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Are the growing levels of neurotoxic and neuro-disruptive chemicals in our food and drink contributing to the youth mental health crisis? A narrative review

Jennifer Jane Newson [a,*], Zoya Marinova [a,b], Tara C. Thiagarajan [a]

[a] Sapien Labs, 1201 Wilson Blvd, 27th floor, Arlington, VA, United States of America
[b] Department of Biology, Lake Forest College, 555 North Sheridan Road, Lake Forest, IL, United States of America

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ABSTRACT

Over the past few decades, there has been a marked and largely unexplained decline in the mental health and wellbeing of young people worldwide. While demographic and life context factors such as employment, education, economic status, life adversity, socialization habits, and physical activity predict mental wellbeing outcomes in older adults with high accuracy, their predictive power diminishes significantly in younger generations. This suggests that additional, underexplored factors are contributing to this decline. One such factor, often overlooked, is the increasing exposure of children and adolescents to neurotoxic and neuro-disruptive chemicals in the food and beverages they consume. These include agricultural and industrial chemicals (e.g., pesticide residues, heavy metals), additives in ultra-processed foods, and packaging-derived contaminants such as microplastics and bisphenols. Here, we provide a narrative review of the associations between exposure to these chemicals and adverse neurodevelopmental and mental health outcomes in youth, considering changes in farming practices, food production, and packaging over recent decades. We also highlight some of the key research challenges of evaluating these impacts and note the lack of attention from the neuroscience and neuroimaging communities. Altogether, the widespread presence of these neurotoxic and neuro-disruptive chemicals in the body and brain, and growing reports of their adverse impacts on behavior, cognition, and mental health in young people, points to the potential for progressive degradation of brain function that poses a grave threat to the future wellbeing of society and underscores the urgent need for increased research, funding, and regulation in this area.

1. An unexplained decline in the mental health and wellbeing of young people

Just over a decade ago, researchers studying psychological wellbeing observed a U-shaped trend with age (Steptoe et al., 2015; Stone et al., 2010). Younger and older adults had the highest wellbeing with a dip in middle age. Today, a growing body of evidence indicates that youth mental health has since steadily declined (Blanchflower et al., 2024c, 2024b; CDC, 2023; Keyes et al., 2019; Marquez and Long, 2021; McCurdy and Murphy, 2024; Twenge et al., 2018; Wiens et al., 2020), with adolescents and young adults in many countries around the world now reporting the lowest mental wellbeing of any age group (Blanchflower, 2025; Blanchflower et al., 2024a; Blanchflower and Bryson, 2025, 2024a, 2024b; Helliwell et al., 2024; Sapien Labs, 2025, 2024, 2022b, 2022a). The Global Mind Project, a large-scale ongoing research program that measures population trends and drivers relating to mental health and wellbeing, shows that, across the internet-enabled world, nearly 50 % of young adults aged 18–24 are mentally distressed or struggling (reflecting the experience of approximately five or more mental health symptoms that would be of clinical concern) compared to less than 10 % of their grandparents' generation (Sapien Labs, 2024). The magnitude of this trend signifies an extraordinary shift in just 15 years. Most prominently, these symptoms include fear and anxiety, unwanted thoughts, feelings of sadness or hopelessness, loss of focus, poor self-image, decreased emotional control, feelings of aggression towards others, and feeling detached from reality (Sapien Labs, 2022b). At the same time, studies have highlighted a significant increase in neurodevelopmental disorder diagnoses in children over the past few

* Corresponding author.
E-mail addresses: jennifer@sapienlabs.org (J.J. Newson), zoya@sapienlabs.org (Z. Marinova), tara@sapienlabs.org (T.C. Thiagarajan).

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decades (Grosvenor et al., 2024; McKechnie et al., 2023; Russell et al., 2022; Xu et al., 2018). However, while researchers generally agree that youth mental health is declining, there is far less clarity on what is causing it, and what to do about it.

The Global Mind Project has previously investigated the contribution of numerous demographic and life context factors, including age, biological sex, educational attainment level, employment status, frequency of physical activity and in-person socializing, the experience of various life traumas and adversities, substance use, and medical conditions, in a large sample of over 400,000 individuals and shown that, together, they predict the mental health status of older adults with high accuracy and precision (95 %+) (Bala et al., 2024). However, their predictive power declines systematically for younger generations to just 67 % for 18–24-year-olds, indicating that other factors are at play. While the rise of smartphones and social media has been shown to contribute to this trend (Braghieri et al., 2022; Orben et al., 2022; Sapien Labs, 2023; Shannon et al., 2022; Twenge and Martin, 2020), this also does not fully explain the extent of the decline. One factor that has seldom been discussed in relation to this decline is the increased number of neurotoxic and neuro-disruptive chemicals that children and young people consume in their food and beverages. These include agricultural and industrialized chemicals (e.g. pesticide residues, heavy metals), additive-containing ultra-processed foods (UPFs), as well as packaging-derived chemicals (e.g. microplastics, bisphenols). While previous studies have discussed the potential role of these chemicals on rising rates of diseases such as obesity and cancer in young people (Phelps et al., 2024; The Consortium for Children's Environmental Health, 2025; Zhao et al., 2023), their potentially disruptive and degenerative effects in the brain have profound and far-reaching consequences for the mental functioning of society. In addition, although these chemicals may affect outcomes in all age groups, children are fundamentally more vulnerable to toxic and disruptive chemicals due to their immature metabolic pathways, the rapid maturation and plasticity of their developing brain, and their lower body weight (Lanphear, 2015; Rauh and Margolis, 2016), with a growing body of evidence demonstrating their harmful impact on mental health in children (Bellinger, 2018; James and OShaughnessy, 2023).

Here we provide a narrative review of the associations between these chemicals and adverse neurodevelopmental and mental health outcomes, and a perspective on their impact in the context of changing trends in the cultivation, manufacturing, and packaging of food and beverages over the past few decades. We also discuss the challenges of researching the impact of environmental chemicals on the developing brain, show how the neuroscience and neuroimaging community has largely ignored this research topic, and highlight the urgent need for more funding and research to understand the risks to the developing brain so that effective regulations can be put in place.

