Early Exposures to Hazardous Chemicals/Pollution and Associations with Chronic Disease – A Scoping Review

July 20, 2011 Videoconference briefing of report prepared for:

Joint CPCHE-OCDPA Initiative on Early Environmental Exposures and Chronic Disease Prevention in Ontario

Kathleen Cooper, Senior Researcher

Canadian Environmental Law Association

Presentation Overview

- Report Part 1 – Context for CPCHE-OCDPA collaboration:
  - Chronic disease in an aging population; multiple and interacting determinants of health
- Report Part 2 - The Evidence
  - Detailed introduction addressing three overarching concepts
  - Five areas of chronic disease - focus on early exposures in context of multiple risk factors.
- Overall observations and conclusions

Part 1 - Context

Chronic disease in an aging population

- *Perfect Storm* (CVD)
- *Rising Tide* (dementia)
- *Global pandemic* of obesity
- *Economic tsunami* (diabetes)
- 1 in 4 will die from cancer (40-45% will get cancer)
- 4-fold increase since 1980 in children with asthma

☐ Projections: multi-millions affected; multi-billions in projected costs

☐ Disproportionately worse health and shorter lifespan among those living in poverty

# Biomonitoring Results Confirm Widespread Exposure to Toxic Chemicals

- ❑ **Biomonitoring**: measures levels of contaminants in blood, urine, breastmilk, expelled air, etc.
- ❑ Results from population-based testing:
  - ❑ Everyone carries many different chemicals.
  - ❑ Levels are higher in children and highest for breast-fed babies at "top of food chain."
  - ❑ **Very low levels, consequences uncertain.**
  - ❑ Results <u>should not deter breastfeeding</u> (always the best food for babies)

Fetus and Child are More Vulnerable

- Higher levels of exposure
  - Children eat, drink and breathe more than adults per unit of body weight
  - Behaviours (e.g., hand-to-mouth activity) increase exposure
  - Toxic substances cross the placenta
- Greater susceptibility to harm:
  - The rapid, dynamic processes of development
  - Immune and detoxification systems are immature
- Poverty increases these risks
- First Nations children likely at greatest risk
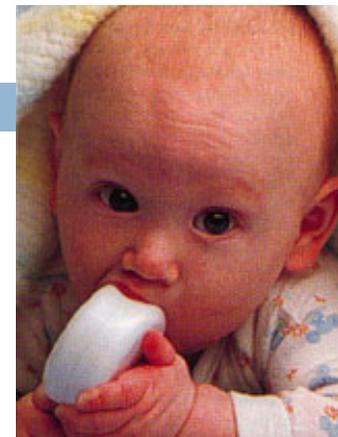
Child Health Problems Associated with Environmental Exposures

Impacts on:

• The respiratory system

• The developing immune system

• The developing brain and nervous system

• Reproduction and child development

• Risk of cancer in children and young adults

• The endocrine system contributing to reproductive/developmental impacts or increased risk for later life cancers

Multiple causes for each and environmental evidence is often incomplete – but, high stakes risks

The worst contaminants are often associated with several of these effects

The Multiple Determinants of Health

Figure 1: Determinants of Health
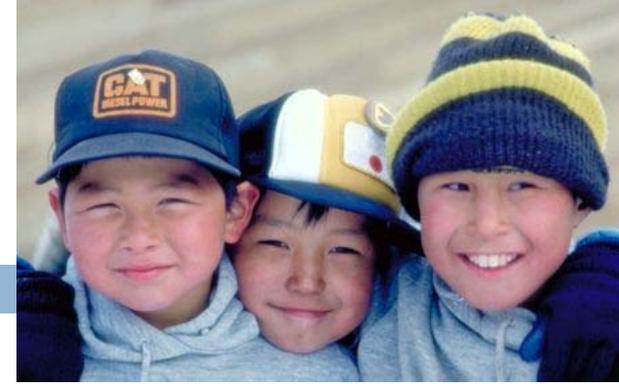

Source: World Health Organization, undated.

