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Pediatric Endocrine-Disrupting Chemical Exposure Produces Irreversible Organizational Injury: Biomarker Evidence Necessitating a Precautionary Public Health Framework

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ABSTRACT

Endocrine-disrupting chemicals (EDCs) possess abilities to interfere with endogenous hormone action, particularly during the sensitive windows of early development. Consequently linked to a spectrum of pediatric disease, including autism spectrum disorder, ADHD, precocious puberty, obesity, thyroid dysfunction, and atopic disease, EDCs now affect hundreds of millions of children and foretell a growing public health crisis. These outcomes are mechanistically rooted in pediatric-specific toxicokinetic and organizational vulnerabilities. Children accumulate disproportionately higher internal EDC due to elevated intake across multiple routes such as putting objects in their mouths, greater intestinal and dermal bioavailability, and immature detoxification systems that significantly extend the internal half-lives of chemicals, exemplified by a hepatic CYP2E1 expression 12,271-fold lower than that of adults and extending chemical half-lives three to ninefold. While adult responses to EDCs are often transient and activational, early-life EDC exposures are organizational, fundamentally altering physiological architecture through epigenetic reprogramming. Mechanisms include aberrant DNA methylation at imprinted loci, histone modification via MLL1-driven H3K4me3 enrichment, and breach of PI3K/AKT and MAPK/ERK phosphorylation thresholds, producing latent hits manifesting at later hormonal surges. Clinically, disruption of the hypothalamic-pituitary-thyroid axis reduces offspring IQ and elevates ADHD and ASD risk, while PPARγ activation and adipokine dysregulation establish an obesogenic phenotype, and neonatal Th2 immune bias drives selective Th1 apoptosis. This paper synthesizes these biomarkers of pediatric susceptibility to inform tailored biomonitoring strategies, incorporating hair, meconium, and cord

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blood matrices, and argues for adoption of the precautionary principle in pediatric environmental health policy.

Keywords: *endocrine-disrupting chemicals (EDCs), pediatric exposure, toxicokinetics, developmental susceptibility, developmental toxicology, prenatal programming, epigenetics, early-life exposure, mechanistic biomarkers, clinical biomarkers, environmental health, metabolic programming*

TOXICOKINETICS AND MATRIX-SPECIFIC BIOMARKERS OF EXPOSURE

Endocrine-disrupting chemicals (EDCs) interfere with hormone action, specifically threatening children through high physiological intake and immature detoxification (Fudvoye et al., 2014). Accurately assessing these risks requires tailoring biological monitoring to a chemical's relative persistence within the developing body (Fudvoye et al., 2014). Children face disproportionate exposure to EDCs due to fundamental physiological and behavioral differences from adults including the relatively higher physiological intake of materials such as water, food, and air (Di Pietro et al., 2023). Infants and toddlers consume more food and water per unit of body surface area (BSA) and body mass than adults, and possess higher ventilation rates (Di Pietro et al., 2023). As these substances all can contain EDCs, children experience a higher internal accumulation of EDCs than do adults in the same environment; specifically around 6 months, there are twofold air intake per body weight, sevenfold more water intake per body surface area, and three to fourfold more caloric intake per body surface area (Ginsberg et al., 2004). Similarly, another difference lies between children and adults' intestinal and dermal bioavailability (Braun, 2017). Pediatric populations exhibit higher intestinal absorption in the stratum corneum, possess a more permeable and thinner skin layer, and have a larger BSA to body mass ratio than do adults (Braun, 2017). Thus, they are subject to higher permeation of EDCs found in textiles or personal care products, currently irreplaceable resources in daily lives (Fernandez et al., 2011). Lastly, children and adults have very distinct behavioral pathways. Children's developmentally appropriate behaviors, including hand-to-mouth and object-to-mouth activities, contribute to a significantly increased ingestion of toxicants (Braun, 2017). These adolescent traits turn into a primary route of exposure for EDCs because modern indoor environments contain accumulated EDCs such as phthalates and flame retardants in household dust and on plastic surfaces (Ghassabian et al., 2022).

