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Environmental influences in the global incidence of attention deficit/hyperactivity disorder and autism spectrum disorder: A longitudinal analysis of historical trends (2010-2021) across 204 countries

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Abstract

Introduction: The global incidence of Attention-Deficit/Hyperactivity Disorder (ADHD) and Autism Spectrum Disorder (ASD) may be shaped by social and environmental factors.
Methods: Historical incidence trends from 204 countries were retrieved from the Global Burden of Disease initiative. Pearson's correlation (for the incidence of ADHD/ASD in 2021) and multivariable regression analyses (for the rate of change in ADHD/ASD incidence between 2010 and 2021) were conducted, adjusting for key socio-economic factors (i.e. purchasing power parity, life expectancy).
Results: The incidence of ADHD in 2021 was negatively correlated with indices of urbanization and industrialization (maximum r 0.359, p <0.001). whereas, the incidence of ASD showed a positive correlation with indicators of suboptimal healthcare delivery (maximum r 0.425, p <0.001). A slower increase in ADHD incidence was associated with reductions in neonaral infections (β 0.010, p 0.007), while improvements in sanitation (β 0.041, p 0.011) were associated with a faster rise in incidence for ASD.

Discussion: These findings suggest that macro-level factors such as urbanization, healthcare infrastructure, and sanitation may play a role in shaping global trends in neurodevelopmental disorders. Understanding these associations may inform public health strategies aimed at early identification and support for affected populations.

Take-home message: The global incidence of Attention-Deficit/Hyperactivity Disorder (ADHD) and Autism Spectrum Disorder (ASD) may be influenced by social and environmental factors. However, future research should more thoroughly account for potential confounders, such as increasing parental age, broader adoption of diverse dietary practices, and evolving diagnostic criteria to better understand the drivers behind these trends.
Key words: Air pollutants; diet and nutrition; endocrine disruptors; neurotoxins; reproductive health.

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INTRODUCTION

Neurodevelopmental disorders represent a spectrum of conditions resulting from complex interplay of biological, psychological and social factors [1]. These biopsychosocial interactions influence the development and maturation of motor, sensory, cognitive, and executive functions, which can ultimately contribute to the emergence of psychiatric or behavioral disorders [2]. While neurodevelopmental conditions are diverse, they often share overlapping biological and psychological overlap [3,4]. However, the incidence and prevalence of these neurodevelopmental conditions are shaped by heterogeneous and multifactorial influences [4,5].

In recent decades, the prevalence and incidence of neurodevelopment disorders, especially Attention Deficit/Hyperactivity Disorder (ADHD) and Autism Spectrum Disorders (ASD) have shown a consistent upward trend in the general population [6]. This increase is the subject of ongoing debate. On one hand, rising rates may reflect improved awareness, and reduced under-diagnosis, and expanded diagnostic criteria [7]. On the other, some argue that the growing burden may also reflect genuine increases in exposure to risk factors beyond improved detection alone [8,9].

Among the most influential contributors to this increase are lifestyle and socio-economic conditions [3]. However, risk factors may also extend beyond the individual to include broader environmental exposure, such as neurotoxic agents [8,10] and prenatal factors related to parental health and lifestyle [11–13]. Growing scientific consensus points to the role of environmental factors, such as ambient air pollution and exposure to industrial chemical agents as contributors to the rising prevalence of neurodevelopmental disorders [8,9,14]. These exposures are believed to influence neurodevelopment through mechanisms including heightened neuroinflammation [15], oxidative stress [16,17], and disruption in neurotransmitter or hormonal regulation [17], potentially mediated by epigenetic modifications [18].

Understanding how environmental factors contribute to the neurodevelopment conditions is a pressing and timley research priority, , especially in the context of of global climate change and its broader health impacts [19–23]. Despite increasing interest in this area, much of the current evidence is derived from cross-sectional or single-country studies [9,14]. To address this gap, the present study leverages global data from the Global Burden of Disease (GBD) initiative to explore environmental and social influences on the incidence of ADHD and ASD, which are the two prominently rising neurodevelopmental conditions at the global level [6].

Aims

The primary aim of this study was to assess the global association between lifestyle factors or environmental exposure, particularly to chemical agents and the incidence of neurodevelopmental conditions, using data from 204 countries included in the Global Burden of Disease (GBD) database.