2. The impact of toxins in our food environment on brain tissue and function

2.1. Agricultural pesticides

In 2022, 3.7 million metric tons of agricultural pesticides were applied to crops worldwide to control pests, weeds, and diseases (FAO, 2024). In turn, these pesticide residues enter the food chain and can be found in bread, fruit, and vegetables, with their concentration varying depending on food type and country (Ahmadi et al., 2024a, 2024b; Claydon, 2017; Environmental Working Group, 2024; Heinrich Böll Foundation et al., 2022; Keklik et al., 2025; Poulsen et al., 2017; Sinha et al., 2012). Over the past few decades, numerous studies and biomonitoring programs have found multiple pesticides (e.g. pyrethroids, organophosphates, neonicotinoids) and/or their metabolites in a range of bodily fluids (e.g., urine, blood/plasma, amniotic fluid, umbilical cord serum, and breast milk) as well as in cerebrospinal fluid and the brain, indicating that they can traverse around the body, and cross both the

Table 1
Cross-section of recent literature showing the presence of pesticides and pesticide metabolites in urine, blood, plasma, cerebrospinal fluid, umbilical cord serum, amniotic fluid, breast milk, and the brain.

	Name of pesticide or pesticide metabolite	References
Urine	*Organophosphates:* chlorpyrifos, 3,5,6-Trichloro−2-pyridinol (TCPy), diazinon, terbufos, dialkyl phosphates (DAPs) metabolites [e.g. diethyl phosphate (DEP), diethylthiophosphate (DETP), dimethylphosphate (DMP) and dimethylthiophosphate (DMTP)], pirimiphos-methyl *Neonicotinoids:* acetamiprid, N-desmethyl-acetamiprid, imidaclothiz, clothianidin, flonicamid, thiamethoxam, dinotefuran *Pyrethroids:* imiprothrin, cypermethrin 3-Phenoxybenzoic acid (3-PBA), 4-Formylphenylboronic acid (4-FPBA), trans 3-(2,2-dichlorovinyl)−2,2-dimethylcyclopropane carboxylic acid (trans-DCCA), *cis*-(2,2-dibromovinyl)−2,2-dimethylcyclopropane−1-carboxylicacid (cis-DBCA) *Organochlorides:* chlorpropham, hexachlorobenzene (HCB) *Carbamates:* chlorpropham	(Bao et al., 2020; Berman et al., 2020; Brahmand et al., 2019; Guzman-Torres et al., 2023; Huen et al., 2012; Ichikawa et al., 2019; Laubscher et al., 2022; Li et al., 2022; Li et al., 2023; Martins et al., 2023; Muñoz-Quezada et al., 2020; Norén et al., 2020; Ottenbros et al., 2023; Simaremare et al., 2020a; Suwannarin et al., 2023; Tang et al., 2024; Zhao et al., 2023)
Blood/Plasma	*Organophosphates:* chlorpyrifos, diazinon, terbufos, *Neonicotinoids:* N-desmethyl-acetamiprid, thiamethoxam, Imidacloprid, dinotefuran, clothianidin *Pyrethroids:* cypermethrin, imiprothrin *Organochlorides:* dichlorodiphenyltrichloroethane (DDT), dichlorodiphenyldichloroethylene (DDE), HCB, pentachlorophenol *Others:* biphenyl, diphenylamine, pyrene	(Han et al., 2023; Huen et al., 2012; Kaya et al., 2022; Laubscher et al., 2022; Qu et al., 2024; Simaremare et al., 2020b; H. Zhang et al., 2022a; Zhang et al., 2023a; Zhao et al., 2023)
Cerebrospinal fluid	*Organophosphates:* Triethyl phosphate (TEP), Tri(2-chloroethyl) phosphate (TCEP), Tri(1-chloro−2-propyl) phosphate (TCIPP), Tri-phenyl phosphate (TPHP), 2-Ethylhexyl di-phenyl phosphate (EHDPP) *Neonicotinoids:* N-desmethyl-acetamiprid, sulfoxaflor, thiamethoxam, imidacloprid *Pyrethroids:* No in-vivo studies identified in humans *Organochlorides:* HCB *Others:* biphenyl, diphenylamine	(Hou et al., 2022; Laubscher et al., 2022; Zhao et al., 2023)
Brain	*Organophosphates:* No in-vivo studies identified in humans *Neonicotinoids:* No in-vivo studies identified in humans *Pyrethroids:* No in-vivo studies identified in humans *Organochloride:* aziridine, benzyloxy bis-trifluoro-methyl aziridine, *Carbamates:* bis-O-methyl oxime, butanol-O-methyl-oxime, methoxy-phenyl-oxime, acetamide oxime (metabolites of aldicarb and dioxocarb) *Other:* 1,2,4 triazolol, 6-chloro-pyridazinone	(Cresto et al., 2023; Dewailly et al., 1999; Louati et al., 2023)
Umbilical cord serum/blood	*Organophosphates:* chlorpyrifos, diazinon *Neonicotinoids:* imidacloprid, *N*-desmethyl-acetamiprid *Pyrethroids:* trans-DCCA cis-DCCA,	(Abdel Hamid et al., 2020; Huen et al., 2012; Kaya et al., 2022; Prahl et al., 2021; Santos et al.,

(*continued on next page*)

Table 1 (*continued*)

