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Environmental Justice and the Health of Children

Current regulation of air quality in Ontario fails to protect children from **synergistic air pollution**, which is the amplification of toxic health effects from mutually interacting air pollutants in the body.

For young girls, combined exposure to different contaminants can disrupt hormonal signalling and trigger **precocious or early puberty**, leading to the harmful premature onset of pubertal development.



Image credit: @Ahmed-adobe.stock.com

The Regulatory Blindspot

- Ontario's provincial regulations of air quality measure the chemicals on the right individually, following the **single pollutant approach** to air assessment.
- Yet, children breathe in contaminants all at the same time. This combined exposure to pollution creates a toxic mix that causes severe, amplified harms our environmental laws do not account for.
- The fair treatment and proactive protection of **at-risk populations** like children requires regulatory frameworks that measure the reality of the air we breathe, rather than exclusively prioritizing economic feasibility over human health.
- Ontario must abandon the single pollutant approach and adopt risk assessment methodologies that measure aggregate, long-term, and synergistic health impacts of air pollution.

Key Air Contaminants:

- **Fine Particulate Matter** ($PM_{2.5}$): a complex mixture of suspended microscopic particles which cause systemic inflammation linked to cardiovascular diseases, respiratory diseases, and infant mortality . $PM_{2.5}$ can carry endocrine disrupting chemicals and heavy metals deep into the lungs and bloodstream.
- **Benzene** (C_6H_6): a volatile organic compound and known carcinogen linked to blood cancers, immune-system degradation, brain damage, and inheritable epigenetic alterations to reproductive health.
- **Nitrogen Dioxide** (NO_2): a respiratory irritant which inflames lung membranes. NO_2 is significantly associated with lung cancer in women via activation of estrogen receptors, and.
- **Ground-level Ozone** (O_3): a corrosive gas that functions like a chemical sunburn inside the lungs. O_3 alters hormone secretion and increases risks of diabetes, menstrual dysregulation, and infertility.

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Reports

The Risk of Particulates to Children



Image credit: @spiritofamerica-adobe.stock.com

A study from Nanjing, China conducted in 2023 analyzed over 2,201 children and found that short-term exposure to high concentrations of fine particulate matter significantly increases risks of precocious puberty. Young girls made up over 96% of diagnosed cases. The oxidative stress caused by fine particulate matter can prematurely trigger release of pubertal hormonal signalling, emphasizing the acute developmental sensitivity of young girls to ambient air pollution.

Synergistic Toxicity and Early Menarche



Image credit: @Семен Саливанчук-adobe.stock.com

A study in Poland from 2022 assessed women's childhood residential exposure to a complex air pollution index comprised of particulate matter, fine particulate matter, benzene, sulphur dioxide, and nitric oxide. Researchers found that high levels of PM_{10} and $PM_{2.5}$ more than tripled the likelihood of a girl experiencing their first menstrual cycle (called menarche) before age 11. The average age of menarche also decreased as the complex air pollution index increased.

The Threat of Proximity to Heavy Traffic



Image credit: @brocreative-adobe.stock.com

A 2016 longitudinal American study revealed that pre-pubertal girls living 150m downwind of a major road reached pubertal milestones several months earlier than girls living in low-traffic areas. This illustrates the developmental burden placed on young girls living in more heavily polluted communities, where concentrated vehicular emissions act as localized endocrine disruptors.

Global Trends in Endocrine Disruption



Image credit: @yos_moes-adobe.stock.com

This South Korean systematic review from 2024 consolidated global studies on health and disease, confirming that exposure to ambient air pollution accelerates pubertal development and lowers the age of menarche. Endocrine-disrupting chemicals and heavy metals bound to fine particulate matter are directly implicated in estrogen receptor activation. The 'toxic cocktail' of different pollutants in our air function as bio-chemical catalysts for premature puberty in young girls worldwide.

