

Mussel Tissue Data

2001

Data, Report

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INTERDEPARTMENTAL MEMORANDUM

Date: October 27, 1999

To: Lee Doggett, Department of Environmental Protection

From: Andrew Smith, SM, ScD, Bureau of Health, Environmental Toxicology Program

Re: Human health assessment on mussel contaminant data from Casco Bay

Cc: Katherine Groves (CBEP/USM), John Sowles (DEP)

At your request, I have conducted a human health assessment of the mussel contaminant data from Casco Bay. The purpose of the human health assessment was to evaluate whether recreational harvesting of mussels and subsequent consumption might result in a significant potential health risk arising from the presence of chemical toxicants in edible tissue. In performing this assessment, I have generally followed the same procedures used in deriving our fish consumption advisories. This memorandum is organized into two sections. Section 1 provides a summary of methods, data, results, discussion and recommendations. Section 2 provides additional details on our derivation of action levels for chemical residues in edible fish and shellfish tissue.

I. Summary of Health Evaluation

Methods: Tissue action levels were derived for the tested chemical contaminants following standard Bureau of Health and USEPA procedures.¹ In the case of threshold toxicants, action levels are set at tissue concentrations that allow consumption of one fish or shellfish meal per week with minimal risk of any deleterious health effects even among sensitive members of the population, based on the use of a USEPA reference dose (RfD). In the case of carcinogens, action levels are set so that consumption of edible tissue at a rate of one 8-ounce meal per week for a lifetime (70 year assumption) would result in a estimated incremental lifetime cancer risk of 1-in-a-100,000. These action levels are then compared to the concentration of chemical contaminants in uncooked fish or shellfish edible tissue. Comparisons are made to the mean and the 95th percentile upper confidence limit of the mean concentration of each chemical

¹ Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories: Vol. 2 Risk Assessment and Fish Consumption Limits, Third Edition. EPA 823-R-99-008, August 1999.

contaminant. Mean concentrations of contaminants in excess of an action level is viewed as evidence of a potential health hazard, though does not necessarily imply that a consumption advisory is warranted. For example, the Bureau of Health is currently considering a policy in which current action levels set on a chemical-by-chemical basis with a incremental cancer risk level of 1-in-100,000 for carcinogens would be used as a trigger for notifying the DEP when fish or shellfish are showing signs of "significant" chemical contamination, while an *aggregate* incremental cancer risk level of 1-in-10,000 based on summing risks of all carcinogens detected in fish would be used as the basis for issuing advisories.

Data: The unit of chemical analysis was a composite sample consisting of approximately 20 (check?) individual mussels. Four composite samples were obtained from eight sampling locations in Casco Bay. The sampling was conducted in 1996 for four stations (Jewell, Back Cove, Quahog, Harraseeket) and 1998 for the remainder (Falmouth, Freeport, New Meadows, Middle Bay stations). Each composite sample was analyzed for a number of elements (notably Pb, Hg, As, Cd), polychlorinated dibenzo-p-dioxins and dibenzofurans, coplanar PCBs, total PCBs, pesticides, and polycyclic aromatic hydrocarbons. Analytical results were generally reported on a wet weight basis for uncooked tissue, with the exception of data for Back Cove where results were reported for both uncooked and cooked tissue. Analytical results reported as nondetect were assumed present at a concentration equal to ½ the detection limit.

Results: Tables 1, 2 and 3 summarize the data on presence of the selected chemical contaminants in edible tissue from mussels collected at the eight sampling sites in Casco Bay. Inspection of these tables reveals the following:

- Levels of the element lead in composite samples collected in Back Cove were slightly above the action level for this neurotoxin.
- Levels of the pesticide dieldrin, a probable human carcinogen, were elevated at all sampling locations except Falmouth and Middle Bay. Levels of dieldrin in mussels collected from Freeport and Harraseeket stations were especially high and may warrant issuance of an advisory upon data confirmation.
- Levels of polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDDs & PCDFs) reported as 2,3,7,8-TCDD Toxic Equivalents were above action levels for both carcinogenicity and non-carcinogenic effects at Freeport, New Meadows, Jewell, Back Cove and Harraseeket. However, the QA/QC surrogate recovery data for all 1996 data was low and highly variable.
- Total PCB levels were elevated in mussels collected from Back Cove and Quahog and less so for Falmouth as well, with the highest level detected approaching 3-times the action level for carcinogenicity. Interestingly, coplanar PCB levels were all very low.
- Only the following PAHs were evaluated for toxicity. Benzo[a]pyrene, benzo[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, chrysene, dibenz[a,h]anthracene, and

indeno[1,2,3-cd]pyrene were evaluated for carcinogenicity using a USEPA toxic equivalency approach relative to the potency of benzo[a]pyrene.¹ The compounds anthracene, fluoranthene, fluorene, and pyrene were evaluated for non-carcinogenic effects using available USEPA RfDs. Of the four compounds evaluated for potential non-carcinogenic effects, none approached levels of concern. With respect to carcinogenicity, toxic equivalent levels were generally no more than twice the action level indicating incremental cancer risks for chronic and frequent intake of mussels of less than 2-in-100,000. The QA/QC data for 1996 indicated relatively poor precision among duplicates.

- Cooked mussels exhibited about a 3 to 5-fold increase in the concentration of lead (and other elements) in tissue when cooked. This increase was presumably due to loss of water and possibly lipid during cooking, based on about a 3-fold increase in percent solids for cooked versus uncooked tissue. In contrast, many of the organic pollutants (though notably not dieldrin) appeared to have significant reductions upon cooking. However, these cooking losses for organic compounds were highly variable and need to be confirmed.

Discussion: By far the most noteworthy observation from a public health perspective was the elevated levels of the banned pesticide dieldrin at a number of the sampling locations. Dieldrin is an organochlorine pesticide that was phased out between 1974 and 1987, and was used mainly on soil dwelling pests and for termite control.¹ Dieldrin is also a product of aldrin metabolism, a structurally similar pesticide which is also no longer used.¹ Dieldrin is considered a probable human carcinogen by the USEPA. Dieldrin levels were especially high for mussels collected at Freeport and Harraseeket, where chronic intake of mussels at a rate of one meal per week is estimated to pose a incremental cancer risk of 1-in-10,000. These data are quite different from the monitoring results that have been obtained for mussels sampled as part of the Gulf of Maine Gulfwatch monitoring program. GulfWatch has tended to report dieldrin levels ranged from <2 to 5 ppb on a dry weight basis.^{2,3} In contrast, levels detected in mussels collected from Freeport and Harraseeket stations were 95 and 155 ppb on a dry weight basis, respectively. The other Casco Bay sampling locations had dieldrin levels of 20 to 40 ppb on a dry weight basis (with the exception of Falmouth which had levels less than 5 ppb). As these results are in apparent contrast to those obtained from GulfWatch, we recommend that an effort be made to confirm these results as soon as it is feasible. As the QA/QC results on precision and accuracy look fairly good for dieldrin, confirmation will likely involve resampling and analysis.

The exceedance of the action level for the neurotoxin lead in the samples from Back Cove is noteworthy, though less surprising. The primary concern here is with consumption by young children. BOH analyses indicate that regular consumption (a meal per week) of mussels

² Evaluation of GulfWatch – 1996: Sixth Year of the Gulf of Maine Environmental Monitoring Plan. The Gulf of Maine Council on the Marine Environment, December (1997), by Chase M, Jones S, Hennigar P, Sowles J, Coombs K, Crawford R, Harding G, Pederson J, Taylor D.

³ Evaluation of GulfWatch – 1995: Fifth Year of the Gulf of Maine Environmental Monitoring Plan. The Gulf of Maine Council on the Marine Environment, December (1996), by Chase M, Coombs K, Crawford R, Harding G, Hennigar P, Jones S, Pederson J, Robinson W, Sowles J, Taylor D.

by young children (ages less than 6 years) could result in a significant chance (>5%) that blood lead would exceed levels of concern (10 µg/dL). Children are more sensitive/vulnerable to the toxic effects of lead because of their actively developing nervous systems and their increased potential for exposure to other lead sources due to hand-to-mouth activity (e.g. lead paint). Importantly, with lead exposure, we are concerned about effects on children exposed over a relatively short period of time as compared to the lifetime exposure typically assumed for carcinogenic effects. Comparison of data from Back Cove with results obtained by GulfWatch indicates that the average lead concentrations in mussels collected from Back Cove are atypical. Average lead levels on a dry weight basis for Back Cove versus GulfWatch was 10 ppb versus 2 ppb, respectively. NOAA has used a lead level in excess of 4.5 ppb on a dry weight basis as atypical in their Mussel Watch survey for trends.⁴

It is not immediately apparent whether the apparent increase in the concentration of lead in mussels when cooked is of concern. The relevance of this observation to public health depends on whether this increase in *concentration* also results in an increase in *mass* of ingested lead. An increase in mass of ingested lead would only occur if the assumed consumption rate for mussels was based on an intake rate of cooked meat, as our data on chemical content is typically uncooked tissue (hence, a inconsistency). Because of the way the action level for lead was derived (via use of EPA's integrated exposure uptake biokinetic "IEUBK" model for lead described below), it will take some investigation into the details of the IEUBK model to determine the underlying data and assumptions used in modeling dietary intake of meat (including fish and shellfish).

In contrast, the cooking loss seen for some of the organic pollutants is more directly relevant as these losses will represent a reduction in the mass of ingested chemicals. However, while some chemicals exhibits substantial losses (50 to 80%), others exhibited either minimal loss or an apparent increase. There is published literature reporting cooking losses for organic pollutants, as well as apparent increases for metals. So the general findings here are consistent with that literature. Indeed, a number of state health agencies assume a default 50% loss of organic chemicals from fish and shellfish tissue due to cooking.⁵ We are just beginning to review these data and assess whether to use a default cooking loss. Additional data on apparent cooking losses and gains would be helpful.

The elevated levels of PCDDs & PCDFs in mussels are of concern as the action level for reproductive & developmental toxicity of 1.8 parts per trillion (ppt) was exceeded (though never by 2-fold) at Freeport, Jewell, Back Cove and Harraseeket stations. Levels at New Meadows were fairly close to the action level. Mussels collected at these five stations also exceeded the cancer action level of 1.5 parts per trillion (ppt). These results are noteworthy in several ways beyond the public health significance. First, the concentrations are considerably higher than

⁴ O'Connor., T.P. and B. Beliaeff. 1995. Recent Trends in Coastal Environmental Quality : Results from the Mussel Watch Project. National Status and Trends Program. NOAA. Silver Spring, MD.

⁵ 1999 American Fisheries Society Forum on Contaminants in Fish., October 18-20, 1999, Discussion group materials prepared by EVS Environment Consultants, Inc., American Fisheries Society, Bethesda, MD.

levels reported in mussel monitoring associated with GulfWatch.² Second, on average 60 to 70 percent of the 2,3,7,8-TCDD toxic equivalents are contributed by the PCDD congener 1,2,3,7,8-Pentachlorinated dibenzo-p-dioxin. This observation is in contrast to what we typically see in fish collected downstream from pulp and paper mills, where toxic equivalents tend to be dominated by the 2,3,7,8-tetrachlorinated dibenzo-p-dioxin and furan isomers. It is also in contrast to mussels collected in the vicinity of the Kennebec River and Penobscot River, where 2,3,7,8-TCDF and OCDD were the only isomers detected.² This may indicate an alternative source of PCDDs and PCDFs is affecting these waters. For example, there are some data indicating that municipal waste incinerators have a tendency to emit pentachlorinated CDDs and CDFs.⁶

Total PCB levels in mussels were above the action level at Falmouth, New Meadows, Back Cove and Quahog sampling stations, with the latter two clearly the highest. The primary concern is with an increase risk of cancer from in the 1 to 3 per 100,000 range assuming chronic and routine consumption. The PCB levels look generally consistent with those obtained by GulfWatch for several Maine stations, with two exceptions. First, levels at Back Cove and Quahog were clearly higher by comparison. Second, the PCB congener 2,2',4,6,6'-Pentachlorobiphenyl (IUPAC# 104) was an important contributor to total PCB levels in Casco Bay mussels but was not for any of the Maine GulfWatch stations.

Evaluation of the data on polycyclic aromatic hydrocarbons (PAHs) was complicated by two factors. First, the 1996 data show relatively poor precision among duplicates. This may in part explain the considerable variability among sample replicates for a given location. Second, some the PAHs coelute preventing unique quantification. This is especially a problem for dibenz[a,h]anthracene (D[a,h]A) and indeno[1,2,3-cd]pyrene (I[1,2,3-cd]P) where the former has a toxic equivalency factor of 1.11 and the latter is 0.055, a 20-fold difference. It was therefore necessary to make an assumption about the relative fractions of these two compounds. A 50% split was assumed. A worst case assumption (i.e., assume all present as the more toxic PAH) would only increase the toxic equivalents by 20 to 30 percent. As the action level was never exceeded by a factor of more than 3, incremental lifetime cancer risk from an assumed intake of mussels from these sites at a rate of 1 meal per week for 70 years is estimated to not exceed 3-in-a-100,000. The levels of PAHs detected on a dry weight basis are somewhat similar to levels reported by GulfWatch for mussels obtained from their Clark Cove and Fort Point sampling locations. However, Casco Bay stations tended to have higher levels of the more carcinogenic potent B[a]P and combined D[a,h]A & I[1,2,3-cd]P PAHs along with others.