Earlier still in development, the primary biological factor underlying fetal and neonatal vulnerability is the deficient expression of the cytochrome P450 (CYP) enzyme system, which functions as a core mechanism for chemical biotransformation (Robinson et al., 2020). During the second trimester, expression of CYP2E1 in the fetal liver is 12,271 times lower than that observed in adults, while CYP1A2 and CYP2B6 levels are reduced

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by factors of 1,528 and 1,224, respectively (Robinson et al., 2020). Because these enzymes are responsible for metabolizing volatile hydrocarbons and herbicides, their markedly reduced presence limits the capacity for effective detoxification early in life (Robinson et al., 2020). As a consequence of this enzymatic immaturity, clearance and elimination processes are substantially delayed in pediatric populations (ATSDR, 2023). With metabolic systems not yet fully developed, the body's ability to deactivate and remove EDCs is significantly compromised, resulting in chemical half-lives that are three to nine times longer in infants than in adults depending on the type of EDCs (Ghassabian et al., 2022). This delay in elimination produces elevated and prolonged circulating concentrations following exposure, the susceptibility further intensified by systemic immaturity beyond hepatic metabolism: infants possess underdeveloped renal systems characterized by lower glomerular filtration rates, which restrict the excretion of unmetabolized EDCs (Singh et al., 2021). In the absence of fully functional enzymatic and excretory defenses, an exposure level considered a "low dose" in adults can therefore constitute a highly toxic internal burden in children (Robinson et al., 2020).

Some EDCs possess short biological half-lives, such as BPA with up to six hours and phthalates with up to 24 hours, and currently for measuring those non-persistent EDCs, urine is considered the gold standard (Braun, 2017). However, because these EDCs are metabolized rapidly, urine samples with short term measurements representing a singular time period may miss chronic exposure. Accordingly, multiple serial samples capturing episodic dietary or personal care product usage are required for accurate assessment (Braun, 2017). Conversely, some EDCs are persistent organic pollutants (POPs) that bioaccumulate in human tissues for years. Examples of POPs are PFAS and PBDEs, possessing long half-lives ranging from 3.8 to 7.3 years (Di Pietro et al., 2023). These chemicals are not easily metabolized and thus can be sufficiently characterized with single samples of persistent reservoirs, such as serum or plasma concentration (Braun, 2017). While urine captures a 24-hour window, hair analysis is emerging as a superior matrix for longitudinal studies because a 6-cm sample can integrate exposure data over six months (Di Pietro et al., 2023). Additionally, biomarkers of fetal exposure are increasingly drawn from meconium and umbilical cord blood, which often reveal higher chemical concentrations in the fetus than in the mother due to transplacental transfer and limited fetal metabolic clearance (Vaiserman, 2014).

In summary, the synergy of high intake and enzymatic immaturity transforms environmental exposures into permanent organizational reprogramming (Di Pietro et al., 2023). Unlike reversible adult shifts, these disruptions fundamentally rewrite physiological blueprints during critical windows (Birnbaum & Miller, 2015). Tailored biomonitoring is required to properly capture the influence of EDCs in consideration of children's unique and vulnerable developmental trajectory (Di Pietro et al., 2023).

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EPIGENETIC AND MECHANISTIC BIOMARKERS UNDERLYING THE ORGANIZATIONAL THRESHOLD

EDCs act as false developmental morphogens, converting transient exposures into permanent modifications by remodeling the system's foundational architecture (Sultan et al., 2025). Mechanistic biomarkers demonstrate how these substances disrupt DNA methylation and the "histone code" to create lifelong gene expression trajectories while breaching critical phosphorylation thresholds that separate healthy signaling from sustained toxicity (Walker, 2016).

