For nearly a century, the effluents from the Sydney Tar Ponds were discharged into Muggah Creek. As this creek opens into the South Arm of Sydney Harbour, this discharge resulted in appreciable levels of polyaromatic hydrocarbons (PAHs) being deposited in the Harbour sediments. Because surveys showed that lobsters captured in the South Arm of the Harbour contained substantial levels of benzo[a]pyrene, a known carcinogen, the lobster fishery in the South Arm of the Harbour was closed in 1982. Prior to the closure, the cancer lifetime risk for those eating lobsters from this area was approximately 3 times greater than the provincial acceptable level. Following the closure, the lifetime cancer risk declined to levels considered to be acceptable. Computed non-cancer hazards were deemed acceptable prior to the closure and decreased following it. A more extensive risk assessment will evaluate the site further and provide valuable information on fisheries decisions for this area.

INTRODUCTION

With industrialization in the 19th century, the rich deposits of coal in Cape Breton Island led to the construction of a steel plant in the city of Sydney. To secure supplies of coke, which played an important role in the steel making process, two coal coking facilities were constructed on the shore of Sydney Harbour beginning in 1901.

A coke-oven is essentially a large chamber where coal is heated and at a specific temperature, undesired tar and gases separate from the desired
coke. For nearly a century the liquid effluents were discharged from the coke ovens into the “Tar Ponds”; from there some portion of the toxic effluents flowed into the harbour (Figure 1). The site in question covers 72 hectares. The coke-oven operation contaminated the ground and surface water with arsenic, lead, and other toxins resulting in an accumulation of some 700,000 tonnes of chemical waste, mostly polycyclic aromatic hydrocarbons (PAHs) [40,000 tonnes of which are polychlorinated biphenyls (PCBs)]. About 24,000 people live in Sydney and a study concluded that cancer rates are highest among those city residents who live within 5 km of the Tar Ponds (Guernsey et al. 2000).

The two coking plants were closed, one in 1981 and the second in 1983. In the early 1980s, the South Arm of the harbour was closed to commercial lobster fishing based on the health risk associated with consuming lobster contaminated with PAHs (Uthe & Musial 1986). Many of these PAHs, such as benzo[a]pyrene (B[a]P), a known carcinogen, were found in the edible tissue of the animal. Lobsters were re-surveyed in 1995 and in addition to the commonly measured PAHs, a multitude of complex unknown peaks were found (King et al 1993). There is still lobster fishing in other parts of the harbour: 52 fishing vessels set as many as 4300 traps per day during the fishing season (Prouse 1994). The seasonal catch amounts to >200,000 lbs.

In order to assess the human health risk involved in consuming contaminated seafood it was necessary to: 1) select a commercial species which is

Fig 1 The Sydney Tar Ponds (Canadian Environmental Assessment Agency 2005).
easy to sample, 2) select a fishery near or in a contaminated site. For this study American lobster (*Homarus americanus*), a highly desirable food stuff, was selected, because it has a large economic commercial value and was easy to sample. The site selected was the South Arm of Sydney Harbour, because it was near the Sydney Tar Ponds. In addition a remote clean second site, St. Margarets Bay of Nova Scotia was selected as a control site.

**MATERIALS AND METHODS**

**Sampling**

*South Arm of Sydney Harbour*

Sampling was carried out in October 2001 on the CCGS Navicula. Grab samples were taken from 20 stations (Figure 2). At each station, when sufficient material was not obtained, the grab sampler was deployed again until the sample size prerequisite was satisfied by the composite sample. Samples of water, suspended particulate matter and sediments were collected at the same time. The samples were homogenized and sub-sampled.

Market-sized lobsters [519±59 grams (with claws, tail and hepatopancreas weighing 29±4 grams)] five to ten animals per site were collected using standard commercial traps in 1982, 1984, 1991 and 1995 (Table 1).
Samples of sediment, water and lobsters were collected in a commercial fishery site near the Head of St. Margarets Bay.

Sample Preparation and Analysis

Sediment samples were prepared according to the Environmental Protection Agency (EPA) method 3540C (1996). Live lobsters were taken to the lab and dissected to remove the digestive gland (hepatopancreas) intact. The digestive glands were prepared according to Uthe and Musial (1986) and King et al. (2003). Early sample extracts (1982 and 1984) were analysed by reverse phase high performance liquid chromatography and in later studies by gas chromatography/mass spectrometry (1991 and 1995).

