European Journal of Epidemiology (2025) 40:981–994
https://doi.org/10.1007/s10654-025-01316-8

COMMENTARY

The autism 'epidemic': misinterpretation, misinformation and conspiracy

Eric Fombonne[1]

Received: 26 August 2025 / Accepted: 16 September 2025 / Published online: 10 October 2025

Abstract

The Secretary of the US Department of Health & Human Services (HHSS) recently claimed that US estimates of the prevalence of autism confirmed the existence of an autism epidemic. Furthermore, HHSS asserted that the autism epidemic was driven by an environmental toxin which he promised to find in the following months. These claims are contradicted by studies showing that progress in the understanding, detection, diagnosis and management of autism have fueled the increasing prevalence. HHSS statements are also in sharp contrast with the opinion of scientists who have monitored the upward trend in autism prevalence in the US and worldwide. In this Commentary, we address sequentially each misconception and misinterpretation proffered by HHSS. We show that changes in the definition of autism, in diagnostic criteria and practices, in case ascertainment in surveys, the inclusion of less severe forms of autism and other contextual factors such as improved awareness, de-stigmatization, advocacy, improved access to service and better insurance coverage have all converged in increasing the prevalence of autism and that presenting the rise on autism prevalence as an epidemic is misleading. Furthermore, given the strong heritability of autism, its genetic and phenotypic heterogeneity and the paucity of leads on environmental risk, the promise to find an environmental toxin causally related to autism in upcoming months appears at best preposterous. We warn about the return of false theories and already debunked hypotheses on the etiology of autism when empirical data are ignored, scientific methodology is dismissed and experts' opinions disdained.

Keywords Autism · Epidemiology · Time trends · Prevalence · Diagnosis · Survey

In April 2025, the Center for Disease Control and Prevention (CDC) released the 10th report from its autism surveillance program launched in the US in the early 2000's [1]. The CDC methodology is based on screening medical/educational records of children age 8, with screen positive children being subsequently confirmed or not as cases. No in-person assessment of participants is performed. For survey year 2022, 16 sites estimated the prevalence of autism spectrum disorder (ASD[1]) among 274,857 US children age 8 (cohort born in 2014). Of these, 8,854 met the surveillance definition for ASD translating into an overall prevalence of 3.22% (95%CI: 3.16–3.29) [2]. This represented an increase of 22%−23% over the prevalence reported in the previous survey year 2020. Increases of about the same magnitude (range: 0.6%−36.3%; median: 18.5%) were found between prior consecutive bi-annual surveys.

The CDC report release was paired with an address of the Health and Human Services new Secretary (R. Kennedy Jr, thereafter HHSS) that contained multiple unfounded claims about the "meaning" of the CDC report and affirmed the undeniable existence of an autism epidemic due to some (unnamed) environmental toxin. In this Commentary, we provide a systematic rebuttal to the misinformation and misconceptions that are at odds with the available empirical evidence. For clarity of presentation, we extracted from HHSS statements eight consubstantial allegations that we employ as headings (bolded, and in quotes when verbatim) the first seven sections that deal with the 'epidemic' interpretation of autism prevalence data. In the last section, we challenge

[1] I use interchangeably ASD and autism in the text.

This article is based in part on a lecture given by the author on May 7, 2025 at Harvard University in the Joint Seminar of the Maternal Child Health Center of Excellence and the Harvard Chan Department of Epidemiology.

✉ Eric Fombonne
fombonne@ohsu.edu

[1] Oregon Health & Science University, Portland, OR, USA

the affirmation that an environmental 'toxin' underlying the 'epidemic' can be found in a matter of months. In rebutting HHSS allegations, we rely on selective autism survey literature, our own experience of having conducted several surveys of autism in different countries, and in the clinical and scientific experience we garnered over the last 40 years.

"The prevalence is 83 times higher… the epidemic is real"

A review of 165 epidemiological surveys conducted since 1966 shows unambiguously a prevalence increase worldwide (Fig. 1) [3][2]. However, the methodology of surveys is highly variable reflecting constraints of local health care and education systems, variable case finding approaches and case definitions, different age groups, and so on. Even in countries where several prevalence estimates are available over time, study methodologies and populations are so variable that between-surveys comparisons are hazardous. Therefore, quantifying the upward trend in a very heterogeneous set of surveys has not been possible.