DNA methylation in humans, generally the silencing of a gene without altering the DNA sequence, involves adding a methyl group to the 5-carbon of cytosine, typically at CpG islands within gene promoters (Vaiserman, 2014). EDCs may induce hypomethylation, a reduction or loss of methyl groups in regions supposed to be methylated, rendering genes inappropriately accessible to transcriptional machinery (Vaiserman, 2014). For instance, in a viable yellow Agouti (Avy) mouse, prenatal BPA exposure induced hypomethylation of the Agouti gene, which resulted in a yellow coat color and a predisposition to obesity and diabetes in adulthood (Walker, 2016). Still, such deleterious effects can be abolished through maternal dietary supplementation with methyl donors; research indicates that reasonable doses of methyl donors include 4.3mg/kg diet for folic acid, 0.53mg/kg diet for Vitamin B12, 5g/kg diet for betaine, and 7.97g/kg diet for choline chloride (Dolinoy et al., 2007).

EDCs such as PI3K/AKT function as estrogen receptor (ER) ligands and can activate rapid cytoplasmic signaling cascades (Walker, 2016). Those cascades phosphorylate and modulate the activity of epigenetic writing enzymes like histone methyltransferases (HMTs) (Walker, 2016). Neonatal exposure to BPA activates the HMT MLL1, leading to enrichment of the active Histone H3 Lysine 4 trimethylation (H3K4me3) mark, capable of enabling active transcription, at transcription start sites of genes associated with prostate cancer (Walker, 2016). These modifications often result in “latent hits”: reprogramming events where basal gene expression remains unchanged but the same genes show an exaggerated and dysfunctional response to secondary hormonal surges, typically during puberty or adult metabolic stress (Walker, 2016).

The balance between phosphate-adding kinases and phosphate-removing phosphatases acts as a critical boundary between healthy physiological states and toxicity. EDCs possess the capacity to alter signal transduction, including the abundance of post-translational modifications and the activity of associated enzymes (La Merrill et al., 2020). Non-traditional dose-responses, such as non-monotonic (U-shaped) curves, suggest that very low concentrations of EDCs can disrupt these signaling pathways through the loss of negative feedback mechanisms, potentially leading to excessive or toxic cellular signals that differ fundamentally from high-dose effects (Schug et al., 2011). EDCs act as "metabolism-disrupting chemicals" that behave as

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diabetogenic substances by interfering with insulin sensitivity (Singh et al., 2021). Proinflammatory signaling pathways triggered by EDCs increase the serine kinase phosphorylation of insulin receptor substrates (IRS-1 or IRS-2), which prevents proper downstream signaling and induces insulin resistance (Bansal et al., 2018). This impaired signaling cascade blocks GLUT4 translocation, thereby reducing glucose uptake in peripheral tissues and predisposing the individual to type 2 diabetes (Di Pietro et al., 2023). Signal transduction pathways often involve proteins with multiple phosphorylation sites that exhibit threshold behavior where cells make all-or-none decisions regarding proliferation, differentiation, or apoptosis (Schug et al., 2011). EDCs can force these thresholds to be crossed prematurely; for example, BPA can activate rapid nongenomic signaling via the PI3K/AKT or MAPK/ERK pathways, which then phosphorylate and modulate the activity of histone methyltransferases like MLL1 (Walker, 2016). These "latent hits" prime genes for an exaggerated, dysfunctional response to later hormonal surges, such as those occurring during puberty, potentially leading to uncontrolled cell growth or cancer later in life (Walker, 2016).

In summary, the transition from activational responses to irreversible, organizational injuries is driven by latent epigenetic hits and breached signaling boundaries (Birnbaum & Miller, 2015). By permanently remodeling epigenetic patterns and disrupting kinase-phosphatase balance, EDCs prime developing tissues for exaggerated pathological responses to later life stressors, establishing a permanent consequence from early-life environmental insults and even indicating the possibility of transgenerational inheritance of adult-onset disease (Vaiserman, 2014).

