Reduction in Inhalation Cancer Risk due to Source Control Measures during the 2008 Beijing Olympics

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Outdoor Air Pollution in China

Associated health care costs in 2003: 157~520 billion RMB (1.2-3.8% of China’s GDP) (World Bank 2007)

~300,000 people die each year from ambient air pollution-associated heart disease and lung cancer (Kahn and Yardley 2007)

~6.5 per million people have lung cancer due to PAH inhalation exposure (Zhang Y et al., 2009)

In Beijing, 1.73% of cancer or 272-309 individual cancer cases were due to PAH inhalation (Yu et al., 2008)
2008 Beijing Olympic Games

- **Beijing Olympic Games:** Aug 8-24
- **Source Control Period:** Jul 20 – Sept 20

- Traffic restricted on alternate days under an even-odd license plate system
- Construction sites closed
- Operation of coal-fired power plants strictly limited
- Additional restrictions on coal-combustion
- Source control measures less strictly implemented
The 2008 Beijing Olympic Games provided a unique case study to investigate the effect of source control measures on the reduction in air pollution, and associated inhalation cancer risk, in a Chinese megacity.
Objectives

- **Study object:** carcinogenic PAHs in Beijing Olympic PM$_{2.5}$ (priority pollutant PAHs + MW 302 PAHs)

- **Method:** estimate the excess inhalation cancer risk during the source control, non-source control, Olympic, and non-Olympic periods

- **Goals:**
  - to assess the effectiveness of source control measures in reducing PAH-induced inhalation cancer risks in a Chinese megacity
  - to compare the estimated inhalation cancer risk posed by MW 302 PAHs with that posed by conventional priority pollutant PAHs.
Experimental


Olympic: 8/8-8/24

Source control: 7/20-9/20

50% phenyl DB-17 column, 60m 0.25mm 0.25µm

Accelerated Solvent Extraction

Solid Phase Extraction
Schubert et al., 2003

MW 302 PAH

85 possible isomers!
MW 302 PAH

Mutagenic potency of MW 302 PAH:

- 33% of the total PAH-derived mutagenicity in the urban dust (SRM 1649) extract (Durant et al., 1998);

- 16% of the total genotoxic response of coal tar-contaminated sediment extracts (Marvin et al., 2000);

- 2-3% of the measured mutagenicity in PM2.5 samples in MA and NY (Pedersen et al., 2005)

85 possible isomers!

Schubert et al., 2003
Most active mutagens reported in human lymphoblast cell lines (Durant et al., 1998):

Schubert et al., 2003
Dibenzopyrenes – potential human carcinogens
- the mutagenic potency of dibenzo[a,l]pyrene on human cells was estimated at 10 to 100 times that of benzo[a]pyrene (Boström et al. 2002)
2008 Beijing Olympic PM2.5 Sample

14 human cell mutagens

Risk Assessment – RPF Approach

• Relative Potency Factor (RPF) – ratio of the compound potency relative to the potency of an index PAH, i.e. benzo[a]pyrene (BaP);

\[ \text{CancerRisk} = \sum_{i=1}^{n} \left( C_{PAH_i} \times RPF_i \right) \times UR_{BaP} \]

• \( UR_{BaP} \) – Inhalation unit risk of BaP
  - "the calculated, theoretical upper limit possibility of contracting cancer when exposed to BaP at a concentrations of one microgram per cubic meter of air for a 70 year lifetime" (OEHHA 1993, 2005)
  - Based on a rodent study: \( 1.1 \times 10^{-6} \) (ng/m\(^3\))\(^{-1} \) (OEHHA, 2005)
  - Based on an epidemiology study: \( 8.7 \times 10^{-5} \) (ng/m\(^3\))\(^{-1} \) (WHO, 2000)
## Risk Assessment – RPF Approach

- RPFs from an EPA draft under review by the Integrated Risk Information System (IRIS) Program *(USEPA 2010)*

<table>
<thead>
<tr>
<th>PAH</th>
<th>Abbreviation</th>
<th>RPF</th>
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<tbody>
<tr>
<td>Anthracene</td>
<td>ANT</td>
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</tr>
<tr>
<td>Benz[a]anthracene</td>
<td>BaA</td>
<td>0.2</td>
</tr>
<tr>
<td>Benzo[b]fluoranthene</td>
<td>BbF</td>
<td>0.8</td>
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<tr>
<td>Benzo[g,h,i]perylene</td>
<td>BghiP</td>
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<td>Benzo[k]fluoranthene</td>
<td>BkF</td>
<td>0.03</td>
</tr>
<tr>
<td>Chrysene</td>
<td>CHR</td>
<td>0.1</td>
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<tr>
<td>Dibenz[a,h]anthracene</td>
<td>DahA</td>
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<tr>
<td>Fluoranthene</td>
<td>FLA</td>
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</tr>
<tr>
<td>Indeno[1,2,3-cd]pyrene</td>
<td>IcdP</td>
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</tr>
<tr>
<td>Phenanthrene</td>
<td>PHE</td>
<td>0</td>
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<tr>
<td>Pyrene</td>
<td>PYR</td>
<td>0</td>
</tr>
<tr>
<td>Benzo[a]pyrene</td>
<td>BaP</td>
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</tr>
<tr>
<td>Dibenzo[a,l]pyrene</td>
<td>DBalP</td>
<td>30</td>
</tr>
<tr>
<td>Naphtho[2,3-e]pyrene</td>
<td>N23eP</td>
<td>0.3</td>
</tr>
<tr>
<td>Dibenzo[a,e]pyrene</td>
<td>DBaeP</td>
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<tr>
<td>Dibenzo[a,i]pyrene</td>
<td>DBaiP</td>
<td>0.6</td>
</tr>
<tr>
<td>Dibenzo[a,h]pyrene</td>
<td>DBahP</td>
<td>0.9</td>
</tr>
</tbody>
</table>

