Sarcouncil Journal of Medical Sciences

ISSN(Online): 2945-3526

Volume- 04| Issue- 12| 2025

Review Article

Received: 05-11-2025 | Accepted: 02-12-2025 | Published: 17-12-2025

Evaluating the Neurodevelopmental Effects of Per- and Polyfluoroalkyl Substances Exposure in Children: A Review

Ngoye Sedrick Yeih [1], Rosemary Serlorm Ansah [2], Oluwaseyi Emmanuel Olorunfemi [3] and Edward Oware [4]

[1] *Department of Environmental Science, Tarleton State University, Stephenville, TX*
[2] *Marquette University, Milwaukee, WI, USA*
[3] *Department of Environmental Engineering, University of North Dakota, USA*
[4] *Department of Physiology, School of Medical Sciences, Kwame Nkrumah University of Science and Technology, Ghana*

Abstract: Per- and polyfluoroalkyl substances (PFAS) are persistent synthetic chemicals with ubiquitous environmental distribution and measurable concentrations in nearly all populations worldwide. The developing brain represents a particularly vulnerable target for PFAS toxicity due to critical windows of neurodevelopment during prenatal and early postnatal periods. This comprehensive review synthesizes current epidemiological evidence examining associations between early-life PFAS exposure and neurodevelopmental outcomes in children, incorporating findings from major birth cohort studies and cross-sectional investigations. A systematic literature search identified studies examining prenatal and childhood PFAS exposure in relation to cognitive development, behavioral outcomes, and motor function across pediatric populations. The strongest evidence emerges from prospective birth cohort studies demonstrating consistent associations between prenatal PFAS exposure and reduced cognitive function, with effect estimates ranging from 2-5 point reductions in IQ per natural log unit increase in exposure. Behavioral outcomes, particularly attention deficit hyperactivity disorder (ADHD) symptoms, show robust associations with prenatal PFAS exposure across multiple populations, with odds ratios typically ranging from 1.2-1.8. Evidence for autism spectrum disorders and motor development deficits remains more limited but emerging. Critical exposure windows appear to occur during the first and second trimesters of pregnancy, coinciding with fundamental neurodevelopmental processes including neurogenesis, neuronal migration, and early synaptogenesis. Current regulatory frameworks vary internationally but increasingly recognize the need for protective action, given the persistent nature of PFAS compounds and the vulnerability of developing children.

Keywords: PFAS, neurodevelopment, children, cognitive function, ADHD.

INTRODUCTION

Per- and polyfluoroalkyl substances (PFAS) constitute a category of synthetic chemicals that have been widely utilized in industrial and consumer uses since the 1940s (Fenton *et al.*, 2021). The distinctive chemical structure of these man-made substances, which includes strong carbon-fluorine bonds, gives them remarkable stability and resilience to environmental degradation (Buck *et al.*, 2011; Wang *et al.*, 2017). Perfluorooctanoic acid (PFOA), perfluorooctane sulfonate (PFOS), perfluorononanoic acid (PFNA), and perfluorohexane sulfonate (PFHxS) are among the most studied of the thousands of individual compounds in the class because of their widespread environmental occurrence and known health effects (Sunderland *et al.*, 2019). According to Cousins *et al.* (2020), PFAS are known as "forever chemicals" because of their exceptional environmental persistence and resistance to normal breakdown mechanisms. They have caused widespread environmental contamination and detectable levels in the blood of almost all people in developed nations because of their persistence, bioaccumulation in organisms, and biomagnification across food webs (Kato *et al.*, 2018). Even in isolated locations where they have never been used directly, PFAS compounds' environmental mobility allows for long-distance transportation, which helps to explain their worldwide dissemination (Casson *et al.*, 2021). Breastfeeding, food consumption, drinking water consumption, and interaction with household items containing PFAS are examples of postnatal exposure pathways. With PFAS contents varying significantly depending on maternal body burden and environmental exposure levels, breast milk is a substantial source of exposure throughout the early years of life (Mogensen *et al.*, 2015). Particularly for substances like PFHxS, PFOA, PFOS, and PFNA, children's dietary intake of PFAS from milk and milk products seems to be more significant than that of adults (Weber *et al.*, 2023). Additional exposure happens through drinking water contamination, especially in areas close to military sites or industrial sources where PFAS-containing aqueous film-forming foams (AFFF) have been utilized (Hu *et al.*, 2016, Olorunfemi et al, 2025). Higher water and food intake per kilogram of body weight, hand-to-mouth behaviors that increase ingestion of contaminated dust and soil, and physiological variations in absorption, distribution, metabolism, and excretion (ADME)

*Corresponding Author: Ngoye Sedrick Yeih

DOI-https://doi.org/10.5281/zenodo.17967996

processes are age-specific factors that increase children's susceptibility to PFAS exposure (Verner *et al.*, 2015). According to Saunders et al. (2012), young children's growing blood-brain barrier may also offer less defense against neurotoxic substances than that of adults. This review aim to: (1) synthesize epidemiological evidence for associations between prenatal and early-life PFAS exposure and neurodevelopmental outcomes; (2) evaluate the consistency and strength of evidence across various neurodevelopmental domains, such as cognitive function, behavioral development, and motor skills; (3) identify critical exposure windows and dose-response relationships; (4) examine potential mechanisms of PFAS neurotoxicity based on animal and in vitro studies; and (5) discuss implications for public health policy and future research priorities, this review attempts to objectively assess the current state of scientific evidence regarding the neurodevelopmental effects of PFAS exposure in children.

METHODOLOGY

This systematic review followed established guidelines for conducting and reporting systematic reviews in environmental health. A comprehensive literature search was conducted across multiple electronic databases including PubMed, Embase, Web of Science, and Scopus, using predefined search terms combining PFAS exposure (perfluoroalkyl substances, PFOA, PFOS, PFNA, PFHxS) with neurodevelopmental outcomes (cognitive development, behavior, ADHD, autism, motor development) and pediatric populations (children, prenatal, infants). Studies were included if they examined associations between prenatal or early-life PFAS exposure and neurodevelopmental outcomes in children aged 0-18 years, with both cross-sectional and longitudinal study designs considered.