A more meaningful approach to quantify the trend in prevalence is to compare studies executed over time in the same population and with the same methodology. The CDC data offer a unique opportunity to that end although the assumption that the methodology of CDC studies has remained identical is not fully verified. First, each survey includes convenience samples that are not representative of the US population; in addition, the number and location of sites as well as the socio-demographic characteristics of the samples has differed substantially across surveys. Second, diagnostic criteria employed in the CDC surveillance case definition changed from DSM-IV to DSM 5 in 2018. Third, whereas the first surveys had a panel of expert clinicians reviewing all individual data abstracted from medical and educational sources to validate case status, this clinical filter was removed in 2018. Case status is now entirely determined by an algorithm based on ASD-related diagnostic statements or codes present in the records. Fourth, in-person clinical assessments had only moderate agreement with the CDC surveillance definition in two small validation studies [4].

Despite these changes and limitations, comparisons of estimates across surveillance years are informative. In survey year 2022 [2], the overall prevalence estimate was 3.22% representing a 5-fold increase in prevalence compared to the 0.66% prevalence in survey year 2000 (Fig. 2). No estimate of the proportion of autism cases missed in any survey and of the sensitivity of the record screening procedure was ever available. Therefore, because this procedure depends on service use, it is very likely that, among other factors, improved access to services over time has resulted in increasing prevalence.

.

HHSS referenced two studies performed in the US decades ago (Fig. 1). Treffert's study [5] identified over a five-year period (1962 to 1967) 280 children ages 3 to 12 (thus born between 1950 and 1964) who had been attending a mix of services with a DSM-II diagnosis of childhood schizophrenia. At the time, there was no specific provision in DSM-II for the diagnosis of autism that, for years, was confounded with that of childhood schizophrenia or psychosis and included in the same DSM category. A record review showed that 69 of them fit the general description of infantile autism, translating into a prevalence of 0.7/10,000 for

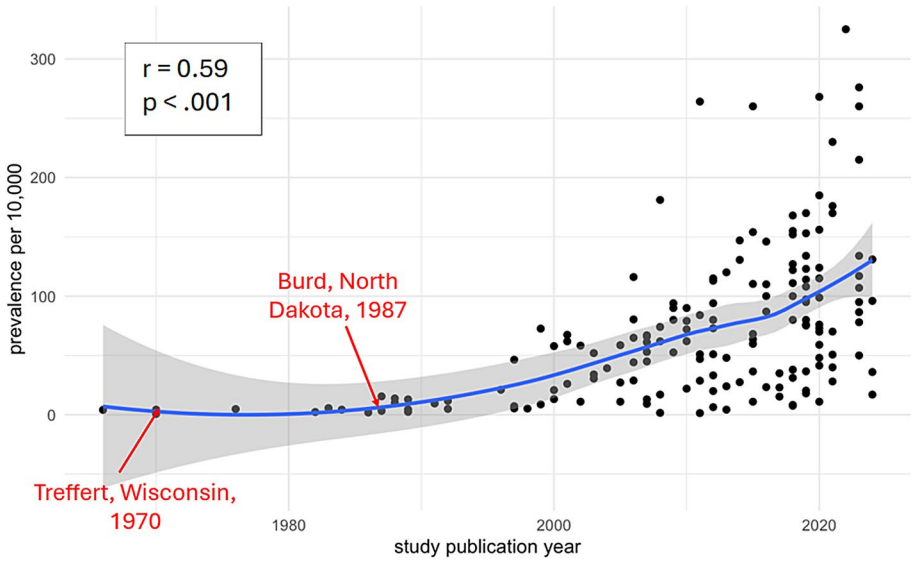

Fig. 1 Prevalence surveys 1966–2024 (165 world studies). Source: [3] Fombonne & MacFarlane (2025)

[2] The last CDC survey [2] is not included in this review nor in Fig. 1.

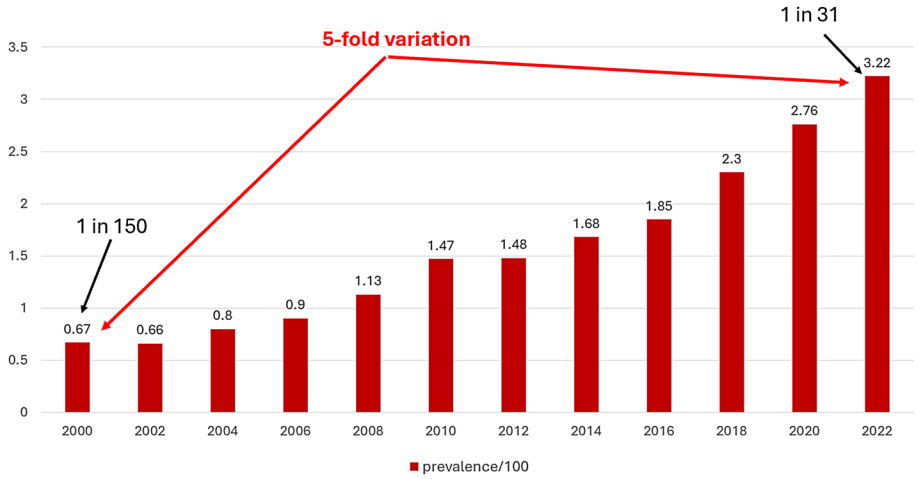

Fig. 2 Autism prevalence in CDC surveys at age 8 (birth cohorts 1992 to 2014). Source: https://www.cdc.gov