	Name of pesticide or pesticide metabolite	References
	transchrysanthemum dicarboxylic acid (t-CDCA), cis−3-(2,2-dibromovinyl)−2,2-dimethylcyclopropane carboxylic acid (c-DBCA), 4-fluoro−3-phenoxybenzoic acid (FPBA), and 3phenoxybenzoic acid (3PBA) *Organochlorides:* DDT, DDE, Hexachlorocyclohexane (HCH), heptachlor *Carbamates:* bendiocarb	2022; Wren et al., 2021; Yang et al., 2021; Zhang et al., 2022b)
Amniotic fluid/ placenta	*Organophosphates:* chlorpyrifos, DAPs (e.g. diethylphosphate, dimethylphosphate, dimethylthiophosphate) *Neonicotinoids:* No in-vivo studies identified in humans *Pyrethroids:* No in-vivo studies identified in humans *Organochlorides:* 2,5-dichlorophenol, pentachlorophenol, DDT, DDD, DDE 2,2′,4,5,5′-Pentachlorobiphenyl (PCB−101), HCH, heptachlor and endrin *Carbamates:* carbofuranphenol Other: naphthol (naphthalene, metabolites) ortho-phenylphenol	(Anand and Taneja, 2020; Barmpas et al., 2020; Bradman et al., 2003; Dusza et al., 2022; Koutroulakis et al., 2014; Rodriguez et al., 2023)
Breast milk	*Organophosphates:* chlorpyrifos, TCPy *Nicotinoids:* thiamethoxam, imidacloprid, clothianidin, acetamiprid-*N*-desmethyl *Pyrethroids:* Cypermethrin, cyhalothrin, permethrin, esfenvalerate/fenvalerate *Organochlorides:* aldrin, dieldrin, endrin, HCB, Endosulfan, HCH, DDT, DDE, DDD, chlordane, heptachlor, mirex, methoxychlor, dechlorane plus	(Al Antary et al., 2021; Brahmand et al., 2019; Chávez-Almazán et al., 2020; Chen et al., 2020; Corcellas et al., 2012; EL-Saeid et al., 2021; Figueiredo et al., 2024; Kao et al., 2019; Kuang et al., 2020; Mehta et al., 2020; Müller et al., 2019; Pan et al., 2020; Santos et al., 2022; Souza et al., 2020; Witczak et al., 2021; Zhang et al., 2023b)

placenta and blood-brain barrier (see Table 1 for a cross-section of recent literature). Concurrently, studies investigating the impact of pesticides on neurodevelopmental outcomes in children and adolescents have shown associations with cognitive, social, and language deficits (Engel et al., 2016; Furlong et al., 2017, 2014; He et al., 2022; Ntantu Nkinsa et al., 2023; Qi et al., 2022; Ramos et al., 2023; Sagiv et al., 2023) as well as altered cortical activation, white matter microstructure, and functional connectivity (Binter et al., 2022; Gao et al., 2024; Sagiv et al., 2024; Van den Dries et al., 2020) [see (Andersen et al., 2022; Elser et al., 2022; James and OShaughnessy, 2023; Reed et al., 2023a; Shekhar et al., 2024) for some recent reviews]. At the neurobiological level, pesticides are also known to be endocrine disruptors and disruptors of the gut-brain axis (Gama et al., 2022; Khoo et al., 2024; Mazuryk et al., 2024; Mnif et al., 2011; Rueda-Ruzafa et al., 2019), and to have various modes of neurotoxicity and neuro-disruption that include targeting nicotinic acetylcholine receptors (e.g. neonicotinoids) (Cimino et al., 2017; Costas-Ferreira and Faro, 2021), voltage-gated sodium channels (e.g. pyrethroids) (Field et al., 2017; Tang et al., 2018), and other neurotransmitter pathways (e.g. per- and polyfluoroalkyl substances (PFAS)-containing pesticides) (Brown-Leung and Cannon, 2022; Nannaware et al., 2024). Taken together, these findings highlight the pervasive presence of pesticide residues in the human body and their potential to interfere with neurodevelopmental processes through multiple biological pathways.

2.2. Heavy metals in groundwater and soil

Heavy metals, such as cadmium, lead, copper, chromium, mercury, manganese, and arsenic are found in trace quantities in the environment with several also being essential minerals. However, they are also released into the environment as byproducts of various industrial processes including mining, smelting, electroplating, waste discharge, chemical manufacturing, and fuel combustion, as well as being present in agricultural pesticides and fertilizers. As a result, they accumulate in soils and seep into surface and groundwater (Alengebawy et al., 2021; European Environment Agency, 2025; Rai et al., 2019; Tóth et al., 2016; Yuan et al., 2021; Zeng et al., 2023; Zhou et al., 2020), entering both the food chain (Angon et al., 2024; European Food SafetyAuthority (EFSA) et al., 2021; Bair, 2022; Godebo et al., 2023; Koch et al., 2022; Rai et al., 2019; Rusin et al., 2021; Scutarașu and Trincă, 2023; Sharma and Nagpal, 2020) and freshwater drinking supplies (Chowdhury et al.,

Table 2
Cross section of recent literature reporting the presence of arsenic, cadmium, lead, and mercury in urine, blood, cerebrospinal fluid, umbilical cord serum, amniotic fluid, breast milk, and the brain. Note: manganese, chromium, and copper are not included as they are essential minerals.

	Arsenic	Lead	Mercury	Cadmium
Urine	(Buekers et al., 2023; Ellingsen et al., 2023; Hudgens et al., 2016; Middleton et al., 2016)	(Kim et al., 2020; Sallsten et al., 2022)	(Castaño et al., 2019; Kim et al., 2020; So et al., 2021)	(Adams and Newcomb, 2014; Lu et al., 2024)
Blood/ Plasma	(Del Rio et al., 2017; Ellingsen et al., 2023; Ettinger et al., 2017; Mullin et al., 2019; Shapiro et al., 2015)	(Akan, 2014; Ericson et al., 2021; Hauptman et al., 2021; Victory et al., 2019)	(Basu et al., 2018; Castaño et al., 2019; Li et al., 2024; Sharma et al., 2019; So et al., 2021; Sun et al., 2021)	(Akan, 2014; Garner and Levallois, 2017; Z. Li et al., 2022; Martins et al., 2020; Yang et al., 2022)
Cerebrospinal fluid	(Wu et al., 2023)	(Kamalian et al., 2023; Vinceti et al., 2017; Wu et al., 2023)	(Vinceti et al., 2017; Wu et al., 2023)	(Vinceti et al., 2017; Wu et al., 2023)
Brain	(Larsen et al., 1979)	(Pamphlett et al., 2023)	(Björkman et al., 2007; O'Donoghue et al., 2020; Pamphlett et al., 2023)	(Lech and Sadlik, 2017)
Umbilical cord serum/Blood	(Ettinger et al., 2017; Hu et al., 2015; Iwai-Shimada et al., 2019; Li et al., 2019; Navasumrit et al., 2019; Xu et al., 2016)	(García-Esquinas et al., 2013; Heiss et al., 2020; Irwinda et al., 2019; Mahdi et al., 2023; Rouzi et al., 2024)	(García-Esquinas et al., 2013; Irwinda et al., 2019; Kozikowska et al., 2013; Sharma et al., 2019)	(García-Esquinas et al., 2013; Hu et al., 2015; Salpietro et al., 2002)
Amniotic fluid/ placenta	(Jalali and Koski, 2018; Johnson et al., 2019; Kocyłowski et al., 2019; Ovayolu et al., 2020)	(Al-Saleh et al., 2011; Kocyłowski et al., 2019; Ovayolu et al., 2020)	(Al-Saleh et al., 2011; Ovayolu et al., 2020)	(Al-Saleh et al., 2011; Kocyłowski et al., 2019; Ovayolu et al., 2020)
Breast milk	(Bzikowska-Jura et al., 2024; Kumar et al., 2024; Linares et al., 2024; Motas et al., 2021; Samiee et al., 2019; Sharafi et al., 2023)	(Bzikowska-Jura et al., 2024; Koyashiki et al., 2010; Mohammadi et al., 2022; Motas et al., 2021; Naspolini et al., 2024; Park et al., 2018; Sharafi et al., 2023)	(Bzikowska-Jura et al., 2024; Cherkani-Hassani et al., 2019; Mohammadi et al., 2022; Motas et al., 2021; Park et al., 2018; Sharma et al., 2019)	(Bzikowska-Jura et al., 2024; Motas et al., 2021; Sharafi et al., 2023; Shawahna et al., 2023)

