environmental Protection Agency method 245.5 with minor revisions. Samples went through a sulfuric acid and nitric acid digestion prior to being analyzed using a graphite furnace atomic absorption spectrophotometer (Hatch and Ott 1968).

Organochlorines

At PACF, the analytical methodology for organochlorines in tissue, including preparation, Soxhlet extraction, and lipid removal followed methods by Cromartie et al. (1975). The silica gel separation of the pesticides from the PCBs was different from the above reference in that four fractions were used instead of three to enable the separation of dieldrin and endrin from the rest of the pesticides. The pesticides in each fraction were separated with a gas-liquid chromatograph, equipped with a ^{63}Ni electron capture detector. The nominal lower limit of detection was 0.01 ppm for pesticides and 0.05 ppm for PCBs based on a 10 g aliquot wet weight.

At GERG, tissue samples were extracted by the National Oceanic and Atmospheric Administration's Status and Trends method (MacLeod et al. 1985) with minor revisions (Brooks

et al. 1989, Wade et al. 1988). Briefly, a tissue sample was homogenized, extracted under a solvent using a Soxhlet apparatus, and the extract purified by silica/alumina and/or Florisil column chromatography to isolate the pesticide/PCB fraction. This fraction was further purified by high performance liquid chromatograph to remove interfering lipids. The quantitative analysis was performed by a capillary gas chromatograph with an electron capture detector.

Total PCBs were determined as Aroclor PCBs at PACF. GERG conducted congener-specific PCB analysis and summed 72 congeners, including co-eluting congeners, to represent total PCBs.

Quality Control/Quality Assurance

QA/QC procedures for sample contaminant analyses followed guidelines established by Rope and Breckenridge (1993) and individual laboratory guidelines. Document control procedures followed Rope and Breckenridge (1993) and all samples were catalogued using the Environmental Contaminants Data Management System. Laboratory control samples consisting of non-blind split samples (one spiked and one non-spiked) were prepared and analyzed with every sample run to estimate within sample variance, test product recovery, and measure analytical errors. Detection limits are provided in Appendix C.

Similar analytical methods for the same group of analytes were used at PACF and GERG. However, differences in equipment, personnel, and procedural modifications specific to the laboratory may have resulted in different detection limits or analyte recoveries. These differences are a source of variation that can influence data interpretation, although it is expected that variation attributed to differences in laboratory methods would be greatest for values at or near detection. To help identify the magnitude of this variation source, we evaluated quality control information such as procedural blanks, duplicates, and matrix spike samples for each catalog. Average spiked matrix recoveries considered acceptable were 80% to 120%, except for PCB congeners, which had acceptable accuracy between 60% and 140%. Duplicate results for inorganic ICP and organic compounds were considered valid if the average relative percent difference between duplicates was 1) 200% for average analyte concentrations at 0 to 2 times the detection limit; 2) 17.3% for concentrations at 2 to 10 times the detection limit; or 3) 8.6% for concentrations greater than 10 times the detection limit. For inorganic chemicals determined with atomic absorption (arsenic, mercury, and selenium) the average relative percent difference between duplicates was 1) 11.5% for average analyte concentrations at 2 to 10 times the detection limit and 2) 5.75% for concentrations at greater than 10 times the detection limit. Duplicate results for PCB congeners were considered valid if the average relative percent difference between duplicates was 1) 40.3% for average analyte concentrations at 2 to 10 times the detection limit and 2) 20.1% for concentrations at greater than 10 times the detection limit. Chemicals outside the specified limits listed in tabulated results are identified.

For samples analyzed at GERG, under catalog number 1060040, inorganic procedural blanks were reported between <-1.48 and 0.28 total µg. The relative percent difference reported for duplicates in this analysis were all within the range considered analytically valid. Boron had low spike recoveries from 61.9% to 72.5% and iron had one high spike recovery (195%), while all other compounds were within the acceptable range.

Samples analyzed for inorganics at PACF, under catalog number 1060042, had reported procedural blanks all below detection levels. Duplicate results show cadmium (liver), copper, and mercury outside the valid relative percent difference. Compounds outside the range of acceptable spike recoveries included boron, barium, iron, mercury, nickel, and strontium. Recovery from the spike recovery matrix (SRM) was low for several ICP elements, especially iron, strontium, vanadium, and zinc. Instrumental analysis was repeated with no improvement for these elements. Insufficient sample was available for reanalysis. The problem may have been isolated to the SRM because spike recoveries were acceptable. Because there is no means of verification, the results for these elements are questionable.

Samples analyzed for inorganics at PACF, under catalog number 1060048, had procedural blanks all ≤1.92 total µg. Results of duplicate quality control show boron, cadmium, and nickel outside the acceptable relative percent difference based on detection limits. Boron and nickel relative percent differences are acceptable for this data group based upon other animal tissue data in the PACF database that has been analyzed for these elements (Brenda Montgomery, Inorganic Analytical Chemist, Analytical Control Facility, Shepherdstown, West Virginia, pers. comm., 2008). The variability of cadmium in the kidney duplicate was slightly higher than normally seen, but this should have no affect on the interpretation of the data. Compounds with invalid spikes are cadmium (kidney), copper, iron, magnesium, and selenium. Lower spike recovery for boron is acceptable based upon previous PACF recovery data for this analyte in this matrix.

PCB results from GERG (catalog 1060040) show procedural blanks ≤0.0007 µg with the exception of PCB 41/64 which had results of 0.003 and 0.004 µg. Duplicates had relative percent differences within the range considered valid with the exception of PCB congeners 99, 138, 41/64, total PCB, heptachlor epoxide, and p,p'-DDE. Delta BHC and endosulfan II had the lowest spike recoveries reported at 68.4% and 40.6%, respectively. Compounds which did not fall into the acceptable bounds for spike recovery were aldrin, alpha BHC, delta BHC, endosulfan II, gamma BHC. PCB congeners 170 and 41/64 showed matrix interference for the majority of samples. Sample numbers 22 and 30 were confirmed by gas chromatography and mass spectrometry for p,p'-DDE.

Samples analyzed for organics at PACF under catalog number 1060042 had procedural blanks all reported at 0 total µg. No duplicate analyses were reported. Spike recoveries for organic compounds were within the acceptable range.

Organic results presented in catalog 1060048 for brain tissue were approved. Procedural blanks were all reported at 0 total µg. Although there was insufficient sample to run duplicate analysis, PACF indicates that this should not affect the interpretation of the data. Aside from mirex (72%) and o,p'-DDT (76%), spike recoveries were within the acceptable range.

Data Analysis

Murres were grouped according to sex, age (adult or juvenile), disposition (alive or dead), and year collected (1995 or 1996). Geometric means and ranges were determined from residue data received from laboratories. Reported means are antilogarithms of transformed (log10) data (Sokal and Rohlf 1981). Values from chemical analyses below detection limits were assigned a value of one-half the lower limit of detection to eliminate values of 0 for computational

purposes, provided the majority of values were above detection limits. In cases where the majority of sample concentrations were below the detection limits, geometric means were not computed. Differences in means among groups were not compared statistically due to inadequate sample size at one or more locations, or an insufficient number of detectable concentrations. However, mean concentrations were graphed for visual interpretation. Geometric mean or maximum contaminant concentrations were compared to values reported in the literature.

Blood parameters were evaluated to determine the general health of the common murres collected. Comparisons were made with adult common murres from Alaska and California (S. Newman, unpubl. data). Descriptive statistics and *t*-tests were performed using SPSS on blood parameters to determine whether hematologic or biochemical blood results differed significantly ($P \leq 0.05$) between groups of murres from different geographical areas.

Concentrations of the various chemical constituents found in murres collected in this study were compared to literature values either found to produce harmful effects in birds or reported to be a threshold level. Much of the literature used for comparison is from the Eisler (1981-1998) synoptic reviews of hazards to fish, wildlife, and invertebrates, and Beyer et al. (1996). Each Eisler report provides a selective review and synthesis of the technical literature on the particular chemical in the environment and its effects primarily on fish, birds, mammals, terrestrial and aquatic invertebrates, and other natural resources. Tables within the main body of this report include data for only inorganic and organic compounds that have the potential to impact seabirds. Complete tables are included in appendices D through H. For consistency, values from the published literature were converted to ppm or ppb as needed and reported herein for comparison to our results.

RESULTS

Blood

1995 Murres

Statistically significant differences between Oregon and California murres were detected for the following parameters: ALT, AST, CK, albumin, globulin, TP, TS, TBili, DBili, glucose, Ca, HCO_3, Cl, and UA. The only statistically significant difference detected between Alaska and Oregon murres was found in CK levels.

Necropsy

Ten reference and 20 die-off birds were examined by the NWHC (see Table 1). Reference birds were in good or fair body condition (Table 1) while most birds collected during the die-off were emaciated. Other than traumatic lesions associated with euthanasia, no consistent significant gross lesions were observed. Stomachs of both reference and die-off birds were predominately empty. Liver and spleen (n = 4) and intestine (n = 5) from tested reference birds were all negative for virology. Common parasites of seabirds were found in both reference and die-off birds and not associated with significant pathology. However, the younger birds did exhibit a somewhat higher prevalence of mild enteric coccidiosis, larval nematodes within the proventriculus, and nematodes attached to the esophageal mucosa. Differences in histopathology

between the reference and die-off birds were not identified. Granulomas were more common in older birds. The most striking difference between juvenile and adult birds was the higher prevalence of pneumonia in the juveniles. No cause for the pneumonia was found on microscopic examination of tissues; possible etiologies could include acute endotoxemia, septicemia, or an acute generalized infection.

1997 Murres

Murres collected in late June 1997 were all necropsied at the NWHC and results are presented in Table 2. All four murres collected during the die-off event were emaciated. One of the murres had undergone postmortem degeneration to the point it was not suitable for further examination. In the remaining three murres, histopathological examination of tissues and bacteriological studies yielded no significant findings. Preliminary testing of a pool of brain material from two of the murres (samples MU97002 and MU97004) produced mortality of embryonated chicken eggs, but further testing yielded no causative agent.

Brain cholinesterase activity in murre sample number MU97004 was within the normal range. No lead (<0.25 ppm wet weight) was present in the liver of murre sample MU97002. Histopathological examination and bacteriological studies of tissues from the three birds suitable for examination yielded no significant findings.

Residue Analysis

Table 3 displays the detection frequency of organics, inorganics and PCBs. Summary statistics for each category of compounds are presented below in Tables 4 through 8. All individuals had greater than 45% detection of inorganic compounds while organics were below 60% detection for all individuals. The highest detection frequency for PCBs was 51%, although most samples were below 26%.

Inorganics

Summary data for inorganic compounds are presented in Tables 4 and 5. Data for juveniles of undetermined sex collected in 1995 are included in Appendix I. In 1996, geometric means were computed only for males collected along the central coast as other groups did not have an adequate number of individuals (Table 5).

A few of the inorganic compounds which are among the more hazardous to wildlife had greater concentration differences among groups. Mean concentrations of arsenic, mercury, nickel and selenium were higher in 1996 dead males compared to 1995 juveniles and adults, although less apparent in mercury and nickel (Table 4). Mean concentrations of cadmium, mercury, and selenium were less in juveniles than male adults; conversely, copper concentrations were higher in juveniles than adults (Table 4).

Organics

Geometric means and ranges of organic compounds are presented in Tables 6 and 7. Data for juveniles of undetermined sex collected in 1995 are included in Appendix I. Of the 24 organic pesticides analyzed, eight had a detection frequency ≥50%: HCB, total PCBs, beta BHC, dieldrin, heptachlor epoxide, oxychlordane, o,p'-DDT (dichlorodiphenyltrichloroethane) and p,p'-DDE. One analyte, p,p'-DDE, was detected in all but two murre samples. Two of these

compounds, dieldrin and o,p'-DDT, had a detection frequency ≥50% in only one group of murres and therefore could not be compared to others. It should be noted that samples were analyzed at two different laboratories, GERG and PACF. GERG provided lower limits of detection and, therefore, produced a greater number of samples with detectable residues.

Appendix B provides the percent lipid from each of the brain samples analyzed for organics. Statistics indicated there was no relationship between concentrations of p,p'-DDE and percent lipid in the brain ($r = 0.16$, $r^2 = 0.026$, $P = 0.32$); therefore, means were not lipid normalized. Brain lipid was also measured at the two different laboratories (Appendix B).

There is no apparent pattern across geometric means of the majority of organic compounds detected (Table 6). However, the mean concentration of p,p'-DDE is substantially higher (>10 fold) in the 1996 adult dead males than in the 1995 groups (Table 6). DDT and its metabolites were highly intercorrelated with PCBs and total PCBs was only correlated with o,p'-DDT ($r = 0.85$, $P < 0.001$) (Appendix J).

PCBs

Seventy-three individual congeners or co-eluting PCBs were analyzed and descriptive statistics are presented in Appendix H. Data from juveniles of undetermined sex collected in 1995 are included in Appendix I. Of the PCBs included in Appendix H, 13 were above the detection limit in over 50% of samples for several of the murre sample groups. Murre sample number 30 had the highest percentage of PCBs detected and the greatest concentration (at least double all other samples) for PCBs 82, 99, 118/108/149, 138,153, 170,180, 187/182/159, and 201. Sample 30 was a dead adult female collected at Seal Rock on August 1, 1995. Most means were less than or equal to 0.01 ppm wet weight. The highest mean PCB concentration was 0.028 ppm wet weight.

DISCUSSION

Blood

Analyses of blood parameters, including specific enzyme levels, were intended to provide evidence of altered organ function that may also relate to contaminant exposure. The blood analyses from juvenile and adult murres for this study do not demonstrate any significant organ pathology.

ALT and AST are enzymes present in the liver, heart, skeletal muscle and kidney, but considered to be the most liver-specific of avian liver enzymes. AST can also be found in the spleen, small intestines, lung and brain depending on the avian family. In the past, these enzymes also have been used to diagnose potential liver disease but current information suggests that ALT might be preferable to assess liver health. For both of these enzymes, Oregon murres had higher mean serum concentrations than California murres. From a clinical perspective, interpretation of both elevations is difficult.