A few comments are in order for arsenic. Mussels collected from Falmouth and Jewell had arsenic levels about two-times the action level of 0.7 ppm. However, as discussed more fully below, interpretation of arsenic levels in seafood is confounded by uncertainty about the fraction of arsenic present as the toxic inorganic form. Most of the arsenic present in seafood

⁶ Estimating Exposure to Dioxin-Like Compounds, Volume III: Site-Specific Assessment Procedures, U.S. Environmental Protection Agency, Office of Research and Development, June 1994, Review Draft, EPA/600/6-88/005Cc (see pp 3-7 to 3-9, and Figure 3-1).

occurs as a relatively non-toxic organic form called arsenobetaine. Data on the fraction of arsenic in mussels present in the inorganic form ranges from 0.5% to 10%, and these data are limited. We have assumed 5% in deriving the action level. Based on the available data, we think it likely that the percent inorganic arsenic is less than 5%, perhaps closer to 1 or 2%. Arsenic levels seen in the mussels collected from Casco Bay are in general agreement with levels reported by NOAA for Mussel Watch.

Need for shellfish consumption advisories: Based on current data, if the Bureau of Health was to issue a shellfish consumption advisory for Casco Bay, it would likely be a warning to limit intake of recreationally harvested mussels from these waters to no more than 1 meal per week or 1 meal every other week. The advisory would be largely driven by the cancer risk posed by dieldrin (representing 50 to 70% of the total cancer risk for 5 of the 8 sampling locations). Dioxins contribute between 7 and 20% of the aggregate risk. PCBs contribute 5 to 47% of the aggregate risk. PAHs contribute 9 to 38% (See Table 4). We do not currently have any data to assess frequency with which mussels are recreationally harvested in Casco Bay and consumed, or the extent to which these areas tend to routinely closed due to bacterial contamination and red tide. Such information would be useful in determining the need for issuing any advisories.

Recommendations:

1. The analytical data (primarily the 1996 data) need to be more carefully reviewed with respect to target data quality objectives. We have noted that some data appear to have relatively poor surrogate percent recovery and high percent differences among duplicates. Ideally, data quality objectives should be clearly defined at the outset and data either rejected if objectives are not met, or subject to strong caveats. Such reviews should occur *prior* to data being submitted to the Bureau of Health for health evaluation. It does not appear that this was done.
2. There is a strong need to confirm dieldrin findings. This organochlorine pesticide is a major determinant of aggregate cancer risk. Concentrations in mussels collected in Casco Bay are considerably greater than levels in mussels collected in other Maine locations as part of the GulfWatch program.
3. Information on the extent of recreational harvesting of mussels in Casco Bay would be helpful in evaluating the need to issue an advisory.
4. Data to confirm an apparent significant (though variable) cooking loss of organic pollutants from mussel tissue would also be helpful in assessing the need for advisories. The apparent increase in metal concentration is also of interest, though relevance has yet to be clearly established.
5. The PCDD and PCDF results suggest a possible connection with municipal solid waste incineration. Studies to investigate this hypothesis appear warranted.

6. Data on fraction of arsenic present as the inorganic form in shellfish would be helpful in improving interpretation of such data.

2. Background Information

A. Standard approach for deriving chemical residue action levels

The generic formula used in deriving tissue action levels is:

$$\text{Action Level} = \frac{(RfD \text{ or } RsD) \times BW}{CR} \quad (1)$$

where *RfD* is the reference dose (for noncarcinogenic compounds), *RsD* is the risk specific dose (for carcinogenic compounds), *BW* is body weight, and *CR* is the contact rate.

RfD and *RsD* are compound specific, being measures of a daily dose thought to be without an appreciable risk of deleterious health effects. For noncarcinogenic compounds, it is assumed that there exists a threshold below which toxic effects will not occur. The *RfD* is an estimate of the threshold for potential sensitive members of the population. For carcinogenic compounds, the default assumption is that for every dose there is some increased risk of cancer (i.e., nonthreshold response). *RsD*'s used by the BOH are the chronic dose thought to result in no more than a one-in-a-hundred thousand (10^{-5}) incremental lifetime cancer risk over background. The primary source for obtaining estimates of either a *RfD* or *RsD* is U.S. EPA's Integrated Risk Information System (IRIS) database.⁷

As for the other inputs into equation (1) above, the assumed body weight for the average adult is 70 kg and for the average woman is 60 kg – the latter used when evaluating risk of reproductive and development toxicants. *CR* is assumed to be 32.4 grams per day, based on a consumption rate of one 8-ounce fish or shellfish meal per week. In the case of carcinogens, it is assumed that this rate of fish consumption occurs over a lifetime (70 years).

B. Use of the IEUBK model in deriving an action level for lead

There was one notable departure from these methods. The action level for lead was derived not by equation (1), but rather using US EPA's Integrated Exposure and Biokinetic (IEUBK) model for lead.⁸ This model was developed to estimate for a hypothetical child or

⁷ Integrated Risk Information System, U.S. Environmental Protection Agency, 1 August 1997 Update. IRIS is an electronic data base containing health risk and US EPA regulatory information on specific chemicals. The health risk information represents the consensus opinion Agency scientists from EPA's Reference Dose Work Group and Carcinogenic Risk Assessment Verification Endeavor Work Group.

⁸ Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK), Version 0.99D. U.S. Environmental Protection Agency, EPA 540-R-93-081 / PB93-963510.

population of children, a plausible statistical distribution of blood lead concentrations centered on the geometric mean blood lead concentration predicted by the model. From this statistical distribution, the model calculates the probability that children's blood lead concentration will exceed a blood level of concern. The IEUBK model has four main components: (1) an exposure model that relates environmental lead concentrations to age-dependent intake of lead into the gastrointestinal tract; (2) an absorption model that relates lead intake into the gastrointestinal tract and lead uptake into blood; (3) a biokinetic model that relates lead uptake in the blood to the concentrations of lead in several organ and tissue compartments; and (4) a model for uncertainty in exposure and for population variability in absorption and biokinetics.

The exposure model considers intake of lead from air, water, soil, dust, diet, and allows the user to incorporate alternate sources (e.g. paint, fish, home grown vegetables, etc). Default concentrations of lead, based on national averages, are assumed for each media, therein accounting for current exposures to lead from all known significant sources. Consideration of background exposure to lead is necessary because of the known exposures from a variety of sources and because current blood lead among some children are already at or near levels of concern. In deriving a fish tissue action level, we modified the IEUBK default inputs for diet by increasing the percent contribution of fish to total intake of meat to be consistent with consuming one meal per week by assuming 10% of meat intake was recreationally harvested mussels (actual meal size is set to be consistent with expected intake for a child). We then used the IEUBK model to compute the lead tissue concentration expected to have no more than a 5% chance that a child's weekly consumption of fish (combined with all other sources of lead) would result in a blood lead level of $> 10 \mu\text{g}$ per 100 ml ($10 \mu\text{g/dl}$). The estimated shellfish concentration was 0.7 ppm (wet weight).

C. Consideration of the fraction of arsenic present in the inorganic form in deriving the tissue action level.

Attention is also called to the derivation of an action level for arsenic as it applies to marine fish and shellfish. Arsenic occurs in marine organisms used as human food mainly in organic forms. Organic arsenic is present chiefly as arsenobetaine, a stable compound which has been shown in a number of studies to be metabolically inert and non-toxic (Edmonds and Francesconi, 1993).⁹ Inorganic arsenic, although a minor component of total arsenic in seafood, is the primary toxicity problem, and the action level derived by BOH for arsenic is based on the toxicity of the inorganic form. It is thus necessary to adjust measured amounts of total arsenic in clams to inorganic arsenic. Based on regression analyses, Edmonds and Francesconi (1993) have argued that at low concentrations of total arsenic in seafood, on *average* 1% is inorganic arsenic. However, there is considerable variability in the data. Actual measured percent inorganic arsenic in the mussel tissue (*Mytilus edulis*) has been reported to range from $< 1\%$ to as much as 10% (Edmonds and Francesconi, 1993). The US FDA has assumed a percent inorganic

⁹ J.S. Edmonds and K.A. Francesconi, Arsenic in Seafoods: Human Health Aspects and Regulations. *Marine Pollution Bulletin*, Vol. 26(12): 665-674.

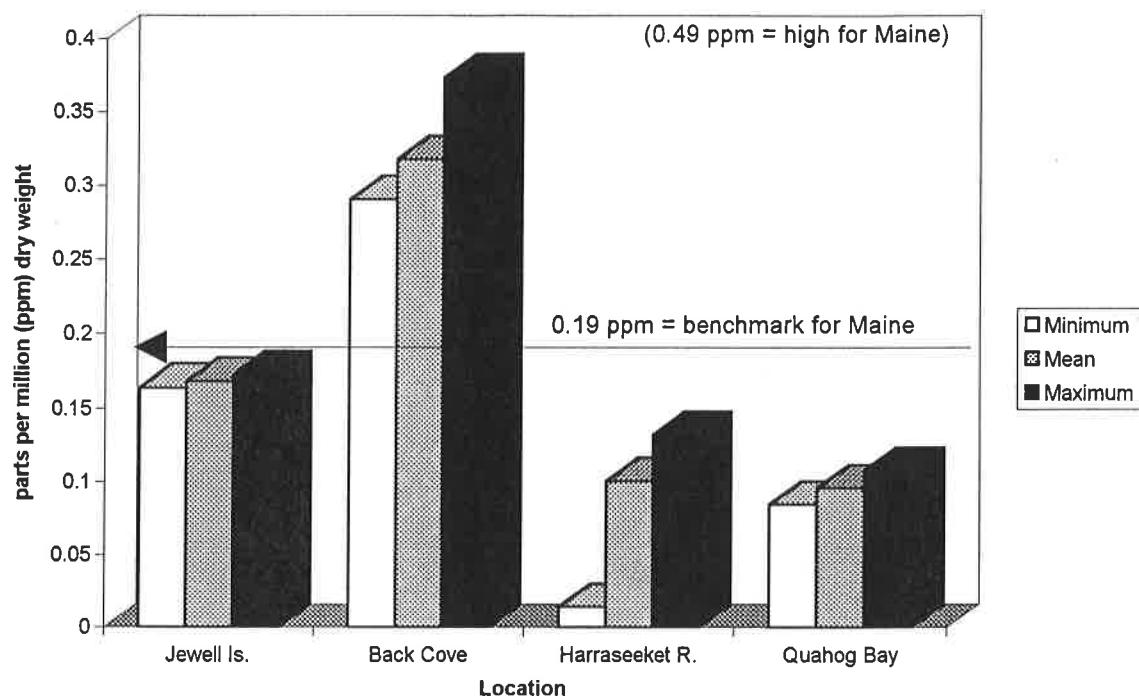
arsenic value of 10% as a conservative health practice when evaluating marine food.¹⁰ In the previous work, BOH staff have assumed a value of 5% of total As present as the inorganic species as being appropriately health protective.

¹⁰ Guidance Document for Arsenic in Shellfish. Center for Food Safety and Applied Nutrition, U.S. Food and Drug Administration, Washington, DC, January (1993).

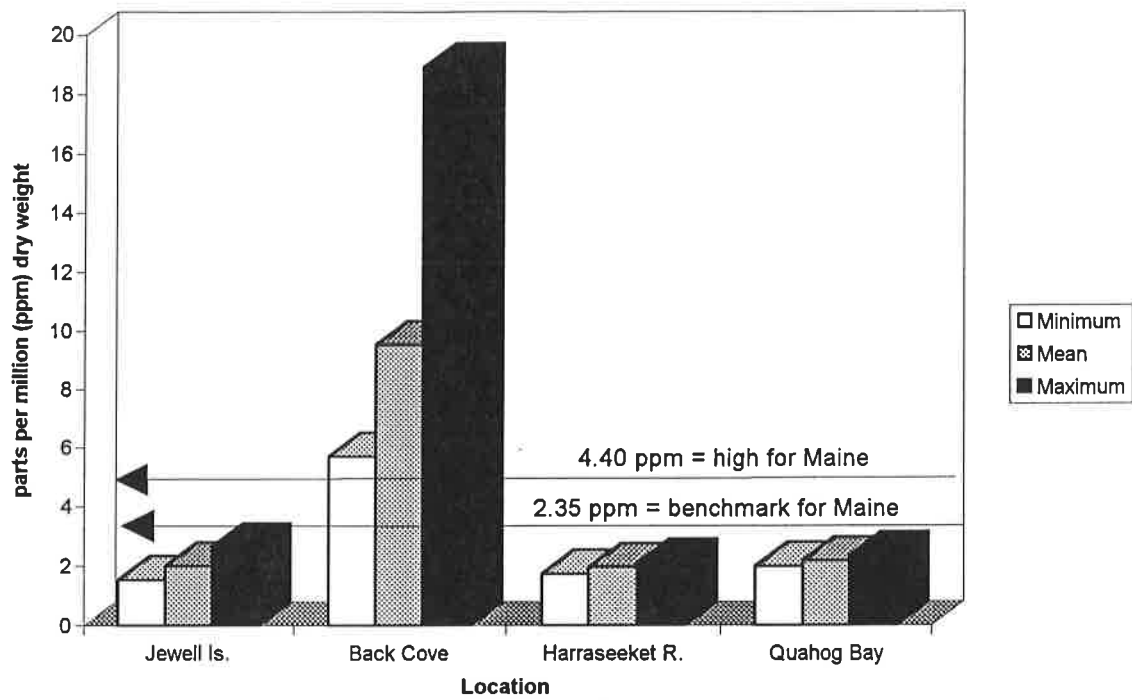
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- Maine Bureau of Health / Environmental Toxicology Program
 - Human Health Assessment of Casco Bay Mussel Data
 - 01/16/01 - DRAFT