2016; Kumar et al., 2023; Santucci and Scully, 2020). For example, data from the LUCAS Topsoil Survey demonstrated that 137,000 km^2 (6 %) of agricultural land in the EU contained concentrations of at least one heavy metal above the guideline value (Tóth et al., 2016), while it is estimated that 94–220 million individuals worldwide may be exposed to high levels of arsenic in their domestic water supply through groundwater sources (Podgorski and Berg, 2020). Although some heavy metals (e.g. chromium, manganese, copper) are essential minerals and therefore deficiencies are associated with adverse health outcomes (Nakamura et al., 2019; Shen et al., 2024), at higher concentrations they are neurotoxic and neuro-disruptive (Bulcke et al., 2017; Feng et al., 2023; Harischandra et al., 2019; Miah et al., 2020; Morris and Levenson, 2017; Wise et al., 2022) and associated with adverse neurocognitive outcomes in both adults and children (Caparros-Gonzalez et al., 2019; J. Chen et al., 2023; Ni et al., 2018; Squitti et al., 2024; Tyler and Allan, 2014). For example, excess manganese exposure arising from industrial waste seepage into drinking water has been associated with structural changes to the brain (Lao et al., 2017), impaired cognitive and motor functioning (Bjørklund et al., 2017; Lucchini et al., 2017), and increased symptoms of attention-deficit/hyperactivity disorder (ADHD) (Schullehner et al., 2020), while excess copper has been associated with symptoms of depression (Chen et al., 2023; Ni et al., 2018).

In addition, heavy metals that are not essential minerals, such as lead, mercury, arsenic, and cadmium, have been detected in numerous body fluids including blood, umbilical cord blood, amniotic fluid, breast milk, and cerebrospinal fluid, as well as in the brain (see Table 2 for a cross section of recent literature). While reported levels vary across studies, there is no known safe level of lead and mercury in children, while for arsenic there is currently no agreed safe level. For cadmium, guidelines vary with the United States Food and Drug Administration (FDA) setting a toxicological reference value (TRV) range for cadmium of 0.21–0.36 μg / kg of body weight / day, while the European Food Safety Authority (EFSA) considers exposure of between 0.3 and 8 μg / kg of body weight / day as a low-level health risk. Concurrently, studies have shown that these heavy metals are metabolically and developmentally neurotoxic (Aaseth et al., 2020; Balali-Mood et al., 2021; Chandravanshi et al., 2021; Gonçalves et al., 2021; Lasley, 2018; Mochizuki, 2019; Sanders et al., 2009; Tolins et al., 2014; Van Wijngaarden et al., 2017; Virgolini and Aschner, 2021) and exposure has been associated with a multitude of adverse mental health outcomes in both children and adults including greater risk of depression and anxiety (Bai et al., 2024; Bouchard et al., 2009; Reuben et al., 2019; Scinicariello and Buser, 2015; L. Zhang et al., 2024a), psychopathology (Ayuso-Álvarez et al., 2019; Reuben et al., 2019), neurodevelopmental disorders [e.g. autism spectrum disorder (ASD), ADHD (Amadi et al., 2022; Ding et al., 2023; Gu et al., 2024)], and cognitive deficits (Althomali et al., 2024; Chen et al., 2023; Ciesielski et al., 2012; Heng et al., 2022; Liu et al., 2025; Stein et al., 2022). For example, exposure to lead in childhood is associated with greater psychopathology in adulthood (Reuben et al., 2019) while maternal exposure to methylmercury through eating contaminated fish and seafood results in transplacental exposure that adversely affects neurodevelopment (Van Wijngaarden et al., 2017). As with pesticides, these findings underscore the pervasive presence of heavy metals in the environment and their capacity to infiltrate the body and brain, contributing to a range of neurodevelopmental and mental health challenges.

2.3. Additives and ingredients in UPFs

A third source of neurotoxic and neuro-disruptive chemicals are the additives and ingredients in convenience UPFs that are typically not found in whole foods (Monteiro et al., 2019). These products range from snacks and breakfast cereals to fizzy drinks, fast food, and ready meals but also include infant formula and baby foods (Dunford and Popkin, 2023). These are intentionally added to food and beverages during manufacturing to improve their flavor, mouthfeel, palatability, and/or shelf life and include emulsifiers, sweeteners, colors, flavorings, and preservatives, with several (e.g. potassium bromate, propylparaben, aspartame, propyl gallate, sodium nitrite) identified as chemicals of health concern (EWG, 2024). Today, adolescents are the highest consumers of UPFs, accounting for 50 %–66 % of adolescent total energy intake in the United Kingdom (UK), United States (US), and Brazil (Chavez-Ugalde et al., 2024; D'Avila and Kirsten, 2017; Wang et al., 2021; Zhang et al., 2022). Studies examining the impact of diets high in UPFs on mental health outcomes in adolescents have shown that higher UPF consumption is associated with poorer mental health as well as higher prevalence of depressive symptoms and internalizing and externalizing problems (Faisal-Cury et al., 2022; Mesas et al., 2022; Reales-Moreno et al., 2022). In addition, maternal diets high in UPFs, saturated fats, and total sugars can adversely affect a child's cognitive development (de de de Lauzon-Guillain et al., 2022; Puig-Vallverdú et al., 2022; Zupo et al., 2024). Similarly, in adult populations, multiple lines of evidence indicate that higher consumption of UPFs are significantly associated with increased symptoms of depression, heightened challenges with emotional/cognitive control, and cognitive decline (Bala et al., 2025; Gomes Gonçalves et al., 2023; Lane et al., 2024, 2022), effects that cannot be explained by differences in exercise habits,

Table 3
Studies reporting the presence of microplastics, bisphenol A, and phthalates in urine, blood, plasma, cerebrospinal fluid, umbilical cord serum, amniotic fluid, breast milk and the brain.