Muscle damage associated with the gunshot collection is the most likely explanation for differences in ALT and AST as well as the higher mean CK (skeletal muscle enzyme) levels

found in the Oregon murres versus the California and Alaska murres. It is interesting to note that although both the Alaska and Oregon murres were collected by gunshot, the Oregon murres still had higher CK levels. This may be explained by Oregon murres exerting themselves more than Alaska murres before being collected, or alternatively, Alaska murres died more immediately after collection compared to Oregon murres. Increased exertion in either scenario could have significantly elevated CK levels in the Oregon birds.

Four parameters which are also related to liver function and provide some indication of immune and nutritional status include albumin, globulins, TP and TS. Oregon birds had statistically lower mean concentrations of all four parameters compared to the California birds. Lower mean concentrations may be suggestive of potential sublethal problems in the population of birds sampled. The white blood cell counts of the Oregon murres were within reference ranges currently being established for common murres and other pelagic species.

Mean calcium concentration was lower in the Oregon murres compared with California murres whereas the mean phosphorus concentration was higher. This may be explained by differences in the sex of birds sampled. The ratio of males to females is unknown in the California sample but the Oregon murres consisted only of males. It is also possible that the sampling date and breeding chronology may have had an effect on levels of these elements and one would expect females near egg laying, or just after egg laying, to have higher calcium concentrations than male murres.

Uric acid concentrations (used to assess renal function in avian species) were lower in Oregon versus California murres. The difference noted was not of clinical significance. Glucose levels in the Oregon murres were significantly lower than California murres, but again, with no clinical relevance. Although the TBili and DBili are statistically different in the California and Oregon birds, this parameter is no longer considered to be diagnostically useful in assessing avian hepatic function.

In evaluating blood analytes, multiple differences (15 of 23 parameters) were observed between murres associated with colonies in California versus Oregon. In contrast, only one of 12 parameters differed between Oregon and Alaska murres. The most likely reason for the minimal number of differences between Oregon and Alaska blood analytes is because these birds were collected prior to blood sampling. In contrast, California murres were live captured, sampled, and released. Trauma associated with collection is the most logical explanation for the observed differences between California samples and Oregon-Alaska samples, but it is also known that local ecological conditions including food resources, contaminants, infectious agents, and other stressors can result in differences in blood analytes among seabird colonies (Newman and Zinkl 1998).

Necropsy

The purpose of conducting histological examinations of tissues from murres collected from the ocean and recovered during die-offs was to directly measure any changes in organ structure, which might be related to exposure to contaminants or to disease states. However, results do not point to any obvious exposure or disease as no differences in histopathology were identified

between the reference and die-off birds. In addition, other than the higher prevalence of pneumonia in the juveniles, there were no apparent differences between juvenile and adult birds.

Of the 10 reference and 20 die-off birds examined by the NWHC, reference birds were in good or fair body condition while most die-off birds were emaciated. However, because the stomachs of both reference and die-off birds were predominately empty, it is possible that reference birds were malnourished and their long-term survival in question.

Residue Analysis

The discussion is limited to those compounds which were detected in over 50% of samples and have the potential to impact bird populations.

Inorganics

Arsenic

In most living organisms, arsenic concentrations are usually low (<1.0 mg/kg fresh weight) (Eisler 1988a). Although arsenic concentrations in marine biota can be elevated, the arsenobetaine form poses little risk to organisms or their consumer (Eisler 1988a). Concentrations of arsenic found in murres collected in this study are <1.0 mg/kg (ppm) fresh weight, except in the 1996 dead adult males in which the geometric mean was 1.55 mg/kg fresh weight and ranged up to 6.5 ppm fresh weight (Tables 4 and 5). Most information presented by Eisler (1988a) regarding impacts to birds relates to dietary studies and therefore cannot be compared to results of liver concentrations.

Living resources are exposed to arsenic through a variety of sources: by way of atmospheric emissions from smelters, coal-fired power plants, and arsenical herbicide sprays; from water contaminated by mine tailings, smelter wastes, and natural mineralization; and from diet, especially from consumption of marine biota (Eisler 1988a). Arsenic is bioconcentrated by organisms, yet not biomagnified in the food chain and its bioavailability and toxic properties are significantly modified by numerous biological and abiotic factors that include physical and chemical form, exposure route, dose, and species.

Cadmium

Eisler (1985) reports that cadmium residues in vertebrate kidney or liver that exceed 10 ppm fresh weight should be viewed as evidence of probable cadmium contamination and concentrations of 200 ppm fresh weight kidney are probably life-threatening to the organism. Murre concentrations detected in this study are above those considered contaminated by Eisler but well below life-threatening. Kidney concentrations in adult males collected in 1995 (alive) and 1996 (dead) have a geometric mean of approximately 15 ppm fresh weight (Table 4). The two dead adult males collected along the south coast in 1996 had kidney concentrations above those considered contaminated (Table 5), as did one adult female collected in 1996 along the southern coast (Table 5) and a dead adult male collected in 1995 (Table 4). All murre concentrations are within what has been found in healthy wild birds.

Cadmium concentrations range from 5 to 35 mg/kg wet weight in the liver of many seabirds (Furness 1996). Cadmium concentrations in healthy wild birds vary widely with mean levels of

<0.1 to 32 mg/kg wet weight in the liver and <0.3 to 137 mg/kg wet weight in the kidney (Walsh 1990; as cited in Furness 1996). Given the enormous natural variation in cadmium accumulation and sensitivity of birds to its toxics effects, Furness (1996) did not suggest any critical levels of cadmium in tissues that are applicable to all birds. However, he does suggest that about 40 mg/kg wet weight in the liver or 100 mg/kg wet weight in the kidney should be considered tentative threshold tissue concentration, above which cadmium poisoning in birds might be expected. However, Furness cautions that among wild birds, such high tissue concentrations of cadmium appear to occur naturally in a small proportion of individuals in some seabird populations and are believed to be harmless by those reporting. Murre cadmium concentrations from this study do not approach those Furness suggests as tentative threshold concentrations.

Of the 1995 and 1996 samples considered contaminated, the adult male group showed the highest concentrations. Juveniles, on the other hand, all had concentrations well below those considered contaminated. Furness (1996) reports that cadmium tends to increase with age in animals and results of our study agree with the many studies which have shown that cadmium concentrations are higher in adults than in juveniles (Hulse et al. 1980, Maedgen et al. 1982, Lock et al. 1992, Wren et al. 1994).

Molluscs have a particular tendency to accumulate large amounts of cadmium and mollusc-eating birds in coastal areas with high inputs of cadmium can be affected (Furness 1996). Although long-lived birds feeding on molluscs may accumulate high concentrations of cadmium, these birds may also have evolved a greater tolerance to cadmium than other non-mollusc eating bird species. Although fish are the primary prey of common murre adults and chicks, adults can feed on other types of prey including squid, molluscs, polychaetes, and fish eggs (Manuwal and Carter 2001).

Diet composition varies considerably between geographic areas and times of year and various factors can affect prey abundance, availability and location on short- and long-term time scales. Information presented by Manuwal and Carter (2001) suggest that if primary prey species become unavailable, murres switch feeding locations or change their diet dramatically. Manuwal and Carter (2001) report that a 3-year study conducted in Oregon found that northern anchovy (*Engraulis mordax*) and rockfish (*Sebastes* spp.) dominated the murre diet throughout the study. In the late 1990s ocean productivity began to increase with signs that adult murres were likely feeding on abundant euphausiids while still bringing fish to their young. Consequently, it appears that unless preferred prey were not available, murres would not choose molluscs as a favored food.

Cadmium concentrations in the liver and kidney of breeding adult birds can vary by a factor of four between different stages in the breeding season as a result of changes in tissue physiology (Walsh 1990; as cited in Furness 1996). Studies have suggested differences in cadmium levels between the sexes in oystercatchers; however, results are not consistent with which sex had the highest concentrations (Hutton 1981, Stock et al. 1989). Furness (1996) also notes that it has been difficult to show an elevation in cadmium concentrations in birds inhabiting environments considered to be polluted by cadmium.

Cadmium is often associated with zinc as they are commercially produced by smelting their mixed ores and are used in industrial processes such as plastic production, electroplating, and the manufacture of alloys and batteries (Furness 1996). Positive correlations have been observed between cadmium and zinc in seabird tissues (Hutton 1981). Sources of cadmium along coastal areas include sewage sludge or smelter or refinery discharges. As Oregon has no smelters or refinery discharges along its coast, either other sources are contributing or the birds are accumulating cadmium outside Oregon. In addition, zinc levels do not appear to be elevated (see *Zinc* below).

Chromium

Although the significance of tissue chromium residues is unclear, Eisler (1986a) reports that tissue levels in excess of 4.0 mg total chromium/kg dry weight should be viewed as presumptive evidence of chromium contamination. Several of the murres collected for this study show concentrations above the 4.0 mg/kg level. Eisler (1986a) states that proposed criteria for the protection of various environmental compartments against chromium are numerous, disparate, and often contradictory which may be attributable to the inability to quantify chemical species and ionic states of chromium. Concentrations of chromium are usually highest at the lowest trophic levels.

Copper

Although numerous copper criteria are proposed for protecting the health of agricultural crops, aquatic life, terrestrial invertebrates, poultry, laboratory rats, and humans, no copper criteria are available for protection of avian wildlife. All studies with birds and copper have evaluated domestic chickens, ducks, or turkeys and extrapolation of results from poultry to wildlife species is not advised given the wide range in sensitivities to copper between species (Eisler 1998a).

In general, birds retain a very small portion of copper and other metals ingested (Bryan and Langston 1992). It is noted by Schroeder et al. (1966) that mammals and birds are 100 to 1,000 times more resistant to copper than other, more primitive animals. Birds from contaminated sites may contain as much as 367 mg/kg dry weight in livers (Eisler 1998a). A few of the livers from murres collected in this study approached this concentration reported by Eisler with the highest concentrations found in juveniles.

In animals, copper interacts with essential and nonessential elements and these interactions may be either beneficial or harmful to the organism (Kirchgessner et al. 1979; as cited in Eisler 1998a). The patterns of copper accumulation, metabolism, and toxicity from these interactions frequently differ from those produced by copper alone. Copper and zinc were found to be positively correlated in kidneys and livers of common murres (Stewart et al. 1994). Copper concentrations were found to be positively correlated with both cadmium and zinc in kidneys of the willow ptarmigan (*Lagopus lagopus*; Wren et al. 1994).

Copper concentrations in tissues of coastal seabirds tend to decrease with increasing age (Eisler 1998a). In New Zealand, younger marine birds showed higher concentrations of copper in livers than adults (Lock et al. 1992). This also seemed apparent in our study. Conversely, Stewart et al. (1994) found that juveniles and adults of common murres from Scotland have similar concentrations of copper in kidney, liver, and muscle.

In avian tissues, season of collection and age can affect copper concentrations. Eisler (1998a) reports that in livers from surf scoters (*Melanitta perspicillata*) from San Francisco Bay, copper concentrations are higher in March than in January; in livers from canvasbacks (*Aythya valisineria*) from Louisiana, concentrations are lower in November than other months; and in primary flight feathers of mallards (*Anas platyrhynchos*) and black ducks (*Anas rubripes*) from the vicinity of a smelter in Sudbury, Ontario, copper concentrations are highest in autumn. Consequently, concentrations found in murres from Oregon in summer may be higher or lower at other times of the year.

Lead

Our study findings do not indicate lead is a major problem for the murres as most groups had no detectable lead. Proposed lead criteria for the protection of natural resources (>2 mg/kg fresh weight) listed by Eisler (1988b) are above those found in the murres collected in this study. Furthermore, concentrations found in these murres are below those reported as background (<2.0 ppm wet weight liver) in waterfowl (Pain 1996).

Eisler (1988b) states that forms of lead other than shot or ingestible lead objects, or routes of administration other than ingestion, are unlikely to cause clinical signs of lead poisoning in birds. Lead shot is a substantial localized source of contamination, primarily in waterfowl habitat including lakes, marshes, and estuaries. Because common murres are seabirds, they sometimes forage in estuarine environments (Csuti et al. 1997) and have potential for exposure to this lead source. Eisler (1988b) found lead residues greatest in older birds. This contrasts with our findings which show juveniles of undetermined sex to have the highest tissue concentrations of lead (Tables 4 and 5 and Appendix I).

Mercury

Eisler (1987) maintains that concentrations in excess of 1.1 mg/kg fresh weight of tissue should be considered as presumptive evidence of an environmental mercury problem. Mercury concentrations found in murre tissue samples collected in this study show adult males with the highest concentrations; 1996 males collected from the central coast exceed levels Eisler (1987) considered evidence of a mercury problem and 1995 males approach this level.

Thompson (1996) notes that, in general, seabirds exhibit higher mercury concentrations than terrestrial birds because of higher mercury burdens encountered in marine ecosystems and, therefore, seabirds are more likely to be able to tolerate higher concentrations of mercury before toxic effects become evident. Thompson (1996) states that non-marine birds with mercury concentrations in the liver and kidney in excess of approximately 20 to 30 mg/kg wet weight were likely to suffer toxic effects and ultimately even death. Yet mercury concentrations far in excess of the 30 mg/kg level have been recorded, particularly in seabirds, which were apparently healthy and reproducing normally (Muirhead and Furness 1988, Honda et al. 1990, Lock et al. 1992). The form of mercury measured in the seabirds reported by Thompson (1996) was predominantly inorganic, suggesting that biotransformation of ingested methylmercury is an important mechanism by which seabirds avoid the toxic effects. The most stable and toxic form of mercury to wildlife is methylmercury (Eisler 1987), which is the form that most top avian and mammalian predators are exposed to through their diet (Thompson 1996).