- All of above child health outcomes result from multiple determinants

- Likewise for chronic disease - well understood risk factors for most

  - Behavioural/"lifestyle" the "Big 3" – diet, exercise, smoking

 - Biomedical factors or intermediate conditions

- **But, not the full story**

Primacy of the Social Determinants of Health (SDOH)

- Worldwide, health follows gradient of socio-economic status

- SDOH referred to as the "causes of the causes"

- WHO re SDOH: ethical imperative to address social justice and health inequities

- Cd'n Medical Assoc. President-elect in November, 2009:

  - "poverty is the greatest predictor of health"

- Canada's Chief MOH and Ontario's MOH – similarly strong statements about primary importance of SDOH

- Inattention to the SDOH can undermine individual behavioural choices to achieve better health, including the inability to adopt such choices at all

Environmental Determinants of Health
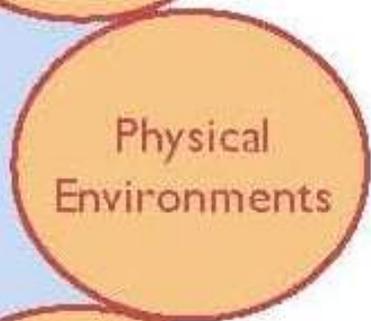
- Indoor and outdoor environments: multiple media and routes of exposure. **Main Focus of Scoping Review**

- Additional highly relevant issues beyond "exposure:"
  - land use planning and built environment: car-dependence, resulting air pollution and climate change, sedentary lifestyles, overweight/obesity
  - Mechanized, centralized and fossil fuel-dependent food production system: major contributor to climate change and the glut of inexpensive sources of unhealthy food

- Severe health impacts of climate change expected in coming decades:
  - catastrophic weather events, extreme heat, increased vector-, food-, and water-borne illnesses and increased air and water pollution, which in turn are anticipated to affect the most fundamental determinants of health - air, food, water and shelter.

Physical Environments

Interacting Environmental and Social Risk Factors

- Environment: cross-cutting determinant
  - similar to income level or gender
  - interacts with other determinants, especially the SDOH
- Examples among low-income communities
  - higher air pollution-related hospitalization and mortality
  - higher exposures via sub-standard housing, use and reuse of older consumer products, canned food, etc.
- Worst effects of climate change predicted to occur among the poor and the elderly
- Early child development – crucial influence on lifelong health; also strongly affected by poverty

Part 2 – The Evidence

- Detailed introduction to address three cross-cutting areas:
  - The Developmental Origins of Health and Disease (DOHaD)
  - Epigenetics – mechanisms underlying DOHaD
  - Evaluating evidence and challenges posed by environmental cases
- Followed by detailed reviews of each of CVD, T2D, AD and PD, Cancer (focus on breast prostate and testicular), and Asthma

The Developmental Origins of Health and Disease (DOHaD)

- The environment (broadly defined) in early life sets the trajectory for lifelong health and well-being.

- Extensive epidemiological, clinical and experimental animal evidence of associations between fetal and neonatal under-nutrition and major risk factors (hypertension, insulin resistance and obesity) for cardiovascular disease, diabetes and the metabolic syndrome in adult life.

- Concept is expanding to include early environmental exposures

Tulip bulb puree was one response to the Dutch famine of 1944

DOHaD Mechanisms: Epigenetics

- Gene-environment interactions which lead to changes in the expression of genes without change in DNA sequences.

- Each cell has same genetic info – widely varied tissues → expression of epigenome.

- Cellular differentiation during development relies strongly on epigenetic vs genetic inheritance. Expression of epigenome in brain development also affected by behaviour and experience.

- Epigenetic processes likely underlie the toxicity of many environmental exposures of concern.
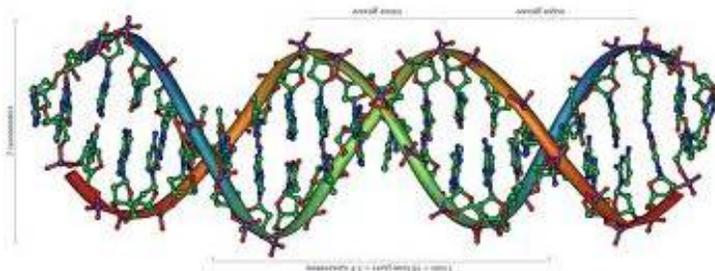
The Causal Puzzle

Recap: Complex and uncertain science

about env'l risks for many reasons

☐   Complex biochemical influences on multiple, inter-related developing systems

☐   Considering the entire life course

☐   Low level, chronic exposure to multiple substances, through diverse media, changing over time and location

☐   Impossible and unethical to run controlled experiments to fully understand env'l risks

Causal webs vs linear systems

☐   Can have many-to-many relationships between variables or complex inter-relationships where "confounders" (e.g., social circumstances) can actually be co-causal factors.