CLINICAL BIOMARKERS OF DEVELOPMENTAL SUSCEPTIBILITY AND DISEASE ENDPOINTS

The translation of molecular initiatory events into clinical pathology relies on identifying physiological markers signalling structural and functional injury (Ghassabian et al., 2022). This section details how EDC-induced shifts in the thyroid axis compromise neurodevelopment, how altered metabolic set points drive an obesogenic phenotype, and how immune imbalances permanently poise the neonate for chronic allergic disease (Braun, 2017).

The hypothalamic-pituitary-thyroid (HPT) axis provides a suite of sensitive biomarkers for cognitive impairment as thyroid hormones (THs) serve essential roles in brain morphogenesis, regulating progenitor cell proliferation, neuronal migration, and myelination (Fudvoye et al., 2014). Even mild, subclinical shifts in maternal thyroxine (T4) or thyroid-stimulating hormone (TSH) during pregnancy is linked to measurable decrements in offspring IQ and elevated risks for ADHD and autism spectrum disorder (ASD) (Fudvoye et al., 2014). The timing or TH availability serves as a temporal biomarker: gestational T4 reductions correlate with deficits in visual processing and motor skills, whereas postnatal reductions correlate with impairments in

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language, attention, and memory (Braun, 2017). EDCs PBDEs, PCBs, and BPA are known to bind to thyroid hormone receptors or alter hormone synthesis and transport and thus interfere with the HPT axis, demonstrated when maternal perchlorate (EDC) levels were negatively associated with neonatal T4 (Cheng et al., 2023). Consequently, monitoring the HPT axis at different life stages, from second trimester through adolescence, allows detection of "latent hits" on neurobehavioral development before clinical symptoms fully manifest (Cheng et al., 2023).

EDCs function as "obesogens" by permanently altering the neuroendocrine systems that govern appetite, energy expenditure, and adipogenesis (Braun, 2017). These alterations lead to a developmentally adapted phenotype where the organism becomes highly efficient at storing energy, predisposing children to rapid weight gain and obesity when calories are abundant ex utero (Hochberg, 2011). A critical clinical biomarker for this process is the activation of PPARγ, a regulator of adipogenesis capable of diverting mesenchymal stem cells(MSCs) away from bone or muscle lineages and toward the production of fat cells (Schug et al., 2011). Additionally, altered levels of adipokines, specifically hyperleptinemia (elevated leptin) and reduced adiponectin, serve as early indicators of insulin resistance and metabolic syndrome (Bansal et al., 2018). Integrating these adipokine levels with neonatal growth velocity and BMI trajectories allows for identification of individuals and high risk for adult-onset type 2 diabetes and non-alcoholic fatty liver disease (NAFLD) (Ghassabian et al., 2022).

The T helper 1/T helper 2 (Th1/Th2) balance serves as a vital biomarker for immune maturation, as early-life EDC exposure can lock the system into a Th2-biased, hyper-reactive state (Basha et al., 2014). In neonates, the Th2 locus is naturally epigenetically hypomethylated or "poised" for rapid activation to prevent maternal rejection, while the Th1-hallmark IFN-γ gene is silenced through hypermethylation (Zaghouani et al., 2009). EDCs exacerbate this imbalance, inducing the expression of a specific heteroreceptor (IL-4Rα/IL-13Rα1) on Th1 cells that is absent in stable adult immune systems (Debock & Flamand, 2014). Upon secondary challenge, the IL-4 produced by dominant Th2 cells binds to this heteroreceptor and triggers selective apoptosis of Th1 memory cells, effectively deleting the body's Th1-mediated defense against pathogens (Debock & Flamand, 2014). This permanent, organizational immune damage is clinically manifested as life-long asthma, allergic rhinitis, and a heightened susceptibility to infections, with the Th1/Th2 cytokine ratio serving as a definitive marker for this developmental vulnerability (Basha et al., 2014).