Mercury occurs naturally in the environment and also can be augmented by significant anthropogenic emissions. According to the Oregon Department of Environmental Quality (2003), mercury is released to the air and water from many regulated point sources within Oregon, including publicly operated treatment works, power plants and manufacturing facilities. Non-point sources of mercury include abandoned gold and mercury mines, as well as air emissions from motor vehicles. In addition, if any of the commonly used products containing mercury (e.g., thermostats, fluorescent lamps, thermometers, automobile switches and dental amalgam) are disposed of improperly, the mercury may ultimately reach the ocean.

Nickel

Tissue concentrations of nickel may not be reliable indicators of potential toxicity in birds because adverse effects, including death, frequently occurred in the absence of elevated tissue nickel concentrations (Outridge and Scheuhammer 1993). For bird monitoring purposes, analysis of kidney, bone, and feathers is most likely to reveal elevated exposure to environmental nickel contamination while nickel concentrations in liver and spleen often do not reflect elevated exposure (Outridge and Scheuhammer 1993). Nonetheless, our study demonstrates liver concentrations of nickel in murres collected along the Oregon Coast.

Nickel concentrations in the organs of most avian wildlife species in unpolluted ecosystems range from about 0.1 to 2.0 mg/kg dry weight and occasionally reach 5.0 mg/kg dry weight (Eisler 1981, as cited in Eisler 1998b; Outridge and Scheuhammer 1993). However, it is reported that nickel residues in avian liver in excess of 3 mg/kg dry weight are sometimes associated with adverse effects in sensitive bird species (Outridge and Scheuhammer 1993). Concentrations of nickel found in murres collected for this study are similar to what Eisler (1998b) reports as nickel concentrations from unpolluted ecosystems, although the highest mean nickel concentration found in dead adult males collected in 1996 exceeded the 3 mg/kg dry weight concentration which has been associated with adverse effects in sensitive bird species.

Selenium

Selenium is an essential trace element that birds need in small quantities. However, the range of dietary concentrations that provides adequate but nontoxic amounts of selenium is narrow compared with ranges for other elements (Heinz 1996). Selenium has several different chemical forms; the four common oxidation states are selenide (-2), elemental selenium (0), selenite (+4), and selenate (+6). Of these, organic selenides pose the greatest hazard and selenomethionine seems to be the most likely form to harm wild birds (Heinz 1996).

Heinz (1996) recommends, based on laboratory and field data, that a concentration greater than 10 ppm wet weight selenium in the liver be considered potentially harmful to the health of young and adult birds via sublethal effects. In addition, he states that wet-weight concentrations exceeding 20 ppm may jeopardize the survival of young and adult birds. Concentrations above about 3 ppm in the liver of laying females may be associated with reproductive impairment. There is a very high risk of embryonic deformity when the population selenium concentration in the liver exceeds 9 ppm wet weight, while populations with means below about 3 ppm wet weight selenium in liver generally did not have many deformed embryos. Mean selenium liver concentrations from murres collected along the Oregon coast are below 5 ppm wet weight. The

highest concentrations were found in adult males where an individual concentration in the liver slightly exceeded 5 ppm. The one adult female that was collected in 1995 showed a concentration of 2.61 ppm, which is just under the 3 ppm liver concentration which may be associated with reproductive impairment.

Zinc

Eisler (1993) states that zinc poisoning usually occurs in birds when the liver or kidney contains >2.1 g/kg (parts per trillion; ppt) dry weight which is substantially higher than the concentrations found in the Oregon birds. However, zinc interacts with numerous chemicals including cadmium, copper, lead and nickel in ways that can greatly differ from those produced by zinc alone (Eisler 1993). Our murre samples had several of these chemicals reported to interact with zinc. However, it is difficult to interpret what significance these chemicals may have in combination with one another.

Organics

DDT and Transformation Products

Concentrations of o,p'-DDT were found in brain tissues of murres collected in 1995 across the various groupings (Table 6). However, other than the dead juvenile females, DDT was only detected in one individual per group. The highest DDT concentration was 28 ppb wet weight with all others less than 4 ppb wet weight.

There are limited data identifying potential sources of DDT for murres collected along the Oregon coast, but it likely accumulated from their diet. Along the west coast of the U.S., common murres range from California to Alaska (Sibley 2000). Therefore, their range is restricted to states where DDT has been banned for decades. Elliott and Noble (1993) state that data on seabirds indicate that some coastal areas are still highly contaminated with residues of persistent organochlorine pesticides even 20 years after severe restrictions on their production and use. The calculated DDT half-life of farm soils from the southern U.S. (low in organic matter) is 5 to 20 years, although this can vary 1,000-fold depending on the past application rate, organic matter content, and cultivation practices (Pelley 2006). Thus, it is possible that the murres are being exposed to historic sources of DDT. If Oregon murres are moving up and down coast during the non-breeding season, they may be accumulating DDT from their prey in hot spots along the west coast.

It is also possible that the source of DDT for these murres' prey is the open ocean. Outside the breeding season, murres winter at sea (National Audubon Society 2007). The pathway for these compounds may be through atmospheric transport or via particulate matter carried by ocean currents. DDT in air and water samples from the North Pacific was higher than in the North Atlantic or the Gulf of Mexico (Elliott and Noble 1993). In most western Pacific countries, agricultural uses of organochlorine pesticides are subject to regulation. Elliott and Noble (1993) report that many Asian countries bordering the North Pacific have banned DDT (China, Japan, Korea); however, in Hong Kong there appears to be continued input of DDT into coastal waters despite imposed restrictions. DDT continues to be used in South Africa for malaria control and agriculture (Lubick 2007). Although there is no direct ocean current that could provide transport to the west coast of the U.S., DDT may be transported atmospherically.

DDE concentrations were detected in brain tissues across all groupings of 1995 murres as well as in the 1996 die-off birds. Die-off birds had the highest concentrations with a geometric mean of 3,800 ppb. Common murres collected in our study had brain residues of DDE that are considerably lower than those reported to cause direct mortality. Stickel et al. (1984b) concluded a strong likelihood of death among four species of wild passerine species tested at brain residues ≥300 μg/g (ppm) wet weight DDE. Blus (1996) states that there are a few possible cases of lethal DDE levels in the brains of wild birds in the U.S. One is the bald eagle (*Haliaeetus leucocephalus*) at a concentration of 385 ppm wet weight (Belisle et al. 1972), a great blue heron (*Ardea herodias*) at 246 ppm wet weight (Call et al. 1976), a black-crowned night-heron (*Nycticorax nycticorax*) at 230 ppm wet weight (Ohlendorf et al. 1981), and American kestrels (*Falco sparverius*) at 213 and 301 ppm of DDE, wet weight (Porter and Wiemeyer 1972).

After an abnormally high common murre die-off in 1969, Scott et al. (1975) collected specimens and found that levels of p,p'-DDE in the brain tissues of dead adults and juveniles were higher than in healthy individuals collected around the same time. In addition, both healthy and dead juveniles had lower concentrations of DDE than corresponding adults. Our results also indicate that dead murres had elevated p,p'-DDE concentrations compared to live murres and that adults had higher concentrations than juveniles. However, this is only a trend as statistical comparisons were not appropriate with our data.

DDD concentrations were detected in a few individuals at concentrations lower than 8.1 ppb wet weight (Table 6). Lethal DDD concentrations in the brain reported by Blus (1996) ranged between 3 to 59 ppm following dosing test birds with DDT, while those given DDD dosages contained an average of 172 ppm at death with an individual lower level of 86 ppm. Stickel et al. (1970) concluded that brain concentrations of ≥65 ppm indicate increasing likelihood of death from DDD poisoning. Therefore, it is apparent that DDD is not a cause of direct mortality in the murres collected for this study.

The emaciated state of specimens collected in this study may suggest a recirculation of DDT and its metabolites. The concept that DDT stored in fat could be recirculated in animals during starvation and metabolism of fat reserves was reported many years ago (Stickel 1968, Van Velzen et al. 1972, Harvey 1967). Stickel (1968) found that in brown-headed cowbirds (*Molothrus ater*) exposed to lethal concentrations of DDT in the diet, the time to mortality was related to beginning body weight with the lightest birds tending to die first and the heaviest dying last. Although these heavier birds lost a greater percentage of their weight before death, they retained a greater percentage of fat in their tissues at death. This implies that if murres had lethal levels of DDT in their body, starvation could have caused them to die earlier. Van Velzen et al. (1972) also provided an experimental demonstration of lethal mobilization of DDT by brown-headed cowbirds and the effects of food deprivation on the distribution and loss of DDT, DDD, and DDE. A dietary dosage of 100 ppm DDT was provided for 13 days, followed by 2 days of full untreated rations and then a 43% of normal food reduction. Results showed that neither weight loss nor dosage alone produced mortality. Prior to weight loss resulting from food restriction, brains of DDT-dosed birds had concentrations of DDT and DDD that were well below the lethal level while the concentrations in the carcasses were high. During the period of food restriction, DDT and DDD brain concentrations had a rapid and pronounced increase to

lethal concentrations and were far higher than concentrations in birds sacrificed immediately before food reduction (Van Velzen et al. 1972). In contrast, DDT residues in carcasses decreased while residues of DDD and DDE increased showing mobilization and redistribution of residues to the brain as well as metabolic changes of DDT. A pilot study with dosage levels of 100, 200, and 300 ppm DDT had birds subjected to two periods of food restriction, immediately after dosage ceased and 4 months later. DDT-dosed birds from all treatment groups died in each period of food restriction. The authors concluded that stored residues of DDT present a lethal hazard to birds during periods of fat mobilization caused by reduced food supply. Harvey (1967) found dying birds which had been fed DDT followed by food withdrawal had brain residues 3 times higher than surviving birds. Other environmental stresses which also reduce fat reserves (reproduction, cold weather, disease, injury, and migration) could have the same effect on birds containing fat residues of DDT/DDE. Although DDT and DDE concentrations in brain tissues of common murres collected in our study were not sufficient to cause mortality, DDT and DDE might have been stored in the fat and remobilized during a period of starvation, possibly increasing morbidity or susceptibility to mortality in these birds.

PCBs

PCBs were widely used throughout the world from about 1930 to 1976, but since 1977 all production in the U.S. has ceased (Eisler and Belisle 1996). Due to their insolubility, resistance to degradation, and high bioaccumulation potential (Rice and O'Keefe 1995), PCBs are ubiquitous and have been identified in environmental samples from polar regions (Eisler and Belisle 1996). Of the 209 PCB isomers and congeners which vary in toxicity and other biological effects (Kannan et al. 1989), about 100 to 150 are represented in compounds that have been used and are widely dispersed (Rice and O'Keefe 1995). A significant part of the toxicity associated with commercial PCB mixtures is related to the presence of the small number of planar congeners. Twenty of the 209 congeners can assume a planar configuration because of the absence of chlorine substitution in the ortho positions (Eisler and Belisle 1996).

PCBs resist bacterial and chemical breakdown and are readily absorbed from water into the fats of plankton which can make their way into fish and then piscivorous birds, accumulating higher concentrations as they become deposited in the body fat as the natural portions of food are metabolized (Hoffman et al. 1996). Eisler (1986b) states that in birds, liver concentrations were highest in birds that feed on fish, followed by those that feed on small birds and mammals, then worm and insect eaters with the lowest found in herbivorous species. Higher total PCB and planar PCB concentrations were found in eggs and tissues of fish-eating seabirds compared to those that fed primarily on invertebrates (Focardi et al. 1988, Gonzalez et al. 1991, Rice and O'Keefe 1995). Fish are the primary prey of common murre adults and chicks (Manuwal and Carter 2001) and, therefore, murres have a high potential to accumulate PCBs in contaminated areas.

Eisler and Belisle (1996) report that among PCB-contaminated sites and in fish-eating species, total PCB concentrations in birds were usually higher in males and in eggs than in livers and that PCBs 138 and 153 tended to predominate in all samples. Similarly, our findings indicate PCB congeners 138 and 153 had the highest concentrations. In our study of Oregon murres, congeners 41/64, 146, 128, 118/108/149, and 60/56 were among the next highest concentrations.

Hoffman et al. (1996) found that PCB brain residue was the most diagnostic tissue residue associated with lethality in adult birds. Eisler and Belisle (1996) report a proposed PCB criteria for the protection of birds as <300 mg/kg fresh weight in the brain, based upon the notation by Bryan et al. (1987) that mortality in birds usually occurs upon the accumulation of 300 ppm PCBs in the brain. Rice and O'Keefe (1995) report that lethality in birds appears to be correlated with brain residue levels and concentrations around 300 ppm appear to be the threshold where toxic effects first begin to appear. Dahlgren et al. (1972a) concluded that a brain residue level of 300 to 400 ppm PCB as Aroclor 1254 was indicative of death due to PCB toxicosis and further study by these authors (Dahlgren et al. 1972b) showed that periodic food deprivation increased brain residues leading to more rapid death. Stickel et al. (1984a) consider 310 ppm in the brain of birds to be diagnostic of PCB-inducted mortality. PCB concentrations in common murres collected in 1969 during a die-off had brain levels averaging 4.0 ppm (Scott et al. 1975). Live birds collected during the same die-off had an average of 3.7 ppm in the brain and a sample collected in 1970 had an average of 1.1 ppm. The highest total PCB concentration detected in our study was 4.0 ppm wet weight with all others <0.50 ppm wet weight. Brain concentrations of PCB from murres collected in our study compared favorably to those from Scott et al. (1975) and were far below those considered to cause toxicosis or mortality.

The most toxic PCB congeners, due to their dioxin-like toxicity, include the non-ortho-chlorinated PCBs 77, 126, and 169 (Hoffman et al. 1996), but only PCB 77 was analyzed in this study (Table 8). There were only two detects among all samples, a reference juvenile male and a dead juvenile female (Appendix H). Of the mono-ortho planar PCBs (105, 114, 118, 123, 156, 157, 167, and 189), only 105, 118, 156, 167, and 189 were included in the chemical analysis. Both di-ortho planar PCBs (170 and 180) were included in the analysis.