Bradford Hill Criteria – problems describing or evaluating multi-causal, complex and dynamic processes

BH Criteria: Strength, consistency, specificity, temporality of the association, biological gradient, reversibility, plausibility and analogy

Examples in the Environmental Health Literature of:

- **bidirectional or reciprocal relationships** (challenges *temporality*)

- **Complex inter-relationships** – removal of "confounders" can remove co-causal factors and weaken and/or misrepresent true associations (challenges *strength*)

- **Complex interrelationships** and **many-to-many relationships** (can create sufficient variability to challenge criteria of *consistency* and *specificity*)

- Where **low dose** and/or **developmental exposure** more important than high dose (can challenge criterion of *biological gradient*)

- Hence, **less weight to criteria generally considered as most important** (strength, consistency, specificity, temporality, and biological gradient)

- **Seek greater weight for analogy and plausibility** – Bradford Hill used fetal e.g.s (thalidomide and rubella virus) to illustrate analogy: evidence to support exercising caution in analogous situations where have less evidence.

- **Combining analogy with biological plausibility**: ability to address large number of environmental contaminants. Use strong evidence from small number of substances as relevant to those less studied (while recognizing need for caution).

Type II Errors Frequent in Environmental Case Studies

Type I – concluding associations exist when not the case

Type II – missing causal associations

Lead – six flaws in study design contributing to Type II errors (Needleman and Bellinger, 1991)

Review of multiple env'l health studies – systematic bias towards Type II errors (Grandjean, 2005)

European Environment Agency, 2001 – multiple examples of Type II errors in environmental case studies (*Late Lessons from Early Warnings* report)

Challenges Arise in How Risk Factors are Defined

- Review of chronic disease evidence via discussion of multiple risk factors and evidence type and strength

- Traditional public health definition of a risk factor:

*an aspect of personal behavior or lifestyle, an environmental exposure, or an inborn or inherited characteristic, that, **on the basis of epidemiologic evidence**, is known to be associated with health-related condition(s) considered important to prevent (emphasis added).*

(Govt of Canada, Chronic Disease Risk Factor Atlas)

- Doesn't sufficiently recognize primary influence of SDOH

- Epidemiological evidence for environmental problems – very difficult; once obtained, can be far too late to achieve prevention or even clean-up in some cases

Disease-focused sections: approach

- Aimed for similar treatment in each

- Prevalence data, and where relevant, trends re incidence

- Risk factors as described by authoritative sources

- Watching for whether and how environmental exposures considered

- All of above as necessary context to describing evidence for environmental exposures as risk factors
  - In adults, brief summary and, again, watching for relevance to early exposure evidence
  - Early exposures – <u>main focus</u>

Cardiovascular Disease (CVD)

- Non-modifiable RF: gender, age, genetic predisposition affecting blood lipids, blood pressure, obesity, insulin resistance and diabetes
- Modifiable RF: considered to account for 90% of risk.
    - abnormal lipids, hypertension, abdominal obesity, diet, smoking, type 2 diabetes, physical inactivity, stress, etc.
- Closer look at psychosocial stress finds stronger influence. If causal, indicates much greater role for stress in CVD than commonly recognized.

"greater stress and corresponding greater risk of CVD when life circumstances were perceived to be beyond personal control"

- Findings support SDOH analysis that "modifiable" RF are inseparable from living conditions; also apparent in higher prevalence of CVD among those in poverty

CVD – Fetal Nutrition and DOHaD concept

- DOHaD - extensive evidence of associations between fetal and neonatal under-nutrition and major risk factors (hypertension, insulin resistance and obesity) for cardiovascular disease, diabetes and the metabolic syndrome in adult life.