In summary, clinical biomarkers allow for detection of organizational injury long before chronic disease endpoints manifest (Birnbaum & Miller, 2015). By identifying specific signatures in the thyroid, metabolic, and immune systems, researchers can predict lifelong trajectories for ADHD, obesity, and asthma (Braun, 2017). These markers demonstrate that early-life environmental insults may shape permanent future disease susceptibility, potentially enabling early treatment (Birnbaum & Miller, 2015).

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EXAMINING HOW TO RESPOND TO EDC'S UNIQUE TOXICOKINETIC AND IMMUNOLOGIC PROPERTIES

Essentially, the fundamental disparity in pediatric health begins with toxicokinetics. As children are highly efficient collectors of environmental toxins due to disproportionate intake of media and immature biotransformation pathways, their vulnerability is incomparable to that of adults (Fudvoye et al., 2014). Additionally, children's markedly lower expression of cytochrome P450 enzymes (such as CYP2E1, which is 12,271 times lower in the fetus) results in internal chemical half-lives that are three to nine times longer than in adults, turning minor exposures into sustained toxic burdens (Zaghouani et al., 2009).

Mechanistically, while adult responses to EDCs are transient, children's responses are organizational: this is because early-life exposures act as "false morphogens" that permanently rewrite the body's structural blueprint (Birnbaum & Miller, 2015). EDCs achieve this through epigenetic reprogramming: altering of DNA methylation and the histone code (Walker, 2016). They also breach phosphorylation thresholds that force cells into irreversible pathological states (Walker, 2016). These alterations function as "latent hits," remaining silent during childhood but priming the individual for exaggerated, dysfunctional responses to future stressors such as the hormonal surges of puberty (Predieri et al., 2022).

The translation of these molecular events results in diverse noncommunicable diseases, including decrements in IQ and neurodevelopment via the thyroid axis, and lifelong phenotypes predisposing to obesity and type 2 diabetes (Braun, 2017). In the immune system, EDCs exploit epigenetic poisoning to cause helper T cell imbalance; specifically, they lock the neonate into a Th2-biased state, leading to selective Th1 cell death and a permanent susceptibility to asthma and allergic disease (Fudvoye et al., 2014).

In measuring EDC influences, evidence directly debunks the myth that "the dose makes the poison": it instead suggests shifting focus toward developmental timing and low-dose mixture effects (Ghassabian et al., 2022). Thus, to protect the unique and vulnerable developmental trajectory of the child, utilizing tailored biomonitoring (hair, cord blood, meconium) and multi-omics integration, future clinical and regulatory strategies must adopt the precautionary principle (Iughetti et al., 2020).

CONCLUSION

In conclusion, endocrine-disrupting chemicals (EDCs) exploit unique pediatric toxicokinetic and organizational vulnerabilities to transform transient environmental exposures into a permanent molecular legacy. Because children fundamentally differ from adults by lacking mature biotransformation systems, with

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fetal hepatic CYP2E1 expression being 12,271 times lower than in adults, chemical half-lives are extended by three to nine times, resulting in highly toxic sustained internal burdens. These toxicants function as "false morphogens" that fundamentally reprogram the body's structural and functional blueprints through stable epigenetic remodeling and the breaching of quantitative phosphorylation thresholds in critical signaling cascades like the PI3K/AKT and MAPK/ERK pathways. Such disruptions create "latent hits" that establish life-long disease trajectories, manifesting later as neurodevelopmental deficits, a metabolically obesity-vulnerable phenotype or a permanent immune Th2-bias driven by selective Th1 apoptosis via the neonatal IL-4Rα/IL-13Rα1 heteroreceptor. Ultimately, the transition from activational adult responses to irreversible pediatric injury requires a paradigm shift toward tailored biological monitoring using longitudinal matrices like hair and cord blood, alongside the prioritization of the precautionary principle in public health policy to protect the child's vulnerable developmental trajectory.

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