Die-offs and Population Trends

Murre die-offs have been occurring for decades along the Pacific Coast. Bayer et al. (1991) report large numbers of murre deaths along the Washington and Oregon coasts since the early 1900s. In the later half of the 20th century, Scott et al. (1975) reported a 1969 die-off that occurred during late July and early August with both adult and young birds affected, but with young birds outnumbering adults two to one. Bayer et al. (1991) report murre mortalities every summer from 1978 through 1990. These die-offs have continued through the 1990s and well into the current decade, and die-offs have occurred in California (Goodfriend 1995, Woolfolk 1995) and Washington (Associated Press 2006).

Attributing the die-offs to one cause is difficult given the geographical expanse of the die-offs and the numerous potential causes. However, one possible cause of the die-offs is a lack of primary productivity in coastal waters causing the forage fish to move offshore and out of reach of the murres (R. Lowe, pers. comm., 2011). The observation that dead birds often show signs of starvation supports depleted food resources as one cause of mortality. A phenomenon known as upwelling has arrived later than normal in the past few years. Upwelling occurs when a flush of cold, nutrient-rich water surges up from the deep ocean along the coast, supporting the food chain that these birds rely upon. Upwellings can result in negative impacts as well; if the upwelling is too strong, the increased nutrients create massive algal blooms followed by oxygen depletion when the algae die, resulting in a "dead zone" (Milstein 2007). Strong upwellings can

also transport plankton, eggs, and larval fish off of the continental shelf where they are lost (R. Lowe, pers. comm., 2011).

Although starvation and lack of food from late upwellings has been suggested as the primary cause of common murre mortalities, other factors could also be involved. Red tide, caused by heavy blooms of the toxic dinoflagellate (*Gymnodinium breve*), is believed to cause massive die-offs of waterfowl and seabirds. Forrester et al. (1977) reported an epizootic that killed several thousand lesser scaup (*Aythya affinis*), which occurred concurrently with the red tide and evidence is suggestive that the red tide was partially responsible. In fall 2009, thousands of birds died along the Washington, Oregon, and California coastlines (Oregon Public Broadcasting 2009) as a result of *Akashiwo sanguine* algae; however, this die-off was not the result of toxicity but rather the algae being whipped into a foam with surfactant properties that decreased the waterproofing of birds' feathers leading to hypothermia (NOAA, National Centers for Coastal Ocean Science 2011).

Mortality events of common murres seem to have a significant impact on the murre population. Carter et al. (2001) reported that the number of murres sampled in Oregon colonies increased from 1988 to 1990. In 1993, ENSO brought conditions harmful to breeding with warm marine waters resulting in complete murre reproductive failure. Prior to this, colony abandonment to this degree had not been reported in Oregon. Murres returned in large numbers in 1994 and increased further in 1995, and the ENSO did not result in large changes in the numbers of breeding murres in Oregon. From 1988 to 1995, the breeding population in Oregon has been relatively stable. Carter et al. (2001) believe that stable murre populations from 1988 to 1995 coincided with a period of relatively low anthropogenic effects before and during this period. However, since 1994 the recovering bald eagle population along the Oregon north and central coast has expanded greatly resulting in large-scale disruption of reproductive efforts of common murres in this area. Bald eagles forage on murres at the colony sites on a daily basis with up to 13 eagles observed simultaneously at Three Arch Rocks. The disturbance from foraging eagles has become so severe that few if any murre chicks are being produced north of Yaquina Head near Newport. Many colony sites have been abandoned and new smaller colony sites are appearing at other locations. The population monitoring for common murres traditionally was conducted by counting birds from aerial photographs taken during the breeding season at these large colony sites; however, the disturbance and abandonment now make this technique unreliable for accurately monitoring the population. Because few murres are being produced along this stretch of coastline, the number of dead juvenile birds found on beaches has declined sharply. Adult murre mortality events continue, but not on an annual basis as in the past. Because of the lack or reproduction, the murre population is believed to be in decline (R. Lowe, pers. comm. 2011).

Although oil spills have not killed large numbers of murres in Oregon, spills in Washington and British Columbia have killed large numbers of murres from Oregon colonies because of the northward movements of Oregon murres after colony departure (Manuwal and Carter 2001). Manuwal and Carter (2001) report that a substantial northward movement of Oregon murres (700,000 breeding birds) occurs into the outer coast of Washington and southern British Columbia in July through September; these murres depart from colonies in Oregon usually between late June and late July. Murres frequented the shelf-edge banks for most of the year and

nearshore coastal waters were used in late summer, according to a study of at-sea murre distribution in 1989 and 1990 (Briggs et al. 1992 and Tyler et al. 1993; as reported in Manuwal and Carter 2001). Generally, only a few murres are present in Oregon outside of the breeding season, primarily off northern Oregon but a small number off southern Oregon. In Washington, two major oil spill events caused murre mortalities in the tens of thousands. In 1988, it is estimated that 30,000 murres were killed by the Nestucca spill and in 1991 between 3,740 and 19,559 were killed during the Tenyo Maru spill (Ford et al. 1991; Tenyo Maru Oil Spill Natural Resource Trustees 2000, as cited in Carter et al. 2001). It is quite likely that a substantial proportion of the birds killed as a result of the Nestucca spill were from Oregon breeding colonies. Warheit (1996, as cited in Carter et al. 2001) estimated between 42% and 61% of murres killed in the Tenyo Maru spill were from Oregon murre colonies.

Results of residue analysis conducted in this study do not indicate any obvious chemical concentrations which would directly cause mortality. However, it is possible that the levels of contaminants contributed to the mortality by lessening the murres ability to withstand stressful conditions. Wiemeyer et al. (1986) postulated that die-offs of kestrels in their studies may have resulted from the additional stress of weight loss which may have resulted from temperature declines, reproduction, or molting. The weight loss could have caused mobilization and redistribution of contaminants within the body going to the brain. Scott et al. (1975) also report that during a common murre die-off along the Oregon coast, p,p'-DDE and PCB concentrations were less than the reported lethal concentrations in birds. However, the authors suggest that environmental stress may have been sufficient to contribute to mortality through effects on metabolism and behavior. Friend and Trainer (1970) showed that ducks fed sublethal concentrations of p,p'-DDT were more susceptible to duck hepatitis virus.

Results of our study may be reflective of contaminant concentrations in marbled murrelets inhabiting Oregon. Marbled murrelets belong to the same family as common murres, Alcidae, and have overlapping distributions along the Pacific Coast (Nelson 1997, Ainley et al. 2002). Foraging areas and prey composition are similar for both birds, although common murres generally tend to feed further offshore (Nelson 1997, Ainley et al. 2002). Given these similar life history patterns, our evaluation of contaminant residues in murres indicates that marbled murrelets may be experiencing similar contaminant exposure and tissue concentrations. If so, tissue contaminant levels may be contributing cumulative stress to the murrelets. Although no murrelet die-offs have been observed, the birds' reduced numbers and unique life history (i.e., nesting far inland) may result in a lack of carcasses being recovered on beaches. If they are experiencing mortalities, they may be dying deep in the coastal old growth forests where they nest and where observations are difficult.

Management Recommendations

Results of this study do not indicate any contaminant concentrations that would directly cause mortality in common murres; however, murres are accumulating some inorganic and organic contaminants to low concentrations that may have sublethal effects. In addition, blood analysis and necropsy results were not suggestive of contaminant exposure or significant disease, but they provided further evidence that starvation from limited food resources is likely a significant contributor to annual die-off events. Further research is warranted to examine murre populations

annually and to perform colony health screens at other geographical locations to determine acceptable variability on blood parameters and changes which indicate potential health problems.

The tissue residue analyses suggest that common murres in Oregon are likely exposed primarily to non-point source pollutants (e.g., those from land runoff or atmospheric deposition), which are not readily controlled through direct management actions. Consequently, the only true management recommendation from this study is to continue ongoing efforts in Oregon to decrease stress on common murres from disturbance by boats, aircraft, and people on foot. Such disturbances increase the potential for breeding colony abandonment and cause general stress that may exacerbate the effects of limited food resources, low levels of environmental contaminants, minor disease states, or any combination of these factors. Given that natural disturbance and predation from bald eagles at north and central Oregon coast murres colonies is now severely impacting murre reproduction, it is imperative that anthropogenic disturbance does not further exacerbate this situation or result in adverse impacts at colonies further south that remain productive.

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Table 1. Necropsy results for 40 common murres collected in 1995 from the central coast of Oregon. Sample numbers in bold indicate birds that were necropsied at the School of Veterinary Medicine, University of California, Davis. Sample numbers in regular type indicate birds that were necropsied by the National Wildlife Health Center, U.S. Geological Survey, Madison, Wisconsin. Age is either J = juvenile or A = adult and sex is either M = male, F = female, or U = unknown. Diagnoses are listed in order of importance. Blanks represent unreported diagnoses.

Murre	Age/ Sex	Body Condition	Final Diagnosis[a] 1	Final Diagnosis 2	Other Diagnostics
Reference					
MU01	A/M	Good			
MU02	J/F	Fair	Euthanasia	Cholangiohepatitis	
MU03	A/M	Good	Euthanasia	Cholangiohepatitis	*Contracaecum*
MU04	A/M	Good			
MU05	A/M	Good			
MU06	A/M	Fair			
MU07	J/U	Fair	Parasites		
MU08	J/U	Good			
MU09	A/M	Fair			
MU10	A/M	Good	Euthanasia	Pneumonia	*Contracaecum*
MU11	J/M	Fair	Euthanasia	Pneumonia	*Contracaecum*, Cestode
MU12	A/M	Fair	Euthanasia	Cholangiohepatitis	Nematode, Cestode
MU13	J/M	Fair	Euthanasia	Cholangiohepatitis	Pneumonia
MU14	J/U	Emaciated	Emaciation	Parasites	Brain hematoma
MU15	J/U	Good	Cranial hematoma		
MU16	J/U	Good	Parasites	Cranial hematoma	
MU17	J/F	Fair	Euthanasia	Cholangiohepatitis	Pneumonia
MU18	A/M	Good	Euthanasia		
MU19	J/M	Good	Euthanasia	Pneumonia	Hepatitis
MU20	A/M	Good	Euthanasia	Cholangiohepatitis	
Die-off					
MU21	J/F	Emaciated	Emaciation	Nematode	Cestode
MU22	A/M	Emaciated	Emaciation	*Contracaecum*	
MU23	J/F	Good	Undetermined		
MU24	J/F	Emaciated	Emaciation	*Contracaecum*	Nematode
MU25	J/M	Emaciated	Emaciation		
MU26	J/M	Emaciated	Emaciation	Pneumonia	*Contracaecum*, *Acuaria*
MU27	J/U	Poor	Hepatitis	Pneumonia	
MU28	J/U	Emaciated	Emaciation	Nematodes	
MU29	J/U	Fair	Trauma	Cestode	Nematode
MU30	A/F	Emaciated	Emaciation		
MU31	J/M	Emaciated	Emaciation	*Contracaecum*	Nematode
MU32	J/F	Emaciated	Emaciation	Pneumonia	*Pasteurella multocida*
MU33	J/M	Emaciated	Emaciation	Pneumonia	
MU34	J/F	Emaciated	Emaciation	Pneumonia	
MU35	J/M	Emaciated	Emaciation	Pneumonia	
MU36	J/F	Emaciated	Emaciation	Pneumonia	
MU37	J/F	Emaciated	Emaciation	Pneumonia	
MU38	J/M	Emaciated	Emaciation		
MU39	J/M	Emaciated	Emaciation	Pneumonia	
MU40	J/M	Emaciated	Emaciation	Pneumonia	

[a]Diagnostic definitions: *Acuaria*–parasitic nematode; cestode–parasitic flatworm; cholangiohepatitis–inflammation of the biliary structures (gallbladder and bile ducts); *Contracaecum*–parasitic nematode; cranial hematoma–accumulation of blood in the skull; emaciation–wasting condition of the body; euthanasia–ending the life of an individual; hepatitis–inflammation of the liver; nematode–roundworm; parasites–organism (e.g., viruses, bacteria, fungi, arthropods, protozoa) that lives on another contributing nothing to the survival of the host; *Pasteurella multocida*–coccobacillus bacteria; pneumonia–lung infection; trauma–serious or critical bodily injury, wound or shock.

Table 2. Necropsy results for four common murres collected in 1997. Diagnoses are in order of importance.

Murre	Age /Sex	Body Condition	Final Diagnosis 1	Final Diagnosis 2	Other Diagnostics
MU97001	A/F	Emaciated	Emaciation		
MU97002	A/M	Emaciated	Emaciation		
MU97003	A/F	Emaciated	Autolyzed	Emaciation	
MU97004	A/M	Emaciated	Emaciation	Parasite Proventriculus	Parasite Intestine

Table 3. Detection frequency (%) in each of the analytical categories for each common murre sample. Location indicates birds were collected on central or southern Oregon coast. Disposition (Disp.) is either alive (A) dead (D) or moribund (M), age is adult (A) or juvenile (J), and sex is male (M) or female (F). NA = not analyzed.