	Microplastics	Bisphenol A	Phthalates
Urine	(Barceló et al., 2023; Massardo et al., 2024; Pironti et al., 2023; Rotchell et al., 2024; Song et al., 2024)	(González et al., 2019; Lee et al., 2018; Lehmler et al., 2018; Shiue, 2014; Vandenberg et al., 2014)	(Bräuner et al., 2022; Carwile et al., 2022; Fruh et al., 2022; Genuis et al., 2012; Shiue, 2014; Smith et al., 2022; Vieyra et al., 2023; Vogel et al., 2023)
Blood/ Plasma	(Barceló et al., 2023; Guan et al., 2023; Leslie et al., 2022; V L Leonard et al., 2024)	(González et al., 2019; Lee et al., 2018; Liu et al., 2017; Vandenberg et al., 2010)	(Bräuner et al., 2022; Genuis et al., 2012; Kolatorova et al., 2018; X. Zhang et al., 2024b)
Cerebrospinal fluid	(Xie et al., 2024)	(Zhang et al., 2024)	(Agin et al., 2020; Zhang et al., 2024)
Brain	(Amato-Lourenço et al., 2024; Campen et al., 2024; Nihart et al., 2025)	(Charisiadis et al., 2018; Geens et al., 2012; van der Meer et al., 2017)	No studies found
Umbilical cord serum/ Blood	(Sun et al., 2024; Zhu et al., 2024)	(Ikezuki et al., 2002; Lee et al., 2018; Liu et al., 2017; Vandenberg et al., 2010)	(Al-Saleh et al., 2024; Hwa et al., 2022; Kolatorova et al., 2018)
Amniotic fluid/ placenta	(Halfar et al., 2023; Ragusa et al., 2021; Tian et al., 2025; Xue et al., 2024)	(Edlow et al., 2012; Ikezuki et al., 2002; Loukas et al., 2023; Pinney et al., 2017; Vandenberg et al., 2010; Yamada et al., 2002; Zbucka-Krętowska et al., 2019)	(Bräuner et al., 2022; Golestanzadeh et al., 2022; Jensen et al., 2012; Katsikantami et al., 2020; Liang et al., 2023; Silva et al., 2004)
Breast milk	(Barceló et al., 2023; Ragusa et al., 2022; Saraluck et al., 2024)	(Lee et al., 2018; Sun et al., 2004; Vandenberg et al., 2010; Zimmers et al., 2014)	(Main et al., 2006)

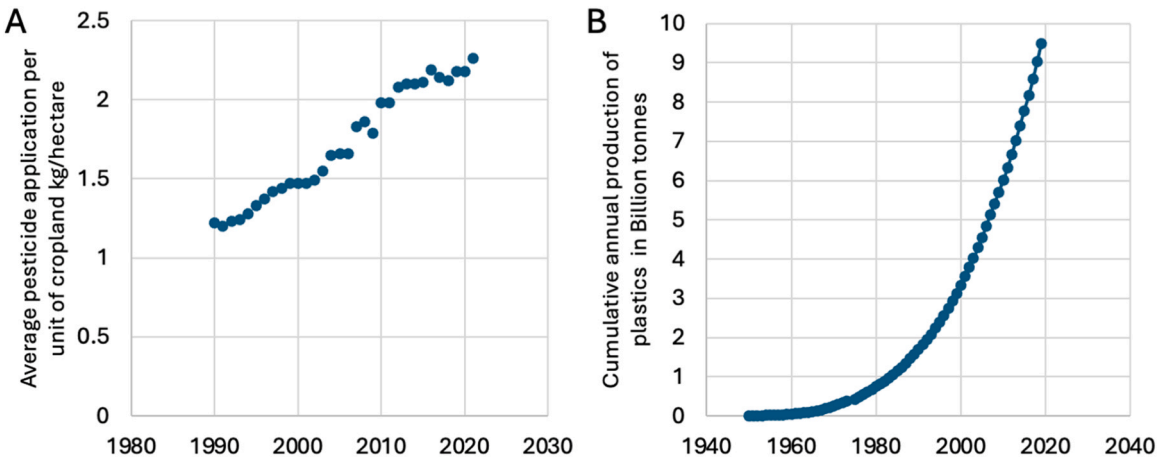

Fig. 1. (A) Average pesticide application per unit of cropland, measured in kilograms per hectare (global estimates) (B) Cumulative annual production of plastics from 1950 to 2019. Source: Our World in Data, (Ritchie et al#, 2023b, 2023a).

income, or life adversity. For example, evidence suggests that up to a third of mental distress experienced in the general population could be associated with UPF consumption, for some demographics and geographies (Bala et al., 2025). At the neurobiological level, studies have shown that frequent UPF consumption disrupts the gut microflora, and in turn the gut-brain axis (Martínez Leo and Segura Campos, 2020; Song et al., 2023) and is associated with myelin degradation (Mannino et al., 2023), structural brain changes (Contreras-Rodriguez et al., 2023), and disruption of reward signalling in the brain [see (Contreras-Rodriguez et al., 2022) for a recent review]. Collectively, these findings highlight how consumption of UPFs and their associated additives can disrupt key neurobiological processes and impact both neurodevelopmental and mental health outcomes.