autism. The second study in North Dakota was performed to validate the newly developed DSM-III category of Pervasive Developmental Disorders (PDD, similar to ASD) and its diagnostic subtypes. Over 200 youths ages 2 to 18 already receiving care and showing autistic symptoms were referred to the authors for in-person evaluation. Of these, 59 children met criteria for PDD and prevalence in the State was estimated to be 3.26/10,000 [6], a figure lower than estimates from four contemporaneous European studies used as a comparison. Both studies relied on children *referred* for services; none examined children with other neurodevelopmental or psychiatric diagnoses, and both were conducted at a time of low awareness about ASD and when little expertise for diagnosing autism was available. It appears that the "83-fold" increase was calculated by dividing the 2020 CDC estimate (2.76%) by the 1987 North Dakota estimate (2.76/0.0326 ≈ 84). This ratio has zero interpretability as it is not comparing like with like; it compares clinic-referred to population-based samples, vastly different age groups (ages 2–18 vs. age 8) and differences in all methodological aspects of these studies confound the comparison. Interestingly, similarly meaningless inflated autism prevalence ratios have long been used by other proponents of the 'epidemic of autism', especially the antivaccine movement that even went to claim a 1,000-fold increase. HHSS also referred to an historical 0.7/10,000 prevalence originating from the Collaborative Perinatal Project that enrolled about 60,000 pregnant mothers from 1959 to 1965, with the final follow-up of offspring at age 8 in 1975. During that interval, there was no agreed upon definition of autism and no standardized tools available to monitor autism reliably; besides, a possible association between offspring autism and loss to follow-up was not evaluated. The 0.7/10,000 figure is therefore anecdotal and should not be used as an historical benchmark for autism prevalence.

Thus, there is no 83-fold increase in autism prevalence. Prevalence of ASD has increased in all countries since the early 1990's but putting a figure on how much it rose is hazardous due to the heterogeneity of survey methods and populations in published studies. Yet, the CDC in the US provided a unique time series of estimates over 22 years that, the variations in methods put aside, can shed some light on the secular change. CDC data show a 5-fold increase in prevalence over 22 years (Fig. 2). This observation does not lend per se to any particular interpretation; and not everything that goes up signals an epidemic.

The relentless increase in prevalence is not due to better detection

The methodology of surveys has improved considerably since the first 1966 study in the UK. Initially, surveys relied on head counts of children already diagnosed and presenting with severe clinical syndromes. Subsequently, case ascertainment improved with children with other diagnoses being scrutinized for possible autism (especially those initially diagnosed with intellectual disability or other neurodevelopmental conditions) allowing detection of cases not previously identified[3]. More recently, case finding methods in surveys added samples drawn from the general school population in order to detect undiagnosed children with milder phenotypes compatible with schooling in typical education settings. All studies found new yet undiagnosed cases of ASD, with this arm of the surveys contributing to about a third of the total number of cases (median: 32% in 11 surveys [3, 7]).

Sensitivity of case finding methods obviously affects prevalence estimation. For example, a comparison of surveys performed in the UK or in the US at the same time and with similar age groups but with different methodologies

[3] In earlier CDC surveys, about 20% of cases meeting the surveillance definition for autism were not previously diagnosed in the community.

showed that, in both countries, lower prevalence proportions were obtained for studies with passive case finding relying on administrative databases (e.g. children already diagnosed in a database/registry) whereas much higher estimates were derived from surveys employing intensive population-based screening techniques resulting in a 6-fold (UK) or 14-fold (US) variation between surveys [8]. Because no passage of time was involved in comparing these surveys, the magnitude of these prevalence gradients could only be attributed to differences in case ascertainment methodology.