Table 2. Age Specific Results from IEUBK Modeling of Blood Lead When Consuming One Meal per Week of Clams			
AGE (years)	Percent Population with Blood Lead Greater Than 10 µg/dL		
	Baseline	Lamson Cove	Peaks Island
1 to 2 years	4%	11%	13%
2 to 3 years	3%	10%	12%
3 to 4 years	3%	9%	11%
4 to 5 years	1%	5%	7%
5 to 6 years	0.5%	4%	5%
6 to 7 years	0.3%	3%	4%

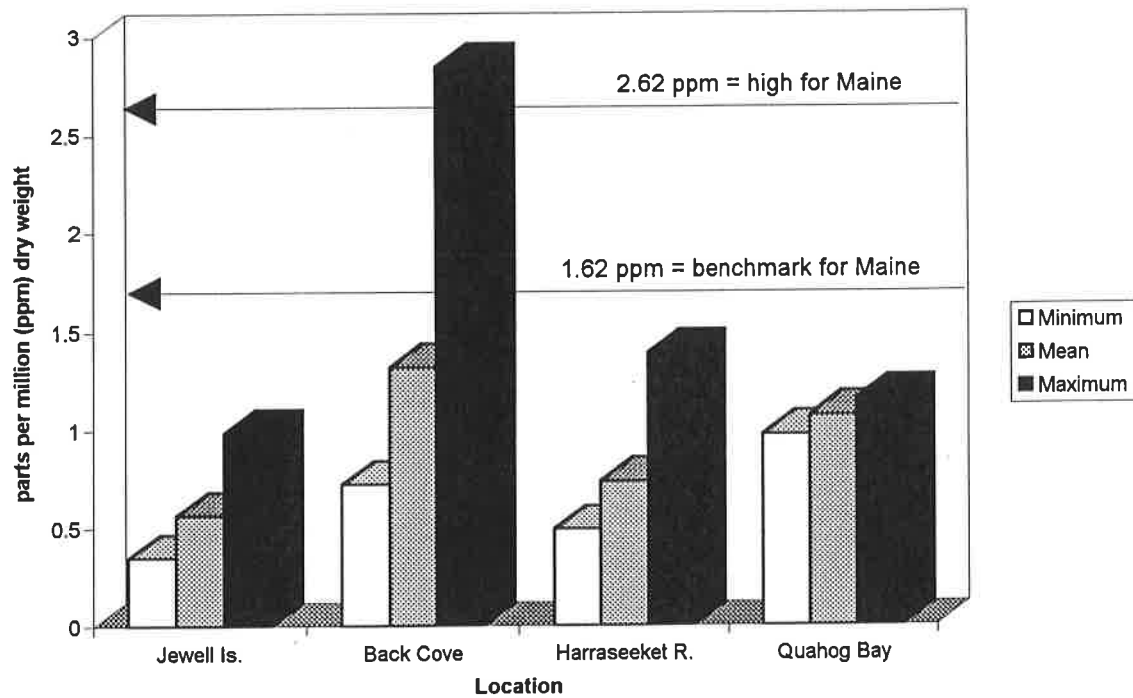
Mercury in Mussel Tissue



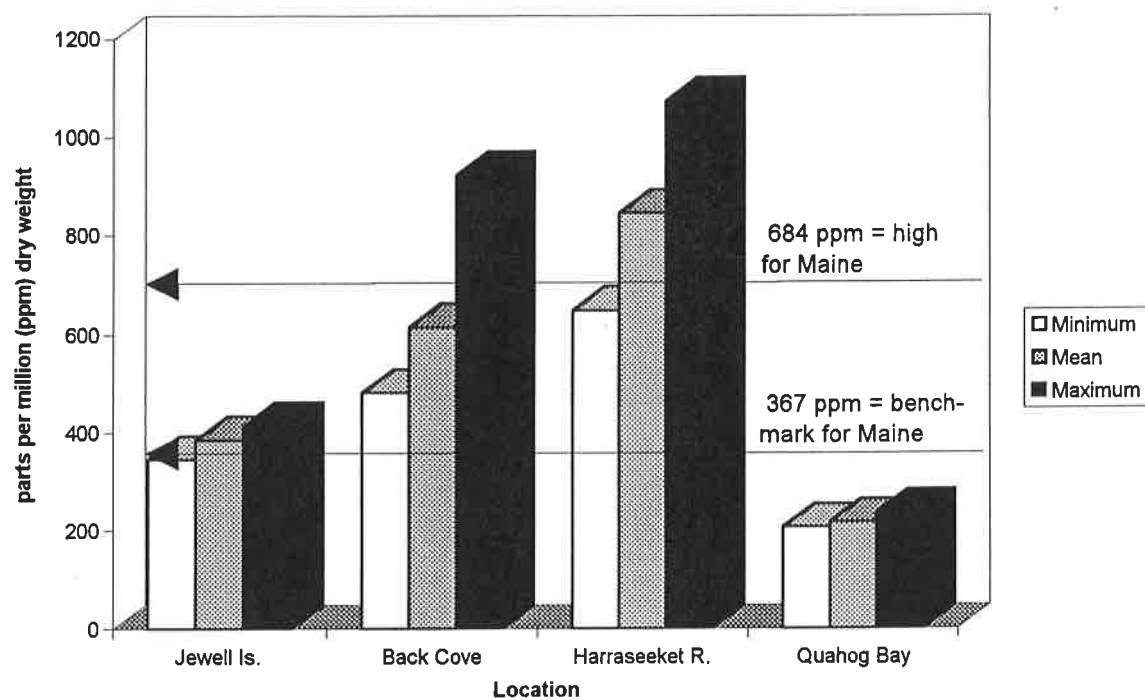
Lead in Mussel Tissue



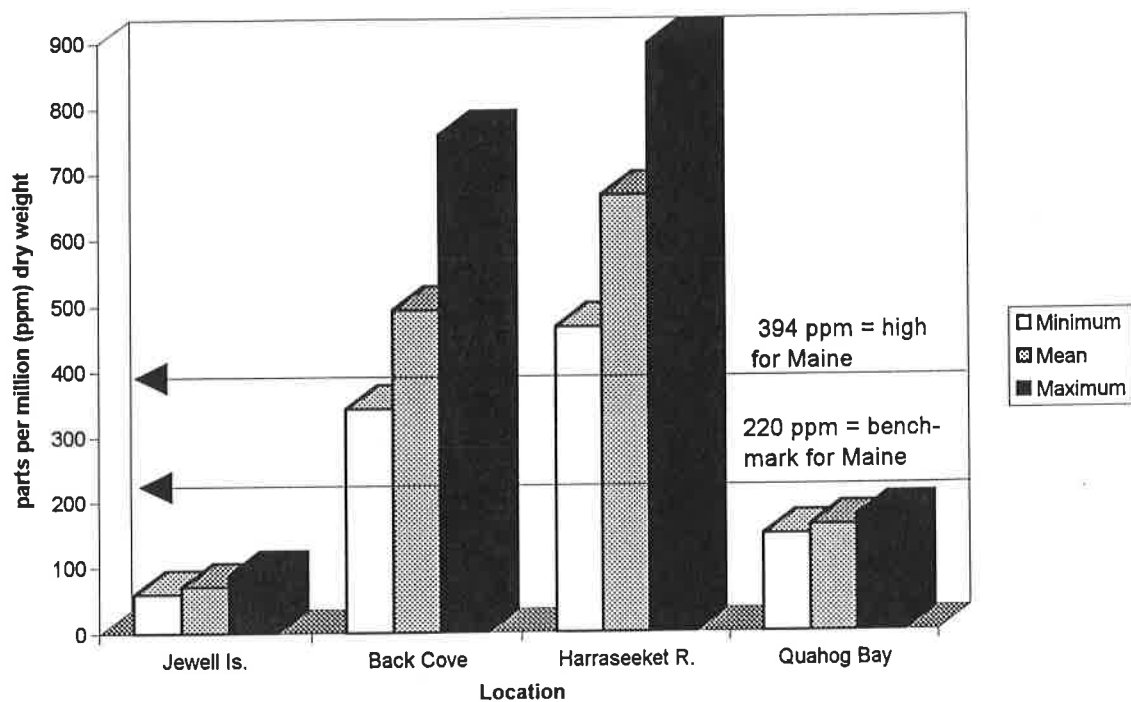
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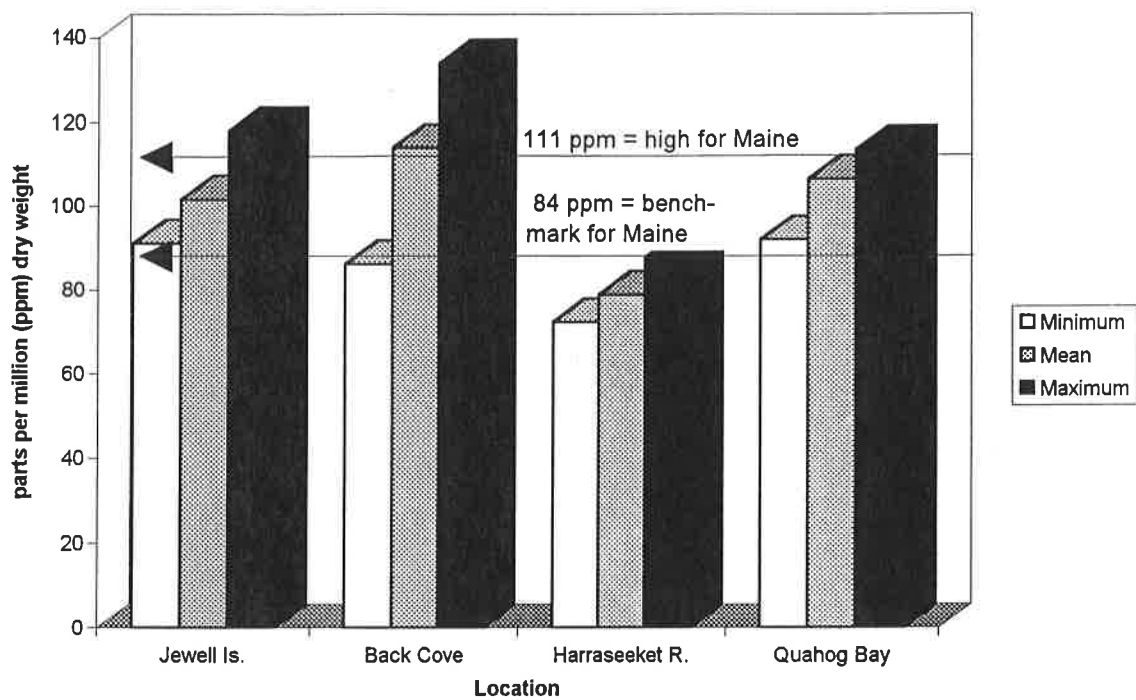
Iron in Mussel Tissue