**12 priority pollutant PAHs**

**5 MW 302 PAHs**
BaP$_{eq}$ Conc. during the Olympics

- **PPAH$_{12}$-BaPeq**
- **302PAH$_5$-BaPeq**

**China SEPA Daily BaP$_{eq}$ Standard (10 ng/m$^3$)**

**Exceedance: 13 of 56 days**

**EU Annual BaP$_{eq}$ Standard (1 ng/m$^3$)**
BaP$_{eq}$ Conc. Reduction

- Individual PAH: 27-78% (p-value ≤ 0.01)
- ΣPPAH$_{17}$-BaPeq: 11.1 → 6.0 ng/m$^3$ (46%)
- ΣPPAH$_{12}$-BaPeq: 9.4 → 4.9 ng/m$^3$ (48%)
- Σ302PAH$_{5}$-BaPeq: 1.7 → 1 ng/m$^3$ (38%)
BaP_{eq} Concentration Profile

- BbF (high conc.)
- DahA (high RPF)
- DBaP (high RPF)
- BaP (high conc.)
Reduction in PM2.5 emission would reduce the concentration of carcinogenic PAHs
Excess cancer risk per million people (based on $UR_{BaP} = 1.1 \times 10^{-6}$ (ng/m$^3$)$^{-1}$)

Source Control Period

Non-Source Control Period

Exhalation Cancer Risk Estimate

Inhalation cancer risk estimate based on $UR_{BaP} = 8.7 \times 10^{-5}$ (ng/m$^3$)$^{-1}$

Bar charts showing excess cancer risk for various compounds during source control and non-source control periods.
Estimated Inhalation Cancer Risk of $\Sigma_{P} PPAH_{17}$:
- @rodent study $UR_{BaP}$: 12.2 → 6.5 per million
- @epidemiology study $UR_{BaP}$: 964 → 518 per million
Excess cancer risk per million people (based on URBaP=1.1E-6 (ng/m³)-1)

Olympic Period

Estimated Inhalation Cancer Risk of ΣPPAH_{17}:
@ rodent study URBaP: 9.4 → 4.8 per million
@ epidemiology study URBaP: 741 → 377 per million

Non-Olympic Period
Estimated cancer risk is 46% lower due to source control measures if they were sustained over time.

The total excess cancer risk would be underestimated by 23% if the 5 MW 302 PAHs were not included in the estimate.
Comparison with Other Studies

586 per million (Liu et al., 2007)
455 per million (Zhang et al., 2009)

Excess cancer risk per million people (based on UR_{BaP}=8.7E-5 (ng/m^3)^{-1})
Limitations

• Additive cancer risk assumption may not be entirely accurate but it’s the currently acceptable approach (OEHHA 2003);

• RPF values are estimated based on toxicological studies that suffer from additional uncertainties but they are the most recently accepted estimated (USEPA 2010);

• Lifetime exposure vs. short duration of source control and Olympic periods;

• Extrapolation of PAH concentration in PM2.5 collected from a single, representative site in Beijing to all of Beijing.
Policy Implications

Controlling vehicle emissions is key to reducing the inhalation cancer risks due to PAH exposure.

Wu et al., 2010

http://www.greenpacks.org/tag/beijing
Policy Implications

Today my car stays home!

• Vehicles restricted by one day per week – a two year demo program in Beijing (Wu et al., 2010)
• Other strategies: development and encouragement of public transportation and more stringent vehicle emission standards (Wu et al., 2010)
Summary

• The lifetime excess cancer risk due to exposure to the 17 carcinogenic PAHs was estimated to range from 6.5 to 518 per million people for the source control period concentrations and from 12.2 to 964 per million people for the non-source control period concentrations;

• This would correspond to a 46% reduction in inhalation cancer risk due to source control measures, if these measures were sustained over time;

• Source control measures, such as those imposed during the 2008 Beijing Olympics, can significantly reduce the inhalation cancer risk associated with PAH exposure in Chinese megacities;
• Benzo[b]fluoranthene, dibenz[a,h]anthracene, benzo[a]pyrene, and dibenzo[a,l]pyrene were the most carcinogenic PAH species measured during the Olympics;

• The total excess inhalation cancer risk would be underestimated by 23% if the 5 MW 302 PAHs were not included in the risk calculation, indicating that MW 302 PAHs are a significant component of the overall inhalation cancer risk.