The impact of case ascertainment has also been consistently *demonstrated* in CDC surveys. In the recent survey [2], there was more than a 5.5-fold difference in prevalence between participating sites (Fig. 3), with an astonishingly high estimate of 5.31% in California (San Diego) compared to a low 0.97% figure for the Texas (Laredo) site. Between-sites variations of comparable magnitude were reported in *all* previous surveys and consistently commented by CDC scientists as reflecting between-areas ascertainment differences as opposed to true differences in incidence. In the recent survey [2], CDC authors wrote: "Differences in the prevalence of children identified with ASD across communities might be due to differences in availability of services for early detection and evaluation and diagnostic practices" and the nature of these differences were readily identified. To illustrate, at the San Diego (California) site, a program (*Get SET Early*) to train pediatricians to pro-actively screen autism in toddlers and refer early to services has been in place for several years; by contrast, the Laredo site in Texas serves a largely Hispanic population with lower income and an older age at diagnosis reflecting barriers to access services. Pennsylvania which had the second highest prevalence has a Medicaid policy financially favorable to children with disabilities. New-Jersey, a site reporting high estimates since 2000, has good support services for children with autism. By contrast, Southern States (such as Alabama) with no such supportive policies had consistently lower prevalence in earlier surveys.

Case ascertainment methodology matters and directly influences prevalence estimates. If a 5-fold difference can arise between sites *within the same* cross-sectional survey (Fig. 3), the 5-fold difference in CDC estimates observed *between surveys* from 2000 to 2022 (Fig. 2) could also reflect the effects of improved case ascertainment in surveyed communities over a 22-year period.

Changes in diagnostic criteria cannot explain the rise

All the evidence points to the contrary. The conceptualization of autism has evolved dramatically since its first description by Kanner in 1943. Initially focused on children with limited or no language, and with profound social impairments associated with rituals and repetitive behaviors, the syndrome subsequently was recognized among children with near-normal language development and intellectual skills in the normal range. Key to this progress was the realization that *delay* in the development was not a condition sine qua non for an autism diagnosis; rather, *qualitative* impairments in key developmental domains (verbal and non-verbal communication, social reciprocity, abnormal patterns of play and activities) were delineated that could occur at whatever developmental level a child was functioning.

These evolving concepts of autism were operationalized in DSM and ICD with diagnostic criteria and algorithms that were revised several times [9]. In DSM-II (1967) and ICD-8 (1968), there was no separate diagnostic category for

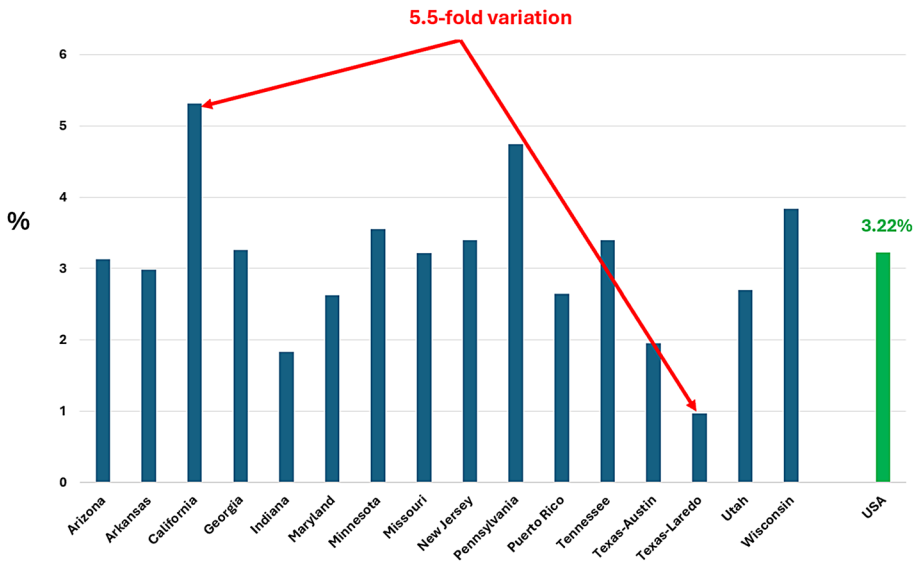

Fig. 3 Prevalence differences across 16 sites in 2022 CDC survey. Source: https://www.cdc.gov

autism that was lumped up with schizophrenia[4]. In 1980, autism was introduced in DSM-III under the PDD umbrella alongside poorly validated subtypes. The DSM-III-R (1987) introduced 16 diagnostic criteria spread across three developmental domains, but the diagnostic algorithm quickly appeared to lack specificity. ICD-10 (1992) and DSM-IV (1994) introduced the new category of Asperger syndrome and adopted a more developmentally oriented and dimensional view of the phenotype that was applicable to different ages and severity levels. This nosography evolution coincided with the development of new diagnostic instruments widely regarded today as gold standards for a diagnostic evaluation. The DSM 5 revision (2013) consolidated symptoms in only two developmental domains (social-communication, restricted repetitive behaviors), added new symptoms (sensory processing abnormalities) and relaxed the age requirement of symptom recognition by age 3. Diagnostic subtypes that were notoriously unreliable were eliminated resulting in one only diagnosis of ASD.