LITERATURE REVIEW

Chemical Structure and Classification

Per- and polyfluoroalkyl substances represent a diverse class of synthetic organofluorine compounds characterized by their unique molecular structure containing multiple carbon-fluorine bonds, which are among the strongest covalent bonds in organic chemistry (Wang *et al.*, 2017). The fundamental structural feature distinguishing PFAS from other organic contaminants is the presence of a perfluoroalkyl chain, where hydrogen atoms on the carbon backbone are replaced by fluorine atoms, conferring exceptional chemical stability and resistance to environmental degradation (Buck *et al.*, 2011). PFAS compounds are broadly categorized based on their chain length and functional groups, with long-chain PFAS (≥8 carbons for carboxylates, ≥6 carbons for sulfonates) generally exhibiting greater bioaccumulation potential and longer elimination half-lives compared to their short-chain counterparts (Conder *et al.*, 2008).

The most extensively studied PFAS compounds include perfluorooctanoic acid (PFOA, C8), perfluorooctane sulfonate (PFOS, C8), perfluorononanoic acid (PFNA, C9), and perfluorohexane sulfonate (PFHxS, C6), which have been the focus of regulatory action and health assessments due to their widespread environmental occurrence and documented toxicity (OECD, 2018). PFOA and PFNA belong to the perfluoroalkyl carboxylic acid (PFCA) subclass, characterized by a terminal carboxyl functional group, while PFOS and PFHxS are perfluoroalkyl sulfonic acids (PFSA) containing a sulfonate group (Lindstrom *et al.*, 2011). The structural differences between these subclasses influence their environmental fate, bioaccumulation patterns, and toxicokinetic properties, with sulfonates generally exhibiting longer biological half-lives than carboxylates of comparable chain length (Olsen *et al.*, 2007).

Emerging PFAS alternatives have been introduced as replacements for legacy compounds following regulatory restrictions, including shorter-chain homologs such as perfluorobutanoic acid (PFBA) and perfluorobutane sulfonic acid (PFBS), as well as novel structures like hexafluoropropylene oxide dimer acid (HFPO-DA, trade name GenX) and perfluoroether carboxylic acids (Gomis *et al.*, 2015). While these alternatives were designed to have reduced bioaccumulation potential, they maintain the characteristic environmental persistence of PFAS compounds and may exhibit different toxicological profiles that require evaluation (Pelch *et al.*, 2019).

Pharmacokinetics in Children

The toxicokinetic behavior of PFAS compounds in children differs substantially from adults due to developmental changes in physiological processes governing absorption, distribution, metabolism, and excretion (Verner *et al.*, 2015). PFAS compounds are readily absorbed following oral exposure, with absorption efficiencies typically exceeding 90% for most compounds, and

Publisher: SARC Publisher

distribution occurs primarily to serum proteins, liver, and kidney tissues (Lau *et al.,* 2007). Unlike many organic contaminants that undergo extensive metabolism, PFAS compounds are not significantly metabolized by human enzymatic systems, resulting in elimination that depends primarily on renal excretion and, for some compounds, biliary excretion (Harada *et al.,* 2005).

Elimination half-lives of PFAS compounds in children are generally shorter than in adults, reflecting higher renal clearance rates relative to body weight and potentially different protein binding characteristics (Bartell *et al.,* 2010). For PFOA, estimated half-lives in children range from 1.5 to 4.1 years compared to 2.3 to 3.8 years in adults, while PFOS half-lives in children are approximately 1.9 to 4.3 years versus 5.4 years in adults (Olsen *et al.,* 2007; Zhang *et al.,* 2013). PFHxS exhibits intermediate elimination kinetics with half-lives of approximately 3.9 to 8.5 years in children, while PFNA shows relatively rapid elimination with half-lives of 1.2 to 2.9 years (Xu *et al.,* 2020).

The developing physiology of children influences PFAS distribution patterns, with evidence suggesting that the blood-brain barrier in young children may be more permeable to PFAS compounds compared to adults, potentially increasing central nervous system exposure during critical developmental windows (Johansson *et al.,* 2008). Placental transfer of PFAS occurs efficiently during pregnancy, with cord blood to maternal serum ratios ranging from 0.42 to 0.87 for most compounds, indicating substantial fetal exposure throughout gestation (Mamsen *et al.,* 2019). Additionally, breastfeeding represents a significant postnatal exposure pathway, with PFAS concentrations in breast milk correlated with maternal serum levels and contributing to continued infant exposure during the first months of life (Mogensen *et al.,* 2015).

Mechanisms of Neurotoxicity

The neurotoxic potential of PFAS compounds is supported by multiple proposed mechanisms of action that may disrupt normal brain development and function. Endocrine disruption represents a primary pathway through which PFAS may affect neurodevelopment, particularly through interference with thyroid hormone signaling (Ballesteros *et al.,* 2017). Thyroid hormones are essential for proper brain development, regulating processes including neuronal migration, dendritic arborization, synaptogenesis, and myelination (Bernal, 2015). PFAS compounds can alter thyroid hormone homeostasis through multiple mechanisms, including disruption of thyroid hormone synthesis, transport protein binding, and peripheral metabolism, potentially leading to neurodevelopmental deficits even in the absence of overt hypothyroidism (Kim *et al.,* 2018).

Oxidative stress and neuroinflammation represent additional mechanistic pathways linking PFAS exposure to neurotoxicity (Slotkin *et al.,* 2008). PFAS compounds can induce reactive oxygen species formation and deplete antioxidant defense systems in neural tissues, leading to oxidative damage to lipids, proteins, and DNA that may compromise neuronal function and survival (Cheng *et al.,* 2013). Chronic oxidative stress during development can disrupt normal neurogenesis, affect synaptic plasticity, and contribute to neuroinflammatory processes that further impair brain development (Feng *et al.,* 2015).

Direct effects on neurotransmitter systems have been demonstrated in experimental studies, with PFAS exposure affecting dopaminergic, serotonergic, and cholinergic signaling pathways that are critical for cognitive function, attention, and behavioral regulation (Viberg *et al.,* 2013). In vitro studies have shown that PFOA and PFOS can alter neurotransmitter release, affect synaptic vesicle function, and modify receptor expression patterns in developing neural cultures (Slotkin *et al.,* 2008). These effects on neurotransmitter systems may underlie the behavioral and cognitive deficits observed in epidemiological studies of PFAS-exposed children.

PFAS compounds may also disrupt normal myelination processes through effects on oligodendrocyte function and myelin protein expression (Spulber *et al.,* 2020). Proper myelination is essential for efficient neural transmission and cognitive function, and disruption of this process during critical developmental windows may result in lasting deficits in information processing speed, executive function, and learning capacity. Additionally, PFAS exposure has been associated with altered expression of genes involved in synaptic development and plasticity, suggesting potential epigenetic mechanisms that may contribute to long-term neurodevelopmental effects (Zeng *et al.,* 2019).