As a secondary aim, the study evaluated the temporal relationship between lifestyle factors and changes in neurodevelopmental disorder incidence over time. , Specifically, it examined the concurrent rate of change in both risk factor exposure and incidence rates between 2010 and 2021 to identify key drivers of global temporal trends.

METHODS

Sample

Global incidence estimates for ADHD and ASD were obtained from the Global Burden of Disease (GBD) study coordinated by the Institute for Health Metrics and Evaluation IHME, 2025b; Murray, 2024). In addition to neurodevelopmental conditions, incidence data were also retrieved for a range of relevant disorders to account for potential comorbidities and public health context. These included: cannabis use disorders; cocaine use disorders; amphetamine use disorders; opioid use disorders; maternal obstructed labor and uterine rupture; neonatal preterm birth; maternal hypertensive disorders; maternal sepsis and other maternal infections; neonatal sepsis and other neonatal infections; maternal abortion and miscarriage; environmental heat and cold exposure; iodine deficiency [24,25].

Summary Exposure Values (SEV) were retrieved for risk factors, namely: ambient ozone pollution; nitrogen dioxide pollution; lead exposure; unsafe water source; unsafe sanitation, occupational exposure to asbestos; arsenic; benzene; beryllium; cadmium; chromium; diesel engine exhaust; formaldehyde; nickel; polycyclic aromatic hydrocarbons; silica; sulfuric acid; trichloroethylene, smoking; alcohol use; suboptimal breastfeeding; vitamin A deficiency; iron deficiency; zinc deficiency; diet low in legumes; diet low in calcium; diet low in fruits; diet low in vegetables; diet low in whole grains; nuts and seeds consumption; milk consumption; diet low in fiber; diet low in seafood omega-3 fatty acids; polyunsaturated fatty acids consumption - PUFAs; diet high in red meat; diet high in processed meat; diet high in sugar-sweetened beverages; diet high in trans fatty acids; diet high in sodium. To reflect trends observed in the last decade [6], incidence rates and SEVs were retrieved for 2010 and 2021.

To account for potential socioeconomic confounders, life expectancy at birth and Gross Domestic Product (GDP) per capita [26] were also retrieved for each country, for both 2010 and 2021. These data were sourced from Gapminder [26,27], which integrates datasets from multiple sources, including the World Development Indicators provided by the World Bank [28].

Measurements and outcomes

The primary outcome measure for this study was the incidence of ADHD and ASD, selected for its greater sensitivity to temporal and environmental influences compared to prevalence. Incidence rates were age standardized to enable comparability across countries and time points [25]. Similarly, Summary Exposure (SEVs) representing population-level exposures to specific risk factors were standardized by age and total population [25]. Gross Domestic Product (GDP) per capita was adjusted using the Purchase Power Capacity (PPP), and reported in constant 2021 international dollars (PPP$2021) to account for inflation and enable valid cross-national comparisons

Drawing from previous literature [9,10,14], and based on data availability, risk factors were grouped into into five major categories, namely: general environmental risk factors (ambient ozone pollution; nitrogen dioxide pollution; lead exposure; environmental heat and cold exposure; unsafe water source; unsafe sanitation), occupational exposure to chemicals (asbestos; arsenic; benzene; beryllium; cadmium; chromium; diesel engine exhaust; formaldehyde; nickel; polycyclic aromatic hydrocarbons; silica; sulfuric acid; trichloroethylene), exposure to drugs (smoking; alcohol use; cannabis use disorders; opioid use disorders; amphetamine use disorders; cocaine use disorders), peripartum factors (maternal obstructed labor and uterine rupture; neonatal preterm birth; maternal abortion and miscarriage; maternal hypertensive disorders; maternal sepsis and other maternal infections; neonatal sepsis and other neonatal infections; suboptimal breastfeeding), and dietary

factors (vitamin A deficiency; iron deficiency; zinc deficiency; iodine deficiency; diet low in legumes; diet low in calcium; diet low in fruits; diet low in vegetables; diet low in whole grains; nuts and seeds consumption; milk consumption; diet low in fiber; diet low in seafood omega-3 fatty acids; PUFA consumption; diet high in red meat; diet high in processed meat; diet high in sugar-sweetened beverages; diet high in trans fatty acids; diet high in sodium).

