Control of Residential Exposures to Environmental Neurotoxins
Environmental Toxins

- Most recognized toxins discovered only after widespread environmental contamination.
- Increasing evidence for adverse effects at levels previously thought to be low.
- Neurotoxicity animal tests may not be sensitive.
- Toxicity tests not done for majority of chemicals, including those recognized as potential neurotoxins.
Residential Hazards

- Lead
- Tobacco Smoke
- Pesticides
Types of Prevention

- Education
- Enforcement
- Engineering

Greater Effectiveness
Types of Prevention

• Education
• Enforcement
• Engineering

Higher Cost
Levels of Prevention

- Primary
- Secondary
- Tertiary
Steps to Prevent Childhood Exposure to Residential Neurotoxins

• Identify sources of exposure

• Identify unacceptable levels of exposures form contributing sources

• Test efficacy and safety of interventions to reduce environmental exposure

• Develop regulations and screening programs
Prevention of Childhood Lead Exposure
Pathways of Childhood Lead Exposure

- Paint Lead
- Soil Lead
- Dust
- Hand
- Blood Lead
- Black Race
- Income
- Play Outside
- Eats Soil
Blood Lead Levels of Children in Monroe County (NY), by Age of Child, 1993
Frequency of Mouthing Behaviors during Early Childhood

Contribution of Various Sources of Lead to Urban Children’s Blood Lead Levels

- Paint Chips: >25 µg/dL
- Lead-contaminated House Dust: 5-25 µg/dL
- Lead ingestion from soil, water and diet: < 5 µg/dL
Contribution of Lead-contaminated Floor Dust to Blood Lead Level by Age

Standards and Interventions for Residential Dwellings
# Effect of Lead Hazard Controls

## Results of Controlled Trials

<table>
<thead>
<tr>
<th>Hazard Control</th>
<th>BPb * (µg/dl)</th>
<th>Age (months)</th>
<th>Change (µg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Charney Dust Control</td>
<td>&gt; 30</td>
<td>15 - 72</td>
<td>- 6.9</td>
</tr>
<tr>
<td>Farfel Abatement</td>
<td>&gt; 29</td>
<td>9 - 72</td>
<td>- 1.9</td>
</tr>
<tr>
<td>Staes Stabilization</td>
<td>&gt; 25</td>
<td>&lt; 72</td>
<td>- 4.0</td>
</tr>
<tr>
<td>Aschengrau Abatement</td>
<td>3-22</td>
<td>&lt; 48</td>
<td>+ 6.5</td>
</tr>
</tbody>
</table>

* Blood lead levels at baseline
Geometric Mean Floor Dust Lead Levels ($\mu$g/ft$^2$) by Abatement Status*

*Farfel AJPH 1990: 80; 1240-1245
**HUD Post Abatement Standards for Lead-Contaminated House Dust**

<table>
<thead>
<tr>
<th>Surface</th>
<th>Standard in µg/ft²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Floors</td>
<td>200 µg/ft²</td>
</tr>
<tr>
<td>Sills</td>
<td>500 µg/ft²</td>
</tr>
<tr>
<td>Troughs</td>
<td>800 µg/ft²</td>
</tr>
</tbody>
</table>
EPA Residential Standards for Lead-Contaminated House Dust

<table>
<thead>
<tr>
<th>Location</th>
<th>Standard</th>
</tr>
</thead>
<tbody>
<tr>
<td>Floors</td>
<td>40 (\mu g/ft^2)</td>
</tr>
<tr>
<td>Sills</td>
<td>250 (\mu g/ft^2)</td>
</tr>
<tr>
<td>Troughs</td>
<td>800 (\mu g/ft^2)</td>
</tr>
</tbody>
</table>
Risk of blood lead levels $\geq 10 \mu g/dl$ by floor dust lead levels ($\mu g/ft^2$)