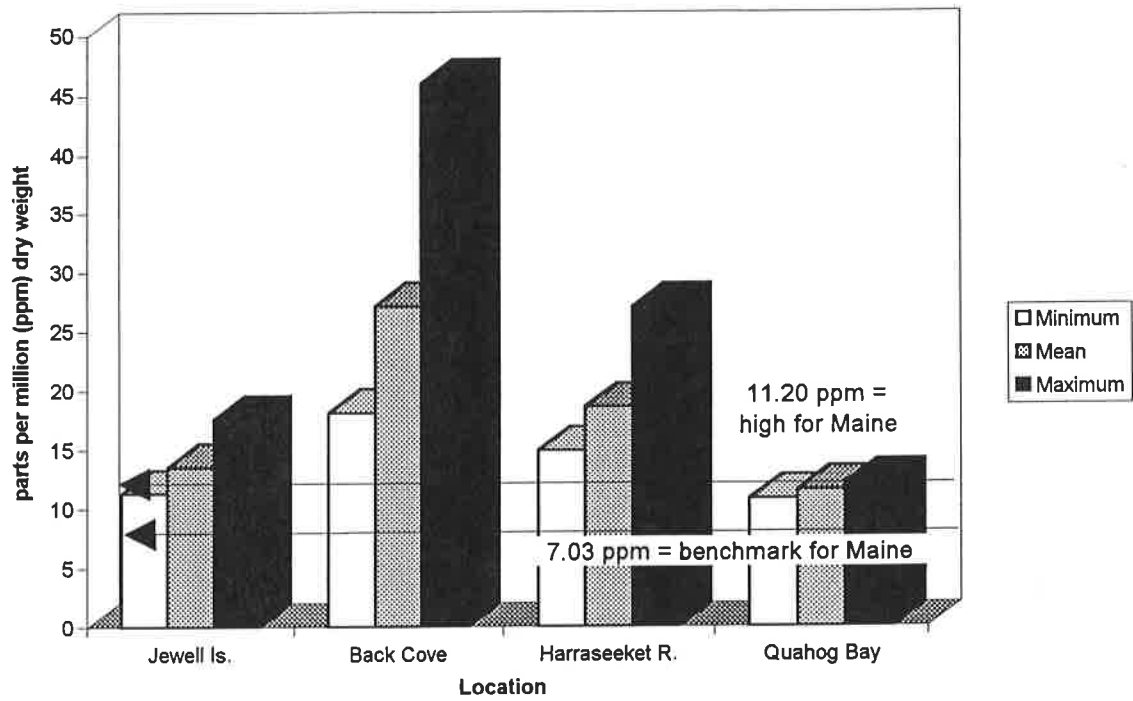
Aluminum in Mussel Tissue



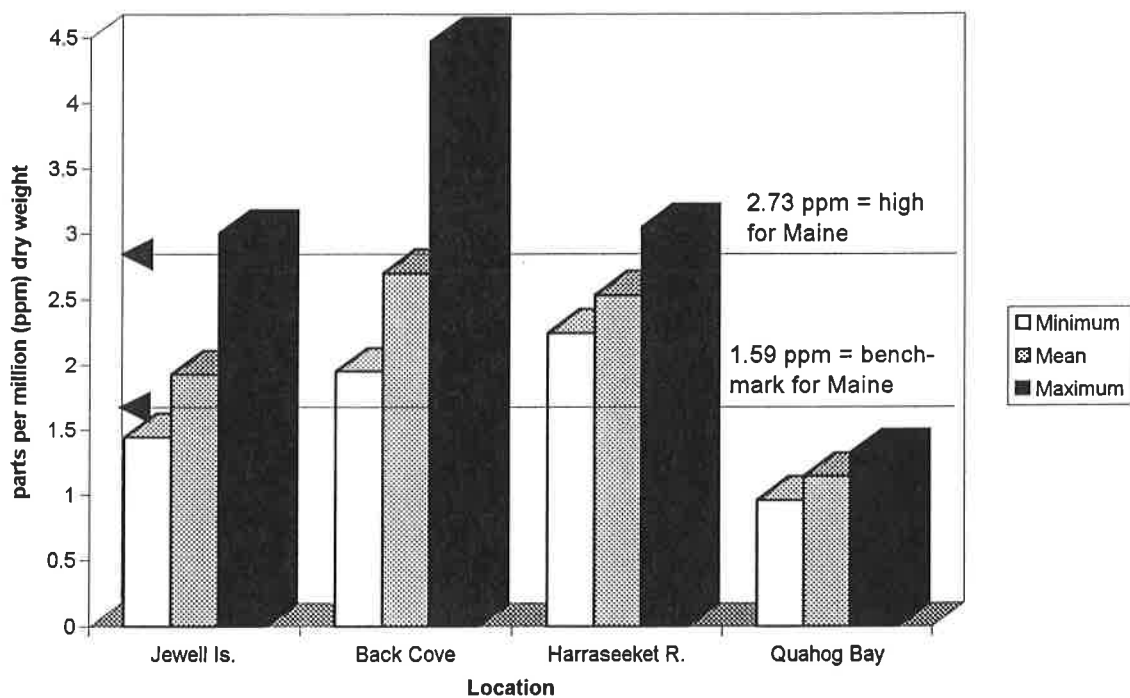
Zinc in Mussel Tissue



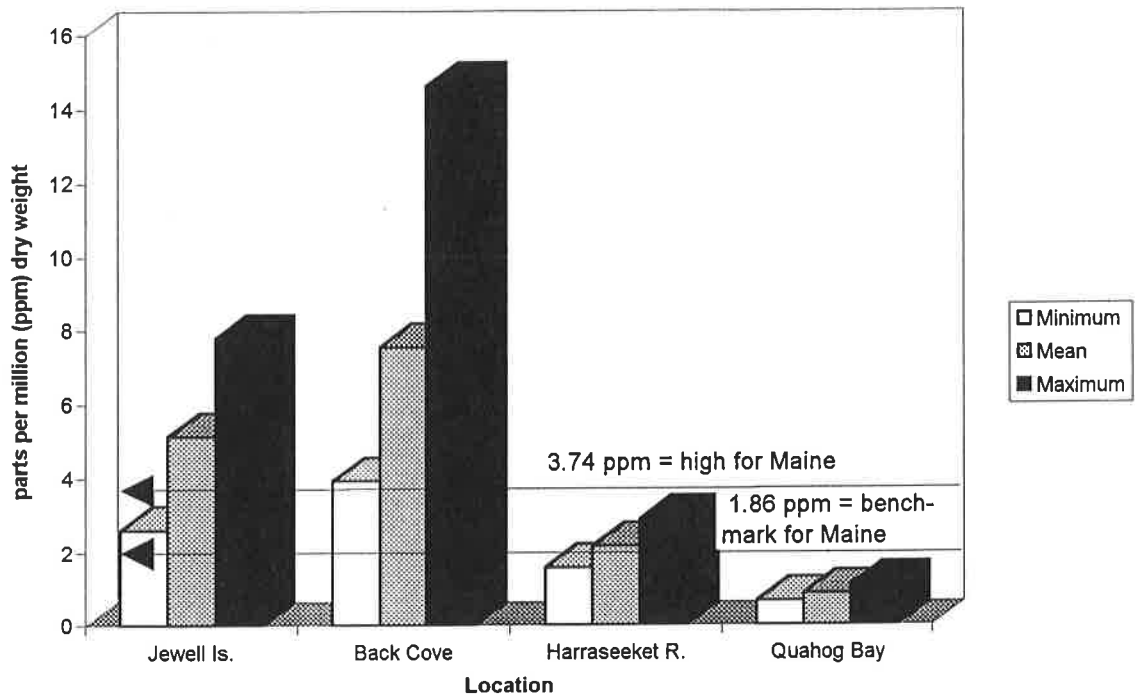
Copper in Mussel Tissue



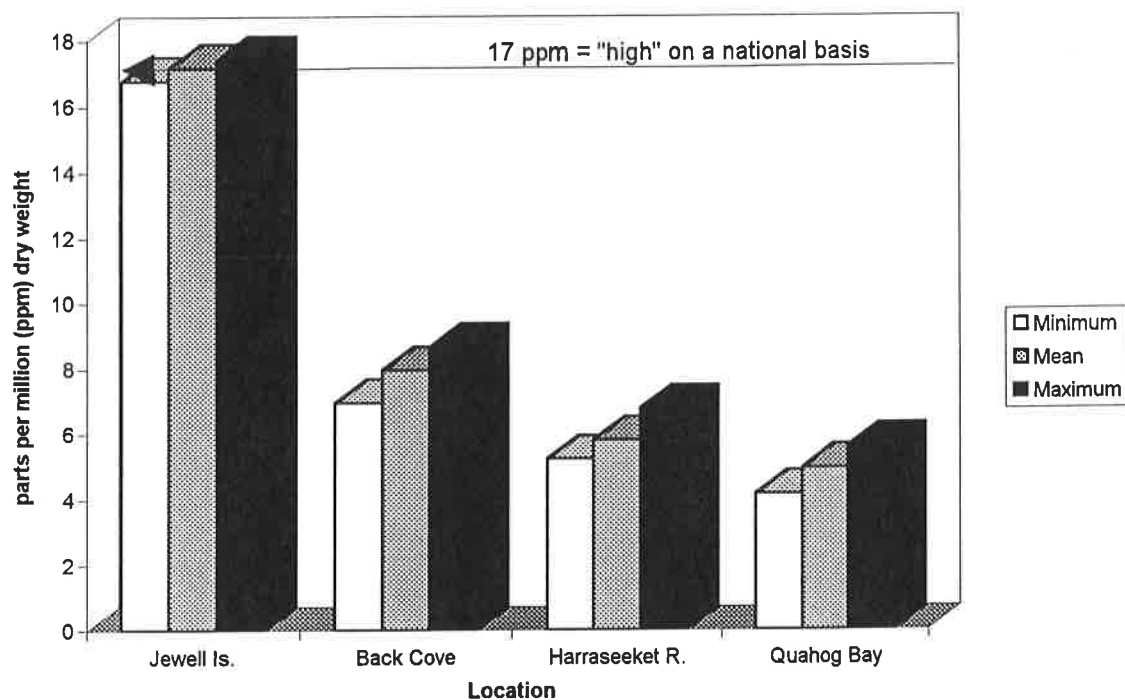
Chromium in Mussel Tissue



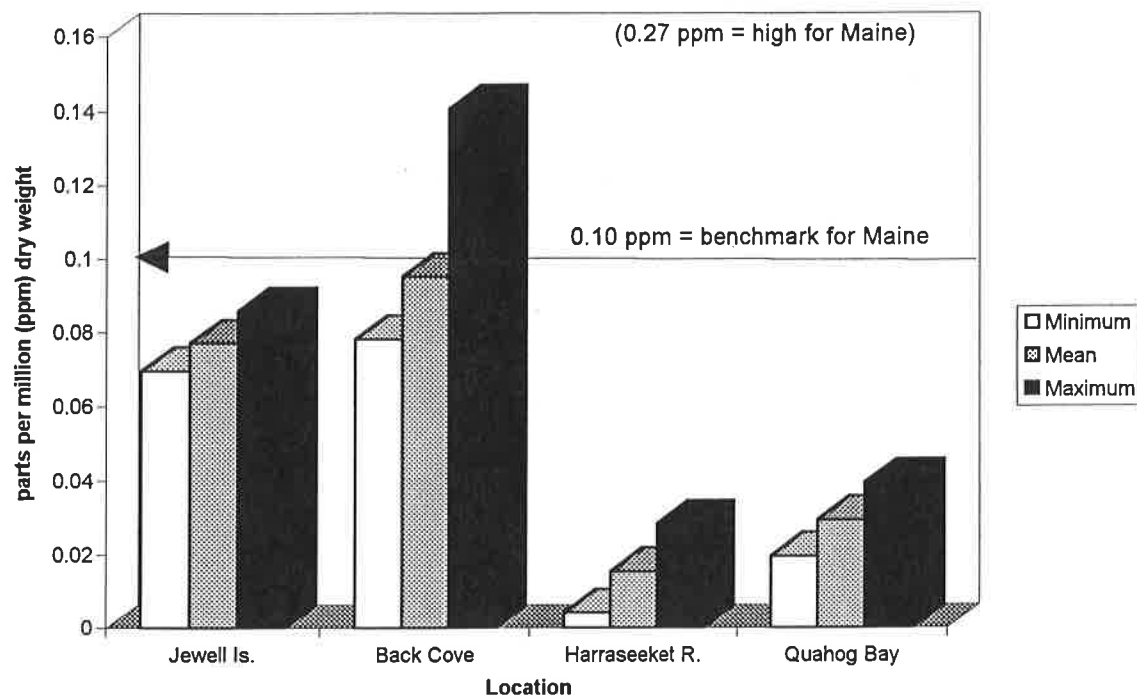
Nickel in Mussel Tissue



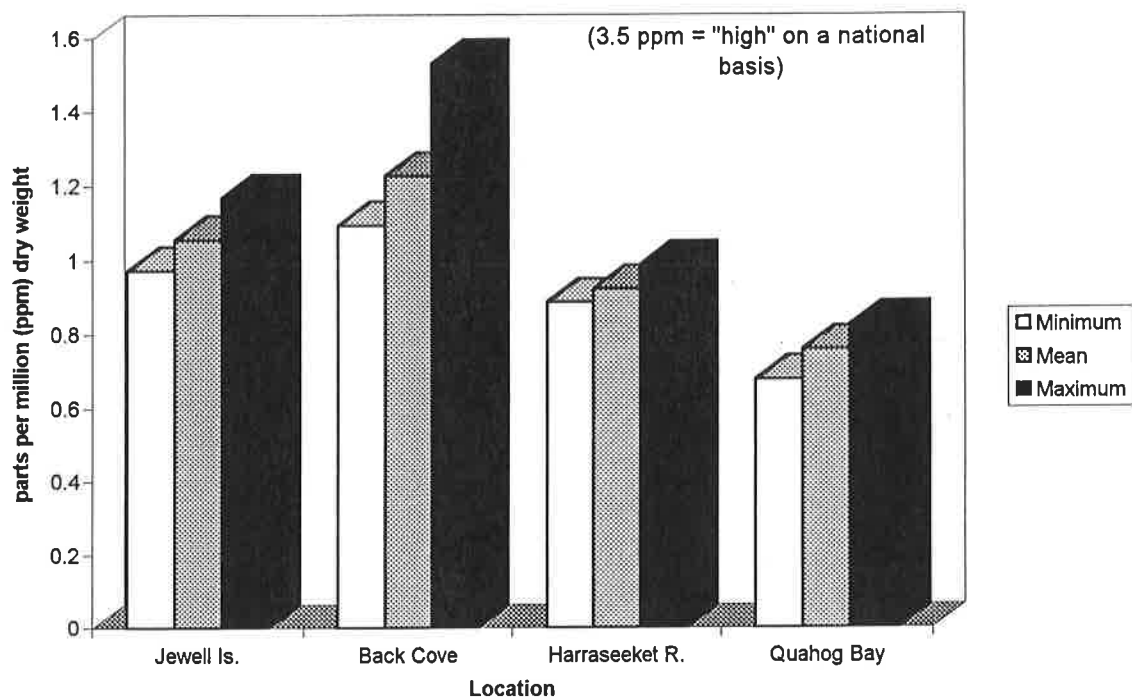
Arsenic in Mussel Tissue



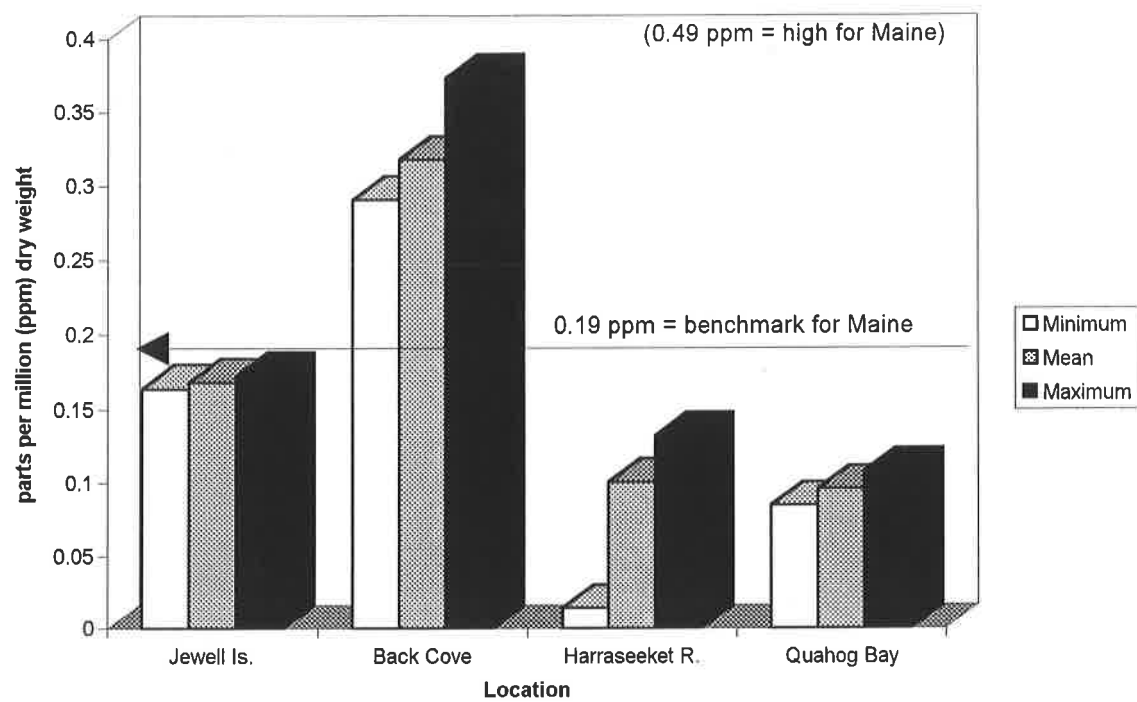
Silver in Mussel Tissue



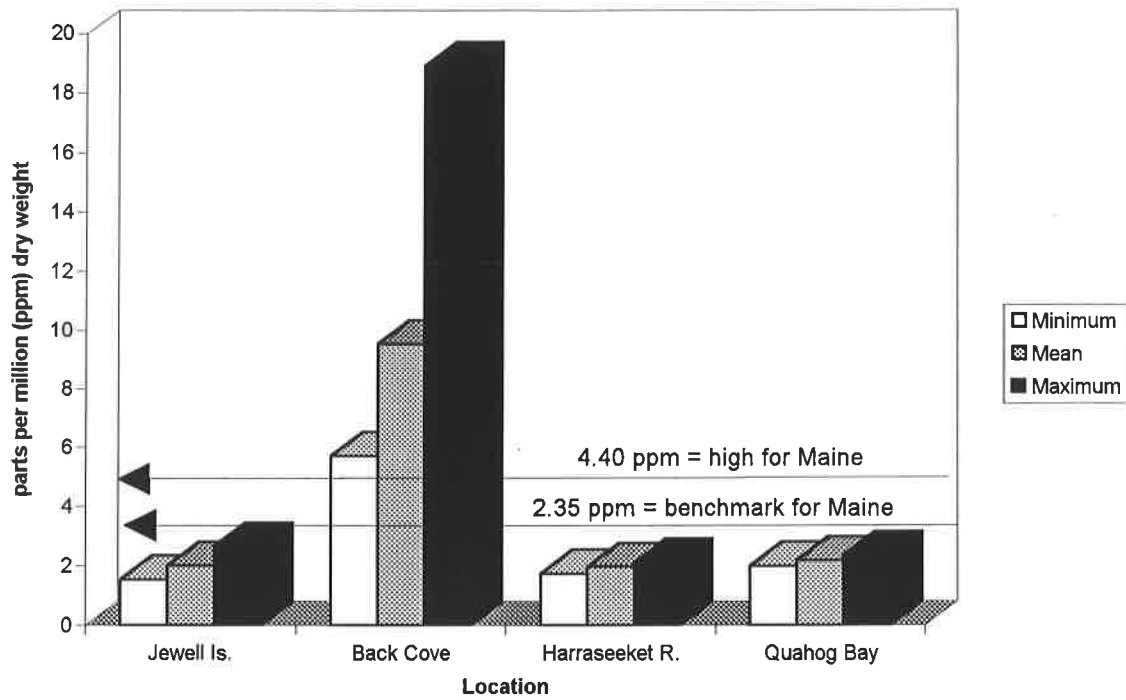
Selenium in Mussel Tissue



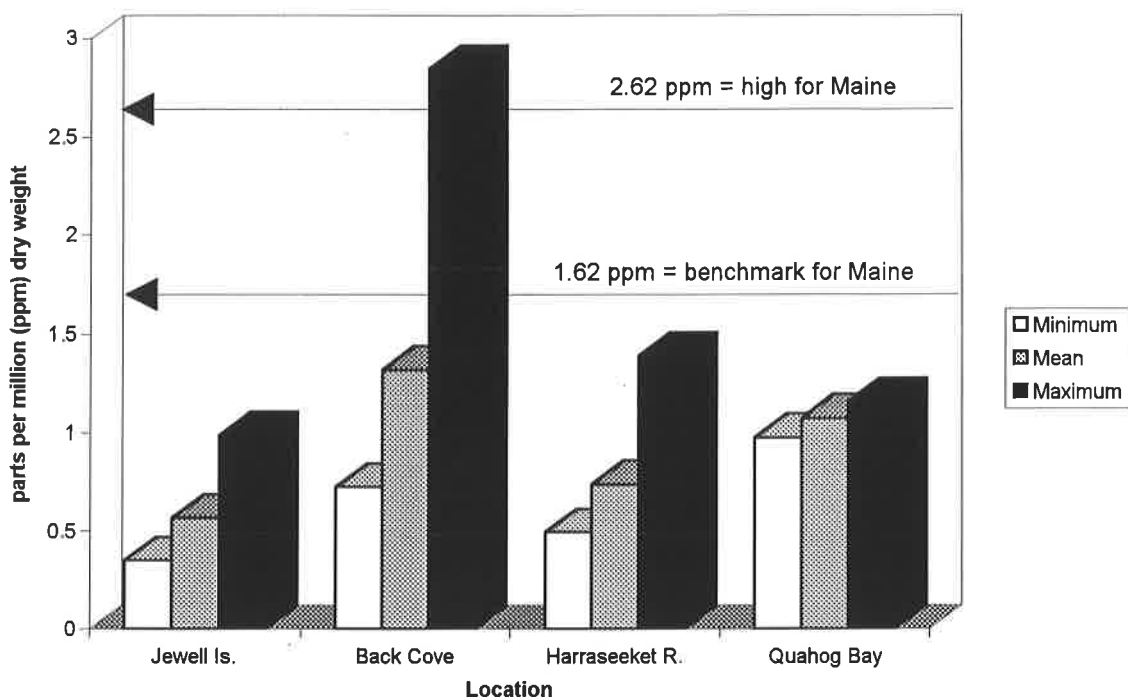
Mercury in Mussel Tissue



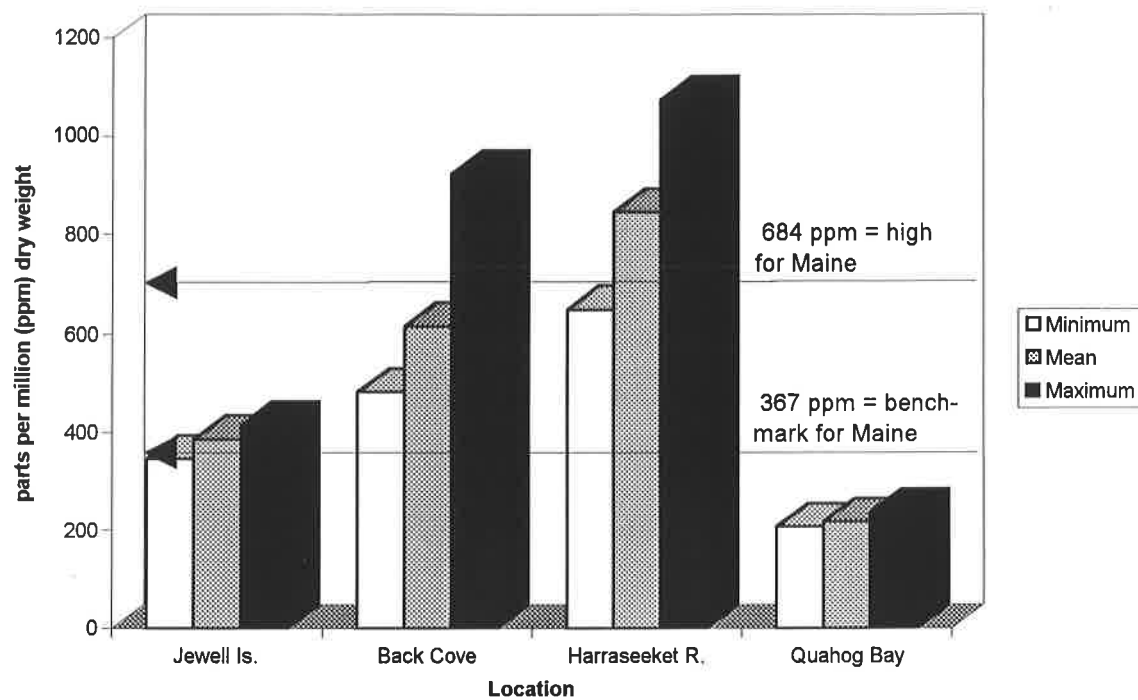
Lead in Mussel Tissue



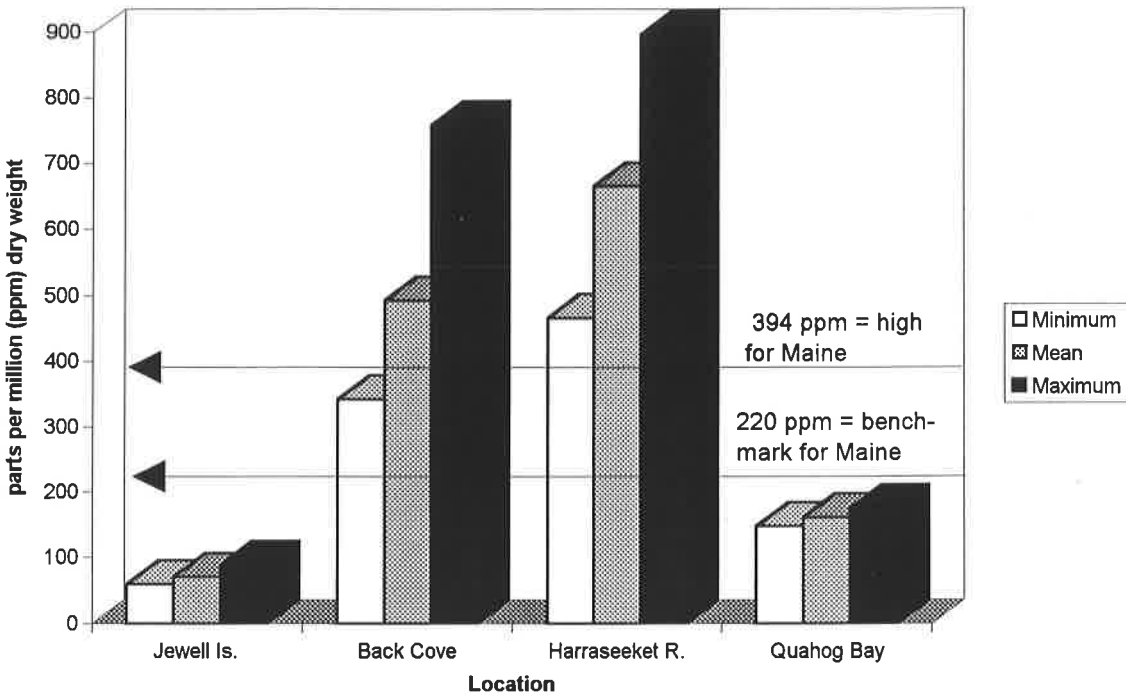
Cadmium in Mussel Tissue



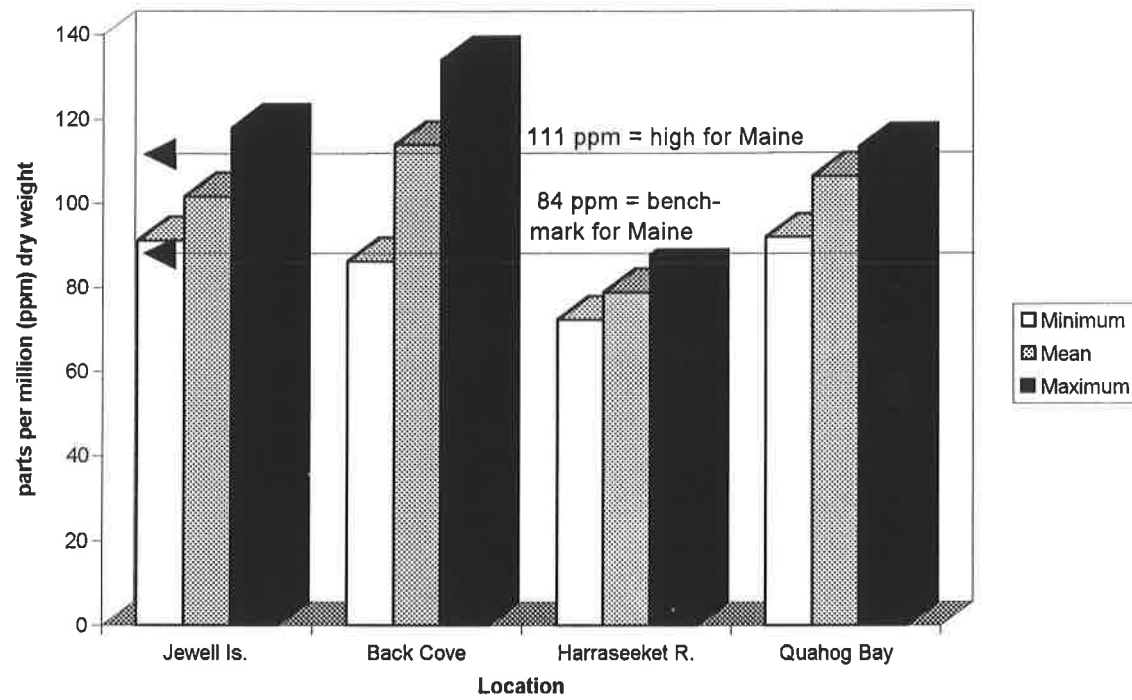
Iron in Mussel Tissue



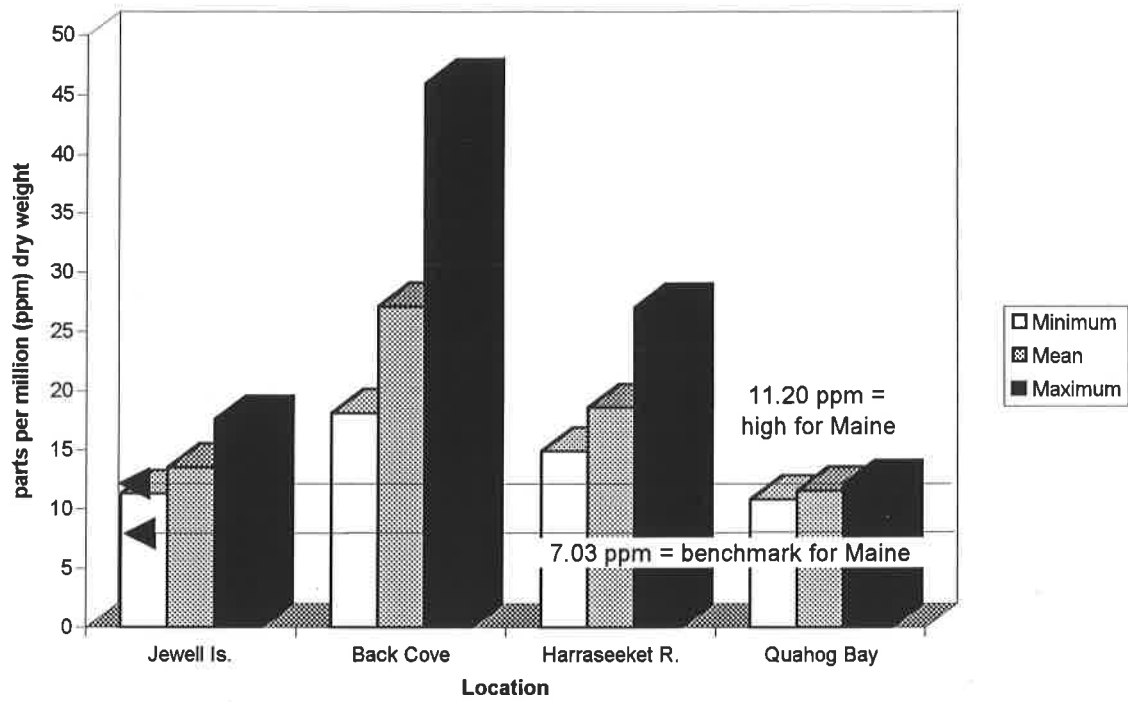
Aluminum in Mussel Tissue



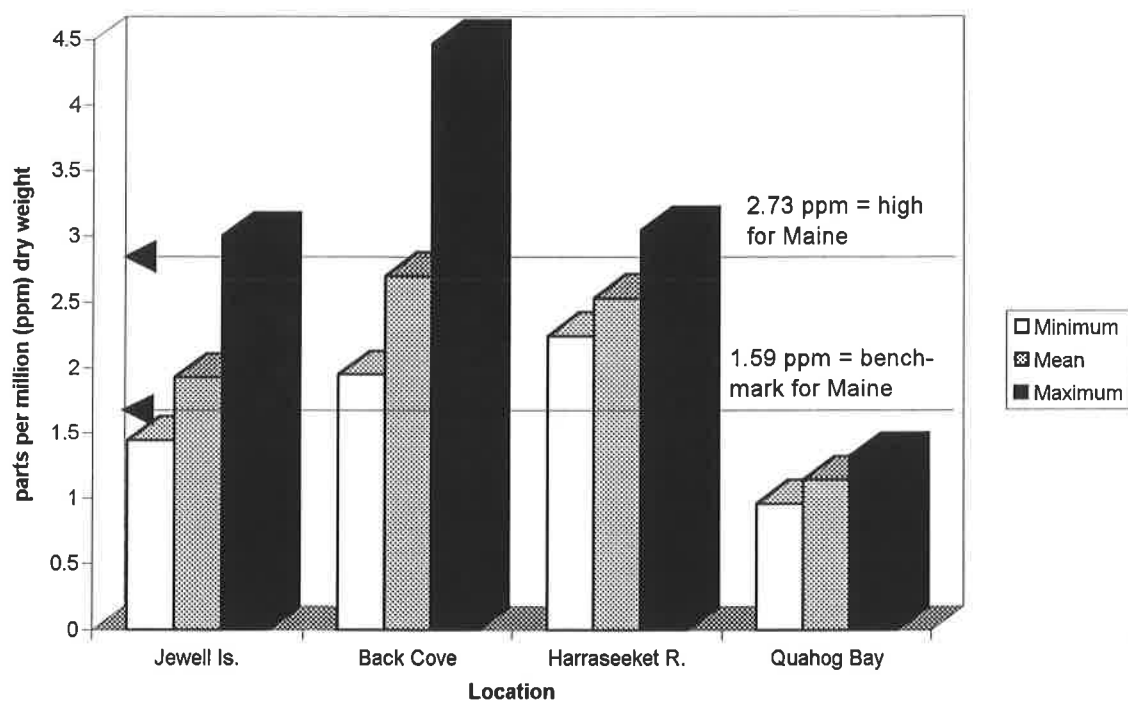
Zinc in Mussel Tissue



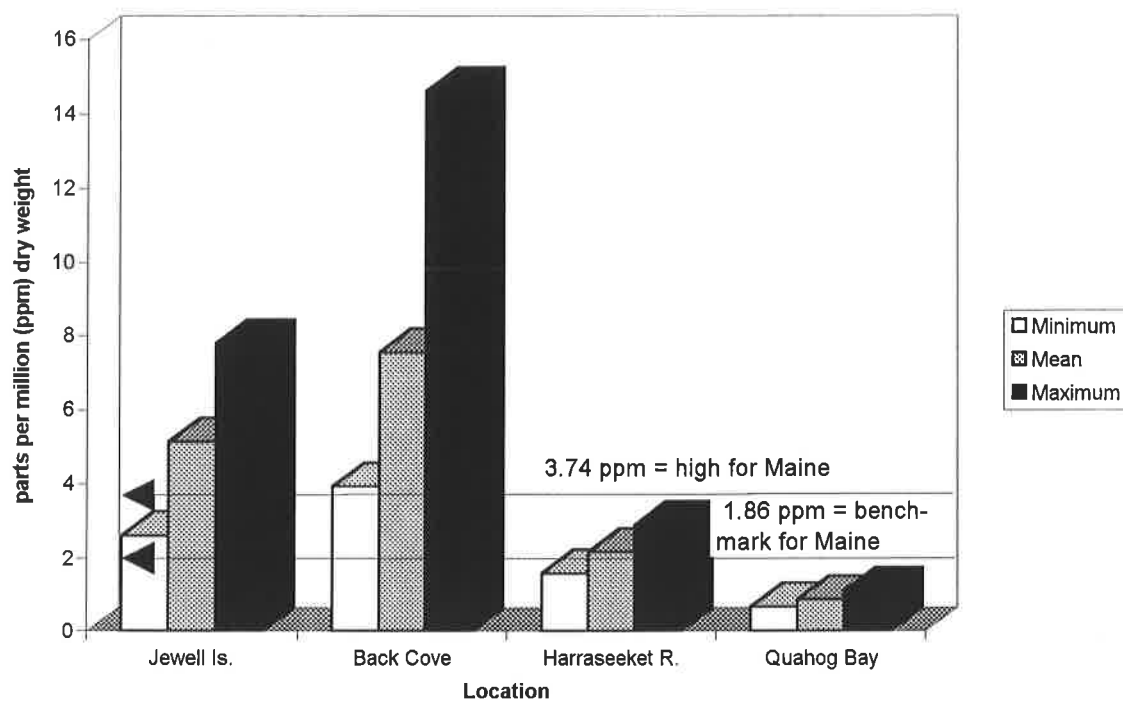
Copper in Mussel Tissue



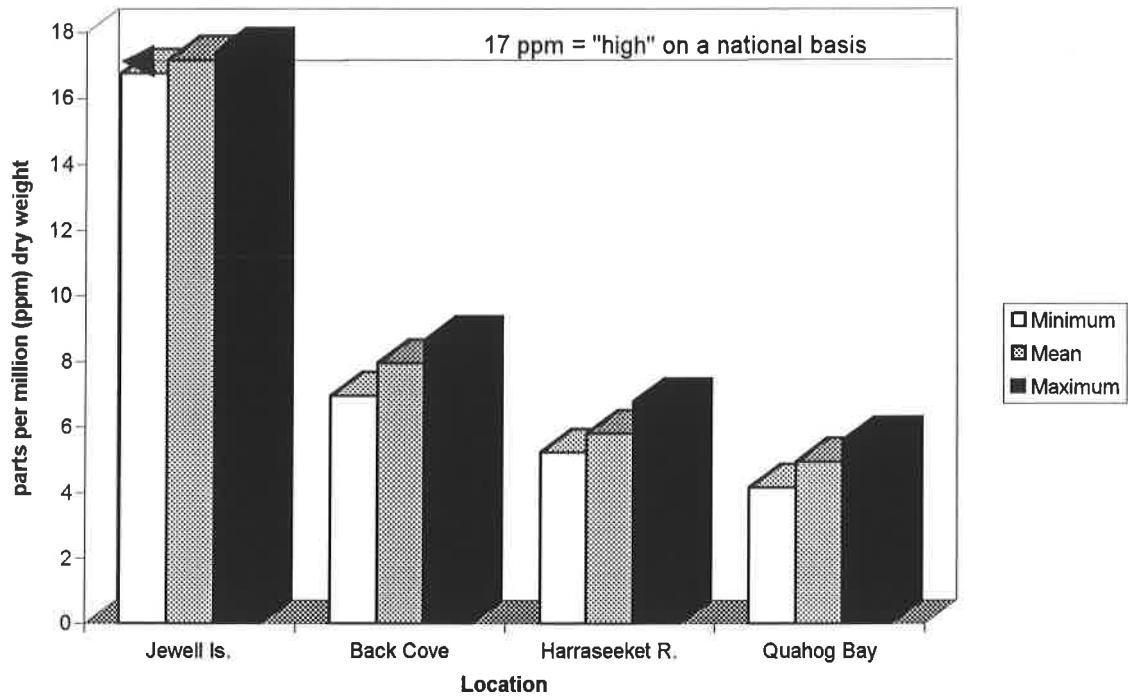
Chromium in Mussel Tissue



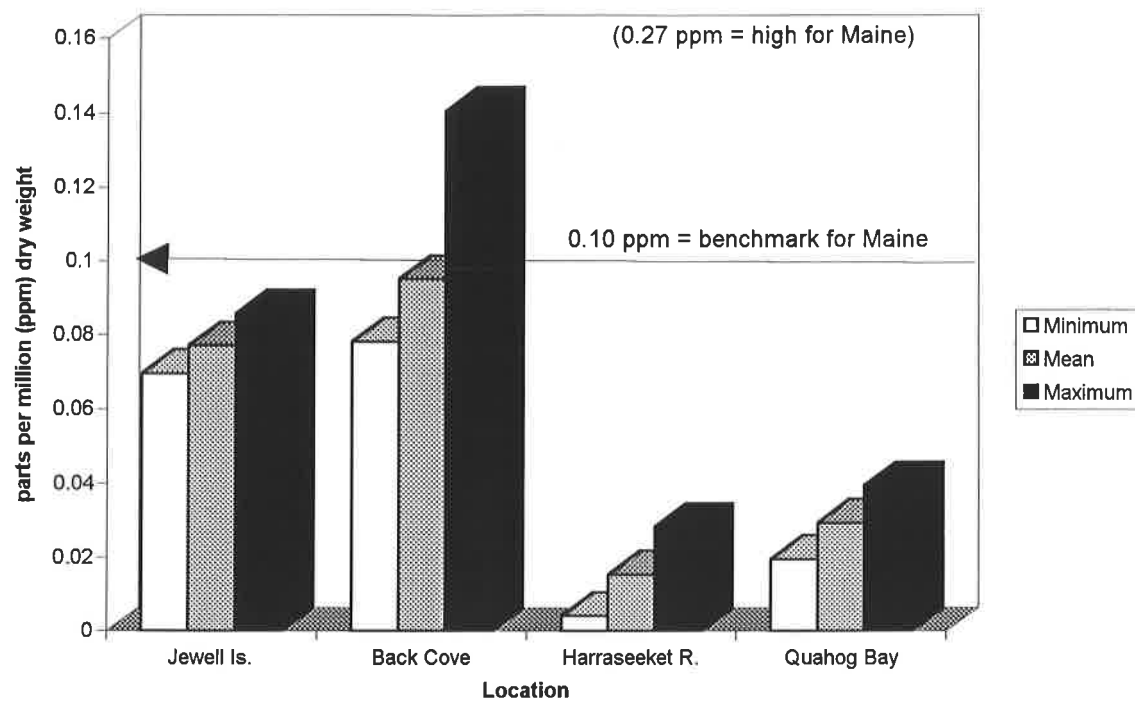
Nickel in Mussel Tissue



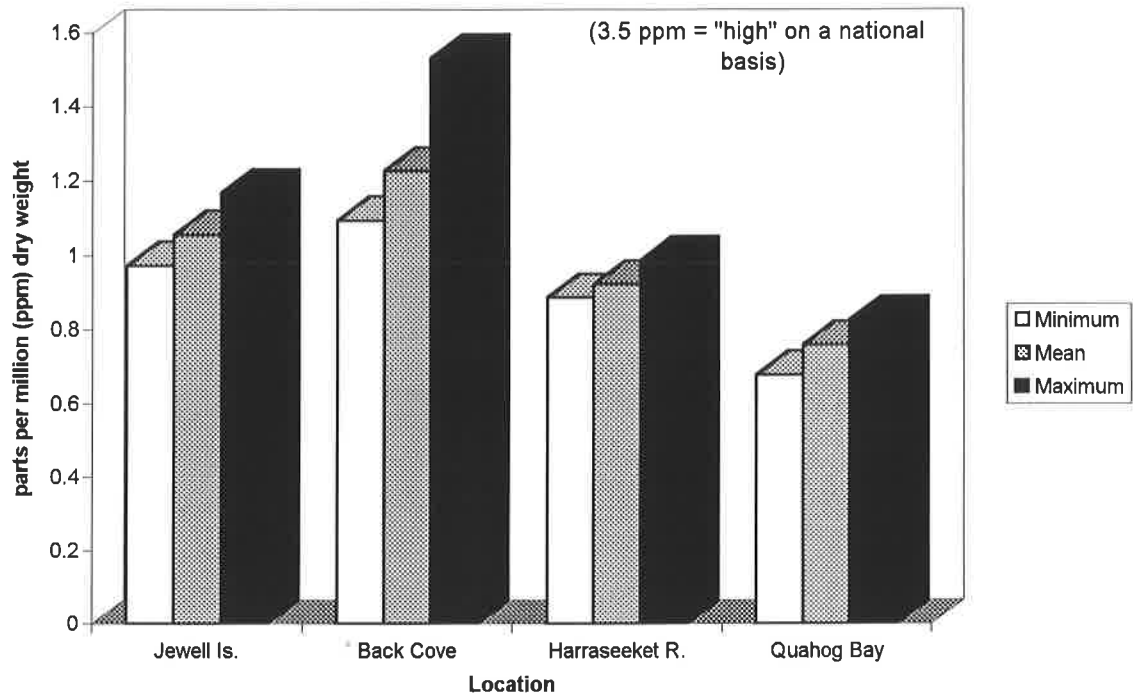
Arsenic in Mussel Tissue



Silver in Mussel Tissue



Selenium in Mussel Tissue



Beverly Bayley-Smith

From: Karen Small [Karen_Small@umit.maine.edu]
Sent: Tuesday, November 05, 2002 2:28 PM
To: bbsmith@usm.maine.edu
Subject: Casco Bay Sample Data



Casco Bay 2001.xls



2001PCPPest CB
wet weight.xls



Casco Bay PAH
2001.xls

Dear Beverly,

Attached are the spreadsheets containing your Casco Bay data. I'm sorry for the mix-up, and for the wait. If you have any questions, please let me know. Thank you very much for your patience.

Take care,

Karen

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2001
mussel
sample
data

2001 Casco Bay Mussels

Sample ID	Hg wet (mg/Kg)	Hg dry (mg/Kg)