Following prior research, several variables were also interpreted as proxies for broader socio-environmental constructs. For instance, ozone and nitrogen dioxide pollution were considered indicators of urbanization and industrialization [29,30], while sub-optimal residential temperature were seen as markers of housing and environmental stress [31]. Diet high in sugar-sweetened beverages, trans-fatty acids and processed meat were used to reflect urban dietary transitions [32,33]. Additionally, suboptimal sanitation, maternal hypertensive disorders, and infectious complications during pregnancy and the neonatal period were classified as indicators of healthcare system limitations [34,35]. However, maternal and neonatal infections may also reflect the effects of urban-industrial environments, such as overcrowding and poor environmental controls [36]. Therefore, these variables were considered in both interpretive categories to reflect their multifactorial relevance.

Statistical methods

First, summary statistics for incidence rates of ADHD and ASD in 2021 were computed and reported (mean, standard deviation, maximum and minimum values). The same was computed and reported for PPP and life expectancy. Then, the change (calculated as a percentage) between 2021 and 1990 was computed and reported for all variables (incidence of ADHD, incidence of ASD, PPP, life expectancy).

For the primary aim, partial correlation coefficients (Pearson r) - adjusted for PPP and life expectancy - were computed for both the incidence in 2021 of ADHD and ASD and all five categories of risk factors. Statistical inflation due to multiple comparisons was addressed by calculating Bonferroni-corrected p-values within each category, rounded to the more conservative value at three digits (minimum p, general environmental: 0.008; occupational exposure: 0.003; exposure to drugs: 0.008; peripartum factors: 0.007; dietary factors: 0.002). Statistical significance was set at Bonferroni $p < 0.05$. For the secondary aim, multivariable regression coefficients were calculated. The dependent variable was represented by either i) ADHD incidence 2021-2010, in percentage or ii) ASD incidence 2021-2010, in percentage. The independent variable was represented by the change in exposure across risk factors, computed as the difference between 2021 and 2010 - in percentages, adjusted for the change in PPP and life expectancy across the same years. Only risk factors with significant correlations in the primary aims were investigated. Statistical significance was set at $p < 0.05$, but coefficients below $p < 0.10$ were also described to support future investigation on identified factors.

Ethics approval

According to Italian legislation, neither approval from the ethics committee nor informed consent from the study populations is required for population-level data acquired from third parties. Data retrieval, data manipulation and data analyses complied with data use agreements of both the Institute for Health Metrics and Evaluation [37], and Gapminder [26,38].

RESULTS

Data across 204 individual countries were retrieved and collated for analysis. On average, both PPP and life expectancy increased globally during the time of investigation (1990-2021). However, the incidence rates of ADHD and ASD did not demonstrate a uniform upward trend across all countries during the same period, indicating regional variability in diagnostic patterns or underlying risk factors. Detailed descriptive statistics are presented in Table 1.

Table 1. Descriptive statistics.

	Mean	Standard Deviation	Minimum	Maximum
ADHD (incidence 2021, age-standardized rates)	66.53/100,000	±39.04/100,000	23.58/100,000	185.04/100,000
ADHD (2021-2010, change %)	0.00%	±0.02%	-0.07%	0.08%
ASD (incidence 2021, age-standardized rates)	19.70/100,000	±3.38/100,000	14.25/100,000	34.52/100,000
ASD (change 2021-2010, %)	0.00%	±0.02%	-0.10%	0.14%
GDP (2021, USD$)	25,163.02 PPP$2021	±29,328.76 PPP$2021	25,163.02 PPP$2021	209,998.80 PPP$2021
GDP (2021-2010, change %)	18.37%	±28.68%	-72.71%	+122.91%
Life expectancy (2021, years)	71.59	±7.33	50.71	85.16
Life expectancy (2021-2010, change %)	2.28%	±7.64%	-4.90%	92.93%

Note: GDP per capita in constant PPP dollars. The unit is constant dollars adjusted for inflation in 2021 (PPP$2021).
Legend: ADHD = Attention Deficit/Hyperactivity Disorder, ASD = Autism Spectrum Disorders, GDP = Gross Domestic Product, PPP = Purchasing Power Parity