### Effect of Dust Control on Children’s Blood Lead Levels

<table>
<thead>
<tr>
<th>Hazard Control</th>
<th>BPb * (µg/dl)</th>
<th>Age</th>
<th>Change (µg/dL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lanphear Education</td>
<td>6.7</td>
<td>12 - 31</td>
<td>-0.55</td>
</tr>
<tr>
<td>Hilts Professional</td>
<td>11.6</td>
<td>32.4</td>
<td>+0.3</td>
</tr>
<tr>
<td>Aschengrau Paint &amp; Repair</td>
<td>16.9</td>
<td>24.5</td>
<td>+1.1</td>
</tr>
<tr>
<td>Rhoads Professional</td>
<td>12</td>
<td>20</td>
<td>-1.9</td>
</tr>
<tr>
<td>Lanphear Education</td>
<td>2.8</td>
<td>6</td>
<td>-0.2</td>
</tr>
</tbody>
</table>

Effect of Dust Control on Blood Lead Concentration by Group Assignment

Limitations of Studies Evaluating Lead Hazard Controls

• Not adjusted for age or seasonal variation
• Often not randomized or lack control group
• Underestimate effect of intervention
• May not be relevant for children who have blood lead levels < 25 µg/dL.
Enroll Women < 16 weeks gestation (n= 400)

Conduct prenatal surveys, collect maternal urine and blood samples for assessing fetal exposure to toxicants

Randomization

Injury Control Group (n = 200)
- 12 - month visit
- 24 - month visit
- 36 - month visit

Lead Hazard Group (n = 200)
- 12 - month visit
- 24 - month visit
- 36 - month visit

Collection of Biomarkers and exposure assessment in early childhood

12, 24 and 36-Month Outcomes
Exposures and Biomarkers for Pesticides, Lead and Cotinine Behavior, Cognition and Executive Function
Hearing and Growth
Tobacco Exposure in Children
Routes of Exposure

- Fetal transfusion
- Respiration
- Ingestion
Salivary Cotinine levels ($\mu$g/mL) by Children’s Age

Urine Cotinine ($\mu$g/mL)

Age of Children

Cotinine Levels by Exposure Source

Median Cotinine Level (ng/ml)

- Mother: 100 ng/ml
- Father: 80 ng/ml
- High: 70 ng/ml
- Moderate: 60 ng/ml
- Low: 40 ng/ml

Community ETS Exposure

Cotinine Levels by Number of Household Smokers

Methods of Control

• Source Elimination
• Source Reduction
  • Filtration
  • Ventilation
  • Containment
Reducing ETS Exposure

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>No.</th>
<th>Age</th>
<th>Cotinine Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Woodward</td>
<td>1987</td>
<td>184</td>
<td>&lt; 3 months</td>
<td>NC</td>
</tr>
<tr>
<td>Chilmonzyk</td>
<td>1992</td>
<td>103</td>
<td>Infancy</td>
<td>NC</td>
</tr>
<tr>
<td>Greenberg</td>
<td>1994</td>
<td>121</td>
<td>&lt; 6 months</td>
<td>NC</td>
</tr>
<tr>
<td>McIntosh</td>
<td>1994</td>
<td>91</td>
<td>1-17 years</td>
<td>NC</td>
</tr>
<tr>
<td>Hovell</td>
<td>2000</td>
<td>108</td>
<td>&lt; 4 years</td>
<td>p = 0.008</td>
</tr>
</tbody>
</table>
Effect of HEPA-CPZ Air Cleaners on Air Nicotine (n=12 per group)

Air nicotine (mcg/m³)

Baseline | 2 weeks | 4 weeks

P < 0.05

Intervention | Control

Aligne CA (unpublished data).
Cincinnati Asthma Prevention (CAP) Study

Enroll Children 6 to 11 years (n= 240)

Conduct asthma surveys, collect serum and hair, PFT’s and expired nitric oxide levels

Randomization

Collection of Biomarkers and exposure assessment

Experimental Group (n = 120)
- 3-month visit
- 6-month visit
- 9-month visit
- 12-month visit

Experimental Group (n = 120)
- 3-month visit
- 6-month visit
- 9-month visit
- 12-month visit

Outcomes
- Exposures and Biomarkers for Tobacco (Cotinine)
- Pulmonary Function, Health Service Utilization,
  Expired Nitric Oxide and Behaviors

HEPA-CPZ Air Cleaners
One Maryland County Moves
To Regulate In-Home Smoking

By The New York Times

BALTIMORE, Nov. 21 — A suburban Washington county has approved a strict antismoking measure that would impose $750 fines against residents if the odor of their smoking irritates neighbors.