RESULTS AND DISCUSSION

Contaminant Concentrations in Environmental Media

Samples of biota, sediment, and water were evaluated. Temporal trends (1986-1995) revealed that PAH concentrations in sediments from 24 of 38 stations had decreased since the closure of the coal coking plants (Ernst et al. 1999). More recent studies (1999, July and October 2000) revealed a continuous downward temporal trend (Lee et al. 2002). The sediments contained substantial PAH concentrations as illustrated in Fig 3. PAHs are insoluble in water; therefore most of the water samples gave results in parts-per-billion levels (ppb) and because the amounts were so small, these data are not included in the figure. Lobster results from 1984 to 1995 for Sydney Harbour and 1995 data for St. Margaret Bay (SMB) are presented in Table 1. The PAH concentrations decreased in lobster (1984-1995) and the temporal trends are similar to those reported by Ernst et al. (1999).

<table>
<thead>
<tr>
<th>Chemical Compound</th>
<th>South Arm Sydney Harbour</th>
<th>St. Margarets Bay</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fluoranthene</td>
<td>15200</td>
<td>12400</td>
</tr>
<tr>
<td>Pyrene</td>
<td>13100</td>
<td>9150</td>
</tr>
<tr>
<td>Benz[a]Anthr</td>
<td>32700</td>
<td>18000</td>
</tr>
<tr>
<td>Chrysene</td>
<td>1030</td>
<td>252</td>
</tr>
<tr>
<td>B[b]Fl</td>
<td>3820</td>
<td>2460</td>
</tr>
<tr>
<td>B[k]Fl</td>
<td>955</td>
<td>640</td>
</tr>
<tr>
<td>B[a]P</td>
<td>1430</td>
<td>930</td>
</tr>
<tr>
<td>B[ghi]Pyl</td>
<td>769</td>
<td>479</td>
</tr>
</tbody>
</table>

Hazard Identification

Screening and Identification of Contaminants of Potential Concern.

Both the Canadian (Canadian Council for Ministers of the Environment 2001) and American (United States Environmental Protection Agency 1999) governments have established marine sediment quality guidelines for 13 individual PAHs. Sediments from the mouth of Muggah Creek (stations 9-11) exceed the Canadian Probable Effects Level (PEL) by 100 to 380 times and American Effects Range-Median (ERM) by 48 to 180 times. In the South Arm sediment concentrations exceeded the PEL by 2.8 to 71 times and the ERM by 1.8 to 34 times (King and Lee 2004). Thus PAHs exceeded both guidelines (King & Lee 2004).

The Canadian standards (maximum acceptable level) of B[a]P toxic equivalency in retail food is 3 ng.kg\(^{-1}\) (Health Canada 2007). Digestive glands of lobsters procured from the South Arm of Sydney Harbour exceeded this level considerably (Table 2).

Identification of Potential Recipients

The recipients were Sydney residents: fishers, tourists, and aboriginal people exposed to the contaminated lobster. The subject group addressed in this study was 20+ years of age.
Exposure Pathways (Monitoring Lobster for Contaminants)

Source.

The Tar Ponds located in Sydney Nova Scotia, contain PAHs, PCBs, and heavy metals in appreciable levels (Ernst et al. 1999, Lee 2002).

Environmental Distribution and Exposure Pathways.

The focus was placed on distribution of contaminants to lobsters. The toxic chemicals that were discharged from the coking plants were rich in PAHs. The solubility of PAHs is low in water and decreases with increasing molecular weight. The log $K_{ow}$ (i.e. octanol/water partition coefficients) ranged from 3.33 to 6.50 (ABB Environmental Services Inc. 1990). PAHs are associated with particulate and dissolved material and tend to be deposited in the sediments. Partition coefficients (from averages for the 1999, July and October 2000 samples) were generated for a number of PAHs and plotted against their log $K_{ow}$, which illustrates that the PAHs partitioning between the water and particles, were at equilibrium based on the regression line approaching linearity (Fig 4). The PAHs were widely distributed in the South Arm and the results were consistent with water/sediment partitioning for PAHs from a pyrogenic source.