2.4. *Microplastics, bisphenols, and phthalates from plastic packaging*

A fourth source of neurotoxic chemicals in food and beverages is their plastic packaging. It has been estimated that, in some markets, approximately 40 % of food and beverages are packaged in plastic (ING, 2019) due to its low cost, versatility, and utility in relation to heat and water resistance, ease of transportation, and elongation of shelf life. However, it is also known that microplastics and other packaging-derived chemicals such as bisphenols (e.g. bisphenol A) and plasticizers (e.g. phthalates) can migrate into the packaged foods and beverages, including fruit, vegetables, meat, seafood, milk formula, and water (Manzoor et al., 2022; Martín-Carrasco et al., 2023; Oliveri Conti et al., 2020; Qian et al., 2024; Udovicki et al., 2022; Vitali et al., 2023). In addition, long-term storage, refrigeration, or heating of food or water in plastic containers can accelerate this migration. For example, a recent study showed that plastic baby food containers can release up to 4 million microplastic and 2 billion nanoplastic particles from only one square centimeter of plastic area within 3 min of microwave heating (Hussain et al., 2023). Correspondingly, microplastics, bisphenols, and phthalates have been found in numerous body fluids (e.g. blood, urine, cerebrospinal fluid, umbilical cord serum, amniotic fluid, and breast milk) as well as in the brain, where they can be present at higher concentrations than in kidney and liver (Campen et al., 2024), and indicating that they are able to traverse both the placenta and blood-brain barrier (Table 3). For example, it has been estimated that eating take-out food 4 −7 times per week can result in a person ingesting 12 −203 pieces of microplastics per week, while an individual consuming an American diet can ingest between 39,000 and 52,000 pieces of microplastics from food and beverages each year (Cox et al., 2019; Du et al., 2020). Studies investigating the neurotoxicity as well as the neurodevelopmental and mental health impacts of these chemicals have, to date, focused mainly on bisphenols (e.g. bisphenol A, C, and F) and phthalates, with only a limited number of studies on microplastics [although see (Prüst et al., 2020)]. They have demonstrated that bisphenols and phthalates act as endocrine disruptors with multifaceted effects. These include mimicking endogenous oestrogen hormones; disrupting androgen and thyroid receptors (Gore et al., 2019; Moriyama et al., 2002); impairing microglial function (Rosin and Kurrasch, 2018); interfering with brain-derived neurotrophic factor (BDNF) signaling (Mustieles et al., 2022), the hypothalamic-pituitary-adrenal (HPA) axis (Giesbrecht et al., 2016), and the gut-brain axis (Balaguer-Trias et al., 2022); modulating epigenetic regulation (Alavian-Ghavanini et al., 2018); exacerbating neuroinflammation (Bjørklund et al., 2024); affecting hippocampal developmental plasticity (Holahan and Smith, 2015); and altering both white matter microstructure (England-Mason et al., 2020) and grey matter volumes (Ghassabian et al., 2023), to name a few. From a neurodevelopmental perspective, exposure to these chemicals, either in childhood or prenatally, has been associated with increased symptoms of ASD and ADHD (Bjørklund et al., 2024; Kim et al., 2022; Oh et al., 2024; Stein et al., 2015) as well as social and cognitive deficits (Engel et al., 2021; Ghassabian et al., 2023; Ham et al., 2024; Miodovnik et al., 2011; Rolland et al., 2023; Shoaff et al., 2023, 2019) [see (Ahn and Jeung, 2023; Costa and Cairrao, 2024; Hyun and Ka, 2024; Radke et al., 2020) for some recent reviews].

3. Growing exposure to environmental toxins

Although inorganic pesticides have been used in agriculture since about the 1940s, their production has expanded rapidly over the past few decades (Fig. 1A) (Carvalho, 2017; FAO, 2024; Heinrich Böll Foundation et al., 2022; Ritchie et al., 2023a). This growth reflects not only increases in the total area of treated farmland (which includes repeat applications to the same area) but also in the number of different active ingredients applied to an area. For example, in the UK, the total treated area increased from 59 million spray hectares in 2000, to 73 million spray hectares in 2016 – a rise of 24 % – while the number of active ingredients increased from 12.8 per hectare in 2000 to 15.9 per hectare in 2016 (Friends of the Earth, 2019). In addition, there has been a shift in the specific pesticides applied. For example, several classes of pesticides have grown in use over the past two decades in response to changing regulatory pressures and disease resistance (Craddock et al., 2019; Umetsu and Shirai, 2020). These include neonicotinoid insecticides (now banned for use in the UK and European Union (EU) but still the most commonly used insecticide globally), PFAS pesticides (Donley et al., 2024; PAN Europe, 2024; Simon-Delso et al., 2015), and pyrethroid pesticides (PAN Europe, 2024; Perry and Moschini, 2020).

For example, between 2000 and 2016 (prior to its ban in 2018), the weight of neonicotinoids applied to all crops in the UK increased from 26,404 kg to 87,704 kg – an increase of 232 % – while French sales of PFAS-containing pesticides tripled from 2008 to 2021 (from 700 to over 2000 tonnes) (PAN Europe, 2024). In the US in 2017, 23 % and 14 % of all approved pesticide active ingredients were organofluorines and PFAS, respectively, with 61 % and 30 % of those being approved in the prior 10 years (Donley et al., 2024). Moreover, more than 50 % of soybean acres and more than 90 % of maize acres are now treated with neonicotinoid pesticides in the US (Perry and Moschini, 2020). These trends have also played out in the presence of pesticide residues found in food. For example, in the EU, the proportions of fruit and vegetables containing residues of PFAS pesticides have risen by 220 % and 247 % between 2011 and 2021, respectively (PAN Europe, 2024), while the number of PFAS pesticides detected in food has increased dramatically from 24 in 2006 to 412 in 2022 (Poulsen et al., 2024).

However, it is not just agricultural practices that have changed over the past few decades. During the same time, there has also been a rise in the manufacturing and consumption of convenience and fast foods in many regions of the globe (Baker et al., 2020; Juul et al., 2022; Juul and Hemmingsson, 2015; Matos et al., 2021; Reardon et al., 2021; Wang et al., 2021) with the UPF market projected to grow by $856.6 billion at a compound annual growth rate (CAGR) of 9 % between 2024 and 2029 (Technavio, 2025). For example, a large-scale study examining trends in UPF consumption in adolescents in the US between 1999 and 2018 showed that the estimated percentage of total energy from UPF consumption increased from 61 % to 67 % whereas the percentage of total energy from consumption of unprocessed or minimally processed foods decreased from 29 % to 23 % (Wang et al., 2021). At the same time, the proportion of food products containing additives that were purchased by households in the US rose significantly from 49.6 % in 2001 to 59.5 % in 2019 while the proportion of purchased baby foods containing additives also increased by 20 % (Dunford et al., 2023). At the same time, the average number of additives in food and beverage products purchased by US households rose significantly from 3.7 in 2001 to 4.5 in 2019 ($p < 0.001$) while the proportion of purchased products containing one, two, or three additives increased by 2 %–5 % between 2001 and 2019, and the proportion of products containing zero additives decreased by approximately 11 % (45 to 34 %) (Dunford et al., 2023). These trends are played out in the global food additive market, which is growing at an annual rate of ~6 % (Grand View Research, 2025; MarketsandMarkets, 2023).