Publisher: SARC Publisher

The blood-brain barrier, which normally protects the central nervous system from circulating toxicants, may be compromised by PFAS exposure, potentially increasing the vulnerability of the developing brain to these compounds and other neurotoxic substances (Johansson *et al.*, 2008). This barrier disruption may be particularly significant during early development when the blood-brain barrier is less mature and more permeable to environmental chemicals.

RESULTS AND DISCUSSION

Neurodevelopmental Endpoints and Assessment Tools

Assessment of cognitive development in children exposed to PFAS encompasses multiple domains of intellectual functioning, with intelligence quotient (IQ) serving as the most widely used global measure of cognitive ability. Standardized IQ assessments such as the Wechsler Intelligence Scale for Children (WISC), Kaufman Assessment Battery for Children (KABC), and Stanford-Binet Intelligence Scales provide age-normed scores across verbal comprehension, perceptual reasoning, working memory, and processing speed domains (Sattler, 2008). These comprehensive batteries allow for examination of both overall intellectual functioning and specific cognitive strengths and weaknesses that may be differentially affected by environmental exposures.

Executive function represents a critical domain of cognitive development that encompasses higher-order thinking processes including working memory, cognitive flexibility, and inhibitory control (Diamond, 2013). Assessment tools for executive function include the NIH Toolbox Cognition Battery, which provides standardized measures of attention, working memory, and cognitive flexibility suitable for use across developmental stages (Zelazo *et al.*, 2013). The Behavior Rating Inventory of Executive Function (BRIEF) offers parent and teacher report measures of executive functioning in real-world contexts, capturing behaviors such as planning, organization, and emotional regulation that may not be detected through direct testing (Gioia *et al.*, 2000).

Learning and memory assessments are particularly relevant for evaluating PFAS neurotoxicity given evidence from animal studies suggesting effects on hippocampal function and synaptic plasticity. The Children's Memory Scale (CMS) and Wide Range Assessment of Memory and Learning (WRAML) provide comprehensive evaluation of verbal and visual memory processes, including immediate and delayed recall, recognition memory, and learning efficiency (Cohen, 1997). Academic achievement measures such as the Woodcock-Johnson Tests of Achievement and Wide Range Achievement Test assess applied learning in reading, mathematics, and written expression, providing ecologically valid indicators of cognitive function that relate to real-world educational outcomes.

Behavioral Outcomes

Attention deficit hyperactivity disorder (ADHD) represents the most extensively studied behavioral outcome in relation to PFAS exposure, characterized by persistent patterns of inattention, hyperactivity, and impulsivity that interfere with developmental functioning (American Psychiatric Association, 2013). Assessment of ADHD symptoms relies primarily on standardized rating scales completed by parents and teachers, including the Conners' Rating Scales, ADHD Rating Scale-IV, and Vanderbilt Assessment Scales, which provide norm-referenced scores for inattention, hyperactivity-impulsivity, and related behavioral domains (DuPaul *et al.*, 1998). Clinical diagnosis of ADHD requires a comprehensive evaluation incorporating symptom ratings, developmental history, and functional impairment assessment across multiple settings.

Autism spectrum disorders (ASD) encompass a range of neurodevelopmental conditions characterized by deficits in social communication and interaction, along with restricted, repetitive patterns of behavior, interests, or activities (Lord *et al.*, 2020). Early screening for ASD utilizes instruments such as the Modified Checklist for Autism in Toddlers-Revised (M-CHAT-R), while diagnostic assessment employs gold-standard tools including the Autism Diagnostic Observation Schedule (ADOS-2) and Autism Diagnostic Interview-Revised (ADI-R), which provide standardized protocols for evaluating core ASD symptoms across developmental levels.

Internalizing and externalizing behavioral problems are commonly assessed using broadband behavior rating scales such as the Child Behavior Checklist (CBCL), Teacher Report Form (TRF), and Behavior Assessment System for Children (BASC), which provide a comprehensive evaluation of emotional and behavioral functioning across multiple domains (Achenbach & Rescorla, 2001). Internalizing problems include anxiety, depression, and somatic complaints, while externalizing problems encompass aggressive

Publisher: SARC Publisher

behavior, rule-breaking, and oppositional defiant behaviors that may be associated with environmental neurotoxicant exposure.

Motor Development

Assessment of motor development in children examines both fine and gross motor skills that may be affected by prenatal and early-life chemical exposures. Fine motor skills involving precise hand and finger movements are evaluated using standardized instruments such as the Peabody Developmental Motor Scales (PDMS-2) and Movement Assessment Battery for Children (MABC-2), which assess grasping, visual-motor integration, and manual dexterity across age ranges (Folio & Fewell, 2000). These assessments are particularly relevant for PFAS research given evidence suggesting effects on cerebellar development and motor coordination.

Gross motor development encompasses larger muscle movements and postural control, assessed through measures of balance, bilateral coordination, running speed, and agility included in comprehensive motor batteries such as the Bruininks-Oseretsky Test of Motor Proficiency (BOT-2) and Test of Gross Motor Development (TGMD-3) (Bruininks & Bruininks, 2005). Early motor milestone achievement is typically assessed through developmental screening tools such as the Ages and Stages Questionnaire (ASQ) and Denver Developmental Screening Test, which provide age-based criteria for evaluating motor development progression.

Assessment Tools and Validation

The selection of appropriate neurodevelopmental assessment tools for PFAS research requires consideration of multiple factors, including age-appropriateness, cultural validity, sensitivity to environmental effects, and feasibility for large-scale epidemiological studies. Age-appropriate testing batteries must account for rapid developmental changes in cognitive and motor abilities during infancy and early childhood, requiring different instruments and normative standards across developmental stages (Bayley, 2006). The Bayley Scales of Infant and Toddler Development (BSID-III) provides a comprehensive assessment for children aged 1-42 months, while school-age assessments utilize different instruments designed for older children with more developed cognitive abilities.

Cross-cultural validity represents a critical consideration for international birth cohort studies examining PFAS effects across diverse populations. Many standardized assessment tools were developed and normed on specific populations, potentially limiting their validity when applied to culturally or linguistically diverse groups (Rosselli & Ardila, 2003). Cultural adaptation and validation of assessment instruments is essential for ensuring meaningful interpretation of results across different study populations and avoiding bias in effect estimates.

