Do Waste Site Risk Assessments Adequately Address Endocrine Disruption Effects?

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Site Management Questions

- **Is our management of endocrine disruption contaminants sufficient?**
- **If not, what should we be doing?**

Overview

- Summary of available information
  - Nature of endocrine disruptors
  - Historical Events and Studies
  - Ambient Exposures
- Endocrine disruptors at waste sites
- Management options

Nature of Endocrine Disruptors

Endocrine Disruptors

EDs can disrupt an endocrine system by:

- Mimicking a natural hormone; or
- Blocking a hormone from a receptor; or
- Directly stimulating or inhibiting the endocrine system

What kinds of Chemicals are Endocrine Disruptors?

Common Waste Site EDs

- Dioxins
- PCBs
- PAHs
- Perchlorate
- Arsenic
- Lead

Ambient EDs

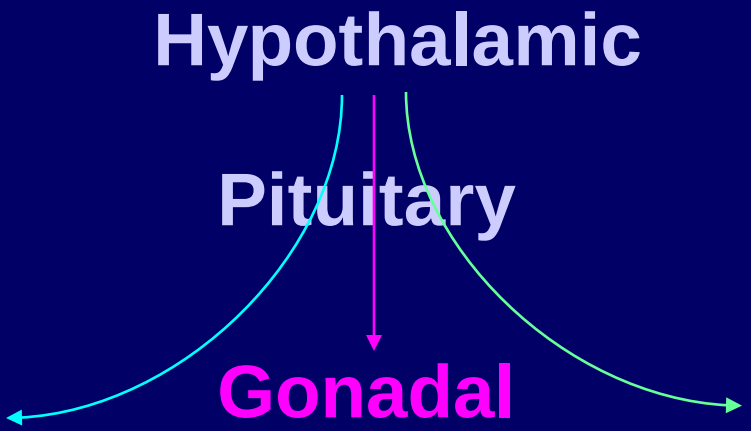
- Plastics
- Surfactants
- Pesticides
- Pharmaceuticals

Endocrine Disruptors: Challenges for Effects Assessment

- Detecting effects may require long study durations, in some cases generations.
- A single endocrine disruptor can act on different endocrine systems.
- Different endocrine disruptors can act on the same endocrine system through the same pathways.
- Different endocrine systems can affect the same biological functions.

Endocrine Organization and Function

Three Examples (Axes)