East End Beach 1	0.0138	0.1727
East End Beach 2	0.0130	0.1730
East End Beach 3	0.0129	0.1747
East End Beach 4	0.0139	0.2255
Mill Creek 1	0.0171	0.1882
Mill Creek 2	0.0179	0.1985
Mill Creek 3	0.0158	0.1828
Mill Creek 4	0.0157	0.1747
Spring Point 1	0.0174	0.1821
Spring Point 2	0.0128	0.1354
Spring Point 3	0.0176	0.1913
Spring Point 4	0.0116	0.1253
Upper New Meadows 1	0.0168	0.1784
Upper New Meadows 2	0.0170	0.1721
Upper New Meadows 3	0.0175	0.1867
Upper New Meadows 4	0.0165	0.1741

Pesticide Report

DEP ID	Mill Creek 1	Mill Creek 2	Mill Creek 3	Mill Creek 4	East End 1	East End 2	East End 3
Sample ID#	01-MUS-45	01-MUS-46	01-MUS-47	01-MUS-48	01-MUS-49	01-MUS-50	01-MUS-51
Extraction ID	1737P	1738P	1739P	1740P	1741P	1742P	1743P
Analytes	A						
PQL (ug/Kg,dry weight)	A						
Hexachlorobenzene	<DL	<DL	<DL	<DL	<DL	<DL	<DL
Lindane	<DL	<DL	<DL	<DL	<DL	<DL	<DL
Heptachlor	<DL	<DL	<DL	<DL	<DL	<DL	<DL
Aldrin	<DL	<DL	<DL	<DL	<DL	<DL	<DL
Heptachlor Epoxide	<DL	<DL	<DL	<DL	<DL	<DL	<DL
2,4-DDE	<DL	<DL	<DL	<DL	<DL	<DL	<DL
Endosulfan I	<DL	<DL	<DL	<DL	<DL	<DL	<DL
Chlordane (a)	<DL	0.01	<DL	0.00	0.01	<DL	<DL
Nonachlor	0.01	0.01	<DL	0.01	0.01	<DL	<DL
4,4-DDE	<DL	<DL	<DL	<DL	<DL	<DL	<DL
Dieldrin	<DL	<DL	<DL	<DL	<DL	<DL	<DL
Endosulfan II	<DL	<DL	<DL	<DL	<DL	<DL	<DL
2,4-DDD	<DL	<DL	<DL	<DL	<DL	<DL	<DL
4,4-DDD	<DL	<DL	<DL	<DL	<DL	<DL	<DL
2,4-DDT	<DL	<DL	<DL	<DL	<DL	<DL	0.02
4,4-DDT	<DL	<DL	<DL	<DL	<DL	<DL	<DL
Mirex	<DL	<DL	<DL	<DL	<DL	<DL	<DL
Sample weight (g, dry weight)	22.4217	23.4023	25.0632	20.6312	20.2170	22.5367	22.0742
% Solids	9.25	9.66	9.43	9.52	7.80	8.42	8.28
Surrogate Recovery (%)	69	88	28.0	66.0	122.00	6	70
Sample weight (g, wet weight)	242.26	265.78	216.7	259.2	266.6	295.84	215.6
The tissue blank is an oil matrix.							
Values below the detection limit are estimated values and should be considered qualitative.							
They are provided for information only.							