In several autism surveys, case status relies upon hospital/special education codes derived from routine clinical diagnoses. While clinical practice has evolved in parallel to the official changes in nosographies, it is not possible to directly evaluate how evolving clinical practices have contributed to the increasing prevalence over the years. Nevertheless, the direct impact of changes in diagnostic criteria on prevalence estimates has been documented in several studies. Thus, in a Northern Finland survey of 152,732 youths aged 3 to 18, authors applied to survey data contrasting diagnostic definitions to evaluate their unique impact on prevalence estimation, everything being maintained constant otherwise [10]. There was a 2.2-fold increase in prevalence when diagnosis changed from 1956 Kanner's criteria to the mid-1990s ICD-10/DSM-IV algorithms. In another study of the national registry in Denmark, 33% of the prevalence increase in 667,000 children born 1980–1991 followed-up to 2011 was due solely to the change from ICD-8 to ICD-10 diagnostic criteria in 1994 when other factors were adjusted for [11]. By contrast, implementation of the more stringent DSM 5 criteria for ASD has resulted in a prevalence decrease of a 15%−20% order of magnitude in research studies [12, 13] although this effect has not been perceptible in community practice and recent CDC surveys.

Overall, there has been a progressive broadening of the definition of autism since the 1980s. Corresponding changes in the operationalization of diagnostic criteria allowed a better recognition of the autism phenotype that exhibits great variability with age and developmental level. When the impact of changes in specific diagnostic criteria and algorithms could be quantified in surveys, more recent diagnostic definitions were generally associated with a positive change in the prevalence estimate which is consistent with the increasing numbers of individuals diagnosed in clinical settings. Comparable historical changes have occurred for other psychiatric and neurodevelopmental conditions (e.g. depression, ADHD)[5].

Diagnostic substitution is not an explanation for the prevalence increase

This phenomenon applies to children previously diagnosed with a non-autism diagnosis who were 'switched' to an ASD diagnosis when new conceptions of autism gained momentum and new supportive interventions became available. First evidence of this phenomenon was reported in an ecological analysis of data from the California Department of Developmental Services (DDS), a network of 21 Centers that provide services for youths with disabilities and autism in that State. From 1987 to 1994, the prevalence of the diagnosis of intellectual disability[6] (ID) decreased over that period to the same extent that diagnoses of autism increased [14]. Using the same DDS dataset, King & Bearman [15] extended this preliminary finding with individual level data showing that 26.4% (95% CI 16.25–36.48) of the increased autism caseload in California between 1992 and 2005 was uniquely associated with this diagnostic switch specifically (from ID to ASD). Shattuck analyzed data on 6- to 11-year-olds from the US special education department between 1994 and 2003 [16]. As the administrative autism prevalence increased 5-fold, the prevalence of ID and learning disabilities diagnoses decreased to a similar extent, and more so in States where the increase in autism categories was the steepest. The author concluded that the autism prevalence findings in US special education data did 'not support the claim of an autism epidemic' [16].

Data on switching from other diagnoses to ASD also exist. For example, a UK follow-up study in young adulthood of children first diagnosed with developmental language disorder showed that, when re-evaluated with two

[4] It is only in 1977 that the *Journal of Autism and Childhood Schizophrenia* changed its name to the *Journal of Autism and Developmental Disorders*, a widely read journal in the autism community.

[5] A good historical parallel with autism is that of Fetal Alcohol Syndrome (FAS) that, perhaps surprisingly, only appeared in 1973 in the international medical literature. FAS was first described as a severe syndrome (with facial dysmorphology, growth deficiency, learning and behavioral difficulties) with a low prevalence (<1%). Progress in the characterization of an extended phenotype led to broader definitions inclusive of milder forms (partial FAS, Alcohol-Related Neurodevelopmental Disorder (ARND), Neurobehavioral Disorder Associated with Prenatal Alcohol Exposure (ND-PAE)) now subsumed in a general category of Fetal Alcohol *Spectrum* Disorders (FASD) with prevalence as high as 5%.