Environmental correlates

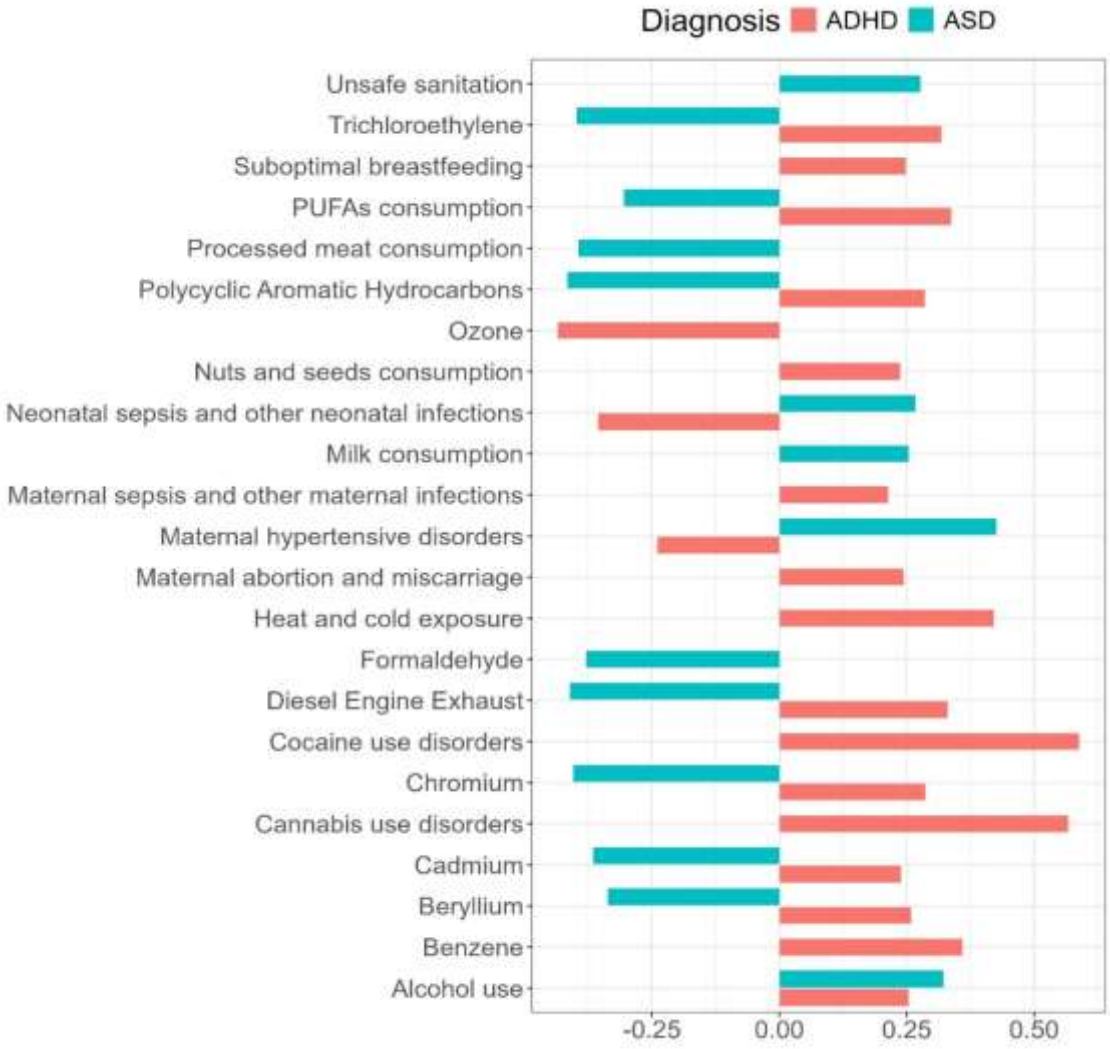
Two divergent profiles of associations were observed for either ADHD or ASD. Occupational exposure to chemical toxins was positively correlated with the incidence of ADHD (benzene r 0.359, p <0.001; beryllium r 0.258, p <0.001; cadmium r 0.239, p 0.002; chromium r 0.286, p <0.001; diesel engine exhaust r 0.330, p <0.001; polycyclic aromatic hydrocarbons r 0.285, p <0.001; trichloroethylene r 0.318, p <0.001), but negatively correlated with the incidence of ASD (beryllium r -0.336, p <0.001; cadmium r -0.365, p <0.001; chromium r -0.404, p <0.001; diesel engine exhaust r -0.411, p <0.001; formaldehyde r -0.379, p <0.001; polycyclic aromatic hydrocarbons r -0.416, p <0.001; trichloroethylene r -0.397, p <0.001). However, both the incidence of ADHD and ASD were correlated with the rate of alcohol use disorders at population level (r 0.254, p <0.001 and r 0.322, p <0.001 - respectively).