Samples marked with an A indicate surrogate recoveries out of range and should be considered estimated values							

Pesticide Report

DEP ID	East End 4	Upper New 1	Upper New 2	Upper New 3	Upper New 4	Spring Pt 1	Spring Pt 2	Spring Pt 3
Sample ID#	01-MUS-52	01-MUS-53	01-MUS-54	01-MUS-55	01-MUS-56	01-MUS-57	01-MUS-58	01-MUS-59
Extraction ID	1744P	1745P	1746P	1747P	1748P	1749P	1750P	1751P
Analytes	A							
Hexachlorobenzene	<DL	<DL	<DL	<DL	<DL	<DL	<DL	<DL
Lindane	<DL	<DL	<DL	<DL	<DL	<DL	<DL	<DL
Heptachlor	<DL	<DL	<DL	<DL	<DL	<DL	<DL	<DL
Aldrin	<DL	<DL	<DL	<DL	<DL	<DL	<DL	<DL
Heptachlor Epoxide	<DL	<DL	<DL	<DL	<DL	<DL	<DL	<DL
2,4-DDE	<DL	<DL	<DL	<DL	<DL	<DL	<DL	<DL
Endosulfan I	<DL	<DL	<DL	<DL	<DL	<DL	<DL	<DL
Chlordane (a)	<DL	0.01	0.01	<DL	<DL	0.02	0.01	0.01
Nonachlor	<DL	0.01	0.01	<DL	<DL	0.02	0.01	0.01
4,4-DDE	<DL	<DL	<DL	<DL	<DL	<DL	<DL	<DL
Dieldrin	<DL	<DL	<DL	<DL	<DL	<DL	<DL	<DL
Endosulfan II	<DL	<DL	<DL	<DL	<DL	<DL	<DL	<DL
2,4-DDD	<DL	<DL	<DL	<DL	<DL	<DL	<DL	<DL
4,4-DDD	<DL	<DL	<DL	0.26	<DL	<DL	<DL	<DL
2,4-DDT	<DL	<DL	<DL	<DL	<DL	0.09	<DL	0.04
4,4-DDT	<DL	<DL	<DL	<DL	<DL	<DL	<DL	<DL
Mirex	<DL	<DL	<DL	<DL	<DL	<DL	<DL	<DL
Sample weight (g, dry w _i)	25.1464	21.7516	25.3021	25.1891	25.2135	19.9381	25.0741	24.9731
% Solids	8.50	10.09	10.35	10.45	10.74	9.18	9.50	9.05
Surrogate Recovery (%)	12	75.0	108.0	113.0	111.0	104.0	111.0	108.0
Sample weight (g, wet w _i)	244.5	241.0	234.8	217.19	275.9	171.7	263.9	275.9
The tissue blank is an oil								
Values below the detection limit								
They are provided for information								
Samples marked with a								

DEP ID	Spring Pt 4	
Sample ID#	01-MUS-60	
Extraction ID	1752P	
Analytes		
Hexachlorobenzene	<DL	
Lindane	<DL	
Heptachlor	<DL	
Aldrin	<DL	
Heptachlor Epoxide	<DL	
2,4-DDE	<DL	
Endosulfan I	<DL	
Chlordane (a)	0.03	
Nonachlor	0.03	
4,4-DDE	<DL	#VALUE!
Dieldrin	<DL	#VALUE!
Endosulfan II	<DL	#VALUE!
2,4-DDD	0.03	
4,4-DDD	<DL	