Cortisol
Corticosterone

Estrogens
Androgens

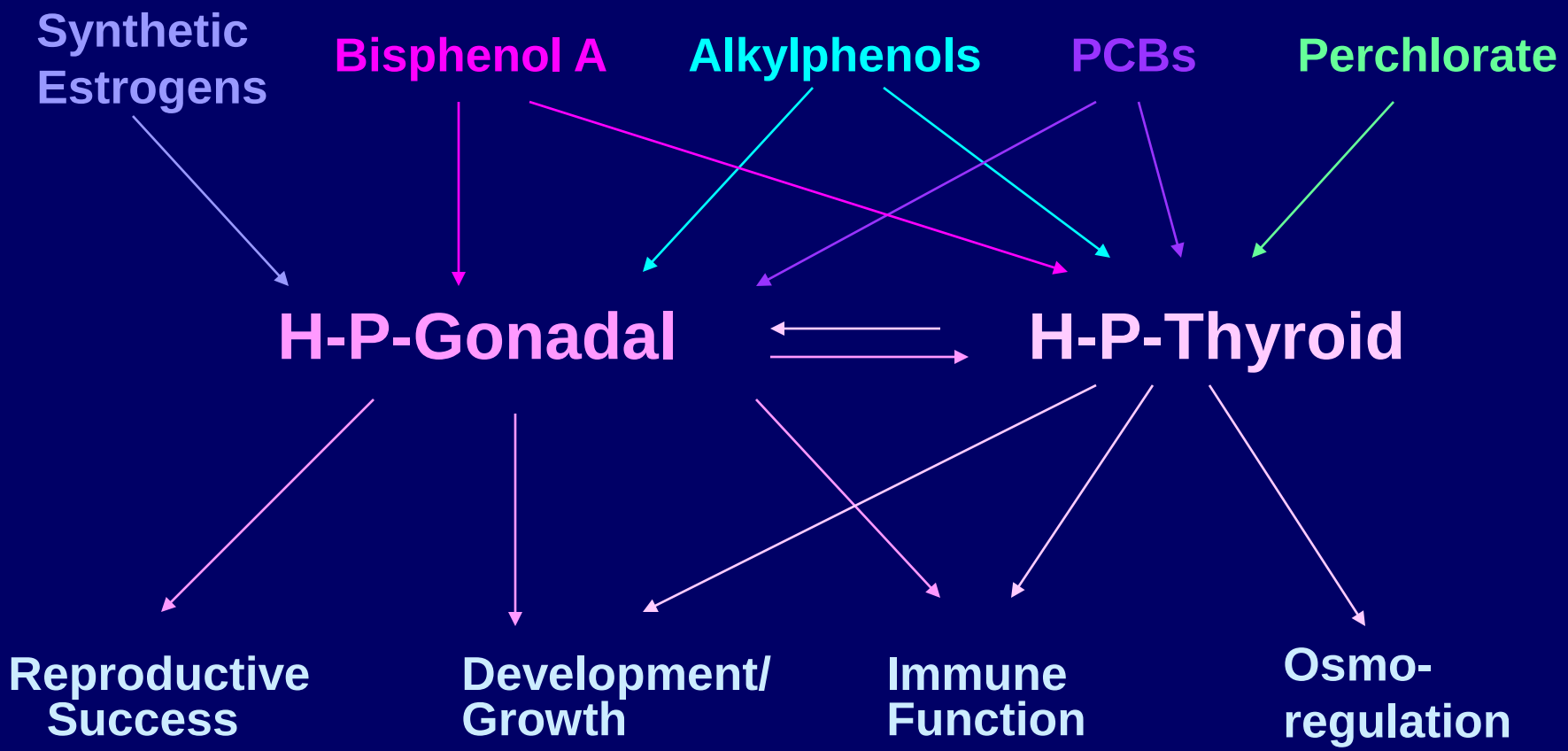
T3 and T4

Metabolism, anti-inflammation, stress responses

Sex characteristics, reproduction, brain, immune system

Development, growth, metamorphosis

Endocrine Disruptor and System Interactions



Historical Events and Studies

Extreme Events Leading to Endocrine Effects

Event/ Location	Maternal Exposure	Effects in offspring
Miscarriage prevention (1950s-60s)	DES, prescribed	F: adenocarcinoma M: cryptorchidia, hypospadias [1]
Japan, Yusho disease (1968)	PCBs in rice oil	Physical abnormalities, Neuropsychological development [2]
Seveso, Italy, explosion (1976)	2,3,7,8-TCDD, dispersed	Altered sex ratios a birth [3]
Taiwan, Yu-cheng (1979)	PCBs in rice oil	Physical abnormalities, Neuropsychological development [4]

Effects on Wildlife from High Exposures

Species	Contaminants	Effects
Eagles, Ospreys (1960s)	DDT	Estrogen function, thin-shelled nonviable eggs[1]
Alligators, L. Apopka (1980s)	OC pesticides, PCBs (past Exposures)	Low testosterone, penis size, gonadal abnormalities[2]
Florida panthers (1990s)	Pesticides and Hg in diet?	Reproductive disorders: cryptorchidia, sperm abnormalities[3]

Ambient Endocrine Disruptors

Untreated Wastewater Contaminants

Pharmaceuticals Plasticizers Pesticides Surfactants

Airborne PAHs and Endocrine Disruptors Effects on Inner-City Children

- Delayed cognitive development
  at 3 years,
  not 1 or 2
- DNA damage
- Impaired fetal growth

Male Reproductive Disorders

In some urban areas:

- Declining sperm counts
- Increasing testicular cancer
- Increasing rates of malformations

Endocrine Effects from Fish Exposures

Location	Exposure	Effects in Children
Lake Michigan, 1980-1981	Maternal fish consumption - PCBs	Decreases in some IQ measures in offspring assoc. with prenatal exposures, up to age 11 yrs [1]
Oswego, NY 1991	Maternal fish consumption - PCBs	Higher cord conc. assoc. with lower visual recognition memory at 6 and 12 months [2]
Akwesasne Mohawk Nation, NY-Ont. Queb.	Mother & child fish consumption & ambient exposure	8 per. PCB cong. conc. in youth serum affect TSH and FT4 in non-breastfed youth [3]
Same as above	Same as above	Estrog. serum PCB conc. assoc. with higher likelihood of menarche in 10-16 yr old females [4]

Ambient Organochlorine Exposures
Neurodevelopment in Children of Exposed Mothers

Location	Exposure Measures	Effects in Offspring
Rotterdam, Netherlands [1]	PCBs, dioxins, & furans in cord and maternal serum	Cognitive development effects up to 3.5 yrs associated with prenatal exposure
Raleigh-Durham, North Carolina, USA [2]	PCBs in cord and maternal serum and milk	Cognitive development effects up to age 2 yrs associated with prenatal exposure
Düsseldorf, Germany [3]	PCBs 138, 153, and 180 in cord serum & milk	Cognitive development effects up to 3.5 yrs associated with prenatal and postnatal exposure

We still do not know . . .