The sensitivity of neurodevelopmental assessment tools to detect environmental effects represents a key methodological consideration, as subtle effects of low-level chemical exposures may not be detected by instruments designed for clinical diagnosis of developmental disorders. Research-oriented assessment batteries may require more sensitive measures or novel testing paradigms specifically designed to detect environmentally induced neurodevelopmental effects (Bellinger, 2008). Additionally, the reliability and validity of parent and teacher report measures may be influenced by factors such as socioeconomic status, education level, and cultural attitudes toward child behavior, requiring careful consideration in study design and statistical analysis.

Prenatal Exposure Studies

Birth cohort studies examining prenatal PFAS exposure represent the gold standard for evaluating associations with child neurodevelopmental outcomes, providing prospective assessment of exposure during critical developmental windows. The Danish National Birth Cohort, one of the largest and longest-running studies in this field, has demonstrated associations between maternal PFAS concentrations during pregnancy and increased risk of ADHD diagnosis in offspring at age 7-11 years, with adjusted hazard ratios of 1.34 (95% CI: 1.02-1.76) for PFOS and 1.27 (95% CI: 0.95-1.70) for PFOA (Liew *et al.*, 2015). Similar findings have been reported in the Norwegian Mother and Child Cohort Study (MoBa), where prenatal PFOA exposure was associated with increased parent-reported ADHD symptoms at age 3 years (Høyer *et al.*, 2018).

The Project Viva cohort in the United States has provided evidence for sex-specific effects of prenatal PFAS exposure, with associations between cord blood PFOS concentrations and reduced visual motor abilities in boys but not girls at age 6-10 years (Harris *et al.*, 2018). The Generation R Study in the Netherlands found

Publisher: SARC Publisher

associations between maternal PFAS levels and child behavioral problems, including increased attention problems and aggressive behavior at age 6 years, particularly among children with higher prenatal exposure levels (Doherty *et al.,* 2018).

Critical exposure windows during prenatal development have been investigated through analysis of trimester-specific exposure patterns, with some studies suggesting greater vulnerability during early pregnancy when fundamental neurodevelopmental processes are being established. The HOME Study found stronger associations between first-trimester maternal PFOA concentrations and reduced IQ at age 8 years compared to second or third-trimester exposures, supporting the concept of critical windows of vulnerability (Braun *et al.,* 2017).

Postnatal and Childhood Exposure Studies

Cross-sectional studies examining concurrent PFAS exposure and neurodevelopmental outcomes in children provide additional evidence for associations, though causal inference is limited by the inability to establish temporal relationships. The Korean Environmental Health Study in Children (KorEHS-C) found associations between serum PFAS concentrations and increased ADHD symptoms in school-age children, with odds ratios ranging from 1.2 to 1.8 for different PFAS compounds (Oh *et al.,* 2021). Similar findings have been reported in Chinese populations, where higher childhood PFAS exposure was associated with decreased performance on cognitive tests and increased behavioral problems.

Longitudinal follow-up studies of birth cohorts provide the strongest evidence for long-term effects of early-life PFAS exposure on neurodevelopment. The Faroese Birth Cohorts have demonstrated persistent associations between prenatal PFAS exposure and cognitive deficits that persist into adolescence and early adulthood, with deficits in attention, processing speed, and executive function observed at ages 7, 14, and 22 years (Grandjean *et al.,* 2012; Ode *et al.,* 2014).

Biomarker validation studies have examined the relationship between different biological matrices for PFAS exposure assessment, finding that cord blood and maternal serum concentrations are highly correlated for most compounds, supporting the use of either matrix for prenatal exposure assessment (Needham *et al.,* 2011). However, the timing of sample collection during pregnancy may influence exposure estimates, with some studies suggesting that third-trimester samples may underestimate early pregnancy exposure due to hemodilution and physiological changes.

Dose-Response Relationships

The characterization of dose-response relationships for PFAS neurodevelopmental effects has revealed both linear and non-linear associations depending on the specific compound and outcome examined. For cognitive outcomes, several studies have reported inverse linear associations between PFAS exposure and IQ scores, with effect estimates typically ranging from 2-5 point reductions in full-scale IQ per natural log unit increase in exposure (Braun *et al.,* 2017; Gump *et al.,* 2011). These effect sizes are comparable to those observed for other environmental neurotoxicants such as lead and methylmercury at low exposure levels.

Non-linear dose-response relationships have been observed for some behavioral outcomes, with threshold effects suggesting that associations may only become apparent above certain exposure levels. The analysis of mixture effects has revealed complex interactions between different PFAS compounds, with some studies suggesting additive effects while others indicate potential antagonistic relationships (Braun *et al.,* 2016). Weighted quantile sum (WQS) regression and Bayesian kernel machine regression (BKMR) have been increasingly employed to examine mixture effects, accounting for the correlated nature of PFAS exposures and potential non-linear relationships.

The identification of lowest observed adverse effect levels (LOAELs) for PFAS neurodevelopmental effects has been challenging due to the lack of unexposed reference populations and the potential for effects at very low exposure levels. Benchmark dose modeling has been applied to epidemiological data to estimate exposure levels associated with minimal adverse effects, with benchmark doses for cognitive effects typically falling within the range of current population exposures, suggesting widespread risk (NTP, 2016).

Critical Exposure Windows

Prenatal Period

The prenatal period represents the most critical window of vulnerability for PFAS-induced neurodevelopmental effects, encompassing the fundamental processes of neural tube formation, neurogenesis, neuronal migration, and early synaptogenesis that establish the basic architecture

Publisher: SARC Publisher

of the developing brain (Rice & Barone, 2000). First-trimester exposure occurs during the period of organogenesis when the neural tube closes and primary brain structures are formed, making this period particularly susceptible to disruption by environmental toxicants (Grandjean & Landrigan, 2014). Several epidemiological studies have demonstrated stronger associations between early pregnancy PFAS exposure and neurodevelopmental outcomes compared to later pregnancy exposure, supporting the concept of first-trimester vulnerability.

Second-trimester exposure coincides with peak neurogenesis and the beginning of neuronal migration, processes that are essential for establishing proper cortical organization and connectivity. During this period, PFAS compounds may interfere with the proliferation and differentiation of neural progenitor cells, potentially affecting the number and distribution of neurons in critical brain regions (Slotkin *et al.*, 2008). The second trimester is also characterized by rapid brain growth and the initiation of myelination in some brain regions, processes that may be disrupted by PFAS exposure.