    - Can be either under-nutrition/over-nutrition (common aspect appears to be insufficient protein)

- Permanent developmental changes (via epigenetic mechanisms) contributing to later pathologies

- Prenatal maternal stress – similar impacts (e.g., reduced nephron formation in fetal kidneys)

- Not implying that all CVD developmental in origin but findings are highly relevant for CVD risk esp. for those living in poverty

CVD and Environmental Exposures

Adults:

☐ AHA and CMA note causal relationship with particulate air pollution and CVD.

☐ Strong evidence for low-level lead exposure (lifelong exposure?) and emerging evidence of associations with bisphenol A (altered insulin signaling)

Early Life Exposures:

☐ Three areas to consider:

    ▪ cardiac birth defects

    ▪ low birth weight, and

    ▪ endocrine disruption

CVD and Early Life Exposures

- cardiac birth defects
  - Air pollution, organic solvents, chlorophenoxy herbicides, halogenated hydrocarbons (DCE and TCE), ionizing radiation, lead, benzene, sulphur dioxide and ETS

- low birth weight (and other impacts on fetal growth)
  - <u>Air pollution, ETS, lead</u>, mercury, organochlorine and organophosphate pesticides, nitrates in drinking water, arsenic, phthalates, brominated flame retardants, and polyfluorinated compounds
  - It may not follow that exposures associated with LBW also contribute to latent CVD but prudent concern warranted

CVD and Early Life Exposures: Endocrine Disruption

Bisphenol A, phthalates, PCBs and lead:

- Adult evidence indicates BPA disrupts insulin signalling. Likewise, prenatal and perinatal exposure associated with permanent alteration of insulin metabolism. Thus, potential risk factor for obesity, metabolic syndrome and sequelae (CVD, cancer, diabetes and Alzheimer's disease)

- Prenatal BPA assoc'd with PCOS in adult women (RF for CVD and T2D)

- Pre- and perinatal exp. to BPA, phthalates and adult exp. to PCBs assoc'd with lower testosterone in men (RF for metabolic syndrome, CVD, and T2D)

- Lead also linked to endocrine disruption via action on the neuroendocrine system involvement in stress responses

Diabetes (Type 2)

- Looked at prevalence and RF for both obesity and diabetes
- Clear indication that is disease of the poor (4x greater at lowest vs highest income bracket; 3 to 5X higher among First Nations than gen'l pop'n)

Risk factors for obesity:
- Excess food intake and insufficient physical activity important RF (context of sedentary lifestyles, changes in built env't, food system and food composition)
- Several other RF equally plausible including:
  - exposure to endocrine disrupting chemicals, intrauterine environment and transgenerational factors, sleep debt, stress, social determinants of health (among others)

Diabetes

Overlapping Risk Factors for Obesity, Metabolic Syndrome, Diabetes and CVD:

- Similar genetic risk factors (as noted for CVD)
- Obesity and diabetes are themselves risk factors for other chronic diseases including:
    - certain cancers, Alzheimer's disease, cognitive impairment, dementia, and CVD
- Hence, RF noted for CVD as well as the DOHaD evidence also relevant to diabetes

Diabetes and Env'l Exposures

Exposure in Adults:

- Air pollution (systemic inflammation and oxidative stress – RF for elevated blood pressure, insulin resistance, and abnormal blood lipids)

- Lead (hypertension)

- Bisphenol A (altered insulin signaling – animal data and limited epi. studies)

- Phthalates (limited epi. evidence of links to measures of obesity and insulin resistance)

- Organophosphate pesticides (limited epi., some animal data of links to altered insulin signaling)

- POPs (organochlorine pesticides, dioxins, PCBs) – epi. and animal data re altered insulin signaling, elevated rates of T2D

Diabetes and Early Env'l Exposures

- Epi. evidence prompted much animal research – seeing disruption of metabolic homeostasis through endocrine pathways with greater risk when exposure occurs in utero or perinatally

- Progression of DOHaD research – greater understanding of epigenetic mechanisms underlying obesity, metabolic syndrome, etc

- Fetal under-nutrition and over-nutrition both relevant – former resulting in later life obesity, etc. whereas latter resulting in neuroendocrine responses that program fat cell development and appetite regulation

  - Implications of latter: childhood and adolescent obesity and thus ability to lead to intergenerational obesity

Diabetes and Early Env'l Exposures

- Low birth weight and endocrine disruption again relevant (hence, repeat of exposures noted for CVD)
- Endocrine disruptors as obesogens
    - Adipose/fat tissue is not inert fat storage but active endocrine tissue dynamically regulating energy expenditure, appetite, food intake and metabolism.
    - Also significantly involved in growth and development with endocrine signaling pathways permanently established during perinatal development.
    - Supported by DOHaD evidence – obesogens act during same prenatal and perinatal periods via the same epigenetic mechanisms
- Suspected obesogens: DES, bisphenol A, phthalates, organotins, PBDEs, polyfluoroalkyl chemicals, and POPs like organochlorine pesticides and PCBs.