- For known disruptors:
  - Critical endocrine effects
  - Threshold concentrations or exposures
  - Mechanism of action

- For observed effects
  - Specific disruptor(s)

Endocrine Disruptors at Hazardous Waste Sites

The Important Questions for Site Managers

- Could endocrine disruptors at sites adversely affect:
  - Wildlife?
  - People?
- At concentrations/exposures below levels of concern for other effects, could endocrine effects occur in:
  - Wildlife?
  - People?

Low-Level Site-Related Exposures Could Affect Human Endocrine Systems

Contaminant/Effect	Endocrine Effect Level	Conventional Effect Level
Serum PCBs - Endometriosis Upstate NY Region affected by Superfund sites[1]	PCB Serum Conc.: >1.36 ng/g tot. >0.4 ng/g est. >0.04 ng/g ant. est	Not measured
Elevated serum PCB, neurodevelopment New Bedford Harbor vicinity, Cord serum vs. distance (No[2]), local dairy (Yes[2]), dredging (Yes[2]), infant attention (Yes[3])	PCB Serum Conc.: Median = 0.4 ng/g Range =0.068 – 18.14 ng/g	Not measured
Lead - Effects on thyroid hormone levels [4] Akwesasne Mohawk children, upstate New York	Med. blood Pb = 1.3 ug/dl Max blood Pb = 4.8 ug/dl	EPA target blood lead level = 10 ug/dl

Current Eco Risk Assessment Practices May Fail to Detect Endocrine Effects on Wildlife at Sites

Effect /Receptor	Endocrine Effect Level	Conventional Measure of Effect
Effects of pyrene in soil on springtails[1] (Lab study)	EC50 = 104 umol/kg soil	LC50 = 741 umol/kg soil
Reproductive effects on turtles in a Cape Cod Pond [2] (Massachusetts Military Reservation)	Qualitative assessment	Not available
Larval survival and metamorphosis in leopard and wood frogs [3] Housatonic River Wetland	Sed. conc: <1mg/kg few effects >20 mg/kg higher rates of effects	Consensus-based TEC is 60 mg/kg
Fish survival and development Housatonic River [3]	Avg. sed. conc. ~10 mg/kg; SW conc. Range = 0.013 – 1.5 ug/L	Population effects (not detected)

Effects Assessment – Phase I Toxicity Results


A


B


C


D


E

Largemouth bass fry:
A – normal
B – head deformity
C – edema
D – vertebral anomaly
E – partially external swim bladder (15-days post-swim up)

- Effects consistent with PCB/dioxin-like toxicity
- Increased effects relative to reference (Threemile Pond)
- Adults –liver; abnormal gonads
- Offspring – reduced survival at swim-up; delayed development; deformities; slower growth

Answers to the Previous Questions

- Could endocrine disruptors at sites adversely affect:
  - Wildlife? Yes
  - People? Probably
- At concentrations/exposures below levels of concern for other effects, could endocrine effects occur in:
  - Wildlife? Yes
  - People? Possibly

The Evidence Suggests:

- Endocrine disrupting chemicals found at waste sites can have effects at environmentally relevant concentrations.

- Endocrine effects have been reported at concentrations that are:
  - lower than risk-based concentrations conventionally derived for site management, and/or
  - consistent with ambient air exposures not normally assessed at waste sites.

Challenges

- For human health risk assessment, endocrine disruption-specific toxicity values are not available.

- For ecological risk assessments, available toxicity values and standard toxicity tests do not generally detect endocrine-related effects.

Options

- Wait for quantitative toxicity values.
- Conduct field/lab studies for wildlife and regional population studies of human exposure in conjunction with the risk assessment.
- Use available information qualitatively in risk assessments.
- Consider available information on reported ED effects of COCs as a factor in risk management decisions as a factor in remedy selection.

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