The bioavailability of [a]P (and other PAHs) is significantly affected by the quality and quantity of dissolved organic matter (Akkanen et al., 2001). Bioavailability of PAHs in sediments is greatly affected by carbon content. Some characteristics that effect binding of hydrophobic organic compounds include aromaticity, hydrophobic acid content, and molecular size (McCarthy et al. 1989, Kukkonen & Oikari 1991, Chin et al. 1997, Haitzer et al. 1999). An increase in any one of the above causes greater binding of organic chemicals. This increased binding causes a reduction in bioavailability (Kukkonen et al. 1990). The strong correlation between total organic

### Table 2: Average PAH and TEQ [a]P concentrations (ng.kg$^{-1}$ wet wt. in lobster tomalley). 1

<table>
<thead>
<tr>
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<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Fluoranthene</td>
<td>13800</td>
<td>4505</td>
<td>330</td>
<td>0.001</td>
<td>14</td>
<td>4.5</td>
<td>0.3</td>
</tr>
<tr>
<td>Pyrene</td>
<td>11225</td>
<td>3025</td>
<td>140</td>
<td>0.001</td>
<td>11</td>
<td>3.0</td>
<td>0.1</td>
</tr>
<tr>
<td>Benz[a]Anthr</td>
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<td>911</td>
<td>47</td>
<td>0.1</td>
<td>2535</td>
<td>91</td>
<td>4.7</td>
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<tr>
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<td>1243</td>
<td>140</td>
<td>0.01</td>
<td>6.4</td>
<td>12</td>
<td>1.4</td>
</tr>
<tr>
<td>B[b]Fl</td>
<td>3140</td>
<td>1313</td>
<td>42</td>
<td>0.1</td>
<td>314</td>
<td>131</td>
<td>4.2</td>
</tr>
<tr>
<td>B[k]Fl</td>
<td>798</td>
<td>671</td>
<td>10</td>
<td>0.1</td>
<td>80</td>
<td>67</td>
<td>1.0</td>
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<tr>
<td>B[a]P</td>
<td>1180</td>
<td>870</td>
<td>23</td>
<td>1.0</td>
<td>1180</td>
<td>870</td>
<td>23</td>
</tr>
<tr>
<td>B[ghi]Pyl</td>
<td>624</td>
<td>353</td>
<td>0.01</td>
<td>6.2</td>
<td>6.2</td>
<td>3.5</td>
<td></td>
</tr>
<tr>
<td>$\Sigma$TEQ-[a]P</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>4147</td>
<td>1182</td>
<td>35</td>
</tr>
</tbody>
</table>

carbon (TOC) and PAH concentrations indicates that there are appreciable quantities of >4 ring PAHs in the sediments and the source is most likely pyrogenic (Fig 5). Contaminated sediments have been shown to be bioavailable as evidenced by their toxicity to amphipods, marine bacteria and their bioaccumulation in clams, *Macoma balthica* (Lee 2002). Previous studies by Tay et al. (1992) showed that sediments from the Sydney Tar Ponds were toxic to marine organisms. The high molecular wt. PAHs (>4 ring) are persistent and have the potential to bioaccumulate in the tissues of some animals and bio-magnify in the food chain. As a result, there is risk to predators, such as lobsters through the water or the consumption of contaminated prey. This risk could be extended to humans by their consumption of contaminated fish or shellfish from the harbour.

The tomalley (hepatopancreas) was the lobster part examined in this study. In order to estimate how many people eat tomalley, a survey of 100 people at a lobster dinner in 2007 was conducted and determined that 90% of the individuals that consumed lobsters ate both the meat (from claws and tails) and the tomalley.

**Exposure Assessment (Estimating Risks to Humans)**

**Benzo[a]pyrene Toxic Equivalent Factors.**

B[a]P is a carcinogen and has been shown to suppress the immune response of mammals (Carlson et al. 2002). B[a]P is a major contributor to the high molecular weight PAH (>4 benzene rings). This compound is particle bound and readily available to be taken up by marine organisms, such as lobsters. B[a]P, once taken up by lobsters and other organisms, undergoes biotransformation in the form of hydroxylation (Stine & Brown...
This results in the production of B[a]P-7,8-diol, which can be further epoxidated to form B[a]P-7,8-diol-9,10 epoxide. This compound is even more reactive with DNA than is the parent compound.

Toxicological data for B[a]P is readily available compared to other PAHs. Even though there is inadequate data to assess the risk of individual PAHs, there is sufficient data for several compounds to approximate cancer potencies to B[a]P (Yender et al. 2002). It is important to note that this approach can only be applied to pyrogenic (combustion of fossil fuels, such as coal tar) sources rather than petrogenic sources (petroleum based), because high molecular weight PAHs, such as B[a]P are not present in detectable concentrations in petrogenic sources. Benzo[a]pyrene toxic equivalent (TEQ) concentrations (individual PAH concentrations multiplied by the B[a]P toxic equivalent factor) were calculated for lobster (Table 2).