Sample Number	Location	Disp.	Age/ Sex	Inorganics (%)	Organics[1] (%)	PCBs	Laboratory[2]
1995							
MU01	Central	A	A-M	75	5	NA	PACF
MU03	Central	A	A-M	55	33	26	GERG
MU04	Central	A	A-M	70	5	NA	PACF
MU05	Central	A	A-M	80	5	NA	PACF
MU06	Central	A	A-M	80	5	NA	PACF
MU09	Central	A	A-M	75	5	NA	PACF
MU10	Central	A	A-M	55	29	17	GERG
MU12	Central	A	A-M	70	21	15	GERG
MU18	Central	A	A-M	55	25	10	GERG
MU20	Central	A	A-M	60	25	15	GERG
MU22	Central	D	A-M	70	46	10	GERG
MU30	Central	D	A-F	65	50	51	GERG
MU11	Central	A	J-M	55	21	11	GERG
MU13	Central	A	J-M	65	21	17	GERG
MU19	Central	A	J-M	55	13	6	GERG
MU25	Central	D	J-M	70	38	24	GERG
MU26	Central	D	J-M	70	17	14	GERG
MU38	Central	D	J-M	50	29	18	GERG
MU31	Central	M	J-M	60	29	18	GERG
MU33	Central	M	J-M	75	38	21	GERG
MU35	Central	M	J-M	70	33	15	GERG
MU39	Central	M	J-M	55	29	18	GERG
MU40	Central	M	J-M	60	29	17	GERG
MU02	Central	A	J-F	50	21	18	GERG
MU17	Central	A	J-F	55[a]	21	13	GERG
MU21	Central	D	J-F	65	58	44	GERG
MU23	Central	D	J-F	75	21	17	GERG
MU24	Central	D	J-F	75	33	22	GERG
MU32	Central	D	J-F	80	25	18	GERG
MU34	Central	M	J-F	70	29	21	GERG
MU36	Central	M	J-F	60	42	24	GERG
MU37	Central	M	J-F	55	21	17	GERG
MU07	Central	A	J-U	60	0	NA	PACF
MU08	Central	A	J-U	55	0	NA	PACF
MU14	Central	A	J-U	45	5	NA	PACF
MU15	Central	A	J-U	50	5	NA	PACF
MU16	Central	A	J-U	60	5	NA	PACF
MU27	Central	D	J-U	65	13	13	GERG
MU28	Central	D	J-U	55	13	7	GERG
MU29	Central	D	J-U	50	8	6	GERG

Table 3 continued.

Sample Number	Location	Disp.	Age/ Sex	Inorganics (%)	Organics[1] (%)	PCBs	Laboratory[2]
1996							
MU41	Central	D	A-M	75	15	NA	PACF
MU42	Central	D	A-M	75	15	NA	PACF
MU43	Southern	D	A-M	80	15	NA	PACF
MU44	Southern	D	A-M	75	15	NA	PACF
MU45	Central	D	A-M	80	25	NA	PACF
MU46	Central	D	A-M	75	25	NA	PACF
MU47	Southern	D	A-F	80	15	NA	PACF
MU48	Southern	D	A-F	85	25	NA	PACF

[1]Detection limits different for GERG and PACF. GERG reported detection limits were 5 ppb less than PACF for all organic chemicals analyzed except aldrin, heptachlor, PCB total, delta BHC and endosulfan. Appendix C reports detection limits.
[2]GERG – Geochemical and Environmental Research Group; PACF – Patuxent Analytical Control Facility
[a]Cadmium detected in kidney but not liver

Table 4. Geometric means and ranges of select inorganic compounds (ppm wet weight) in liver samples (and kidney for cadmium) from common murres collected in 1995 and 1996 from the central Oregon coast. No live adult females were collected because only adult males are on the water at this time of year as they accompany the juveniles. Results of juveniles of indeterminate sex collected in 1995 (5 alive and 3 dead) are included in Appendix I. Samples collected in 1996 from the central Oregon coast during a die-off were all adults and in Table 5 are compared to other murres collected from the southern Oregon coast.

	1995							**1996**
	Alive			**Dead**				**Dead**
	Adult	**Juvenile**		**Adult**		**Juvenile**		**Adult**
	M	**M**	**F**	**M**	**F**	**M**	**F**	**M**
	n = 10	n = 3	n = 2	n = 1	n = 1	n = 8	n = 7	n = 4
Arsenic	0.454 0.0431-13.7	0.524 0.738-2.94	NC[1] 0.448-2.83[2]	NC 0.438	NC 0.897	0.564 0.291-1.08	0.689 0.39-1.60	1.55 0.373-6.50
Cadmium Liver	2.13 1.48-4.12	0.125 0.0546-0.193	NC 0.0839	NC 1.11	NC 4.43	0.316 0.0494-0.680	0.436 0.0913-2.43	2.69 1.63-4.06
Cadmium Kidney	15.1 9.33-24.6n	0.267 0.126-0.446	NC 0.084-0.181	NC 19.2	NC 8.71	0.553 0.0605-1.45	0.724 0.200-3.59	15.5 **E**[3] 12.7-20.9
Chromium	0.642 0.0788-7.30	ND[4] ≤0.398	ND ≤0.398	ND ≤0.398	ND ≤0.398	NC 0.211	NC 0.227-0.841	2.65 0.623-8.14
Copper	7.34 5.86-9.46	19.9 10.3-35.7	NC 4.05-23.9	NC 21.5	NC 19.6	31.8 9.96-118	35.7 14.4-94.3	4.00 **E** 1.88-13.8
Lead	NC 0.22-0.261	ND ≤0.398	ND ≤0.398	ND ≤0.398	ND ≤0.398	ND ≤0.398	ND ≤0.398	ND ≤0.398
Mercury	0.811 **E** 0.255-1.97	0.159 **E** 0.155-0.164	NC 0.154-0.193	NC 1.28	NC 0.747	0.250 0.191-0.316	0.253 0.123-0.402	1.60 1.13-2.73
Nickel	0.411 **E** 0.0720-5.85	0.916 **E** 0.0668-0.167	NC 0.183	NC 0.134	NC 0.208	0.142 0.0549-0.578	0.0837 0.0567-0.281	1.35 0.326-4.97
Selenium	2.40 1.36-3.68	0.934 0.902-0.967	NC 0.733-0.783	NC 3.84	NC 2.61	1.65 0.930-2.67	1.51 0.816-2.40	4.15 **E** 3.33-5.52
Zinc	34.3 **E** 29.3-39.7	26.8 **E** 25.1-28.2	NC 23.3-31	NC 45.7	NC 45.1	33.3 27.0-42.1	31.2 23.2-36.8	25.9 11.4-53.1

[1]NC – Not calculated because majority of samples were non-detect or there were inadequate number of detectable concentrations to compute a mean.
[2]Only the range is reported when n ≤ 2 or when the mean was not calculated due to majority of non-detects.
[3]Estimated concentration based on quality assurance/quality control spike recoveries outside the acceptable range.
[4]ND – Non-detect. Detection limits are included in Appendix C.

Table 5. Geometric means and ranges of select inorganic compounds (ppm wet weight) in liver samples (and kidney for cadmium) from adult dead common murres collected in 1996 from the Oregon coast. No females were collected on the central coast.

	Central Coast	Southern Coast	
	M	M	F
Compound	n = 4	n = 2	n = 2
Arsenic	1.55	NC[1]	NC
	0.373-6.50	0.175-0.233[2]	0.470-0.629
Cadmium	2.69	NC	NC
Liver	1.63-4.06	2.48-2.62	2.54-6.28
Cadmium	15.5 **E**[3]	NC	NC
Kidney	12.7-20.9	13.4-14.5 **E**	6.69-25.4 **E**
Chromium	2.65	NC	NC
	0.623-8.14	6.37-12.4	8.39-23.9
Copper	4.00 **E**	NC	NC
	1.88-13.8	15.3-17.4 **E**	2.84-3.68 **E**
Lead	ND[4]	ND	NC
	≤0.398	≤0.398	0.234
Mercury	1.60	NC	NC
	1.13-2.73	2.4-2.5	1.34-2.00
Nickel	1.35	NC	NC
	0.326-4.97	5.16-6.53	5.66-9.72
Selenium	4.15 **E**	NC	NC
	3.33-5.52	2.65-3.41 **E**	2.84-4.60 **E**
Zinc	25.9	NC	NC
	11.4-53.1	35.7-38.4	32.8-38.4

[1]NC – Statistics not calculated because majority of samples were non-detect or there were inadequate number of detectable concentrations to compute a mean.

[2]Concentrations are reported as the geometric mean and range, unless $n \leq 2$. Only the range is reported when $n \leq 2$ or when the mean was not calculated due to majority of non-detects.

[3]**E** – Estimated concentration based on quality assurance/quality control spike recoveries outside the acceptable range.

[4]ND – Non-detect. Detection limits included in Appendix C.

Table 6. Geometric means and ranges of select organic compounds in brain tissue from common murres collected in 1995 and 1996 from the central Oregon coast. Results are reported in ppb wet weight. No live adult females were collected because only adult males are on the water at this time of year as they accompany the juveniles. Results of juveniles of indeterminate sex collected in 1995 (5 alive and 3 dead) are included in Appendix I. Samples collected in 1996 from the central Oregon coast during a die-off were all adults and in Table 7 are compared to other murres collected from the southern Oregon coast.

	1995							**1996**
	Alive			**Dead**				**Dead**
	Adult	**Juvenile**		**Adult**		**Juvenile**		**Adult**
Organic Compound	**M**	**M**	**F**	**M**	**F**	**M**	**F**	**M**
	n = 10	n = 3	n = 2	n = 1	n = 1	n = 8	n = 7	n = 4
HCB	5.1 4.1-11	2.5 0.86-6.5	NC[1] 3.1-6.3[2]	NC 35	NC 44	16 10-26	11 5.2-19	120 91-120
PCB total	55 25-280	64 41-100	NC 65-120	NC 400	NC 4000	150 120-260	180 120-490	ND[3] <50
beta BHC	NC 2.0-4.6	NC 2.9	NC 1.6-3.3	NC 10	NC 31	5.2 1.7-9.7	4.5 3.4-12	32 5.0-110
Dieldrin	NC 2.3	ND ≤10	ND ≤10	NC 5.1	NC 20	2.1 0.92-3.6	NC 1.8-6.7	ND ≤10
Endrin	ND ≤10	NC 2.8	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10
o,p'-DDD	ND ≤10	ND ≤10	ND ≤10	NC 3.7	NC 8.1	ND ≤10	NC 3.2	ND ≤10
o,p'-DDE	NC 4.6	ND ≤10	ND ≤10	ND ≤10	NC 3.7	ND ≤10	NC 2.1-2.5	NC 29-36
o,p'-DDT	NC 2.7	ND ≤10	ND ≤10	NC 7	NC 28.0	NC 3	1.8 0.85-3.6	ND ≤10
p,p'-DDD	ND ≤10	ND ≤10	ND ≤10	NC 3.4	ND ≤10	NC 2.2	ND ≤10	ND ≤10
p,p'-DDE	51 18-180	24 6.9-62	NC 29-110	NC 1,100	NC 3,100	150 97-240	180 110-330	3,800 3,300-5,300
p,p'-DDT	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10

[1]NC – Statistics not calculated because majority of samples were non-detect or there were an inadequate number of detectable concentrations to compute a mean.

[2]Only the range is reported when n ≤ 2 or when the mean was not calculated due to majority of non-detects.

[3]ND – Non-detect. Detection limits included in Appendix C.

Table 7. Geometric means and ranges of select organic compounds in brain tissue from common murres collected in 1996 from the Oregon coast. Results are reported in ppb wet weight. All samples were from dead adult murres collected during beach surveys.

	Central Coast	Southern Coast	
	M	M	F
Compound	n = 4	n = 2	n = 2
HCB	120	NC[1]	NC
	91-120	85-87[2]	70-790
PCB total	ND[3]	ND	ND
	≤50	≤50	≤50
beta BHC	32	NC	NC
	5.0-110	5.0-30	5.0-75
Dieldrin	ND	ND	ND
	≤10	≤10	≤10
Endrin	ND	ND	ND
	≤10	≤10	≤10
o,p'-DDD	ND	ND	ND
	≤10	≤10	≤10
o,p'-DDE	NC	ND	ND
	29-36	≤10	≤10
o,p'-DDT	ND	ND	ND
	≤10	≤10	≤10
Oxychlordane	NC	ND	NC
	25-37	≤10	0.030
p,p'-DDD	ND	ND	ND
	≤10	≤10	≤10
p,p'-DDE	3,800	NC	NC
	3,300-5,300	2,200-3,100	2,700-3,100
p,p'-DDT	ND	ND	ND
	≤10	≤10	≤10
trans-Nonachlor	ND	ND	ND
	≤10	≤10	≤10

[1] NC – Statistics not calculated because majority of samples were non-detect.
[2]Only the range is reported when n ≤ 2 or when the mean was not calculated due to majority of non-detects.
[3]ND – Non-detect. Detection limits included in Appendix C

Table 8. Identification of non-, mono-, and di-ortho planar PCBs analyzed in common murre tissues. Non-ortho-chlorinated PCBs 77, 126, and 169 are the most toxic; however, murre tissue samples in our study were only analyzed for PCB congener 77.