[6] The term 'mental retardation' in use then has been now replaced with 'intellectual disability'.

autism standardized diagnostic tools, 34% met ASD criteria on both measures and 66% on at least one measure [17]. Authors commented *"some children who would nowadays be diagnosed unambiguously with autistic disorder had been diagnosed with developmental language disorder in the past."* In British Colombia (Canada), autism eligibility codes in 4- to 9-year-olds increased from 12.3/10,000 in 1996 to 43.1/10,000 in 2004; 32.8% of this increase in prevalence was accounted by children assigned initially a different special education code and later switched to an autism code [18]. There is also evidence from some US studies that autistic children from minorities and underserved groups are often initially misdiagnosed before receiving an ASD diagnosis. In a sample of 406 Medicaid-eligible children diagnosed with autism, 54.2% were misdiagnosed at their first visit in specialty care at a mean age of 6.7 years, with ADHD and other diagnoses [19]. Furthermore, misdiagnosis was 2.6 times more frequent in black children compared to white children, with behavioral diagnoses (conduct disorder, oppositional defiant disorder) being even more frequent among black children, likely accounting for a later age at diagnosis and missed treatment opportunities. Many other studies have shown that known high levels of psychiatric comorbidity in autism mask the autism symptomatology and account for late diagnoses in people with various psychiatric diagnoses.

Thus, a substantial proportion of individuals currently diagnosed with ASD were previously diagnosed with other neurodevelopmental and psychiatric diagnoses which may account to up to a third of prevalence increase in some studies. Therefore, new autism diagnoses do not necessarily represent new incident cases. Much how individuals with autism existed unrecognized before Kanner described it, autism in individuals diagnosed today has often been overshadowed by other diagnoses.

The prevalence increase is not due to the inclusion of milder forms of autism, "most cases are now severe"

This is inaccurate. As explained above, the proportion of individuals diagnosed with autism but without ID has soared in both clinical and epidemiological samples since the 1990s. The best evidence showing that prevalence increased as less severe forms of autism were detected and included in surveys comes from our review of 74 epidemiological surveys with available IQ data performed worldwide since 1966 [3]. There was a strong correlation between the survey publication year and the proportion of survey participants without ID (Fig. 4; rho = 0.48, $p < 0.001$); the proportion of survey participants with associated ID declined on average from 70% to 37.9% over the 1966–2024 period (Fig. 4).

In the 2022 CDC survey [2], the proportion of participants with ID was 39.6% which accords well with our review. The proportion of participants with ASD and ID has fluctuated in CDC surveys between 44.6% in survey year 2002 and 30.1% in 2012. However, an examination of the contribution of autism without ID to the trend in prevalence is difficult due to the exclusion of sites with high number of missing data, the variation in the socio-demographic composition of samples across sites and over time and the association of ID with specific race and ethnicity characteristics. However, sites with higher prevalence (Utah, New Jersey) consistently had higher proportions of participants without ID. In the recent survey, the proportion of participants

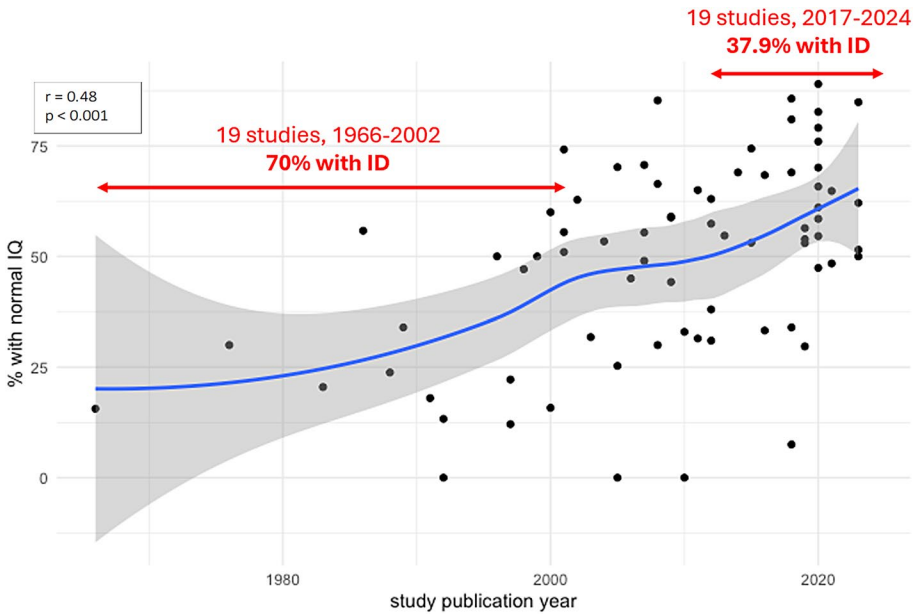

Fig. 4 Autism and intellectual disability over time.
Source: [3] Fombonne & MacFarlane (2025)