Alzheimer's Disease

Non-modifiable RF – advancing age, gender and genetics

"Modifiable" – continuum of common RF for obesity, metabolic syndrome, T2D, CVD, vascular dementia and AD.

- Hence, the same biomedical and behavioural risk factors noted for CVD as well as the additional risk factors noted for obesity and diabetes, all relevant to AD.

- Notable importance: those that contribute to inflammation and oxidative stress and thence to disrupted insulin signaling

- Some researchers call AD "diabetes of the brain"

- Poverty, stress, limited physical activity and poor nutrition. Again, concern is upregulation of inflammation and oxidative stress.

Environmental Exposures and Alzheimer's disease

- Continuum of other common RF for CVD, obesity, and T2D – thus, env'l exposures may also be relevant for AD
- Adult exposures with evidence of assoc with AD:
  - Lead (could be via lifetime exposure), air pollution, PCBs and other POPs
- Early exposures
  - Continuum of shared risk factors – thus, may also be relevant for AD
  - DOHaD concept and epigenetic mechanisms are also involved (healthy brain aging results from a lifelong continuum beginning with healthy brain development, including via epigenetic influences, and creation of brain reserve.)
- Early life exposures with more direct evidence of possible associations: air pollution and lead

Cancer

- Not one disease but many with diverse risk factors for each

- Again, "big three" RF plus SDOH – major contributions

- Evolving perspectives on environmental factors, gene-env't interactions, and on theories of carcinogenesis itself

- Key role of epigenetics:

  - Epigenetic mechanisms and events are increasingly seen as central to the understanding of how most cancers develop and progress.

  - This knowledge also indicates epigenetic mechanisms as centrally involved in early life events that can lead to later life cancer.

Cancer

- Focus on three cancers (breast, prostate and testicular) with high prevalence, rising incidence and where is evidence of environmental contribution, particularly early exposures

- Two overall mechanisms apparent: direct but delayed causation and increased sensitivity to later exposures

- Very limited review of adult evidence; rather summarized the detailed evidence about greater cancer susceptibility when exposures occur in early life

Early Life Exposures and Breast Cancer

☐ Strongest evidence from suspected endocrine disrupting properties of dioxins, PCBs, and most of the persistent organochlorine pesticides such as DDT, its metabolite DDE, as well as dieldrin, aldrin, heptachlor and chlordane.

☐ Other xenoestrogenic substances, like Bisphenol A, are implicated, on the basis of animal studies.

☐ More limited evidence: alkylphenols, several metals, phthalates, parabens, UV filter components of sunscreens and the food additives bovine somatotropin (rBST) and zeranol.

☐ Postulated mechanism: permanent epigenetic changes (during mammary gland development in utero) that alter later susceptibility, often before and during puberty, to other factors that can initiate breast cancer.

Early Life Exposures and Prostate Cancer

Adults:

☐ Occup. exp. indicates increased prostate cancer risk for some pesticides, PCBs, arsenic and cadmium. Endocrine disruption mechanisms suspected. (Hormonal disruption suspected in prostate cancer RF more generally)

Early life exposure:

☐ Endocrine-disrupting modes of action from the synthetic hormone DES and bisphenol A.

☐ Likely mechanism: Again, permanent epigenetic changes (during prostate gland development in utero) that alter later susceptibility to prostate cancer.

☐ Also similar to BPA breast cancer evidence - low-dose studies seeing biphasic or U-shaped dose-response curve

Early Life Exposures, Testicular Dysgenesis Syndrome, and Testicular Cancer

☐ TDS: unifying hypothesis for common fetal origin of four effects on the male reproductive system

- ◻ birth defects cryptorchidism (undescended testicles) and hypospadias (defect in male urinary tract)
- ◻ poor semen quality
- ◻ later development of testicular cancer.