The incidence of ADHD was also positively correlated with the rage of cannabis and cocaine use disorders (r 0.567 p <0.001, r 0.588 p < 0.001 - respectively). For what concerns dietary factors, the incidence of ADHD was positively correlated with the rate of consumption of nuts and seeds (r 0.237, p 0.002) and PUFAs (r 0.337, p <0.001). The incidence of ADHD was also positively correlated with the rate of maternal miscarriages (r 0.243, p 0.002) and suboptimal breastfeeding (r 0.248, p 0.001). The incidence of ADHD was negatively correlated with indices of urbanization and industrialization, namely ozone exposure (r -0.432, p <0.001), sub-optimal residential temperatures (r 0.420, p <0.001) and the incidence of maternal infections (r 0.213, p 0.006), but negatively correlated with the incidence of neonatal infections (r -0.355, p <0.001).

The incidence of ASD was positively correlated with indices of sub-optimal healthcare

delivery, namely sub-optimal sanitation (r 0.277, p <0.001), the incidence of maternal hypertensive disorders (r 0.425, p <0.001) and, in contrast to ADHD, the incidence of neonatal infections (r 0.267, p <0.001). For what concerns dietary factors, the incidence of ASD was positively correlated with the rate of consumption of milk (r 0.253, p 0.001), processed meat (r 0.394, p <0.001) and, in contrast to ADHD, negatively correlated with the rate of consumption of PUFAs (r -0.305, p <0.001). See Supplementary Materials Table S1a-e for further details. Figure 1 shows the correlation between risk factors and incidence of ADHD or ASD (year of reference: 2021).

Figure 1. Correlation between risk factors and incidence of ADHD and ASD (year of reference: 2021). Values represent partial Pearson r coefficients, adjusted for PPP and life expectancy.



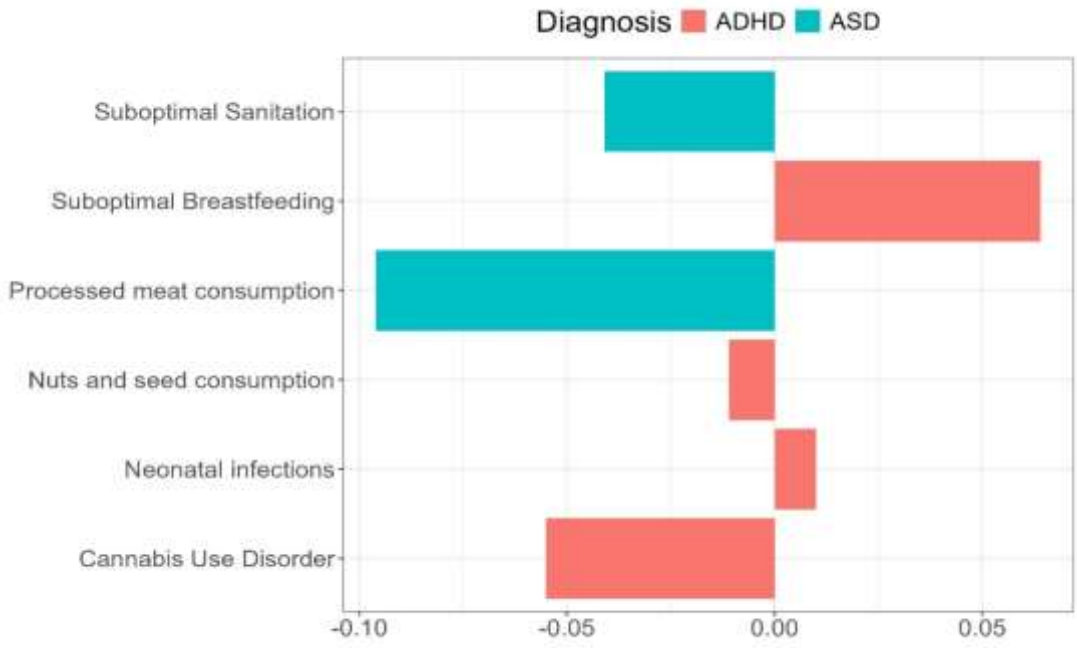
Concurrent trends

The growth in incidence for ADHD at the global level was positively associated with rates of neonatal sepsis and other neonatal infections (β 0.010, p 0.007) - indicating that concurrent improvements at the global level in reducing neonatal infection were also associated with a slower growth in incidence for ADHD. The growth in incidence for ADHD at the global level was negatively associated with the rate of change in cannabis use disorders (β -0.055, p 0.001) – indicating that increases in cannabis abuse were associated with a slower growth in incidence of ADHD. Two other factors were border-line significant, namely suboptimal breastfeeding (β 0.064, p 0.052), and nuts and seeds consumption (β -0.011, p 0.086) – indicating that improvements in suboptimal breastfeeding and a lower consumption of nuts and seeds were associated with a slower growth in incidence for

ADHD (Figure 2).