Relatedly, this rise in consumption of plastic-wrapped convenience foods with longer shelf-lives and fast foods in single-use plastic containers means that food and beverages increasingly come into contact with plastic during their manufacturing, transportation, and storage, increasing the risk of human exposure. For example, a recent report from Environmental Defense in Canada found that more than 70 % of products in Canada's produce and baby food aisles are packaged in plastic, with the amount of baby food packaged in plastic increasing by 6 % between 2022 and 2024 (Environmental Defense, 2024). In addition, an estimated average of 13 billion microplastic particles are released daily into waterways in the US via municipal wastewater (Mason et al., 2016), while 63,000–430,000 and 44,000–300,000 tons of microplastics are estimated to be added to farmlands in Europe and North America, respectively, each year, further contributing to exposure (Nizzetto et al., 2016). With plastic use and waste production continuing to rise at an unprecedented rate [Figure1B (Geyer et al., 2017; Ritchie et al., 2023b)], the cumulative and persistent nature of plastic exposure in food systems and the environment suggests that human exposure to plastic-derived chemicals will only intensify in the coming decades.

In addition, while this review focuses on food and beverages, it is important to acknowledge that children and adolescents are also exposed to numerous other neuro-disruptive chemicals in daily life, including from clothing, air pollution, toys, and personal care products, whose combined and poorly understood interactions may further impact brain development (Ageel et al., 2024; Fadaei, 2023; Reuben et al., 2021; The Consortium for Children's Environmental Health, 2025; Tung et al., 2024; Wang et al., 2019; Xia et al., 2022).

4. An urgent need to fill knowledge gaps and overcome methodological challenges

This rapid and cumulative exposure to a growing range of toxins that begins in gestation, along with growing evidence of their neurotoxic and neuro-disruptive impacts on the brain, support the hypothesis that our food environment is progressively degrading neural and metabolic function in the brain, with cascading consequences for psychological and cognitive health. In addition, the heightened vulnerability of children to these toxic and disruptive chemicals (Lanphear, 2015; Rauh and Margolis, 2016) puts them at the forefront of these risks. This positions the toxicity of our food environment and systems as a key candidate contributing to the rapid decline in various facets of mental health and wellbeing in young people (Blanchflower, 2025; Blanchflower et al., 2024c; CDC, 2023; Helliwell et al., 2024; Keyes et al., 2019; Sapien Labs, 2025). It may also account for some of the unexplained components highlighted in previous research (Bala et al., 2024). While the links between environmental exposures and chronic health conditions such as cancer, obesity, and diabetes are better established (Phelps et al., 2024; The Consortium for Children's Environmental Health, 2025; Zhao et al., 2023), diseases such as cancer (where early onset under age 40 has increased by an estimated 79 % between 1990 and 2019) are typically perceived as a binary outcome, either present or absent, and often considered a future risk. In contrast, the progressive degradation of the brain and nervous system functioning, driven by increasing exposure to neurotoxic and neuro-disruptive chemicals, poses an immediate and pervasive public health issue that is an existential threat to the very fabric of human society and its progress.

To tackle this growing public health crisis, there are still major gaps in our basic knowledge that need to be filled and multiple challenges to overcome. For example, we still know very little about how the presence of micro- and nanoplastics in the body and brain impact neurodevelopmental and mental health outcomes. There are also many methodological challenges that currently stand in the way of progress. To start with, simply carrying out these kinds of studies is challenging due to the sheer number of different chemicals and metabolites involved from farm to table; the rapid metabolism of some chemicals; poor visibility of the chemicals actually added (intentionally or unintentionally) to a product; the fact that chemicals are typically present in mixtures and not individually; the costs, complexities, and practicalities of assaying and analyzing the levels of these chemicals in biological samples; the complexities of determining human exposure levels when they are often from multiple sources over a lifetime; quantifying the disruption of neural systems when they do not necessarily result in cell death; and relating exposures to neurobiological and behavioral outcomes. For example, assays for the detection and quantification of most toxins are not readily available in commercial labs, particularly for plastics and PFAS in human blood, and are expensive at several hundred dollars per sample. In addition, within the existing literature, there is currently an extensive variety of methodologies used to measure the impacts of environmental toxins and neurotoxic chemicals. For example, studies trying to understand the impacts of pesticides on children vary in terms of sampling approaches (e.g. 1-spot vs 3-spot sampling during different trimesters of pregnancy), the demographic and clinical profile of the children in the study, the storage and analysis of samples, the specific outcomes studied, and the assessments and questionaries used [e.g. (Reed et al., 2023b)]. This lack of standardization makes it hard to aggregate across studies (which often have relatively small sample sizes), gives inconsistent results, and causes studies to be ignored by regulators due to methodological weaknesses. For example, a number of studies investigating the impacts of pesticides, heavy metals, bisphenols, and phthalates on neurodevelopmental outcomes report null or

Table 4
The number of articles with the following search terms anywhere in the article published over the past 10 years (January 01, 2015–December 31, 2024) in top ranking neuroscience, neuroimaging, and psychiatry journals.