2,4-DDT	0.10	
4,4-DDT	<DL	
Mirex	<DL	
Sample weight (g, dry wt)	15.9300	
% Solids	9.28	
Surrogate Recovery (%)	71.0	
Sample weight (g, wet w	171.7	
The tissue blank is an oil		
Values below the detecti		
They are provided for inl		
Samples marked with a		

PAH Report

DEP ID		Mill Creek #1	Mill Creek #2	Mill Creek #3
Sample ID#		01-MUS-45	01-MUS-46	01-MUS-47
Extraction ID		1725P	1726P	1727P
Analytes	DL (ug/Kg weight)	a,b	b	b
naphthalene	1.0	<DL	<DL	<DL
1-methyl naphthalene	1.0	<DL	<DL	<DL
2-methylnaphthalene	1.0	<DL	<DL	<DL
biphenyl	1.0	<DL	<DL	<DL
2,6-dimethylnaphthalene	1.0	<DL	<DL	<DL
acenaphthylene	1.0	<DL	<DL	<DL
acenaphthene	1.0	<DL	<DL	<DL
2,3,5-trimethylnaphthalene	1.0	<DL	<DL	<DL
fluorene	1.0	<DL	<DL	<DL
phenanthrene	1.0	1.98	<DL	<DL
anthracene	1.0	<DL	<DL	<DL
1-methylphenanthrene	1.0	1.32	<DL	<DL
fluoranthrene	1.0	6.45	2.71	1.22
pyrene	1.0	4.46	2.05	0.96
benz(a)anthracene	1.0	3.64	1.15	<DL
chrysene	1.0	2.15	1.28	<DL
benzo(b)fluoranthene	2.0	<DL	<DL	<DL
benzo(k)fluoranthene	*			
benzo(a) pyrene	2.0	<DL	<DL	<DL
benzo(e)pyrene	2.0	<DL	<DL	<DL
perylene	2.0	<DL	<DL	<DL
ideno(1,2,3-cd)pyrene	2.0	<DL	<DL	<DL
dibenz(a,h)anthracene	**			
benzo(g,h,i)perylene	2.0	<DL	<DL	<DL
% Lipids		0.34	0.81	0.48
Sample weight (g, dry weight)		32.5	28.8	33.5
% Solids		9.25	9.66	9.43
Surrogates				
p-Terphenyl	10 ug/ml	6.05	11.5	7.03
% surrogate recovery	65-135	60.5	115	70.3
* Benzo(k)fluoranthrene coelutes with Benzo(b)fluoranthrene.				
** Dibenz(a,h)anthracene coelutes with ideno(1,2,3-cd)pyrene.				
Values below the detection limit are estimated values and should be considered qualitative.				
They are provided for information only.				
An a indicates surrogate recovery that is out of bounds, all data for these samples should be considered esti				
A b indicates blank spike recovery that is out low for naphthalene, acenaphthylene, and acenaphthene. These values sh				