with ID was 28.4% in the state with the highest prevalence (California) whereas it was 80.0% in the site with the lowest prevalence (Texas-Laredo) [2]. Other US studies have shown that the rise in prevalence was due to inclusion in samples of less severe forms of autism. In a study investigating the rise of cases in California between 1990 and 2006 [20], it was found that 56% of the increase was due to the inclusion of less severe forms of autism. In a study of US adults age 18 and over enrolled in Medicaid, prevalence increased from 0.42% in 2011 to 0.95% in 2019; in 2019, 46% of over 403,000 enrollees with autism also had claims for intellectual disability, down from 62% in 2011 [21]. Therefore, in adult samples too, the increasing prevalence reflects higher proportions of clinical presentations with lesser severity.

Another informative approach to examine the relationship between the prevalence trend and severity of the phenotype has focused on the most functionally impaired individuals with autism. A re-analysis of 20,135 children aged 8 surveyed at 15 CDC sites examined trend for the period 2000–2016 in the prevalence of "profound autism". Profound autism is a new descriptive terminology focusing on the most severe end of the spectrum and defined in this study by being non-verbal or minimally verbal and/or with an IQ < 50 [22]. Overall, individuals with profound autism represented 26.7% of all cases. While prevalence of both non-profound autism and profound autism increased over that time frame, the increase was steeper for non-profound autism again demonstrating that recognition of less severe forms of autism is an important driver of the overall prevalence trend.

Thus, there is ample evidence that the identification and diagnosis of autism among individuals with good language skills and intellectual functioning in the normal range has been and still is an important contributor to the increasing prevalence, both in children and in adults. These epidemiological trends mirror the clinical experience acquired in every country over the last decades.

If this is not an epidemic, we should see more adults with autism

The question of where the adults with autism are has been examined in many studies that consistently showed that adults with autism were and still are undiagnosed, or misdiagnosed and disproportionately found in segregated settings (psychiatric institutions, prisons) with little recognition and support for their needs. For example, in a London forensic psychiatric hospital, undiagnosed cases of Asperger syndrome were reported at a much higher frequency than expected suggesting a three-fold increase of Asperger prevalence among incarcerated male adults [23]. Likewise, a survey of 761 individuals diagnosed with ID and residing in a long-stay mental handicap hospital in the Greater London area identified 4% of residents who were newly diagnosed with autism [24]. More recently in the US, an ASD prevalence of 9.9% was reported among 141 adults (mean age: 52 years) civilly committed at one Pennsylvania state psychiatric hospital over a period of 3 years [25]. Almost all adults meeting criteria for ASD had a diagnosis of schizophrenia, and none had been previously diagnosed with autism; 64.3% had co-occurring ID, 28.6% had a history of seizures, and they had been institutionalized for 17.6 years on average. Thus, many adults meeting current definitions and criteria for autism have not been diagnosed in their early life and were misdiagnosed with other psychiatric and/or neurodevelopmental diagnoses.

Prevalence surveys in adult samples have just started. In comparing adult to children prevalence data, the well-established increased mortality associated with autism must be borne in mind [26] although mortality should only have a modest impact on overall prevalence. In a 2007 national UK survey, investigators estimated ASD prevalence to be around 1.1% in all age groups of this adult epidemiological sample [27, 28]. More recently, US data have shown that adult diagnoses are increasing at a fast pace. In a study of over 12 millions electronic health records of the Mental Health Research Network, an increase in ASD diagnosis rates from 2011 to 2022 was found in all age groups [29]; the increase among adults aged 26–44 was relatively steeper than that observed for children ages 5 to 12 even though the prevalence in adult cohorts remained lower than that in younger cohorts. In a large study of Medicaid enrollees in the US, ASD prevalence increased in all age groups from 2011 to 2019, ranging from + 195% in the 26–34 age group to + 45% in the 55–64 age group [21]. In 2019, the prevalence of autism was 0.95% in all age groups combined, with strong cohort effects illustrated by prevalence differences between the 18–34-year-olds (>1.5%) and the 55 + year olds (< 0.4%). As indicated above, the upward trend in prevalence was strongly correlated with a decrease in the prevalence of co-occurring ID.

It is therefore inaccurate to affirm that autism is not seen among adults. Repeated studies have found adults meeting diagnostic criteria for autism in clinical settings, segregated environments and epidemiological samples who were either undiagnosed or misdiagnosed with ID or other psychiatric conditions. There is evidence that autism prevalence among adults is going upward, a trend that parallels that in youth studies although adult estimates remain at a lower level. The increase in adult prevalence is associated with clinical profiles with lesser severity. Of note, the rise in autism diagnoses among adults has unequivocal interpretation. Since

one does not develop autism in adult life, new valid adult diagnoses necessarily apply to individuals who had autism but were unrecognized previously and cannot represent new incident cases[7]. Collectively, these studies demonstrate prior systematic underdiagnosis among adults, especially in older birth cohorts. Autism was there but not seen.