Two factors were also significantly associated with the growth of incidence for ASD at the global level, namely suboptimal sanitation (β -0.041, p 0.011) and a high consumption of processed meat (β -0.096, p 0.008) – indicating that improvements in sanitation were related to faster growth in incidence of ASD at the global level. By contrast, increased consumption of processed meat was related to a slower growth in incidence of ASD.

Figure 2. Regression coefficients indicating associations between global changes in selected risk factors and growth in ADHD and ASD incidence rates from 2010-2021 (adjusted for PPP and life expectancy).



DISCUSSION

This study provides novel insights into global patterns of environmental and lifestyle exposures associated with the incidence of neurodevelopmental disorders, particularly ADHD and ASD. Leveraging data from the GBD study across 204 countries, our analysis underscores the multifactorial drivers of these conditions at the population level. The findings reveal nuanced, condition-specific associations between risk factors and incidence trends, suggesting distinct mechanistic pathways through which environmental exposures may shape neurodevelopment.

Environmental correlates of ADHD

ADHD incidence was positively correlated with occupational exposure to neurotoxic chemicals such as benzene, cadmium, and diesel exhaust. These agents, known to contribute to oxidative stress, endocrine disruption, and neuroinflammation [39-41], may impair early brain development [10-17]. We also observed strong positive correlations between ADHD and rates of alcohol, cannabis, and cocaine use disorders. These substances may disrupt brain development directly [42,43], affect epigenetic regulation [44], or reflect broader social and healthcare system vulnerabilities [45].

Interestingly, while cannabis use disorder was positively correlated with ADHD incidence, an increase in cannabis abuse was associated with a slower growth in ADHD incidence over time. One interpretation is that rising cannabis use may increase the risk of psychotic disorders [46-48], potentially diverting diagnostic attention from underlying ADHD symptoms [49].

ADHD incidence also showed positive associations with maternal miscarriage rates, suggesting potential links to structural or prenatal healthcare challenges. Conversely, a negative association was found with neonatal infections, reinforcing the importance of the prenatal period over the perinatal window in shaping ADHD risk [50,51]. Improvements in neonatal infection rates and (borderline significant) gains in breastfeeding practices were both associated with slower growth in ADHD incidence, further underscoring the relevance of early-life health and nutrition interventions.

Environmental correlates of ASD

In contrast to ADHD, ASD incidence was negatively correlated with occupational exposure to neurotoxins.. This divergence suggests that different neurodevelopmental trajectories may be influenced by distinct environmental mechanisms. While repetitive behaviors are shared diagnostic features across both conditions [52-55], ASD is more strongly defined by impairments in social communication, sensory integration, and restricted interests [3,56]. Evidence linking carcinogen exposure to ASD largely stems from animal models [55,57.58]. The opposing patterns of association between chemical exposures and ADHD/ASD may therefore reflect diagnostic distinctions and neurobiological specificity [59].

Perinatal factors such as neonatal infections, maternal hypertensive disorders, and suboptimal sanitation were positively associated with ASD, pointing to the influence of early-life health environments on ASD risk [60,61]. These findings also support hypotheses that excessive hygiene or sterile early environments may alter immune development, potentially contributing to ASD risk [62].

Dietary factors and neurodevelopment

Dietary patterns, such as higher consumption of nuts and seeds, PUFAs, and a higher reliance on breastfeeding were positively associated with ADHD incidence, further underscore the role of diet and nutrition in mitigating neurotoxic effects and promoting cognitive health [32]. This positive association may be interpreted as possibly exerted through the promotion of optimal microbiota composition [63], a lower production of pro-inflammatory cytokines [64] and a lower synthesis of dopamine/serotonin precursors [65,66]. Although only border-line significant, improvements in promoting breastfeeding and a shift towards lower consumptions of nuts and seeds were also associated with slower growth in incidence for ADHD, indicating the potential for societal and policy interventions to shape the incidence of neurodevelopmental conditions.