☐ Multiple animal studies demonstrate these effects from endocrine disrupting chemicals. Strong evidence that testicular cancer is initiated during fetal development.

☐ Some pesticides, certain phthalates, perfluorochemicals and bisphenol A may all disrupt fetal testes development and are implicated in the development of TDS.

☐ Analogy important here given emerging evidence for multiple substances and indication of mixture effects

Asthma

- Host, genetic, and environmental risk factors for asthma with multiple interactions.
- E.g., genetic susceptibility for asthma or allergies onset can be influenced by multiple gene-environment and gene-gene interactions, as well as epigenetic mechanisms.
- Immune system development and lifelong lung function determined in utero and during early life.
- DOHaD evidence again relevant: Lifelong lung function also affected by low birth weight, undernutrition and other factors.

Environmental Risk Factors for Asthma

☐ Multiple environmental risk factors for asthma onset/triggers: biological allergens, childhood viral infections, environmental tobacco smoke, multiple indoor and outdoor pollutants, socioeconomic status and stress, as well as nutritional factors, gut colonization (which influence immune system development), and obesity.

- ◻ Outdoor air pollutants, such as Criteria Air contaminants and many PAHs, acid vapours and aerosols, and diesel exhaust are associated with asthma onset and are triggers of asthma attacks.

- ◻ Indoor air pollutants (some overlap): products of combustion such as NO2 and CO, formaldehyde, and numerous volatile organic compounds (VOCs) arising from consumer products such as cleaning agents, laundry, and personal care products.

Environmental Risk Factors for Asthma

☐ Evidence of associations between preterm birth and air pollution, which in turn affects lung development. (additional substances associated with preterm birth or LBW thus also of concern for asthma)

☐ Air pollutants also impact the immune system. Immune response in asthma onset and as asthma trigger.

☐ Phthalates and bisphenol A - heighten lung inflammation

☐ Perfluorinated compounds - limited evidence of immunotoxicity

Table 1: Summary of Early Environmental Exposures Suspected or Associated with Chronic Diseases, Conditions or Risk Factors
(*NOTE: important detail about strength of evidence is lost with this aggregation – see specific sections of report for more detail*)

Cardiovascular disease (CVD)	• Lead, smoking, particulate air pollution, • Substances associated with cardiac birth defects • Substances associated with low birth weight • Endocrine disrupting substances affecting insulin signalling (BPA and phthalates), adult dev'mt of polycystic ovarian syndrome (BPA and other EDCs), lower testosterone levels in adults (BPA, phthalates, PCBs), and dysfunction in HPA-axis and stress response (lead).
Cardiac Birth Defects	• Ambient air pollution (specifically carbon monoxide and ozone), organic solvents in dyes, lacquers and paints (specifically halogenated hydrocarbons including trichloroethylene and dichloroethylene), chlorophenoxy herbicides, trihalomethanes, additional pesticides, ionizing radiation, lead, benzene, sulphur dioxide, ETS.
Low Birth Weight	• Air pollution – CACs (particularly sulphur dioxide and particulates), maternal smoking, ETS, PAHs, lead, mercury, arsenic, OC and OP pesticides, nitrates in drinking water, phthalates, BFRs, polyfluorinated compounds.
Obesogens	• Endocrine disrupting substances suspected as obesogenic (BPA, phthalates, organotins, PBDEs, polyfluoroalkyl compounds, OC pesticides, PCBs) • Human adenovirus 36, phytoestrogens, glycyrrhetinic acid (sweetener)
Type 2 Diabetes	• Prudent to assume that known continuum of common risk factors across CVD, metabolic syndrome and diabetes likely extends to early environmental exposures • Substances associated with low birth weight • Endocrine disrupting substances relevant to CVD • Suspected obesogens

Table 1 _continued_: Summary of Early Environmental Exposures Suspected or Associated with Chronic Diseases, Conditions or Risk Factors
(_NOTE: important detail about strength of evidence is lost with this aggregation – see specific sections of report for more detail_)