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Developmental Vulnerabilities by the Numbers

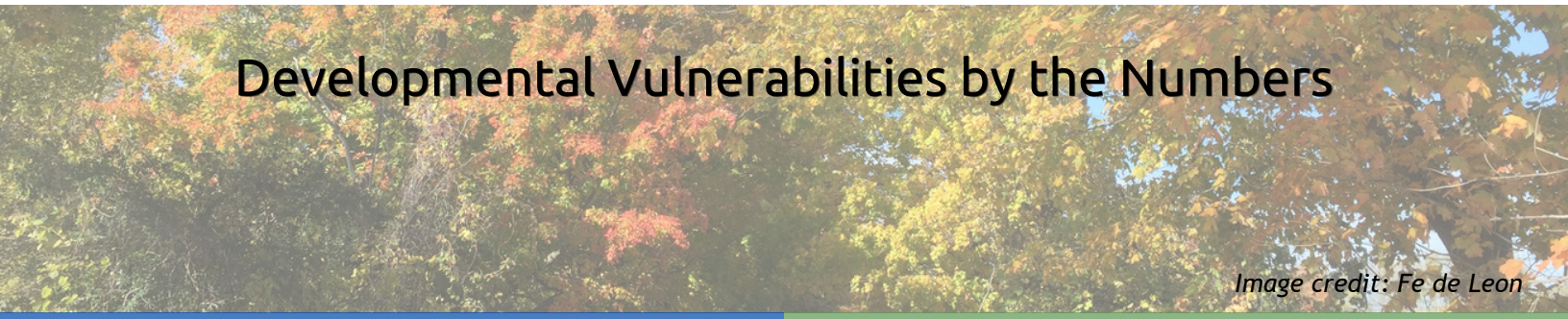

Image credit: Fe de Leon

The Gender Disparity

96.6%



Disproportionate impact: the percentage of precocious puberty cases overwhelmingly occurred in young girls (2,127 girls compared to 74 boys) in the Nanjing ambient air pollution study.

The Age of Onset

7.47 Years



The alarmingly low average age of children diagnosed with with precocious puberty linked to fine particulate matter exposure.

The Accelerated Timeline

-0.78 Years



Young girls living in high pollution areas reached menarche earlier than those living in low-exposure zones. This exemplifies developmental acceleration caused by synergistic air pollution.

The Proximity Threat

150 Metres



Living within this distance of a major road or higher is a primary predictor for some of the earliest signs of pubertal development. Young girls living in high-traffic areas reach pubertal milestones several months faster than their peers.

Notes:

- Existing studies are on children assigned female at birth. Throughout this fact sheet, girls generally refers to cis-gender girls unless otherwise noted in the source material.
- While precocious puberty is more common in girls, it is not exclusive to them.
- There is evidence that pollutants may be increasing precocious puberty, and while most research is on menarche, there is some evidence it may effect children of any sex/gender.

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Problems and Recommendations

The Core Problems

- **The Single Pollutant Approach:** provincial regulations measure and manage industrial pollution one chemical at a time. This isolates pollutants on paper, entirely ignoring the synergistic, amplifying toxic effects that occur when these chemicals mix in the ambient air.
- **The Single-Source Problem:** relying on generalized data from sparsely distributed air monitoring stations provides only a snapshot of air composition. It fails to capture the fluctuating, transboundary reality of air pollution.
- **Exemptions over Health:** site-specific standards, technical standards, and abatement plans function as state-sanctioned permissions to pollute.

The Path Forward

- **Implement Hazard Indexing (HI):** shift to a cumulative risk assessment framework that sums the individual hazard quotients of multiple chemicals to better evaluate aggregated risks to public health from air pollution.
- **Utilize PBPK Modelling:** adopt physiologically based pharmacokinetic (PBPK) modelling to actively simulate how mixtures of chemicals are absorbed and metabolized by at-risk populations, rather than solely relying on adult male clinical data.
- **Fund Community-level Biomonitoring:** build long-term databases of biomarkers for sensitive populations like children, pregnant women, and occupation workers.