In contrast, ASD was positively correlated with the consumption of milk and processed meat, while negatively correlated with PUFAs. PUFA-related immune modulation may then act similarly to what was previously hypothesized for other environmental factors, that is shaping neurodevelopmental trajectories towards either ASD (generally pro-inflammatory: processed meat, low PUFAs) or ADHD (generally anti-inflammatory: nuts and seeds, high PUFAs). The positive correlation between milk consumption and ASD could be explained through several mechanisms: including milk-related peptides [67]; gut permeability [68]; microbiota modifications [69]; immune, allergic and inflammatory modulation [70]. The positive correlation between processed meat and ASD may also reflect the role played by nitrites or nitrates - which can lead to the formation of neurotoxic compounds, including nitrosamines, and thus contribute to oxidative stress and neuroinflammation in ASD [71]. However, a shift towards higher consumption of processed meat was related to a slower growth in incidence of ASD.

Future studies might shed light on this result, considering that although the confounding role of socio-economic factors was controlled in all analyses, potential influences by structural factors might still ensue. The authors, for instance, posit that these results may cautiously be interpreted in light of recent evidence, describing a role for western dietary patterns as influencing the incidence of neurodevelopmental disorders [72].

Study limitations

Conducting analyses on population-level data, rather than individual-level data, restricts the potential to establish causal relationships or account for individual variability in risk factors. While the analysis provides valuable insights into global and historical trends, population-level data can not capture the nuanced interactions between individual, societal and environmental factors. Future

research utilizing individual-level longitudinal data will be essential to refine these associations and further elucidate the underlying mechanisms driving the incidence of ADHD and ASD.

For what concerns observed associations, occupational exposure to carcinogens may also be interpreted as indicating indirect effects of urbanization and industrialization as driving ADHD incidence – although no metric of urbanization or industrialization were here employed. Although all analyses were adjusted for socioeconomic confounders, urbanization and healthcare institutionalization may still be associated with a higher awareness of neurodevelopmental conditions, and thus drive an increase in incidence [7].

Observed associations, although assessed by longitudinal methods, may not fully describe causal directions, nor fully account for confounding factors, such as parental age and/or individual-level dietary practices, while solid evidence is arising on both these factors as influencing the incidence of either ADHD or ASD [72,73]. Therefore, future studies are warranted to more comprehensively account for these variables and reduce the risk of residual confounding in interpreting the findings.

CONCLUSION

The current study provides critical evidence on environmental factors associated with the incidence of ADHD and ASD. The distinct association profiles association identified for ADHD and ASD suggest potentially different mechanistic pathways through which environmental factors exert their influence. These findings underscore the importance of targeted public health strategies that address condition-specific risk factors to mitigate the growing burden of neurodevelopmental disorders, while future studies might explore how to best improve health and educational outcomes in neurodivergent children, possibly increasing awareness in parents and the general population [74-82]. Furthermore, the observed trends highlight the potential long-term impact of historical shifts in urbanization and sanitation, reinforcing the need for interdisciplinary research that bridges environmental health, developmental neuroscience, and public policy to better understand and address these complex, evolving risks.

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Supplementary Materials

Table S1a. Correlation between risk factors and incidence of ADHD and ASD (2021), environmental factors.

	Attention-deficit/ hyperactivity disorder	**Autism spectrum disorders**
Ambient ozone pollution	r -0.435*	r 0.029
Nitrogen dioxide pollution	r -0.187	r 0.080
Lead exposure	r 0.024	r -0.002
Environmental heat and cold exposure	r 0.420*	r 0.035
Unsafe water source	r 0.074	r -0.052
Unsafe sanitation	r -0.156	r 0.277*

Note. Conditioned on variables: life expectancy in 2021, PPP$2021.
Legend: * = Column-wise correction for multiple comparisons, Bonferroni $p < 0.05$; r = Pearson r coefficient; PPP$2021 = GDP per capita in constant PPP dollars. The unit is constant dollars adjusted for inflation in 2021

Table S1b. Correlation between risk factors and incidence of ADHD and ASD (2021), occupational factors.