Non-ortho planar PCBs	Included
77	+
81	
126	
169	
Mono-ortho planar PCBs	
105	+
114	
118	+
123	
156	+
157	
167	+
189	+
Di-ortho planar PCBs	
170	+
180	+

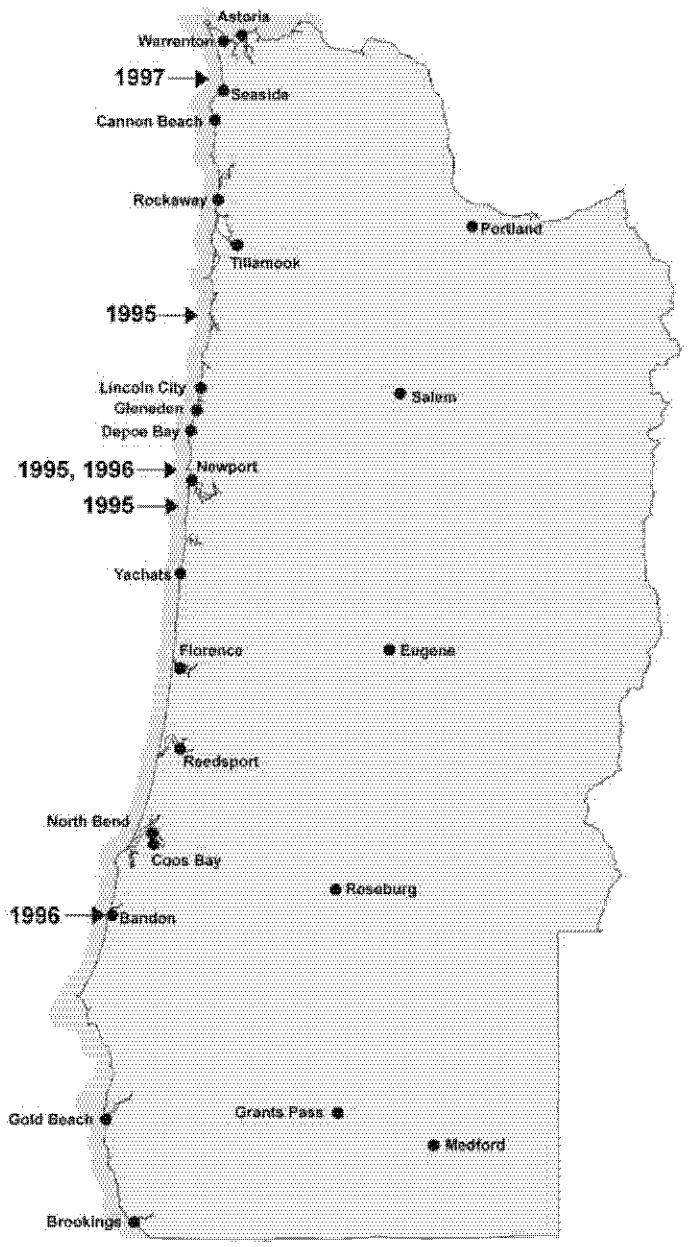

Figure 1. Map of Oregon coast indicating approximate locations of collection sites and year collected.

Appendix A. Sample information.

Disp-disposition; A-alive, D-dead, M-moribund; Age/Sex: A-adult, J-juvenile, M-male, F-female, U-unknown; Necropsy: NWHC-National Wildlife Health Center, U.S. Geological Survey, Madison, Wisconsin; SVM-School of Veterinary Medicine, University of California, Davis. Association was left blank when there was no chick associated with the adult. Necropsy and Catalog entries of None indicate those samples which did not go through necropsy or chemical analysis. Catalog 1060040 for inorganic and organic constituents analyzed at Geochemical and Environmental Research Group (GERG), College Station, Texas. Catalog 1060042 and 1060048 for inorganic and organic constituents analyzed at Patuxent Analytical Control Facility (PACF), Laurel, Maryland.

1995

Sample Number	Location	Disp	Date	Age-Sex	Association	Catalog	Necropsy
MU0110c	3-4 mi. W of Yaquina H.	A	7/12/95	A-M	Chick not collected	1060042	SVM
MU0210c	3-4 mi. W of Yaquina H.	A	7/12/95	J-F	MU0310c	1060040	NWHC
MU0310c	3-4 mi. W of Yaquina H.	A	7/12/95	A-M	MU0210c	1060040	NWHC
MU0410c	3-4 mi. W of Yaquina H.	A	7/12/95	A-M		1060042	SVM
MU0510c	3-4 mi. W of Yaquina H.	A	7/12/95	A-M		1060042	SVM
MU0610c	3-4 mi. W of Yaquina H.	A	7/12/95	A-M	MU0710c	1060042	SVM
MU0710c	3-4 mi. W of Yaquina H.	A	7/12/95	J-U	MU0610c	1060042	SVM
MU0810c	3-4 mi. W of Yaquina H.	A	7/12/95	J-U	MU0910c	1060042	SVM
MU0910c	3-4 mi. W of Yaquina H.	A	7/12/95	A-M	MU0810c	1060042	SVM
MU1010c	3-4 mi. W of Yaquina H.	A	7/12/95	A-M	MU1110c	1060040	NWHC
MU1110c	3-4 mi. W of Yaquina H.	A	7/12/95	J-M	MU1010c	1060040	NWHC
MU1210c	3-4 mi. W of Yaquina H.	A	7/12/95	A-M	MU1310c	1060040	NWHC
MU1310c	3-4 mi. W of Yaquina H.	A	7/12/95	J-M	MU1210c	1060040	NWHC
MU1410c	3-4 mi. W of Yaquina H.	A	7/12/95	J-U	Adult not collected	1060042	SVM
MU1510c	3-4 mi. W of Yaquina H.	A	7/12/95	J-U	Adult not collected	1060042	SVM
MU1610c	3-4 mi. W of Yaquina H.	A	7/12/95	J-U	Adult not collected	1060042	SVM
MU1710c	3-4 mi. W of Yaquina H.	A	7/12/95	J-F	MU1810c	1060040	NWHC
MU1810c	3-4 mi. W of Yaquina H.	A	7/12/95	A-M	MU1710c	1060040	NWHC
MU1910c	3-4 mi. W of Yaquina H.	A	7/12/95	J-M	MU2010c	1060040	NWHC
MU2010c	3-4 mi. W of Yaquina H.	A	7/12/95	A-M	MU1910c	1060040	NWHC
MU2110c	3-4 mi. W of Yaquina H.	D	7/12/95	J-F		1060040	NWHC
MU2210c	Seal Rock	D	7/14/95	A-M		1060040	NWHC
MU2310c	Seal Rock	D	7/14/95	J-F		1060040	NWHC
MU2410c	Seal Rock	D	7/14/95	J-F		1060040	NWHC
MU2510c	Seal Rock	D	7/14/95	J-M		1060040	NWHC
MU2610c	Seal Rock	D	7/17/95	J-M		1060040	NWHC
MU2710c	Seal Rock	D	7/17/95	J-U		1060040	NWHC
MU2810c	3-4 mi. W of Yaquina H.	D	7/22/95	J-U		1060040	NWHC
MU2910c	3-4 mi. W of Yaquina H.	D	7/22/95	J-U		1060040	NWHC
MU3010c	Seal Rock	D	8/01/95	A-F		1060040	NWHC
MU3110c	Yaquina Head	M	8/06/95	J-M		1060040	NWHC
MU3210c	S. of Seal Rock	D	8/08/95	J-F		1060040	NWHC
MU3310c	Lincoln County	M	8/17/95	J-M		1060040	NWHC
MU3410c	Lincoln County	M	8/17/95	J-F		1060040	NWHC
MU3510c	Lincoln County	M	8/17/95	J-M		1060040	NWHC
MU3610c	Pacific City, Tillamook Co.	M	8/19/95	J-F		1060040	NWHC
MU3710c	Lincoln County	M	8/20/95	J-F		1060040	NWHC
MU3810c	Lincoln County	D	8/21/95	J-M		1060040	NWHC
MU3910c	Lincoln County	M	9/10/95	J-M		1060040	NWHC
MU4010c	Seal Rock	M	9/20/95	J-M		1060040	NWHC

Appendix A. Continued.

1996

Sample Number	Location	Disp	Date	Age-Sex	Catalog	Necropsy
MU41	Newport	D	6/27/96	A-M	1060048	None
MU42	Newport	D	6/27/96	A-M	1060048	None
MU43	Bandon	D	6/29/96	A-M	1060048	None
MU44	Bandon	D	6/29/96	A-M	1060048	None
MU45	Newport	D	6/27/96	A-M	1060048	None
MU46	Newport	D	6/27/96	A-M	1060048	None
MU47	Bandon	D	6/29/96	A-F	1060048	None
MU48	Bandon	D	6/29/96	A-F	1060048	None

1997

Sample Number	Location	A/D	Date	Age Sex	Catalog	Necropsy
MU97001	Gearhart, Clatsop Co.	D	6/30/97	A-F	None	NWHC
MU97002	Gearhart, Clatsop Co.	D	6/30/97	A-M	None	NWHC
MU97003	Gearhart, Clatsop Co.	D	6/30/97	A-F	None	NWHC
MU97004	Gearhart, Clatsop Co.	D	6/30/97	A-M	None	NWHC

Appendix B. Percent lipid and moisture.

Percent brain lipid for common murre samples collected in 1995 from the central Oregon coast. Disposition: A-alive, D-dead, M-moribund; Age: A-adult, J-juvenile; Sex: M-male, F-female. Percent moisture was left blank when there was insufficient sample mass to conduct analysis. Catalog 1060040 analyzed at Geochemical and Environmental Research Group (GERG), College Station, Texas. Catalog 1060042 analyzed at Patuxent Analytical Control Facility (PACF), Laurel, Maryland.

Sample Number	Brain Lipid (%)	Moisture (%)	Disposition	Age-Sex	Catalog
MU01	7.60		A	A-M	1060042[1]
MU02	2.11	75.5	A	J-F	1060040
MU03	2.17	71.3	A	A-M	1060040
MU04	7.26		A	A-M	1060042
MU05	7.07		A	A-M	1060042
MU06	7.61		A	A-M	1060042
MU07	6.21		A	J-U	1060042
MU08	5.97		A	J-U	1060042
MU09	7.17		A	A-M	1060042
MU10	1.46	79.0	A	A-M	1060040
MU11	1.54	81.0	A	J-M	1060040
MU12	2.61	80.3	A	A-M	1060040
MU13	2.49	79.6	A	J-M	1060040
MU14	6.50		A	J-U	1060042
MU15	5.68		A	J-U	1060042
MU16	5.90		A	J-U	1060042
MU17	6.09	76.1	A	J-F	1060040
MU18	1.73	78.1	A	A-M	1060040
MU19	1.34	68.1	A	J-M	1060040
MU20	15.9	75.5	A	A-M	1060040
MU21	1.79	69.9	D	J-F	1060040
MU22	1.1	77.8	D	A-M	1060040
MU23	22.0	75.2	D	J-F	1060040
MU24	1.32	74.2	D	J-F	1060040
MU25	1.32	78.8	D	J-M	1060040
MU26	0.9	75.1	D	J-M	1060040
MU27	1.17	82.2	D	J-U	1060040
MU28	1.42	69.0	D	J-U	1060040
MU29	4.61	64.4	D	J-U	1060040
MU30	1.49	80.5	D	A-F	1060040
MU31	4.17	83.0	M	J-M	1060040
MU32	1.83	81.1	D	J-F	1060040
MU33	1.75	81.1	M	J-M	1060040
MU34	7.94	82.0	M	J-F	1060040
MU35	1.92	75.4	M	J-M	1060040
MU36	1.05	77.9	M	J-F	1060040
MU37	1.23	82.1	M	J-F	1060040
MU38	1.86	77.9	D	J-M	1060040
MU39	1.81	78.8	M	J-M	1060040
MU40	1.26	78.4	M	J-M	1060040

[1] Samples in catalog 1060042 were of insufficient mass to conduct analysis of percent moisture.

Appendix C. Detection limits.

Compound	Detection Limit (ppm wet weight)
Aluminum	0.980-3.18
Arsenic	0.0667-0.165
Boron	0.396-2.48
Barium	0.198-0.796
Beryllium	0.0197-0.0796
Cadmium-kidney	0.00342-0.248
liver	0.0198-0.329
Chromium	0.0991-0.398
Copper	0.0991-0.398
Iron	0.980-3.18
Lead	0.0991-0.398
Mercury	0.0991-0.0490
Magnesium	0.980-3.18
Manganese	0.198-0.796
Molybdenum	0.396-1.59
Nickel	0.0991-0.398
Selenium	0.0991-0.165
Strontium	0.0991-0.398
Vanadium	0.0991-0.398
Zinc	0.198-0.796

	Detection Limit (ppb wet weight)	
Compound	PACF	GERG
Aldrin	<5	<5
HCB	≤10	<5
Heptachlor	<5	<5
PCB congeners – all	NA[1]	3.0-10
PCB total	<50	<50
alpha BHC	≤10	<5
alpha chlordane	≤10	<5
beta BHC	≤10	<5
cis-Nonachlor	≤10	<5
delta BHC	<5	<5
Dieldrin	≤10	<5
Endosulfan	<10	<10
Endrin	≤10	<5
gamma BHC	≤10	<5
gamma Chlordane	≤10	<5
Heptachlor epoxide	≤10	<5
Mirex	≤10	<5
o,p'-DDD	≤10	<5
o,p'-DDE	≤10	<5
o,p'-DDT	≤10	<5
Oxychlordane	≤10	<5
p,p'-DDD	≤10	<5
p,p'-DDE	≤10	<5
p,p'-DDT	≤10	<5
trans-Nonachlor	≤10	<5

[1]Not applicable

Appendix D. Geometric means and ranges of inorganic compounds (ppm wet weight) in liver samples (and kidney for cadmium) from common murres collected in 1995 and 1996 from the central Oregon coast. No live adult females were collected because only adult males are on the water at this time of year as they accompany the juveniles. Results of juveniles of indeterminate sex collected in 1995 (5 alive and 3 dead) are included in Appendix E. M represents males and F represents females. Samples collected in 1996 during the die-off were all adults.