Equations and Risk Calculations.

Adult Lobster Consumer Exposure Assumptions

| Lobster tissue concentration (mg/kg w/w) | TC | TEQ B[a]P (Table 2) |
| Cancer slope factor (per mg/kg-day) | CSF | 0.11 |
| Reference Dose factor (mg/kg-day) | RfD | NV |
| Consumption rate-daily average (g/day) | CR | 6 |
| Relative absorption factor (oral) | RAF | 1 |
| Exposure frequency (days/yr) | EF | 365 |
| Exposure duration (yr) | ED | 50 |
| Body weight (individual average in kg) | BW | 70 |
| Conversion Factor (kg/g) | CF | 0.001 |
| Averaging time-car. (days) | ATc | 25550 |
| Averaging Time-noncar. (days) | ATnc | 18250 |

Fig 5  Correlation plot of PAH concentration against Total Organic Carbon
**Intake Calculations.**

\[
\text{Intake} = \frac{\text{TC} \times \text{CR} \times \text{EF} \times \text{ED} \times \text{RAF} \times \text{CF}}{\text{BW} \times \text{AT}}
\]

**Before Fishery Closure**

Sydney (intake) = 
\[
(4.147 \text{mg.kg}^{-1}) \times (6 \text{g/day}) \times (365 \text{ days/yr}) \times (50 \text{ yr}) \times (1) \times (0.001 \text{kg/g}) \times (70 \text{kg}) \times (25550 \text{days})
\]

Intake (carcinogen B[a]P) = \frac{\text{(454.1 mg)}}{1788500 \text{ kg/day}} = \text{2.54} \times 10^{-4} \text{ mg/kg/day}

**After Fishery Closure**

Sydney (intake) = 
\[
(1.182 \text{mg.kg}^{-1}) \times (6 \text{g/day}) \times (365 \text{ days/yr}) \times (50 \text{ yr}) \times (1) \times (0.001 \text{kg/g}) \times (70 \text{kg}) \times (25550 \text{days})
\]

Intake (carcinogen B[a]P) = \frac{\text{(129.4 mg)}}{1788500 \text{ kg/day}} = \text{7.24} \times 10^{-5} \text{ mg/kg/day}

SMB (intake) = 
\[
(0.035 \text{ mg.kg}^{-1}) \times (6 \text{g/day}) \times (365 \text{ days/yr}) \times (50 \text{ yr}) \times (1) \times (0.001 \text{kg/g}) \times (70 \text{kg}) \times (25550 \text{days})
\]

Intake (carcinogen B[a]P) = \frac{\text{(3.83 mg)}}{1788500 \text{ kg/day}} = \text{2.14} \times 10^{-6} \text{ mg/kg/day}

**Risk Estimate.**

Risk = \text{intake (Carc)} \times \text{cancer slope factor}

Risk (Sydney before closure) = \text{0.000254 mg/kg/day x 0.11 per mg/kg/day) = 2.79 x 10^{-5}}

Risk (Sydney after closure) = \text{0.0000724 mg/kg/day x 0.11 per mg/kg/day) = 7.96 x 10^{-6}}

Risk (SMB) = \text{0.0000021 mg/kg/day x 0.11 per mg/kg/day) = 2.31 x 10^{-7}}
Hazard Estimate.

Hazard=intake (non-carc)/reference dose factor

RDF are present for 3 compounds only, benzo[ghi]perylene (B[ghi]Pyl), fluoranthene (Fl), and pyrene (P). A total hazard estimate will be calculated summing the estimated hazard from these compounds.

Sydney Site (Before Closure)

Hazard (B[ghi]Pyl) = \( \frac{0.000254 \text{mg/kg/day}}{0.004 \text{mg/kg/day}} = 0.064 \)

Hazard (Fl) = \( \frac{0.000254 \text{mg/kg/day}}{0.04 \text{mg/kg/day}} = 0.0064 \)

Hazard (P) = \( \frac{0.000254 \text{mg/kg/day}}{0.03 \text{mg/kg/day}} = 0.0085 \)

Total Hazard = 0.079

Sydney Site (After Closure)

Hazard (B[ghi]Pyl) = \( \frac{0.0000724 \text{mg/kg/day}}{0.004 \text{mg/kg/day}} = 0.018 \)

Hazard (Fl) = \( \frac{0.0000724 \text{mg/kg/day}}{0.04 \text{mg/kg/day}} = 0.0018 \)