PAH Report

DEP ID		Mill Creek #4	East End Beach #1
Sample ID#		01-MUS-48	01-MUS-49
Extraction ID		1728P	1721P
Analytes	DL (ug/Kg weight)	b	b
naphthalene	1.0	<DL	<DL
1-methyl naphthalene	1.0	<DL	<DL
2-methylnaphthalene	1.0	<DL	<DL
biphenyl	1.0	<DL	<DL
2,6-dimethylnaphthalene	1.0	<DL	<DL
acenaphthylene	1.0	<DL	<DL
acenaphthene	1.0	<DL	<DL
2,3,5-trimethylnaphthalene	1.0	<DL	<DL
fluorene	1.0	<DL	<DL
phenanthrene	1.0	<DL	2.01
anthracene	1.0	<DL	<DL
1-methylphenanthrene	1.0	<DL	<DL
fluoranthrene	1.0	1.09	6.72
pyrene	1.0	0.83	5.40
benz(a)anthracene	1.0	<DL	2.70
chrysene	1.0	<DL	3.47
benzo(b)fluoranthene	2.0	<DL	1.75
benzo(k)fluoranthene	*		
benzo(a) pyrene	2.0	<DL	<DL
benzo(e)pyrene	2.0	<DL	2.01
perylene	2.0	<DL	<DL
ideno(1,2,3-cd)pyrene	2.0	<DL	<DL
dibenz(a,h)anthracene	**		
benzo(g,h,i)perylene	2.0	<DL	<DL
% Lipids		0.51	0.76
Sample weight (g, dry weight)		30.3	27.4
% Solids		9.5	7.80
Surrogates			
p-Terphenyl	10 ug/ml	8.82	10.86
% surrogate recovery	65-135	88.2	108.6
* Benzo(k)fluoranthrene coelutes with Benzo(b)fluoranth			
** Dibenz(a,h)anthracene coelutes with ideno(1,2,3-cd)py			
Values below the detection limit are estimated values and			
They are provided for information only.			
An a indicates surrogate recovery that is out of bounmated values.			
A b indicates blank spike recovery that is out low for naphould be considered estimated values.			

PAH Report

DEP ID		East End Beach #2	East End Beach #3
Sample ID#		01-MUS-50	01-MUS-51
Extraction ID		1722P	1731P
Analytes	DL (ug/Kg weight)	b	b
naphthalene	1.0	<DL	<DL
1-methyl naphthalene	1.0	<DL	<DL
2-methylnaphthalene	1.0	<DL	<DL
biphenyl	1.0	<DL	<DL
2,6-dimethylnaphthalene	1.0	<DL	<DL
acenaphthylene	1.0	<DL	<DL
acenaphthene	1.0	<DL	<DL
2,3,5-trimethylnaphthalene	1.0	<DL	<DL
fluorene	1.0	<DL	<DL
phenanthrene	1.0	1.23	0.78
anthracene	1.0	<DL	<DL
1-methylphenanthrene	1.0	<DL	<DL
fluoranthrene	1.0	5.85	5.30
pyrene	1.0	4.58	3.81
benz(a)anthracene	1.0	1.95	1.34
chrysene	1.0	3.03	2.91
benzo(b)fluoranthene	2.0	1.26	<DL
benzo(k)fluoranthene	*		
benzo(a) pyrene	2.0	<DL	<DL
benzo(e)pyrene	2.0	<DL	<DL
perylene	2.0	<DL	<DL
ideno(1,2,3-cd)pyrene	2.0	<DL	<DL
dibenz(a,h)anthracene	**		
benzo(g,h,i)perylene	2.0	<DL	<DL
% Lipids		0.46	0.74
Sample weight (g, dry weight)		27.7	26.8
% Solids		8.42	8.28
Surrogates			
p-Terphenyl	10 ug/ml	9.30	9.15
% surrogate recovery	65-135	93	91.5
* Benzo(k)fluoranthrene coelutes with Benzo(b)fluoranth			
** Dibenz(a,h)anthracene coelutes with ideno(1,2,3-cd)py			
Values below the detection limit are estimated values and			
They are provided for information only.			
An a indicates surrogate recovery that is out of bound			
A b indicates blank spike recovery that is out low for naph			

PAH Report

DEP ID		East End Beach #4	Upper New Meadows # 1
Sample ID#		01-MUS-52	01-MUS-53
Extraction ID		1729P	1732P
Analytes	DL (ug/Kg weight)	b	b
naphthalene	1.0	<DL	<DL
1-methyl naphthalene	1.0	<DL	<DL
2-methylnaphthalene	1.0	<DL	<DL
biphenyl	1.0	<DL	<DL
2,6-dimethylnaphthalene	1.0	<DL	<DL
acenaphthylene	1.0	<DL	<DL
acenaphthene	1.0	<DL	<DL
2,3,5-trimethylnaphthalene	1.0	<DL	<DL
fluorene	1.0	<DL	<DL
phenanthrene	1.0	1.75	<DL
anthracene	1.0	<DL	<DL
1-methylphenanthrene	1.0	<DL	<DL
fluoranthrene	1.0	11.75	1.05
pyrene	1.0	9.81	0.99
benz(a)anthracene	1.0	3.94	<DL
chrysene	1.0	5.05	<DL
benzo(b)fluoranthene	2.0	2.79	<DL
benzo(k)fluoranthene	*		
benzo(a) pyrene	2.0	<DL	<DL
benzo(e)pyrene	2.0	2.57	<DL
perylene	2.0	<DL	<DL
ideno(1,2,3-cd)pyrene	2.0	<DL	<DL
dibenz(a,h)anthracene	**		
benzo(g,h,i)perylene	2.0	<DL	<DL
% Lipids		0.49	0.96
Sample weight (g, dry weight)		31.5	31.3
% Solids		8.50	10.09
Surrogates			
p-Terphenyl	10 ug/ml	9.26	12.38
% surrogate recovery	65-135	92.6	123.8
* Benzo(k)fluoranthrene coelutes with Benzo(b)fluoranth			
** Dibenz(a,h)anthracene coelutes with ideno(1,2,3-cd)py			
Values below the detection limit are estimated values and			
They are provided for information only.			
An a indicates surrogate recovery that is out of bound			
A b indicates blank spike recovery that is out low for naph			

PAH Report

DEP ID		Upper New Meadows # 2	Upper New Meadows # 3
Sample ID#		01-MUS-54	01-MUS-55
Extraction ID		1723P	1733P
Analytes	DL (ug/Kg weight)	b	a,b
naphthalene	1.0	<DL	<DL
1-methyl naphthalene	1.0	<DL	<DL
2-methylnaphthalene	1.0	<DL	<DL
biphenyl	1.0	<DL	<DL
2,6-dimethylnaphthalene	1.0	<DL	<DL
acenaphthylene	1.0	<DL	<DL
acenaphthene	1.0	<DL	<DL
2,3,5-trimethylnaphthalene	1.0	<DL	<DL
fluorene	1.0	<DL	<DL
phenanthrene	1.0	<DL	<DL
anthracene	1.0	<DL	<DL
1-methylphenanthrene	1.0	<DL	<DL
fluoranthrene	1.0	<DL	<DL
pyrene	1.0	<DL	<DL
benz(a)anthracene	1.0	<DL	<DL
chrysene	1.0	<DL	<DL
benzo(b)fluoranthene	2.0	<DL	<DL
benzo(k)fluoranthene	*		
benzo(a) pyrene	2.0	<DL	<DL
benzo(e)pyrene	2.0	<DL	<DL
perylene	2.0	<DL	<DL
ideno(1,2,3-cd)pyrene	2.0	<DL	<DL
dibenz(a,h)anthracene	**		
benzo(g,h,i)perylene	2.0	<DL	<DL
% Lipids		0.44	0.65
Sample weight (g, dry weight)		36.4	35.6
% Solids		10.35	10.45
Surrogates			
p-Terphenyl	10 ug/ml	8.43	5.81
% surrogate recovery	65-135	84.3	58.1
* Benzo(k)fluoranthrene coelutes with Benzo(b)fluoranth			
** Dibenz(a,h)anthracene coelutes with ideno(1,2,3-cd)py			
Values below the detection limit are estimated values and			
They are provided for information only.			
An a indicates surrogate recovery that is out of bound			
A b indicates blank spike recovery that is out low for naph			

PAH Report

DEP ID		Upper New Meadows # 4	Spring Point #1	Spring Point #2
Sample ID#		01-MUS-56	01-MUS-57	01-MUS-58
Extraction ID		1730P	1734P	1724P
Analytes	DL (ug/Kg weight)	b	b	b
naphthalene	1.0	<DL	<DL	<DL
1-methyl naphthalene	1.0	<DL	<DL	<DL
2-methylnaphthalene	1.0	<DL	<DL	<DL
biphenyl	1.0	<DL	<DL	<DL
2,6-dimethylnaphthalene	1.0	<DL	<DL	<DL
acenaphthylene	1.0	<DL	<DL	<DL
acenaphthene	1.0	<DL	<DL	<DL
2,3,5-trimethylnaphthalene	1.0	<DL	<DL	<DL
fluorene	1.0	<DL	<DL	<DL
phenanthrene	1.0	<DL	<DL	<DL
anthracene	1.0	<DL	<DL	<DL
1-methylphenanthrene	1.0	<DL	<DL	<DL
fluoranthrene	1.0	0.75	2.69	2.14
pyrene	1.0	<DL	1.54	1.41
benz(a)anthracene	1.0	<DL	1.33	0.88
chrysene	1.0	<DL	1.57	1.11
benzo(b)fluoranthene	2.0	<DL	<DL	<DL
benzo(k)fluoranthene	*			
benzo(a) pyrene	2.0	<DL	<DL	<DL
benzo(e)pyrene	2.0	<DL	<DL	<DL
perylene	2.0	<DL	<DL	<DL
ideno(1,2,3-cd)pyrene	2.0	<DL	<DL	<DL
dibenz(a,h)anthracene	**			
benzo(g,h,i)perylene	2.0	<DL	<DL	<DL
% Lipids		1.21	0.69	0.44
Sample weight (g, dry weight)		34.6	33.8	26.2
% Solids		10.74	9.18	9.50
Surrogates				
p-Terphenyl	10 ug/ml	9.62	10.11	10.01
% surrogate recovery	65-135	96.2	101.1	100.1
* Benzo(k)fluoranthrene coelutes with Benzo(b)fluoranth				
** Dibenz(a,h)anthracene coelutes with ideno(1,2,3-cd)py				
Values below the detection limit are estimated values and				
They are provided for information only.				
An a indicates surrogate recovery that is out of bound				
A b indicates blank spike recovery that is out low for naph				

PAH Report

DEP ID		Spring Point #3	Spring Point #4
Sample ID#		01-MUS-59	01-MUS-60
Extraction ID		1735P	1736P
Analytes	DL (ug/Kg weight)	b	b
naphthalene	1.0	<DL	<DL
1-methyl naphthalene	1.0	<DL	<DL
2-methylnaphthalene	1.0	<DL	<DL
biphenyl	1.0	<DL	<DL
2,6-dimethylnaphthalene	1.0	<DL	<DL
acenaphthylene	1.0	<DL	<DL
acenaphthene	1.0	<DL	<DL
2,3,5-trimethylnaphthalene	1.0	<DL	<DL
fluorene	1.0	<DL	<DL
phenanthrene	1.0	<DL	<DL
anthracene	1.0	<DL	<DL
1-methylphenanthrene	1.0	<DL	<DL
fluoranthrene	1.0	2.53	2.15
pyrene	1.0	1.61	1.32
benz(a)anthracene	1.0	1.45	0.74
chrysene	1.0	1.29	1.48
benzo(b)fluoranthene	2.0	<DL	<DL
benzo(k)fluoranthene	*		
benzo(a) pyrene	2.0	<DL	<DL
benzo(e)pyrene	2.0	<DL	<DL
perylene	2.0	<DL	<DL
ideno(1,2,3-cd)pyrene	2.0	<DL	<DL
dibenz(a,h)anthracene	**		
benzo(g,h,i)perylene	2.0	<DL	<DL
% Lipids		0.65	0.73
Sample weight (g, dry weight)		24.9	32.5
% Solids		9.05	9.28
Surrogates			
p-Terphenyl	10 ug/ml	10.04	8.32
% surrogate recovery	65-135	100.4	83.2
* Benzo(k)fluoranthrene coelutes with Benzo(b)fluoranth			
** Dibenz(a,h)anthracene coelutes with ideno(1,2,3-cd)py			
Values below the detection limit are estimated values and			
They are provided for information only.			
An a indicates surrogate recovery that is out of bound			
A b indicates blank spike recovery that is out low for naph			