	1995							**1996**
	Alive			**Dead**				**Dead**
	Adult	**Juvenile**		**Adult**		**Juvenile**		**Adult**
	M	**M**	**F**	**M**	**F**	**M**	**F**	**M**
	n = 10	n = 3	n = 2	n = 1	n = 1	n = 8	n = 7	n = 4
Aluminum	ND[1] ≤3.18	ND ≤3.18	ND ≤3.18	ND ≤3.18	ND ≤3.18	ND ≤3.18	NC[2] 2.12-5.78[3]	ND ≤3.18
Arsenic	0.454 0.0431-13.7	0.524 0.738-2.94	NC 0.44 8-2.83	NC 0.438	NC 0.897	0.564 0.291-1.08	0.689 0.39-1.60	1.55 0.373-6.50
Boron	1.05 **E**[4] 0.315-3.87	ND ≤2.48	ND ≤2.48	ND ≤2.48	ND ≤2.48	ND ≤2.48	NC 1.02-1.18	3.01 2.00-6.98
Barium	NC **E** 0.605	ND ≤0.796	ND ≤0.796	ND ≤0.796	ND ≤0.796	ND ≤0.796	ND ≤0.796	ND ≤0.796
Beryllium	ND ≤0.080	ND ≤0.080	ND ≤0.080	ND ≤0.080	ND ≤0.080	NC 0.0291-0.0642	NC 0.0622	ND ≤0.080
Cadmium Liver	2.13 1.48-4.12	0.125 0.0546-0.193	NC 0.0839	NC 1.11	NC 4.43	0.316 0.0494-0.680	0.436 0.0913-2.43	2.69 1.63-4.06
Cadmium Kidney	15.1 9.33-24.6n	0.267 0.126-0.446	NC 0.084-0.181	NC 19.2	NC 8.71	0.553 0.0605-1.45	0.724 0.200-3.59	15.5 **E** 12.7-20.9
Chromium	0.642 0.0788-7.30	ND ≤0.398	ND ≤0.398	ND ≤0.398	ND ≤0.398	NC 0.211	NC 0.227-0.841	2.65 0.623-8.14
Copper	7.34 5.86-9.46	19.9 10.3-35.7	NC 4.05-23.9	NC 21.5	NC 19.6	31.8 9.96-118	35.7 14.4-94.3	4.00 **E** 1.88-13.8
Iron	281 **E** 198-375	89.0 **E** 47.5-129	NC 53.3-59.5 **E**	NC 909 **E**	NC 3300**E**	183 **E** 67.4-396	202 **E** 82.9-416	655 **E** 255-1,620
Lead	NC 0.22-0.261	ND ≤0.398	ND ≤0.398	ND ≤0.398	ND ≤0.398	ND ≤0.398	ND ≤0.398	ND ≤0.398
Mercury	0.811 **E** 0.255-1.97	0.159 **E** 0.155-0.164	NC 0.154-0.193	NC 1.28	NC 0.747	0.250 0.191-0.316	0.253 0.123-0.402	1.60 1.13-2.73
Magnesium	221 190-234	198 192-209	NC 204-221	NC 318	NC 248	184 148-244	215 161-302	169 **E** 61.6-371
Manganese	4.46 3.45-5.96	4.75 4.58-4.96	NC 3.42-5.20	NC 2.76	NC 2.56	3.81 3.05-5.96	3.04 2.28-4.37	2.02 1.08-3.40
Molybdenum	0.873 0.329-1.95	0.272 0.267-0.276	ND ≤1.59	NC 0.587	NC 1.71	0.453 0.225-1.10	0.737 0.198-1.72	NC 4.60

	1995							1996
	Alive			Dead				Dead
	Adult	Juvenile		Adult		Juvenile		Adult
	M	M	F	M	F	M	F	M
	n = 10	n = 3	n = 2	n = 1	n = 1	n = 8	n = 7	n = 4
Nickel	0.411 **E** 0.0720-5.85	0.916 **E** 0.0668-0.167	NC 0.183	NC 0.134	NC 0.208	0.142 0.0549-0.578	0.0837 0.0567-0.281	1.35 0.326-4.97
Selenium	2.40 1.36-3.68	0.934 0.902-0.967	NC 0.733-0.783	NC 3.84	NC 2.61	1.65 0.930-2.67	1.51 0.816-2.40	4.15 **E** 3.33-5.52
Strontium	0.239 0.0788-0.849	0.102 0.0668-0.227	ND ≤0.398	NC 1.73	NC 0.256	0.105 0.0520-0.645	0.264 0.0496-1.21	0.721 0.528-1.21
Vanadium	NC 0.296-0.592 **E**	0.211 **E** 0.146-0.372	NC 0.421	NC 0.438	ND	0.191 0.0549-0.465	0.202 0.0589-0.414	ND ≤0.398
Zinc	34.3 **E** 29.3-39.7	26.8 **E** 25.1-28.2	NC 23.3-31	NC 45.7	NC 45.1	33.3 27.0-42.1	31.2 23.2-36.8	25.9 11.4-53.1

[1]ND – Non-detect. Detection limits are included in Appendix C.

[2]NC – Not calculated because majority of samples were non-detect or there were inadequate number of detectable concentrations to compute a mean.

[3]Only the range is reported when n ≤ 2 or when the mean was not calculated due to majority of non-detects.

[4]**E** – Estimated concentration based on quality assurance/quality control spike recoveries outside the acceptable range.

Appendix E. Geometric means and ranges of inorganic compounds (ppm wet weight) in liver and kidney (cadmium only) from adult dead common murres collected in 1996 from the Oregon coast. No females were collected on the central coast.

	Central Coast	Southern Coast	
	Male	Male	Female
Compound	n = 4	n = 2	n = 2
Aluminum	11.8 7.27-26.7	NC[1] 12.4-16.1[2]	NC 11.5-11.9
Arsenic	1.55 0.373-6.50	NC 0.175-0.233	NC 0.470-0.629
Boron	3.01 2.00-6.98	NC 3.42-4.59	NC 1.53-1.54
Barium	ND[3] ≤0.796	ND ≤0.796	ND ≤0.796
Beryllium	ND ≤0.0796	ND ≤0.0796	ND ≤0.0796
Cadmium Liver	2.69 1.63-4.06	NC 2.48-2.62	NC 2.54-6.28
Cadmium Kidney	15.5 **E**[4] 12.7-20.9	NC 13.4-14.5 **E**	NC 6.69-25.4 **E**
Chromium	2.65 0.623-8.14	NC 6.37-12.4	NC 8.39-23.9
Copper	4.00 **E** 1.88-13.8	NC 15.3-17.4 **E**	NC 2.84-3.68 **E**
Iron	656 **E** 255-1620	NC 643-1,900 **E**	NC 828-1,010 **E**
Lead	ND ≤0.398	ND ≤0.398	NC 0.234
Mercury	1.60 1.13-2.73	NC 2.4-2.5	NC 1.34-2.00
Magnesium	169 **E** 61.6-371	NC 419-601 **E**	NC 303-388 **E**
Manganese	2.02 1.08-3.40	NC 3.51-3.52	NC 4.37-5.40
Molybdenum	0.530 0.274-1.20	NC 8.06	NC 0.802-0.883
Nickel	1.35 0.326-4.97	NC 5.16-6.53	NC 5.66-9.72
Selenium	4.15 **E** 3.33-5.52	NC 2.65-3.41 **E**	NC 2.84-4.60 **E**
Strontium	0.721 0.528-1.21	NC 2.14-4.06	NC 1.02-1.32
Vanadium	ND ≤0.398	ND ≤0.398	ND ≤0.398
Zinc	25.9 11.4-53.1	NC 35.7-38.4	NC 32.8-38.4

[1]NC – Statistics not calculated because majority of samples were non-detect or there were inadequate number of detectable concentrations to compute a mean.

[2]Only the range is reported when n ≤ 2 or when the mean was not calculated due to majority of non-detects.

[3]ND – Non-detect. Detection limits included in Appendix C.

[4]**E** – Estimated concentration based on quality assurance/quality control spike recoveries outside the acceptable range.

Appendix F. Geometric means and ranges of organic compounds in brain tissue from common murres collected in 1995 and 1996 from the central Oregon coast. Results are reported in ppb wet weight. No live adult females were collected because only adult males are on the water at this time of year as they accompany the juveniles. Results of juveniles of indeterminate sex collected in 1995 (5 alive and 3 dead) are included in Appendix E. M represents males and F represents females. Samples collected in 1996 from the central Oregon Coast during a die-off were all adults and in Appendix G are compared to other murres collected from the southern Oregon coast.

	1995							1996
	Alive			Dead				Dead
	Adult	Juvenile		Adult		Juvenile		Adult
Organic Compound	M	M	F	M	F	M	F	M
	n = 10	n = 3	n = 2	n = 1	n = 1	n = 8	n = 7	n = 4
Aldrin	ND[1] <5.0	ND <5.0	ND <5.0	ND <5.0	ND <5.0	ND <5.0	ND <5.0	NA[2]
HCB	5.1 4.1-11	2.5 0.86-6.5	NC[3] 3.1-6.3[4]	NC 35	NC 44	16 10-26	11 5.2-19	120 91-120
Heptachlor	ND <5.0	ND <5.0	ND <5.0	ND <5.0	ND <5.0	ND <5.0	ND <5.0	NA
PCB total	55 25-280	64 41-100	NC 65-120	NC 400	NC 4,000	150 120-260	180 120-490	ND <50
alpha BHC	NC 3.2-4.5 **E**[5]	NC 2.7 **E**	NC 4.4 **E**	ND ≤10	ND ≤10	NC 3.5-5.4 **E**	ND ≤10	ND ≤10
alpha Chlordane	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10
beta BHC	NC 2.0-4.6	NC 2.9	NC 1.6-3.3	NC 10	NC 31	5.2 1.7-9.7	4.5 3.4-12	32 5.0-110
cis-Nonachlor	ND ≤10	ND ≤10	ND ≤10	ND ≤10	NC 7.9	NC 2.3	NC 2.4-16	ND ≤10
delta BHC	ND <5.0	ND <5.0	ND <5.0	ND <5.0	ND <5.0	ND <5.0	ND <5.0	NA
Dieldrin	NC 2.3	ND ≤10	ND ≤10	NC 5.1	NC 20	2.1 0.92-3.6	NC 1.8-6.7	ND ≤10
Endosulfan	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	NA
Endrin	ND ≤10	NC 2.8	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10
gamma BHC	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10
gamma Chlordane	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	NC 2.5	NC 3.9	ND ≤10
Heptachlor Epoxide	NC 2.0-2.1	NC 3.1	ND ≤10	NC 7.4	NC 15	2.0 0.79-2.9	1.9 0.85-8.7	ND ≤10

Organic Compound	1995							1996
	Alive			Dead				Dead
	Adult	Juvenile		Adult		Juvenile		Adult
	M	M	F	M	F	M	F	M
	n = 10	n = 3	n = 2	n = 1	n = 1	n = 8	n = 7	n = 4
Mirex	ND ≤10	ND ≤10	ND ≤10	NC 4.4	NC 8.0	ND ≤10	NC 2.8	ND ≤10
o,p'-DDD	ND ≤10	ND ≤10	ND ≤10	NC 3.7	NC 8.1	ND ≤10	NC 3.2	ND ≤10
o,p'-DDE	NC 4.6	ND ≤10	ND ≤10	ND ≤10	NC 3.7	ND ≤10	NC 2.1-2.5	NC 29-36
o,p'-DDT	NC 2.7	ND ≤10	ND ≤10	NC 7.0	NC 28	NC 3.0	1.8 0.85-3.6	ND ≤10
Oxychlordane	NC 4.0-4.1	NC 3.5	NC 4.1	NC 13	NC 28	4.6 3.3-7.2	4.6 3.1-9.7	NC 25-37
p,p'-DDD	ND ≤10	ND ≤10	ND ≤10	NC 3.4	ND ≤10	NC 2.2	ND ≤10	ND ≤10
p,p'-DDE	51 18-180	24 6.9-62	NC 2.9-110	NC 1,100	NC 3,100	150 97-240	180 110-330	3,800 3,300-5,300
p,p'-DDT	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10
trans-Nonachlor	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	NC 1.9-8.5	ND ≤10

[1]ND – Non-detect. Detection limits included in Appendix C

[2]NA – Not analyzed

[3]NC – Statistics not calculated because majority of samples were non-detect or there were inadequate number of detectable concentrations to compute a mean.

[4]Only the range is reported when n ≤ 2 or when the mean was not calculated due to majority of non-detects.

[5]**E** – estimated concentration based on quality assurance/quality control spike recoveries outside the acceptable range.

Appendix G. Geometric means and ranges of organic compounds in brain tissue from common murres collected in 1996 from the Oregon coast. Results are reported in ppb wet weight. All samples were from dead adult murres collected during beach surveys.

	Central Coast	Southern Coast	
	Male	Male	Female
Compound	n = 4	n = 2	n = 2
Aldrin	NA[1]	NA	NA
HCB	120	NC[2]	NC
	91-120	85-87[3]	70-790
Heptachlor	NA	NA	NA
PCB total	ND[4]	ND	ND
	<50	<50	<50
alpha BHC	ND	ND	ND
	≤10	≤10	≤10
alpha Chlordane	ND	ND	ND
	≤10	≤10	≤10
beta BHC	32	NC	NC
	5.0-110	5.0-30	5.0-75
cis-Nonachlor	ND	ND	NC
	≤10	≤10	47
delta BHC	NA	NA	NA
Dieldrin	ND	ND	ND
	≤10	≤10	≤10
Endosulfan	NA	NA	NA
Endrin	ND	ND	ND
	≤10	≤10	≤10
gamma BHC	ND	ND	ND
	≤10	≤10	≤10
gamma Chlordane	ND	ND	ND
	≤10	≤10	≤10
Heptachlor Epoxide	ND	ND	ND
	≤10	≤10	≤10
Mirex	ND	ND	ND
	≤10	≤10	≤10
o,p'-DDD	ND	ND	ND
	≤10	≤10	≤10
o,p'-DDE	NC	ND	ND
	29-36	≤10	≤10
o,p'-DDT	ND	ND	ND
	≤10	≤10	≤10
Oxychlordane	NC	ND	NC
	25-37	≤10	0.030
p,p'-DDD	ND	ND	ND
	≤10	≤10	≤10
p,p'-DDE	3,800	NC	NC
	3,300-5,300	2,200-3,100	2,700-3,100
p,p'-DDT	ND	ND	ND
	≤10	≤10	≤10
trans-Nonachlor	ND	ND	ND
	≤10	≤10	≤10

[1]NA – Not analyzed

[2]NC – Statistics not calculated because majority of samples were non-detect.

[3]Only the range is reported when n ≤ 2 or when the mean was not calculated due to majority of non-detects.

[4]ND – Non-detect. Detection limits included in Appendix C.

Appendix H. Geometric means and ranges of PCB congener concentrations in brain tissue from common murres collected in 1995 from the central Oregon coast. Results are reported in ppb wet weight. For PCBs where all groups are listed as ND (non-detect; e.g., congener 129), detection limit is ≤10 ppb wet weight. M represents males and F represents females. No live adult females were collected because only adult males are on the water at this time of year as they accompany the juveniles. Among dead juveniles, there were three of undetermined sex which are included in Appendix I.

