Environmental Health: Issues and Impact

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Module #17: Environmental Health: Issues and Impact

Part 2: Toxics and Children’s Health
Lead and Mercury

EH-8 Reduce blood lead levels in children to human health and the environment posed by EH-8.2
EH-9 Minimize the risks hazardous sites
EH-20 Reduce exposure to selected environmental chemicals in the population, as measured by blood and urine concentrations of the substances or their metabolites
Environmental Exposures to Lead

- Lead has been released into the environment from: Factory and automobile emissions
- Contamination from mining and smelting
- Lead-based paint with deterioration, demolition or renovation
- Incineration of lead-containing waste
- Much of lead produced today is recovered from secondary sources of lead scrap
- Battery recycling important exposure in developing countries; electronic recycling potentially big exposure
Common LEAD Sources for U.S. children

Older Housing—Dust, Soil and Paint Chips Especially Older, low-income, Deteriorated Housing

Oral, Mobile Child PLUS Old and Deteriorated housing with deferred Maintenance

EQUALS

Lead Exposure and Elevated Blood Lead Level (=> 5 µg/dL)

- Philadelphia Housing, 2000 Census: 590,071 occupied units; 59% owner-occupied units; 41% rental-occupied; ~94% built pre-1978; 75% pre-1960; 42% pre-1940
- American Housing Survey, Phila, 2009: 91.6% pre-1978
- US Housing: 2002 HUD Survey: 25% of US housing (24 million units) with LBP hazards: deteriorated paint, lead in dust or bare soil (Jacobs et al, 2002)
Where Lead Is Found

Different surfaces in a home pose varying lead-paint risks. Friction from opening and closing windows can turn hazardous exterior paint into lead dust, which can be ingested by people inside. If intact, large interior surfaces, like walls and ceilings, are far less likely to pose a danger.

Where Lead Is Found in the Home:

- **Very frequently**
- **Frequently**
- **Occasionally**

SOURCE: DEPARTMENT OF HOUSING AND URBAN DEVELOPMENT
Common LEAD Sources For U.S. Children-Other Sources

- Ethnic Cosmetics/Remedies/Foods, Ceramic Cookware—specifically with diverse Latino and Asian populations
- Consumer Products-Toys, Lunch Boxes, Jewelry etc.
- Water from older plumbing systems
- Industry: point sources Smelters, mines

LEAD exposures- for Children

• **INGESTION**- lead is swallowed after putting contaminated dust or soil into the mouth, or paint chips or other lead-containing object

• **INHALATION**- breathing lead fumes or dust when lead is heated or sanded

• **TRANSPLACENTAL TRANSFER**- lead freely passes from mother to fetus during pregnancy at roughly the same blood lead levels
Biological Impact Pathway of Lead on Cognitive Function

**Sources**
- Paint
- Toys
- Contaminated Soil
- Air

**Presence**
- Home
- Other
- Buildings
- Outdoors

**Exposure**
- Individual children

**Dose**
- At Central nervous system

**Health Effect**
- Decreased cognitive function and other neurological effects

**Modifiers**
- Housing quality
- Paint deterioration
- Other factors
- Other transport

- Hand-to-mouth activity
- Crawling/floor contact
- Pica
- Respiration
- Play locations
- Other behaviors

- Age
- Iron deficiency
- Calcium deficiency
- Other pharmacokinetic factors

- Age
- Malnutrition
- Folate intake
- Low birth weight
- Home learning environment
- School experience
- Maternal education level
- Socioeconomics status (SES)
- Environmental enhancement
- Health status
- Health-related quality of life
- Genetic and epigenetic variation
- Other does-response modifiers
- Other environmental exposures
Children

Lead Concentration in Blood (µg Pb/dL)

150
Death

100
Encephalopathy

Frank Anemia

Nephropathy

Frank Anemia

Decreased Longevity

Colic

50
Hemoglobin Synthesis

Peripheral Neuropathies

Hemoglobin Synthesis

Fertility (Men)

Nephropathy

Vitamin D Metabolism

Systolic Blood Pressure (Men)

Hearing Acuity

Erythrocyte Protoporphyrin

(Men)

Nerve Conduction Velocity

Erythrocyte Protoporphyrin

10
Hypertension (?)

Increased Function

Decreased Function

- Effects in children generally occur at lower blood lead levels than in adults.
- The developing nervous system in children can be affected adversely at blood lead levels of less than 10 µg/dL.
Children and Lead exposure

Overview

- One of major environmental problems for U.S. children

- Acute exposure can affect many body systems, particularly the Central Nervous System (CNS); **CAN BE FATAL** with encephalopathy

- Children are usually asymptomatic or have subtle neurodevelopmental changes
Children and Lead exposure Overview