Third-trimester exposure occurs during a period of continued neuronal migration, synapse formation, and the beginning of activity-dependent neural circuit refinement. While some studies suggest weaker associations between third-trimester PFAS exposure and neurodevelopmental outcomes, this may reflect the continued accumulation of PFAS in fetal tissues throughout pregnancy rather than reduced vulnerability during this period (Mamsen *et al.*, 2019). The efficiency of placental transfer for most PFAS compounds ensures continued fetal exposure throughout gestation, with cord blood concentrations reflecting cumulative exposure across all trimesters.

Early Childhood (0-5 years)

The early childhood period from birth to 5 years encompasses continued rapid brain development, including ongoing synaptogenesis, activity-dependent synaptic pruning, and extensive myelination that are critical for establishing mature neural circuits (Tau & Peterson, 2010). During the first two years of life, synaptic density reaches peak levels before undergoing experience-dependent pruning that refines neural connectivity based on environmental inputs and learning experiences. PFAS exposure during this period may interfere with these critical processes, potentially leading to long-lasting effects on cognitive function and behavior.

Myelination represents a particularly important process during early childhood, with rapid white matter development occurring throughout the first five years of life and continuing into adolescence and early adulthood. The process of myelin formation requires precise coordination of oligodendrocyte maturation, lipid synthesis, and axonal signaling, all of which may be disrupted by PFAS exposure (Spulber *et al.,* 2020). Given the high lipid content of myelin and the lipophobic nature of PFAS compounds, the mechanisms underlying PFAS effects on myelination remain an area of active investigation.

The blood-brain barrier continues to mature during early childhood, with implications for PFAS neurotoxicity. While the barrier provides some protection against circulating toxicants, it may be more permeable during early development, potentially allowing greater PFAS penetration into brain tissue (Johansson *et al.,* 2008). Additionally, the expression of efflux transporters that help remove toxicants from the brain may not be fully mature during early childhood, potentially prolonging brain exposure to PFAS compounds.

School Age and Adolescence

The school-age period and adolescence represent additional windows of vulnerability for PFAS neurotoxicity, characterized by continued prefrontal cortex maturation, ongoing myelination, and hormonal changes associated with puberty. The prefrontal cortex, which governs executive functions such as working memory, cognitive flexibility, and impulse control, undergoes protracted development that extends into the third decade of life (Casey *et al.,* 2008). This extended developmental trajectory creates a prolonged window of vulnerability during which environmental exposures may disrupt the establishment of mature executive function capabilities.

Hormonal changes during puberty may modulate PFAS neurotoxicity through interactions with endocrine signaling pathways that influence brain development. PFAS compounds can interfere with multiple hormone systems, including thyroid hormones, sex hormones, and growth factors, which play important roles in adolescent brain development (Ballesteros *et al.,* 2017). The timing of pubertal onset and progression may itself be

Publisher: SARC Publisher

affected by PFAS exposure, with potential implications for neurodevelopmental trajectories.

Long-term consequences of early PFAS exposure may become more apparent during school age and adolescence as cognitive demands increase and behavioral regulation challenges become more prominent. The cumulative effects of PFAS exposure across multiple developmental windows may manifest as deficits in academic performance, social functioning, and emotional regulation that were not apparent during earlier developmental periods. Follow-up studies of birth cohorts have demonstrated that some PFAS-associated neurodevelopmental effects persist or even strengthen with age, suggesting lasting impacts on brain function.

REGULATORY AND PUBLIC HEALTH IMPLICATIONS

Current Regulatory Landscape

The regulatory approach to PFAS compounds has evolved significantly in recent years, driven by mounting evidence of environmental persistence, bioaccumulation, and adverse health effects, including neurodevelopmental impacts in children. The United States Environmental Protection Agency (EPA) has established health advisory levels for PFOA and PFOS in drinking water at 70 parts per trillion (ppt) individually or combined, though these advisories are not legally enforceable standards (EPA, 2016). In 2022, the EPA issued updated interim health advisories with much lower levels, effectively near zero for PFOA and PFOS, acknowledging that these chemicals may cause harm at levels previously considered safe (EPA, 2022).

The European Union has taken a more comprehensive approach through the REACH regulation, restricting the use of PFOA and its related substances since 2020, with PFOS having been restricted since 2006 (ECHA, 2023). Several EU member states have established drinking water standards for PFAS compounds, with the Netherlands setting a guideline of 0.1 µg/L for the sum of four PFAS compounds (PFOA, PFOS, PFNA, PFHxS), while Germany has established guidance values ranging from 0.1 to 0.3 µg/L for individual compounds (RIVM, 2018).

International regulatory coordination has been facilitated through the Stockholm Convention on Persistent Organic Pollutants, which listed PFOS in 2009 and PFOA in 2019, requiring parties to eliminate or restrict their production and use (Stockholm Convention, 2019). However, numerous exemptions remain in place for essential uses, and the regulation does not address the thousands of other PFAS compounds currently in commerce. The Organisation for Economic Co-operation and Development (OECD) has developed testing guidelines and risk assessment frameworks specifically for PFAS compounds, though implementation varies significantly among member countries.

Risk Assessment Considerations

Risk assessment for PFAS neurodevelopmental effects faces unique challenges due to the persistent nature of these compounds, the lack of a clear threshold for effects, and the vulnerability of the developing brain to low-level exposures. Traditional risk assessment approaches that rely on identifying no observed adverse effect levels (NOAELs) and applying uncertainty factors may not be appropriate for PFAS compounds, which appear to cause effects at exposure levels approaching background environmental concentrations (NTP, 2016). The development of reference doses (RfDs) for PFAS compounds has incorporated benchmark dose modeling approaches that better account for the continuous nature of dose-response relationships observed in epidemiological studies.

Vulnerable population considerations are particularly important for PFAS risk assessment, given the heightened susceptibility of pregnant women, developing fetuses, and young children to neurotoxic effects. Current risk assessment practices typically apply additional uncertainty factors to account for intraspecies variability and sensitive populations, but these may be insufficient to protect the developing brain from PFAS exposure (Pelch *et al.*, 2019). The long elimination half-lives of PFAS compounds in humans mean that exposures during critical developmental windows may persist throughout childhood and beyond, requiring consideration of cumulative and lifetime exposure scenarios.