Journal	pesticides	"heavy metals"	"ultra-processed foods"	"food additives"	microplastics	"bisphenol A"	"endocrine disruptors"	Estimated total number of published articles on all topics
Journal of Neuroscience	16	20	0	4	0	9	0	9445
Neuroimage	3	11	0	0	1	0	1	8690
Nature Neuroscience	2	5	1	1	1	1	3	2588
Neuron	13	6	0	1	1	4	0	4542
Brain	18	10	0	1	1	0	0	4275
Cerebral Cortex	2	8	0	2	1	4	2	4328
Human Brain Mapping	2	0	0	0	0	0	0	4016
Trends in Neurosciences	9	5	0	0	0	2	1	973
World Psychiatry	4	5	0	0	0	0	0	1071
The Lancet Psychiatry	27	2	2	0	0	1	0	2940
JAMA Psychiatry	6	4	0	1	0	1	1	2462
American Journal of Psychiatry	6	0	0	0	0	0	0	1966
Journal of the American Academy of Child and Adolescent Psychiatry	6	1	0	3	0	1	1	1933

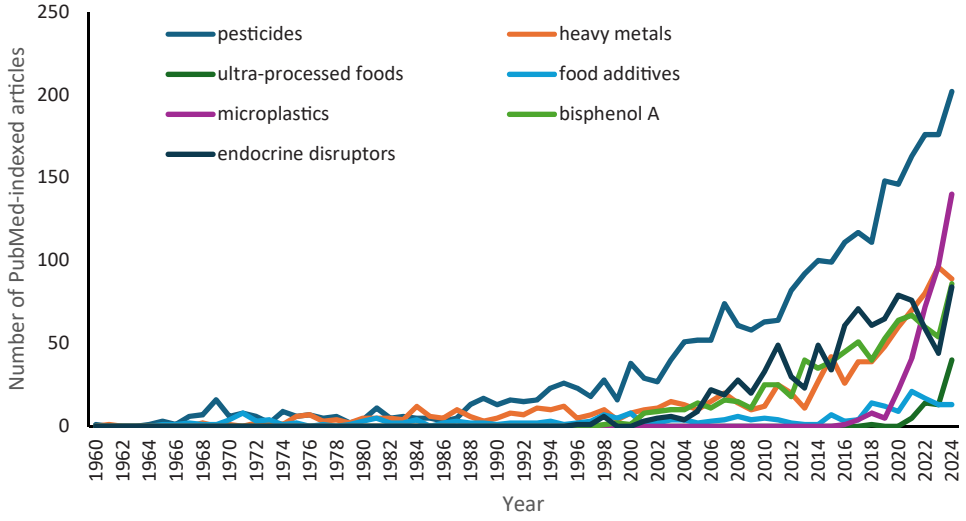

Fig. 2. Number of PubMed-indexed articles from 1960 to 2024 with each of the key terms (pesticides, "heavy metals", "ultra-processed foods", "food additives", microplastics, "bisphenol A", "endocrine disruptors") AND "neurotoxicity" OR "neurodevelopment" OR "brain" OR "mental health" OR "psychopathology" anywhere in the text.

inconsistent results [e.g. (Andersen et al., 2021; Boffetta et al., 2024; Cunha et al., 2023; Fage-Larsen et al., 2024; Gascon et al., 2015; Hall et al., 2022; Islam et al., 2022; Jensen et al., 2019; Joyce et al., 2022; Manley et al., 2022; Praveena et al., 2020; Sagiv et al., 2022; Tsuji et al., 2015)].

It is also of relevance that, to date, this topic has been largely ignored by the neuroscience, neuroimaging, and psychiatric communities, including funding bodies and journals. For example, Table 4 shows the number of articles containing relevant search terms relating to these chemicals in leading neuroscience, neuroimaging, and psychiatry journals, highlighting the minimal number of published studies on these topics in these journals over the past 10 years. Without increased engagement of the neuroscience community, progress in this area will be hindered, as their expertise is vital for investigating underlying mechanisms and evaluating the impact on brain function and behavior. In contrast, a growing number of articles on these topics have been published over the past decade (Fig. 2; Supplementary Table 1), primarily in journals focusing on neurotoxicity, the environment, or nutrition. Greater recognition of these gaps in mainstream neuroscience and mental health research is essential to ensure a more comprehensive understanding of the implications of these chemicals on brain function and mental health.

To help inform public policy and regulation, the many gaps in our understanding must be filled and multitude of methodological challenges must be overcome to obtain more conclusive evidence on the impacts of these chemicals on the brain. Although there are several large-scale national and regional biomonitoring initiatives and consortiums that track the presence of these chemicals in the general population [e.g. (Birnbaum et al., 2012; Marx-Stoelting et al., 2023; National Center for Environmental Health., 2022; Patisaul, 2020; Vorkamp et al., 2023)], they rarely combine understanding of neurophysiological and mental health outcomes and are insufficient for sounding the alarm on critical exposure levels, establishing pre-market testing guidelines and informing robust regulatory processes and guidance. In addition, given that exposure to toxins can vary substantially across geographies, this calls for more geographically distributed monitoring.

5. A call for action

There is an urgent need for further research on the impacts of these chemicals on the brain and mental health outcomes to show consistent evidence of the risks so that regulatory action can be taken. This includes

(i) systematic demonstration of the impacts on brain tissue and structure; (ii) larger scale cross-sectional studies in humans that assess the relationship between various toxin exposures, brain physiology, and functional deficits; (iii) longitudinal studies across the lifespan, particularly with respect to understanding the consequences of early childhood exposure; and (iv) replication of studies to resolve conflicting evidence. These insights will not only help address this growing public health crisis but will also expand our mechanistic understanding of the brain and its environment and shed light on fundamental neurophysiological processes that shape cognition and behavior.

Altogether, the potential consequences of a progressive deterioration of brain function poses a grave threat to the future wellbeing of society and requires a paradigm shift in research, regulation, and funding. In addition, it is imperative that the many challenges of studying these exposures does not deter research progress. To accelerate research and understanding of these impacts, funding bodies must prioritize large-scale, transdisciplinary research initiatives that engage neuroscientists, mental health professionals, environmental toxicologists, nutritional scientists, and policymakers. Future studies should systematically investigate the long-term neurodevelopmental effects of these exposures using standardized methodologies and large cohort designs and incorporating multi-level assessments spanning cellular mechanisms, molecular biomarkers, neuroimaging, and behavioral outcomes. Additionally, global biomonitoring efforts must be expanded to establish critical exposure thresholds, inform regulatory frameworks, and guide pre-market safety assessments for chemicals used in food production and packaging. Addressing this critical gap in research is essential for safeguarding the brain health and mental wellbeing of future generations before the consequences become irreversible.

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Declaration of Competing Interest

None declared.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.neubiorev.2025.106290.

Data availability

No data was used for the research described in the article.

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