- Chronic, low-level exposure can lead to subtle neurodevelopmental effects

- Sometimes not recognized until older grades requiring higher-level cognitive functions

- These can impact on children’s future education and employment status (as well as lead to societal costs)

- Newer studies indicate NO THRESHOLD for effects: NO LEAD IS GOOD LEAD
What does the Literature tell us about effects of LEAD Exposure

- Inverse relationship between indices of lead exposure and IQ (cognition)

  About 4-8 points as BLL increases to 10 µg/dL and 1-5 points as BLL reaches 20 µg/dL

- Decrease in proficiency in basic academic skills (math, reading), decreased school achievement and poor organizational skills

- Association with attention, learning, behavioral (distractibility and hyperactivity) problems

(REF: Lidsky and Schneider, 2006; Bellinger 2004; AAP 2005; Lanphear et al, 2005; Jusko et al, 2008; Canfield, 2003; CDC 2005; Binns, 2007)
2012, CDC: Changing the Blood Lead Level of Concern (10 µg/dL) to Reference Value (5 µg/dL)

Reference value replaces 1991 level of concern; 97.5th percentile of two cycles (4 years) of NHANES data

To be updated every four years

This is not a level for toxicologic effects in an individual, but the level at which clinical and public health interventions are recommended

Primary prevention interventions: have the greatest potential for success
NHANES estimates and 95% confidence intervals of prevalence of blood lead >=5 among U.S. children aged 1-5, 1999-2008
Mercury -
What is it and where it comes from

- Forms of Mercury - Elemental, Inorganic and Organic
- Methylmercury

- Sources of Mercury
- Natural sources
- Anthropogenic
  - Combustion
  - Manufacturing
  - Mining
  - Re-mobilization

Exposure to Mercury

- Environmental Effects
  - Ecosystems
    - aquatic
    - terrestrial
  - Bioaccumulation

- Health Effects
  - Consumption of fish
  - Dental amalgams
  - Spills, Products, Airborne Mercury
  - Minamata Disease

In the U.S., Power Plants Emit:

- 13% of the NO_x
- 60% of the SO_2
- 60% of the arsenic
- 30% of the nickel
- 20% of the chromium
- 50% of the mercury
- Over 50% of many acid gases

Sources: NEI Trends Data (2009) and IPM (2010) (SO_2, NO_x). A proposed toxics rule modeling platform, based on inventory used for 2005 NATA (Hg); Inventory used for 2005 NATA (other toxics)
Biotransformation of mercury

- Inorganic Mercury
  - Discharge
  - Bio methylation
    - Methyl-Mercury (Me-Hg)
      - Bioaccumulation
        - Me-Hg in edible fish
          - Exposure
        - Me-Hg in Humans
          - Bioaccumulation
            - Ambient Water Sediments
              - Discharge
Mercury health effects w/ increasing urinary concentrations $\geq 20 \mu g/L$

- 20 – 100 – abnormal psychometric test results, nerve conduction velocities
- 100 – 500 – tremor, memory loss, irritability, depression, other CNS abnormalities, renal impairment, (peripheral neuropathy?)
- 500 – 1000 – overt CNS disorder, stomatitis, renal failure, severe tremor, perip. neuropathy

Ref: ATSDR, 1999
Methylmercury in Fish

- Bacteria biotransform elemental mercury into methyl mercury (Me-Hg)
- Mollusks, crustaceans, plankton, etc., eat bacteria
- Me-Hg poorly eliminated; concentrates up the food chain... biggest and oldest predators at the top of ecosystem have the highest concentrations
- Me-Hg is distributed evenly throughout the fish, not changed by cooking
Minamata Bay, Japan

- 1953 - outbreak of “polio-like disease” in coastal fishing villages
- Coincided w/ startup of acetaldehyde production at coastal factory using mercuric oxide as a catalyst
- Stray cats went crazy, died, after eating fish
- 1968 - mercury identified as cause of 2000 disease victims
- Water levels Me-Hg = 400+ ppb
Minamata disease - infants

Health Effects

- Mental retardation
- Abnormal reflexes, ataxia, involuntary movements
- Cerebral palsy
- Developmental delays — some didn’t walk until age 7
How much Hg is toxic?


Reference Dose (RfD) recommended to EPA is 0.1 micrograms/kg/day

(“safe” daily intake to avoid toxicity)

To comply with EPA reference dose:
- Do not eat fish with levels > 1 part per million (ppm)
- Limit fish with levels > 0.2 ppm to once per week
Do vaccines containing thimerosal cause autism?

Some parents say “yes;” science says “no”

Hviid A, et al. Association between thimerosal-containing vaccine and autism (Denmark registry) JAMA October 1, 2003;290:1763-6 - totally negative


May 2004AAP, CDC - no association

Paul Offitt, book, 2008 - no association