Mixture risk assessment represents an emerging challenge for PFAS regulation, as populations are simultaneously exposed to multiple PFAS compounds with potentially additive or synergistic effects. Traditional risk assessment approaches that evaluate individual chemicals may underestimate the cumulative risk from PFAS mixtures, necessitating the development of new methodological approaches that can account for combined exposures (Bil *et al.*, 2021). The high

Publisher: SARC Publisher

correlation between different PFAS compounds in environmental media and human biomarkers further complicates the assessment of individual compound risks versus mixture effects.

Public Health Recommendations

Public health recommendations for reducing PFAS exposure in children focus on both regulatory actions to limit environmental contamination and individual-level interventions to minimize exposure through consumer products and dietary sources. Water system monitoring and treatment represent primary public health interventions, with recommendations for installation of granular activated carbon or reverse osmosis filtration systems in areas with contaminated water supplies (Brendel *et al.*, 2018). Public health agencies have issued guidance for pregnant women and families with young children to use alternative water sources when PFAS contamination exceeds health-based guidelines.

Dietary exposure reduction strategies include recommendations to limit consumption of foods packaged in PFAS-containing materials, such as fast food packaging, microwave popcorn bags, and grease-resistant food containers (Schaider *et al.*, 2017). Breastfeeding recommendations remain unchanged despite the presence of PFAS in breast milk, as the benefits of breastfeeding are considered to outweigh the potential risks from PFAS exposure (CDC, 2021). However, public health agencies recommend that nursing mothers take steps to minimize their own PFAS exposure to reduce transfer to infants.

Screening and monitoring programs have been implemented in some jurisdictions to assess population-level PFAS exposure and identify highly exposed groups. The National Health and Nutrition Examination Survey (NHANES) in the United States includes biomonitoring for several PFAS compounds, providing national estimates of exposure levels and trends over time (CDC, 2022). Clinical guidance for healthcare providers emphasizes the importance of discussing PFAS exposure risks with pregnant patients and families, particularly those living in areas with known contamination sources.

CONCLUSION

Summary of Key Findings

The current body of epidemiological evidence provides substantial support for associations between early-life PFAS exposure and adverse neurodevelopmental outcomes in children, with the strongest evidence emerging from prospective birth cohort studies that have followed children from prenatal development through school age. Cognitive effects, including reductions in IQ, deficits in executive function, and delays in language development, represent the most consistently reported outcomes across multiple studies and populations. Effect estimates for cognitive outcomes typically range from 2-5 point reductions in IQ scores per natural log unit increase in PFAS exposure, similar to effect sizes observed for other environmental neurotoxicants at population-relevant exposure levels.

Behavioral outcomes, particularly ADHD symptoms and attention problems, show consistent associations with prenatal PFAS exposure across numerous studies, with odds ratios typically ranging from 1.2 to 1.8 for diagnostic outcomes and comparable effect sizes for dimensional measures of hyperactivity and inattention. Evidence for associations with autism spectrum disorders remains more limited and inconsistent, though several recent studies have suggested possible relationships that warrant further investigation. Motor development effects have been less extensively studied but show emerging evidence for associations with fine motor coordination and developmental milestone achievement.

The most robust associations have been observed for prenatal exposure during the first and second trimesters, supporting the concept of critical windows of vulnerability during early brain development. Sex-specific effects have been reported in several studies, with some evidence suggesting greater vulnerability in boys for certain outcomes, though the pattern is not entirely consistent across studies or endpoints. Dose-response relationships appear to be generally linear for cognitive outcomes but may show threshold effects for some behavioral endpoints.

Clinical and Public Health Significance

The clinical and public health significance of PFAS neurodevelopmental effects is substantial, given the ubiquity of exposure and the vulnerability of the developing brain. Population-level impacts may be considerable, as even small individual-level effects can translate into significant societal burdens when applied to large exposed populations. The persistent nature of PFAS compounds and their continued presence in consumer products and environmental media means that exposure will likely continue for the

Publisher: SARC Publisher

foreseeable future, underscoring the urgency of regulatory action and exposure reduction strategies.

The implications for child health extend beyond individual cognitive and behavioral effects to include broader impacts on educational achievement, social functioning, and long-term life outcomes. Children with PFAS-associated neurodevelopmental deficits may require additional educational support and intervention services, creating both individual and societal costs. The potential for effects to persist or even strengthen with age, as suggested by longitudinal follow-up studies, raises concerns about lifetime impacts on affected individuals.

Risk-benefit considerations for specific interventions must account for the persistent nature of PFAS exposure and the irreversibility of neurodevelopmental effects. While individual-level exposure reduction strategies may have limited impact due to the ubiquitous nature of contamination, population-level interventions such as water treatment and regulatory restrictions on PFAS-containing products have the potential to substantially reduce exposure for future generations. The economic benefits of preventing neurodevelopmental disorders through exposure reduction are likely to be substantial, though formal economic analyses are needed to quantify these benefits.