Alzheimer's disease	• Prudent to assume that known continuum of common risk factors across CVD, metabolic syndrome, diabetes and Alzheimer's disease likely extends to early environmental exposures • Air pollution, lead
Parkinson's Disease	• If link to obesity confirmed, prudent to consider obesogens as environmental risk factors • Air pollution, certain pesticides (maneb and paraquat, OC pesticides)
Developmental Neurotoxicity	• Lead, mercury, arsenic, manganese, organic solvents, OP and OC pesticides, PAHs, ETS, PCBs, phthalates, BPA, dibutyltin, PBDEs, triclosan, artificial food colours and additives.
Cancer •Breast •Prostate •Testicular •Other Cancers	• _Breast cancer_: ionizing radiation, benzene and organic solvents, 1.3-butadiene, aromatic amines, BPA, phthalates, parabens, alkylphenols, PAHs, OC and triazine pesticides, PBDEs and other POPs, metals, tobacco and ETS, vinyl chloride, ethylene oxide. (See also Table 6.) • _Prostate cancer_: Synthetic hormones in food production, BPA • _Testicular cancer_: Maternal exposure to several POPs • _Testicular Dysgenesis Syndrome_: EDCs with anti-androgenic action (phthalates, some pesticides, BPA) • _Other Cancers_: particulate air pollution, radon, multiple pesticides, chlorination byproducts, cadmium, aromatic amines, PAHs, diesel exhaust, smoking and ETS, dioxin, ionizing radiation, vinyl chloride, some paints and solvents, cell phone use (see also Table 5)
Respiratory disease (asthma)	• Substances associated with low birth weight • Smoking and ETS, aeroallergens, indoor and outdoor air pollution including all the CACs (ozone, CO, PM_{10} and $PM_{2.5}$, nitrogen dioxide, sulphur dioxide, many different VOCs), multiple hazardous air pollutants (PAHs, aldehydes, acide vapours and aerosols, diesel exhaust), formaldehyde, VOCs, phthalates, aldehydes, isocyanates, anhydrides, cadmium, hexavalent chromium, manganese, nickel, benzene, dibutyl phthalate, dioxins, PCBs, metals (esp. lead), some pesticides, BPA, perfluorinated compounds

Overall Observations

- Increasing evidence that early environmental exposures are associated with certain chronic diseases.

- Numerous knowledge gaps and uncertainties remain. Yet, exposures continue with high stakes risks.

- Continuum of shared well-known risk factors across obesity, metabolic syndrome, CVD, T2D and AD points to likelihood that environmental risk factors are also shared, including for other early life risks such as low birth weight.

- Chemicals/pollutants most frequently implicated include air pollutants (mainly the CACs but also other toxic air pollutants and ETS) metals, especially lead, bisphenol A and phthalates, pesticides, solvents and persistent organic pollutants (PBDEs, PCBs, OC pesticides).

CPCHE-OCDPA Areas of Common Ground

Shared goals – health promotion, disease prevention

Four overall conclusions about our collective chronic disease prevention work

- ☐ Prevention focus on behavioural/lifestyle risk factors is too narrow

- ☐ The evidence challenges how evidence is typically evaluated

- ☐ Developmental origins concept – solid evidence and rapidly expanding to include early life exposures

- ☐ Low birth weight, nutrition issues (esp. evidence of impacts on insulin signalling): of key impt'ce and overlap between CPCHE and OCDPA

Solid foundation built for further collaborative work on chronic disease prevention

Acknowledgments and Thanks:

- To CELA, for allowing me to spend two years on this report
- To CPCHE and OCDPA for the opportunity to write it
- To CPCHE-OCDPA project advisory committee
- The Trillium Foundation and the Ontario Public Health Association
- Randee Holmes and the MaRS Centre for organizing videoconference
- To several key people for ongoing peer review, collaboration and advice:
  - Lynn Marshall, Environmental Health Institute of Canada, Ontario College of Family Physicians / CPCHE
  - Erica Phipps, CPCHE
  - Brian Stocks, Ontario Lung Association / OCDPA
  - Franca Ursitti, Ontario Public Health Association / CPCHE
  - Loren Vanderlinden, Toronto Public Health / CPCHE
  - Bruce Lanphear, Simon Fraser University
  - Robert Dales, University of Ottawa

- Full report posted to www.cela.ca and www.healthyenvironmentforkids.ca