	Attention-deficit/ hyperactivity disorder	**Autism spectrum disorders**
Occupational exposure to asbestos	r 0.136	r 0.212
Occupational exposure to arsenic	r 0.150	r 0.017
Occupational exposure to benzene	r 0.359*	r -0.189
Occupational exposure to beryllium	r 0.258*	r -0.336*
Occupational exposure to cadmium	r 0.239*	r -0.365*
Occupational exposure to chromium	r 0.286*	r -0.404*
Occupational exposure to diesel engine exhaust	r 0.330*	r -0.411*
Occupational exposure to formaldehyde	r 0.221	r -0.379*
Occupational exposure to nickel	r 0.123	r -0.060
Occupational exposure to polycyclic aromatic hydrocarbons	r 0.285*	r -0.416*
Occupational exposure to silica	r 0.076	r -0.072
Occupational exposure to sulfuric acid	r 0.213	r -0.153
Occupational exposure to trichloroethylene	r 0.318*	r -0.397*

Note. Conditioned on variables: life expectancy in 2021, PPP$2021.
Legend: * = Column-wise correction for multiple comparisons, Bonferroni $p < 0.05$; r = Pearson r coefficient; PPP$2021 = GDP per capita in constant PPP dollars. The unit is constant dollars adjusted for inflation in 2021

Table S1c. Correlation between risk factors and incidence of ADHD and ASD (2021), drugs.

	Attention-deficit/ hyperactivity disorder	**Autism spectrum disorders**
Smoking	r -0.159	r 0.015
Alcohol use	r 0.254*	r 0.322*
Cannabis use disorders	r 0.567*	r -0.062
Opioid use disorders	r -0.106	r 0.096
Amphetamine use disorders	r 0.063	r 0.077
Cocaine use disorders	r 0.588*	r 0.146

Note. Conditioned on variables: life expectancy in 2021, PPP$2021.
Legend: * = Column-wise correction for multiple comparisons, Bonferroni $p < 0.05$; r = Pearson r coefficient; PPP$2021 = GDP per capita in constant PPP dollars. The unit is constant dollars adjusted for inflation in 2021

Table S1d. Correlation between risk factors and incidence of ADHD and ASD (2021), maternal and peripartum factors.

	Attention-deficit/ hyperactivity disorder	**Autism spectrum disorders**
Maternal obstructed labor and uterine rupture	r 0.148	r -0.073
Neonatal preterm birth	r 0.002	r -0.069
Maternal abortion and miscarriage	r 0.243*	r 0.006
Maternal hypertensive disorders	r -0.239*	r 0.425*
Maternal sepsis and other maternal infections	r 0.213*	r -0.047
Neonatal sepsis and other neonatal infections	r -0.355*	r 0.267*
Suboptimal breastfeeding	r 0.248*	r 0.132

Note. Conditioned on variables: life expectancy in 2021, PPP$2021.
Legend: * = Column-wise correction for multiple comparisons, Bonferroni $p < 0.05$; r = Pearson r coefficient; PPP$2021 = GDP per capita in constant PPP dollars. The unit is constant dollars adjusted for inflation in 2021

Table S1e. Correlation between risk factors and incidence of ADHD and ASD (2021), dietary factors.

	Attention-deficit/ hyperactivity disorder	**Autism spectrum disorders**
Vitamin A deficiency	r -0.098	r 0.235
Iron deficiency	r -0.183	r 0.026
Zinc deficiency	r 0.017	r -0.107
Iodine deficiency	r -0.172	r 0.131
Diet low in legumes	r -0.048	r 0.100
Diet low in calcium	r -0.131	r 0.018
Diet low in fruits	r -0.188	r 0.125
Diet low in vegetables	r 0.104	r -0.072
Diet low in whole grains	r 0.034	r -0.136
Nuts and seeds consumption (diet low in nuts and seeds, reversed)	r 0.237*	r -0.194
Milk consumption (diet low in milk, reversed)	r -0.052	r 0.253*
Diet low in fiber	r 0.114	r -0.126
Diet low in seafood omega-3 fatty acids	r -0.127	r 0.195
PUFAs consumption (diet low in PUFAs, reversed)	r 0.337*	r -0.305*
Red meat consumption	r 0.117	r 0.211
Processed meat consumption	r-0.187	r -0.394*
Sugar-sweetened beverages consumption	r 0.180	r 0.016
Trans fatty acids consumption	r -0.019	r -0.119
Diet high in sodium	r -0.149	r 0.061

Note. Conditioned on variables: life expectancy in 2021, PPP$2021.
Legend: * = Column-wise correction for multiple comparisons, Bonferroni $p < 0.05$; r = Pearson r coefficient; PPP$2021 = GDP per capita in constant PPP dollars. The unit is constant dollars adjusted for inflation in 2021