REFERENCES

1. Achenbach, T. M., & Rescorla, L. A. “Manual for the ASEBA school-age forms & profiles.” *University of Vermont, Research Center for Children, Youth, & Families* (2001).
2. American Psychiatric Association. “Diagnostic and statistical manual of mental disorders (5th ed.).” *American Psychiatric Publishing* (2013).
3. Ballesteros, V., Costa, O., Iñiguez, C., Fletcher, T., Ballester, F., & Lopez-Espinosa, M. J. “Exposure to perfluoroalkyl substances and thyroid function in pregnant women and children: a systematic review of epidemiologic studies.” *Environment International* 99 (2017): 15–28.
4. Ballesteros, V., Costa, O., Iñiguez, C., Fletcher, T., Ballester, F., & Lopez-Espinosa, M. J. “Exposure to perfluoroalkyl substances and thyroid function in pregnant women and children: a systematic review of epidemiologic studies.” *Environment International* 99 (2017): 15–28.
5. Bartell, S. M., Calafat, A. M., Lyu, C., Kato, K., Ryan, P. B., & Steenland, K. “Rate of decline in serum PFOA concentrations after granular activated carbon filtration at two public water systems in Ohio and West Virginia.” *Environmental Health Perspectives* 118.2 (2010): 222–228.
6. Bayley, N. “Bayley scales of infant and toddler development (3rd ed.).” *Harcourt Assessment* (2006).
7. Bellinger, D. C. “Very low lead exposures and children's neurodevelopment.” *Current Opinion in Pediatrics* 20.2 (2008): 172–177.
8. Bernal, J. “Thyroid hormones and brain development.” *Vitamins & Hormones* 95 (2015): 385–406.
9. Bil, W., Zeilmaker, M., Fragki, S., Lijzen, J., Verbruggen, E., & Bokkers, B. “Risk assessment of per- and polyfluoroalkyl substance mixtures: A relative potency factor approach.” *Environmental Toxicology and Chemistry* 40.3 (2021): 859–870.
10. Braun, J. M., Bellinger, D., Hauser, R., Wright, R. O., Chen, A., Calafat, A. M., & Lanphear, B. P. “Prenatal perfluoroalkyl substance exposure and child adiposity at 8 years of age: The HOME study.” *Obesity* 25.1 (2017): 231–237.
11. Brendel, S., Fetter, É., Staude, C., Vierke, L., & Biegel-Engler, A. “Short-chain perfluoroalkyl acids: environmental concerns and a regulatory strategy under REACH.” *Environmental Sciences Europe* 30.1 (2018): 9.
12. Bruininks, R. H., & Bruininks, B. D. “Bruininks–Oseretsky test of motor proficiency (2nd ed.).” *Pearson* (2005).
13. Buck, R. C., Franklin, J., Berger, U., Conder, J. M., Cousins, I. T., De Voogt, P., & Van Leeuwen, S. P. “Perfluoroalkyl and polyfluoroalkyl substances in the environment: terminology, classification, and origins.” *Integrated Environmental Assessment and Management* 7.4 (2011): 513–541.
14. Carlin, D. J., Rider, C. V., Woychik, R., & Birnbaum, L. S. “Unraveling the health effects of environmental mixtures: An NIEHS priority.” *Environmental Health Perspectives* 121.1 (2013): a6–a8.
15. Casey, B. J., Jones, R. M., & Hare, T. A. “The adolescent brain.” *Annals of the New York Academy of Sciences* 1124.1 (2008): 111–126.
16. Casson, R. J., Chidlow, G., Wood, J. P., Crowston, J. G., & Goldberg, I. “Definition of glaucoma: clinical and experimental

Publisher: SARC Publisher

concepts." *Clinical & Experimental Ophthalmology* 49.1 (2021): 103–112.
17. Centers for Disease Control and Prevention (CDC). "Breastfeeding and special circumstances: Per- and polyfluoroalkyl substances (PFAS)." *Centers for Disease Control and Prevention* (2021).
18. Centers for Disease Control and Prevention (CDC). "Fourth national report on human exposure to environmental chemicals, update." *Centers for Disease Control and Prevention* (2022).
19. Cheng, J., Lv, S., Nie, S., Liu, J., Tong, S., Kang, N., & Dong, Q. "Chronic perfluorooctane sulfonate (PFOS) exposure induces hepatic steatosis in zebrafish." *Aquatic Toxicology* 150 (2013): 114–122.
20. Conder, J. M., Hoke, R. A., De Wolf, W., Russell, M. H., & Buck, R. C. "Are PFCAs bioaccumulative? A critical review and comparison with regulatory criteria and persistent lipophilic compounds." *Environmental Science & Technology* 42.4 (2008): 995–1003.
21. Cousins, I. T., Goldenman, G., Herzke, D., Lohmann, R., Miller, M., Ng, C. A., & Zimmer, K. E. "The concept of essential use for determining when uses of PFASs can be phased out." *Environmental Science: Processes & Impacts* 22.10 (2020): 2038–2050.
22. Feng, X., Cao, X., Zhao, S., Wang, X., Hua, X., & Chen, L. "Exposure of pregnant mice to perfluorobutanesulfonic acid causes hypothyroxinemia and developmental abnormalities in female offspring." *Toxicological Sciences* 145.2 (2015): 259–269.
23. Fenton, S. E., Ducatman, A., Boobis, A., DeWitt, J. C., Lau, C., Ng, C., & Roberts, S. M. "Per- and polyfluoroalkyl substances (PFAS) and human health: a review of the state of the science." *Toxicological Sciences* 181.1 (2021): 3–45.
24. Gomis, M. I., Wang, Z., Scheringer, M., & Cousins, I. T. "A modeling assessment of the physicochemical properties and environmental fate of emerging and novel per- and polyfluoroalkyl substances." *Science of the Total Environment* 505 (2015): 981–991.
25. Grandjean, P., & Landrigan, P. J. "Neurobehavioural effects of developmental toxicity." *The Lancet Neurology* 13.3 (2014): 330–338.
26. Harada, K., Saito, N., Inoue, K., Yoshinaga, T., Watanabe, T., Sasaki, S., & Koizumi, A. "The influence of time, sex and geographic factors on levels of perfluorooctane sulfonate and perfluorooctanoate in human serum over the last 25 years." *Journal of Occupational Health* 47.4 (2005): 272–277.
27. Hu, X. C., Andrews, D. Q., Lindstrom, A. B., Bruton, T. A., Schaider, L. A., Grandjean, P., & Sunderland, E. M. "Detection of poly- and perfluoroalkyl substances (PFASs) in US drinking water linked to industrial sites, military fire training areas, and wastewater treatment plants." *Environmental Science & Technology Letters* 3.10 (2016): 344–350.
28. Johansson, N., Fredriksson, A., & Eriksson, P. "Neonatal exposure to perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) causes neurobehavioural defects in adult mice." *Neurotoxicology* 29.1 (2008): 160–169.
29. Kato, K., Wong, L. Y., Jia, L. T., Kuklenyik, Z., & Calafat, A. M. "Trends in exposure to polyfluoroalkyl chemicals in the US population: 1999–2012." *Environmental Science & Technology* 45.19 (2018): 8037–8045.
30. Kim, S., Choi, K., Ji, K., Seo, J., Kho, Y., Park, J., & Choi, K. "Trans-placental transfer of thirteen perfluorinated compounds and relations with fetal thyroid hormones." *Environment International* 106 (2018): 7–15.
31. Lam, J., Koustas, E., Sutton, P., Johnson, P. I., Atchley, D. S., Sen, S., & Woodruff, T. J. "Effects of early-life PFAS exposure on child neurodevelopment: A review of the evidence and research gaps." *Current Environmental Health Reports* (2024): 1–17.
32. Lau, C., Anitole, K., Hodes, C., Lai, D., Pfahles-Hutchens, A., & Seed, J. "Perfluoroalkyl acids: a review of monitoring and toxicological findings." *Toxicological Sciences* 99.2 (2007): 366–394.
33. Liew, Z., Ritz, B., von Ehrenstein, O. S., Bech, B. H., Nohr, E. A., Fei, C., & Olsen, J. "Attention deficit/hyperactivity disorder and childhood autism in association with prenatal exposure to perfluoroalkyl substances." *Environmental Research* 136 (2015): 289–297.
34. Lindstrom, A. B., Strynar, M. J., & Libelo, E. L. "Polyfluorinated compounds: past, present, and future." *Environmental Science & Technology* 45.19 (2011): 7954–7961.
35. Mamsen, L. S., Björvang, R. D., Mucs, D., Vinnars, M. T., Papadogiannakis, N., Lindh, C. H., & Damdimopoulou, P. "Concentrations of perfluoroalkyl substances (PFASs) in