Hazard (P) = \( \frac{0.0000724 \text{mg/kg/day}}{0.03 \text{mg/kg/day}} = 0.0024 \)

Total Hazard = 0.022

St. Margarets Bay Site

Hazard (B[ghi]Pyl) = \( \frac{0.0000021 \text{mg/kg/day}}{0.004 \text{mg/kg/day}} = 0.00053 \)

Hazard (Fl) = \( \frac{0.0000021 \text{mg/kg/day}}{0.04 \text{mg/kg/day}} = 0.000053 \)

Hazard (P) = \( \frac{0.0000021 \text{mg/kg/day}}{0.03 \text{mg/kg/day}} = 0.00007 \)

Total Hazard = 0.00065
Risk Characterization

*Integrating Hazard and Exposure Data.*

The current risk to humans was estimated based on consumption of lobster contaminated with PAHs using the TEQ B[a]P concentrations to estimate the risk. Average adults that consume lobster are estimated to do so twice annually while heavy consumers eat lobsters about 6 to 10 times annually (Conestoga-Rovers & Associates 2003). A serving was estimated to be ¼ of a kilogram; therefore heavy consumers eat about 1.4 to 2.3 kg annually. The result was 6 g/day/yr for a person considered to be a heavy eater. Taking into consideration the consumer exposure and using the intake and risk estimate equations, a cancer risk was determined for adults (20+ years) consuming lobster procured from the South Arm of Sydney Harbour and St. Margarets Bay. A total cancer risk estimate of $7.96 \times 10^{-6}$ and $2.31 \times 10^{-7}$ (see risk estimate section for calculations) for lifetime consumers of Sydney and St. Margarets Bay lobsters respectively were calculated. The provincial regulatory guidelines accept an incremental lifetime cancer risk of $1 \times 10^{-5}$. Thus the values calculated in this study were well below the acceptable guidelines.

Prior to the closure of the Coke Ovens, the estimated incremental lifetime cancer risk was $2.79 \times 10^{-5}$, approximately 3 times higher than the provincial acceptable levels, hence the closure of the South Arm to commercial fishing. The non-cancer hazard (source of potential damage or adverse health effects from non-carcinogenic PAHs) calculated in all cases, is well below the acceptable target hazard level of 0.2. The data calculated in this study was comparable to estimates calculated by Conestoga-Rovers & Associates (2003).

Uncertainty in the Approach.

There are several areas of uncertainty to consider. The analyses are focused on the use of previous data sets (1985-1995). The regulatory guideline for acceptable cancer risk varies from province to province in Canada (i.e. BC, Alberta and the Atlantic provinces accept an incremental lifetime cancer risk of $1 \times 10^{-5}$, while Ontario uses $1 \times 10^{-6}$). Provincial statistical approaches for exposure calculations also vary; some use maximum concentrations, some use average concentrations, others use 95% upper confidence limit of the mean and yet others use 90 or 95% values of the available data (Health Canada 2004). There are also varying toxic equivalent factors for B[a]P (Yender et al. 2002). However, this does not appear to affect the results in this study significantly. The average concentrations used in this study were based on data collected from 1984 to 1995. As the variance in the data set was small after the closure of the fishery, using averages is adequate for this data set. There would have to be a significant change in concentration (at least 10 fold) in order to cause a significant change (10 fold) in the risk estimates.

Another uncertainty involves the assumption that PAHs are the primary stressor affecting benthos. Other chemicals, such as heavy metals, PCBs,
and alkyalted PAHs are present in detectable concentrations and the accumulated effects may increase the cancer risk and non-cancer hazard estimates from consuming lobster from Sydney Harbour.

**CONCLUSIONS**

The sediments and lobster PAH concentrations decreased after the closure of the coal coking plants. This decreasing temporal trend in environmental compartments is consistent with the computed carcinogenic risk from PAHs, which according to the NS provincial regulatory guidelines had decreased from unacceptable in 1982 to acceptable in 1995. TEQ B[a]P were adequate for determining the overall PAH risk, as there is no data to access the risks for all the individual PAHs in this study. The non-cancer hazards calculated in all cases were below the acceptable target hazard level. The clean St. Margarets Bay control site was a suitable reference site for background levels of PAHs in the province of Nova Scotia. Considerations concerning the fisheries in the South Arm would require a more comprehensive risk assessment in order to address the more recent effects from other contaminants and perhaps extend future studies to other commercial species of fish to evaluate the safety of annual consumption of fish from Sydney Harbour.

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