Douglas M. Duncan, a Democrat who is the county executive in Montgomery County, Md., said today that he planned to sign the measure, which the County Council approved 6 to 2 on Tuesday. Mr. Duncan originally proposed a bill to regulate indoor air quality that had exempted tobacco smoke, but the council added it to the measure.

Under the bill, tobacco smoke would be treated the same as pollutants like asbestos, radon, molds and pesticides. Smokers could face fines of as much as $750 a violation, and landlords or condominium associations could also face fines for failing to equip buildings with adequate ventilation.

"Montgomery County has been a leader on air-quality issues for many years, so it is fitting that we once again are breaking new ground by enacting tough indoor air-quality standards," Mr. Duncan said.

But opponents, including civil libertarians and some council members, called the antismoking provision an infringement on the rights of residents who smoke in their homes.

"It certainly does smack of Big Brotherism and invasion of privacy," said Michael L. Subin, a Democratic council member who voted against the measure. "Government has now entered the four walls to your home to say you can be fined for doing something that your neighbor doesn't like."

Patrick Lacefield, a council spokesman, defended the measure, saying it was intended to protect the health of the county’s residents.

"We were looking to revise a 25-year-old clean-air law, and 25 years ago, people were smoking in hospitals, and train stations and airports," he said. "Second-hand tobacco smoke is very dangerous and is a known health hazard. The idea was if somebody could complain about someone sending dioxide or benzene their way, why couldn’t they also be able to complain about smoke?"

Under terms of the bill, county environmental inspectors would investigate complaints. Fines would be imposed, the measure’s supporters said, only after warnings and suggestions to resolve complaints through steps that might be as simple as opening a window.

Critics argued that no evidence exists showing that smoke from a neighboring home poses a risk.

"The government should not be able to come in and say you can’t use lots of garlic or perfume in your own home, and the government shouldn’t be able to say you can’t smoke in your own home because of the smell," said Arthur Spitzer, the legal director of the American Civil Liberties Union’s area branch.

Brendan McCormick, a spokesman for Philip Morris USA, called the measure the most restrictive he had ever heard of.

"We understand that people are bothered by being around smoke and, in public places, we support reasonable restrictions," Mr. McCormick said. "But when the legislation restricts smoking in your own home, that’s going too far."
Rates of Cigarette Consumption and Predicted Rates without the Tobacco Control Program
Limitations of Research on ETS Exposure

- Inconsistent measures of exposure.
- Often relied on parental report for ETS exposure and behavioral outcomes.
- Small sample size
- Limited to educational interventions.
Implications For Prevention

• Emphasis to broaden beyond treatment and education to include passive reduction of exposure to environmental tobacco smoke.

• Empirically-derived health-based standards are needed for environmental tobacco smoke.

• Randomized trials to assess if hazard controls are effective in reducing children’s exposure to environmental tobacco smoke, including improvements in neurobehavioral outcomes.
Pesticides
Organophosphosphate Metabolites in Children’s Urine, Seattle WA

Lu C. Env Health Persp 2002:109:299-303
Failure of Toxicity Testing

• Of the 3,000 high production volume chemicals, 75% lack even the most basic toxicity tests. ¹

• Of the 140 registered pesticides EPA considers to be neurotoxic, the majority have not been tested for developmental neurotoxicity. ¹

• Animal testing may not be sensitive enough to protect humans. ²

• We lack animal models for important human skills, such as reading.

Steps to Prevent Childhood Exposure to Residential Pesticides

• Developmental neurotoxicity tests for all new chemicals or pesticides.

• Test children’s exposure to new agents by measuring biomarkers and potential adverse effects prior to marketing.

• Post-marketing surveillance of pesticide toxicity.
Implications for Prevention of Children’s Exposure to Residential Toxins

• Emphasis to shift from screening or diagnosing children with disease to preventing exposure.

• Empirically-derived health-based standards for settled dust and indoor air are needed.

• Randomized trials to assess if controls are effective in preventing children’s exposure and any adverse effects.

• Studies to examine adverse effects of toxins at lower levels and for pesticides.
“Until effective standards for the domestic environment are devised, it is likely that children will continue to be employed as biological indicators of substandard housing.”

Donald Barltrop, 1974
"Until effective standards for the domestic environment are devised, it is likely that children will continue to be employed as biological indicators of substandard housing."