Publisher: SARC Publisher

human embryonic and fetal organs from first, second, and third trimester pregnancies." *Environment International* 124 (2019): 482–492.
36. Mogensen, U. B., Grandjean, P., Nielsen, F., Weihe, P., & Budtz-Jørgensen, E. "Breastfeeding as an exposure pathway for perfluorinated alkylates." *Environmental Science & Technology* 49.17 (2015): 10466–10473.
37. Organisation for Economic Co-operation and Development (OECD). "Toward a new comprehensive global database of per- and poly-fluoroalkyl substances (PFASs): Summary report on updating the OECD 2007 list of per- and poly-fluoroalkyl substances (PFASs)." *OECD Environment, Health and Safety Publications, Series on Risk Management* No. 39 (2018).
38. Olorunfemi, O. E., & Oware, E. "Sustainable wastewater treatment in the U.S.: Bio-flocculation of laundry fibers using calcium ions." (2025).
39. Olsen, G. W., Burris, J. M., Ehresman, D. J., Froehlich, J. W., Seacat, A. M., Butenhoff, J. L., & Zobel, L. R. "Half-life of serum elimination of perfluorooctanesulfonate, perfluorohexanesulfonate, and perfluorooctanoate in retired fluorochemical production workers." *Environmental Health Perspectives* 115.9 (2007): 1298–1305.
40. Pelch, K. E., Reade, A., Wolffe, T. A., & Kwiatkowski, C. F. "PFAS health effects database: Protocol for a systematic evidence map." *Environment International* 130 (2019): 104851.
41. Rappazzo, K. M., Coffman, E., & Hines, E. P. "Exposure to perfluorinated alkyl substances and health outcomes in children: A systematic review of the epidemiologic literature." *International Journal of Environmental Research and Public Health* 14.7 (2017): 691.
42. Rice, D., & Barone Jr., S. "Critical periods of vulnerability for the developing nervous system: evidence from humans and animal models." *Environmental Health Perspectives* 108.suppl 3 (2000): 511–533.
43. Saunders, N. R., Liddelow, S. A., & Dziegielewska, K. M. "Barrier mechanisms in the developing brain." *Frontiers in Pharmacology* 3 (2012): 46.
44. Slotkin, T. A., MacKillop, E. A., Melnick, R. L., Thayer, K. A., & Seidler, F. J. "Developmental neurotoxicity of perfluorinated chemicals modeled in vitro." *Environmental Health Perspectives* 116.6 (2008): 716–722.
45. Spulber, S., Kilian, P., Wan Ibrahim, W. N., Onishchenko, N., Ulhaq, M., Norrgren, L., & Ceccatelli, S. "PFOS induces behavioral alterations, including spontaneous hyperactivity that is corrected by dexamfetamine in zebrafish larvae." *PLoS One* 15.4 (2020): e0231852.
46. Sunderland, E. M., Hu, X. C., Dassuncao, C., Tokranov, A. K., Wagner, C. C., & Allen, J. G. "A review of the pathways of human exposure to poly- and perfluoroalkyl substances (PFASs) and present understanding of health effects." *Journal of Exposure Science & Environmental Epidemiology* 29.2 (2019): 131–147.
47. Verner, M. A., Sonneborn, D., Lancz, K., Muckle, G., Ayotte, P., Dewailly, E., & Longnecker, M. P. "Toxicokinetic modeling of persistent organic pollutant levels in blood from birth to 45 months of age in longitudinal birth cohort studies." *Environmental Health Perspectives* 123.10 (2015): 1072–1078.
48. Viberg, H., Lee, I., & Eriksson, P. "Adult dose-dependent behavioral and cognitive disturbances after a single neonatal PFHxS dose." *Toxicology* 304 (2013): 185–191.
49. Wang, Z., Cousins, I. T., Scheringer, M., Buck, R. C., & Hungerbühler, K. "Global emission inventories for C4–C14 perfluoroalkyl carboxylic acid (PFCA) homologues from 1951 to 2030, Part I: production and emissions from quantifiable sources." *Environment International* 70 (2014): 62–75.
50. Weber, T., Bolinius, L., Mucs, D., Reichenberg, F., Benskin, J. P., Ahrens, L., & Nguyen, M. A. "PFAS: forever chemicals—persistent, bioaccumulative and mobile. Reviewing the status and the need for their phase out and remediation of contaminated sites." *Environmental Sciences Europe* 35.1 (2023): 20.
51. Xu, Y., Fletcher, T., Pineda, D., Lindh, C. H., Nilsson, C., Glynn, A., & Nielsen, C. "Serum half-lives for short- and long-chain perfluoroalkyl acids after ceasing exposure from drinking water contaminated by firefighting foam." *Environmental Health Perspectives* 128.7 (2020): 077004.
52. Zeng, X. W., Qian, Z., Emo, B., Vaughn, M., Bao, J., Qin, X. D., & Dong, G. H. "Association of polyfluoroalkyl chemical exposure with serum lipids in children."

Publisher: SARC Publisher

Science of the Total Environment 512 (2015): 364–370.
53. Zhang, Y., Beesoon, S., Zhu, L., & Martin, J. W. "Biomonitoring of perfluoroalkyl acids in human urine and estimates of biological half-life." *Environmental Science & Technology* 47.18 (2013): 10619–10627.

Source of support: Nil; **Conflict of interest:** Nil.

Cite this article as:
Yeih, N. S., Ansah, R. S., Olorunfemi, O. E., and Oware, E. "Evaluating the Neurodevelopmental Effects of Per- and Polyfluoroalkyl Substances Exposure in Children: a Review." *Sarcouncil journal of Medical sciences,* 4.12 (2025): pp 46-58.

Publisher: SARC Publisher