PCB Congener[1]	Alive			Dead			
	Adult	Juvenile		Adult		Juvenile	
	M	M	F	M	F	M	F
	n = 5	n = 3	n = 2	n = 1	n = 1	n = 8	n = 7
101	NC[2] 4.1	ND[3] ≤10	ND ≤10	NC 6.8	NC 19	NC 4.0	NC 6.8-12[4]
105	NC 5.5	ND ≤10	ND ≤10	NC 8.9	NC 40	NC 4.8	NC 3.3-6.2
107/108/144	ND ≤10	ND ≤10	ND ≤10	ND ≤10	NC 0.009	ND ≤10	ND ≤10
110/77	ND ≤10	NC 3.8	ND ≤10	ND ≤10	ND ≤10	ND ≤10	NC 5.4
118/108/149	3.6 1.8-22	2.6 1.6-6.5	NC 9.0	NC 39	NC 240	9.6 6.9-23	11 7.2-31
128	ND ≤10	ND ≤10	ND ≤10	NC 4.4	NC 500	ND ≤10	NC 9.9
129	ND	ND	ND	ND	ND	ND	ND
136	ND	ND	ND	ND	ND	ND	ND
137	ND ≤10	ND ≤10	ND ≤10	ND ≤10	NC 0.21	ND ≤10	NC 0.0035-0.013
138	9.2 5.0-33	4.0 1.7-9.4	NC 5.0-13	NC 520	NC 520	17 13-32	20 12-55
141	ND	ND	ND	ND	ND	ND	ND
146	2.8 1.8-13	2.2 1.6-4.0	NC 5.9	NC 23	NC 220	6.7 4.6-14	8.4 5.6-21
149	ND ≤10	ND ≤10	ND ≤10	ND ≤10	NC 7.0	ND ≤10	NC 5.0
15	ND	ND	ND	ND	ND	ND	ND
151	ND	ND	ND	ND	ND	ND	ND
153	11 4.9-48	4.4 1.7-9.1	NC 5.9-19	NC 34	NC 820	24 17-48	28 20-84
156/171/202	ND ≤10	ND ≤10	ND ≤10	NC 7.8	NC 41	NC 4.7	NC 3.8
158	ND ≤10	ND ≤10	ND ≤10	NC 2.9	NC 49	ND ≤10	NC 6.5
16/32	ND	ND	ND	ND	ND	ND	ND

PCB Congener[1]	Alive			Dead			
	Adult	Juvenile		Adult		Juvenile	
	M	M	F	M	F	M	F
	n = 5	n = 3	n = 2	n = 1	n = 1	n = 8	n = 7
167	ND ≤10	ND ≤10	ND ≤10	ND ≤10	NC 31	ND ≤10	ND ≤10
170	8.9 **E**[5] 7.6-14	7.0 **E** 6.2-8.5	NC 7.4-8.6 **E**	NC 17 **E**	NC 150 **E**	6.1 **E** 2.1-12	8.6 **E** 5.2-18
172	ND ≤10	ND ≤10	ND ≤10	NC 8.2	NC 47	ND ≤10	NC 3.9-4.4
174	ND	ND	ND	ND	ND	ND	ND
177	ND ≤10	ND ≤10	ND ≤10	NC 13	NC 51	ND ≤10	NC 6.6
178	ND ≤10	ND ≤10	ND ≤10	NC 5.7	NC 43	ND ≤10	NC 5.8
18	ND	ND	ND	ND	ND	ND	ND
180	3.8 1.8-22	1.6 1.4-1.7	NC 6.1	NC 7.6	NC 480	10 7.5-19	10 5.6-33
183	NC 5.2-5.5	ND ≤10	ND ≤10	NC 9.3	NC 100	NC 3.5-8.7	NC 4.6-11
185	ND	ND	ND	ND	ND	ND	ND
187/182/159	3.7 1.9-17	2.7 1.6-7.2	NC 6.3	NC 60	NC 420	8.8 6.0-14	12 6.4-28
189	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	NC 6.9
191	ND ≤10	ND ≤10	ND ≤10	ND ≤10	NC 8.7	ND ≤10	ND ≤10
194	ND ≤10	ND ≤10	ND ≤10	NC 7.9	NC 81	ND ≤10	NC 4.4
195	ND ≤10	ND ≤10	ND ≤10	NC 3.7	NC 410	ND ≤10	ND ≤10
196	NC 5.3	ND ≤10	ND ≤10	NC 7.2	NC 9.2	ND ≤10	NC 3.7-5.9
200	ND ≤10	ND ≤10	ND ≤10	NC 2.8	NC 29	ND ≤10	ND ≤10
201	6.5 4.9-11	3.4 1.7-5.9	NC 4.1-6.3	NC 18	NC 130	4.2 1.6-8.0	6.8 4.6-11
205	ND	ND	ND	ND	ND	ND	ND
206	ND ≤10	ND ≤10	ND ≤10	NC 5.6	NC 34	ND ≤10	ND ≤10

PCB Congener[1]	Alive			Dead			
	Adult	Juvenile		Adult		Juvenile	
	M	M	F	M	F	M	F
	n = 5	n = 3	n = 2	n = 1	n = 1	n = 8	n = 7
209	ND ≤10	ND ≤10	ND ≤10	ND ≤10	NC 15	ND ≤10	ND ≤10
22	ND	ND	ND	ND	ND	ND	ND
24	NC 5.1	NC 3.7	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10
25	NC 5.9	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10
26	ND	ND	ND	ND	ND	ND	ND
28	NC 6.3	ND ≤10	ND ≤10	NC 3.7	NC 6.4	ND ≤10	ND ≤10
29	ND	ND	ND	ND	ND	ND	ND
33	ND	ND	ND	ND	ND	ND	ND
37/42	ND	ND	ND	ND	ND	ND	ND
40	ND	ND	ND	ND	ND	ND	ND
41/64	3.9 **E** 1.8-8.9	11 **E** 5.6-15	NC 7.0-9.8 **E**	NC 1.3 **E**	NC 240 **E**	22 **E** 6.2-100	14 **E** 1.8-100
44	ND	ND	ND	ND	ND	ND	ND
45	ND	ND	ND	ND	ND	ND	ND
46	ND	ND	ND	ND	ND	ND	ND
47/48	NC 6.4	ND ≤10	ND ≤10	ND ≤10	NC 15	ND ≤10	NC 0.0053
49	NC 3.7	ND ≤10	NC 3.1	ND ≤10	ND ≤10	ND ≤10	NC 4.1
50	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	NC 0.0049
52	NC 4.9-5.6	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10
60/56	6.5 5.6-7.6	5.8 4.4-8.5	NC 4.5-5.8	NC 6.9	NC 18	9.4 5.7-17	8.3 5.4-14
66	NC 6.2	ND ≤10	ND ≤10	NC 7.7	NC 27	ND ≤10	NC 4.5
7	NC 7.3	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10
70	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	ND ≤10	NC 5.3
74	NC 5.0	ND ≤10	ND ≤10	NC 110	NC 13	ND ≤10	NC 3.8

PCB Congener[1]	Alive			Dead			
	Adult	Juvenile		Adult		Juvenile	
	M	M	F	M	F	M	F
	n = 5	n = 3	n = 2	n = 1	n = 1	n = 8	n = 7
8	7.1 6.2-9.4	5.4 5.0-6.0	NC 5.1-6.8	NC 5.5	ND ≤10	2.3 1.6-5.3	3.1 1.5-6.6
82	2.8 1.8-5.9	1.6 1.4-1.7	NC 5.4	ND ≤10	NC 86	9.8 3.4-17	10 5.5-36
83	ND	ND	ND	ND	ND	ND	ND
84	ND	ND	ND	ND	ND	ND	ND
85	ND	ND	ND	ND	ND	ND	ND
87	ND	ND	ND	ND	ND	ND	ND
88	ND	ND	ND	ND	ND	ND	ND
92	NC 4.2	ND ≤10	ND ≤10	NC 11	NC 77	NC 4.3-7.5	NC 4.0-7.0
97	ND	ND	ND	ND	ND	ND	ND
99	3.3 **E** 1.8-12	2.1 **E** 1.6-3.2	NC 4.8-5.7 **E**	NC 7.1 **E**	NC 120 **E**	5.7 **E** 2.1-10	6.8 **E** 4.9-17

[1]Multiple PCB congeners reported as a group when individual congeners could not be resolved.
[2]NC – Mean not calculated because majority of samples were non-detect or there were inadequate number of detectable concentrations to compute a mean.
[3]ND – Non-detect. Detection limits are included in Appendix C.
[4]Only the range is reported when n ≤ 2 or when the mean was not calculated due to majority of non-detects.
[5]**E** – Estimated concentration based on quality assurance/quality control spike recoveries outside the acceptable range.

Appendix I. Geometric means and ranges of inorganic compounds (ppm wet weight) in liver tissue (and kidney for cadmium) and organic compounds (ppb wet weight) in brain tissue from juvenile common murres of undetermined sex collected in 1995 along the central Oregon coast.

	Juvenile	
Inorganic Compound	**Alive** n = 5	**Dead** n = 3
Aluminum	ND[1]	ND
Arsenic	0.419 0.132-0.997	1.80 1.30-3.20
Boron	NC[2] 1.74-7.93	ND
Barium	ND	ND
Beryllium	ND	ND
Cadmium Liver	1.45 0.253-63.1	NC 0.9-1.8
Cadmium Kidney	0.649 0.376-0.805	1.05 0.200-2.50
Chromium	NC 0.553	ND
Copper	51.0 16.6-110	78.8 15.6-278
Iron	310 134-1970	442 213-679
Lead	1.58 0.783-2.60	ND
Mercury	ND	0.853 0.420-1.24
Magnesium	812 726-917	965 674-1340
Manganese	13.8 12.7-14.4	10.7 8.42-12.8
Molybdenum	ND	NC 2.83
Nickel	ND	NC 0.680
Selenium	2.93 2.70-3.11	4.49 2.50-7.40
Strontium	NC 0.668-1.48	NC 2.22-2.48
Vanadium	ND	NC 2.04
Zinc	89.1 66.7-119	106 67.3-147

Appendix I. Continued.

Organic Compound	Juvenile	
	Alive	**Dead**
	n = 5	n = 3
Aldrin	NA[3]	ND
HCB	ND	NC 5.5-8.1
Heptachlor	NA	ND
PCB total	ND	110 94-120
alpha BHC	ND	ND
alpha Chlordane	ND	ND
beta BHC	ND	ND
cis-Nonachlor	ND	ND
delta BHC	NA	ND
Dieldrin	ND	ND
Endosulfan	NA	ND
Endrin	ND	ND
gamma BHC	ND	ND
gamma Chlordane	ND	ND
Heptachlor Epoxide	ND	ND
Mirex	ND	ND
o,p'-DDD	ND	ND
o,p'-DDE	ND	ND
o,p'-DDT	ND	ND
Oxychlordane	ND	ND
p,p'-DDD	ND	ND
p,p'-DDE	32 5.0-200	70 31-100
p,p'-DDT	ND	ND
trans-Nonachlor	ND	ND

Appendix I. Continued.

PCB Congener [5]	Juvenile (Dead) n = 3
101	ND
105	ND
107/108/144	ND
110/77	ND
118/108/149	NC 5.9
128	ND
129	ND
136	ND
137	ND
138	NC 9.5-11.7
141	ND
146	ND
149	ND
15	ND
151	ND
153	14 11-18
156/171/202	ND
158	ND
16/32	ND
167	ND
170	ND
172	ND
174	ND
177	ND
178	ND
18	ND
180	NC 7.2
183	ND
185	ND
187/182/159	NC 6.4
189	ND
191	ND
194	ND
195	ND
196	ND
200	ND
201	NC 9.8
205	ND
206	ND
209	ND

PCB Congener [5]	Juvenile (Dead) n = 3
22	ND
24	NC 6.3-12.1
25	ND
26	ND
28	ND
29	ND
33	ND
37/42	ND
40	ND
41/64	260 180-350
44	ND
45	ND
46	ND
47/48	ND
49	ND
50	ND
52	ND
60/56	19 13-25
66	ND
7	ND
70	ND
74	ND
8	ND
82	NC 6
83	ND
84	ND
85	ND
87	ND
88	ND
92	ND
97	ND
99	ND

[1]ND – Non-detect. Detection limits are included in Appendix C.
[2]NC – Mean not calculated because majority of samples were non-detect or there were inadequate number of detectable concentrations to compute a mean.
[3]Only the range is reported when $n \leq 2$ or when the mean was not calculated due to majority of non-detects.
[4]NA – not analyzed.
[5]Multiple PCB congeners reported as a group when individual congeners could not be resolved.

Appendix J. Correlation matrix (*r* value/P value) of select organochlorine contaminants in brain tissue from common murres collected in 1995 and 1996 from the Oregon coast. Bold values are statistically significant at $P \leq 0.05$.

	PCB total	**o,p'-DDD**	**o,p'-DDE**	**o,p'-DDT**	**p,p'-DDD**	**p,p'-DDE**
o,p'-DDD	0.30/ 0.81					
o,p'-DDE	-0.06/ 1.00	**0.46/ 0.02**				
o,p'-DDT	**0.86/ 0.00**	**0.74/ 0.00**	0.20/ 1.00			
p,p'-DDD	-0.17/ 1.00	**0.88/ 0.00**	**0.50/ 0.01**	0.35/ 0.28		
p,p'-DDE	0.22/ 1.00	**0.57/ 0.00**	**0.67/ 0.00**	**0.46/ 0.02**	**0.49/ 0.01**	
p,p'-DDT	-0.18/ 1.00	**0.86/ 0.00**	**0.52/ 0.00**	**0.33/ 0.51**	**0.98/ 0.00**	**0.47/ 0.01**