Method artifacts explain "at most 25% of the rise"

It is unclear how this 25% figure was arrived at. In support, HHSS distinguished one specific study that examined how lower age at diagnosis and lesser clinical severity contributed to the rise of autism in California [20] but misquoted its findings. The authors found that "changing age at diagnosis can explain a 12% increase, and inclusion of milder cases, a 56% increase" in the rise of autism prevalence between 1990 and 2006 in the DDS network; they further concluded that "other artifacts have yet to be quantified, and as a result, the extent to which the continued rise represents a true increase in the occurrence of autism remains unclear" [20]. As shown above, studies show that methodological changes could account for a much higher proportion than 25% when their impact could be quantified.

In addition to aforementioned factors, other changes in awareness, early detection, access to intervention and services organization most certainly contributed to some part (albeit difficult to quantify) of the upward trend observed worldwide.

Increased awareness and de-stigmatization

World awareness about autism changed dramatically in the last 30 years, with large initiatives being credited to nonprofit organizations such as Autism Speaks that have advocated efficiently worldwide for individuals with autism and their families. However, knowledge and awareness about autism is still low in many countries albeit evolving rapidly due to widespread use of internet and social media. In China, autism first appeared in the medical literature in 1983, and first prevalence estimates were low around 0.1% [30]; recent dissemination of evidence-based practices and improved awareness and service access resulted in much higher detection of autism with prevalence estimates going up in more recent surveys (e.g. 0.7% [31]). In France, surveys of special education records in the 1970–80s yielded low estimates (e.g. 0.16% in [32]) when psychoanalytically oriented psychiatrists restricted the autism diagnosis to those without language and with the most severe presentations[8]; recent studies performed with updated methods yielded higher prevalence estimates (e.g. 0.73% in [33]). In South Korea, two decades ago, autism was rarely diagnosed in clinics and mostly seen as a stigmatizing hereditary disorder that compromised the pedigree, reputation, and marriage prospects on both sides; many children were misdiagnosed with "Reactive attachment disorder", a psychiatric diagnosis associated with institutional rearing and/or maternal care deprivation. Yet, the first population survey relying on rigorous research assessments reported a high prevalence in that population [34]. Only recently, in Central and South America, has awareness of autism increased in both professional and lay audiences with population surveys now yielding prevalence estimates consistent with those from other countries [35, 36]. Yet, there are many world regions where awareness is still low, autism considered to be very rare and prevalence information lacking.

Early detection and screening for autism

After studies charted the early developmental trajectories of autism in infants and toddlers and with the development of early intensive interventions, several programs were developed to improve the detection of first signs of autism, promote early referrals for comprehensive developmental evaluations, decrease the age of diagnosis and thereby ameliorate the outcome. The CDC "Learn the Signs. Act early" (LTSAE) program [37] is an example of promotion of developmental monitoring by caregivers of their offspring using paper forms or smartphone apps and developmental screening by health professionals from birth to age 3. Several reliable and valid screening tools to screen for autism have been developed and are now widely used in clinical practice. In 2007, new US guidelines from the American Academy of Pediatrics (AAP) mandated pediatricians to systematically screen toddlers during well visits at 18 and 24 months of age with one of the available screeners [38]. Other countries such as Canada or the UK have not recommended universal screening but encourage developmental surveillance and autism-specific secondary screening for children presenting with developmental delay, behavioral problems, genetic syndromes or being at heightened risk (e.g. siblings of autistic children) [39].

There is evidence that the implementation of these various monitoring programs has resulted in earlier detection

[7] Symptom recognition before age 3 was a mandatory diagnostic criterion in DSM-IV, ICD-10 and before. In DSM 5 and ICD-11, it was removed (mostly to not deny the diagnosis among older individuals for whom evidencing an early onset may sometimes be difficult). However, DSM 5 stipulates that "symptoms are typically recognized during the 2nd year of life (12 to 24 months of age) and ICD-11 indicates that "The onset of the disorder occurs during the developmental period – typically in early childhood".

[8] When a child developed some language, he was considered to have reached a 'post-autistic' state (sic.)

and diagnosis, and has contributed to the increasing prevalence. In an effort to evaluate the AAP recommendation for universal screening, a Utah study of 36,233 toddlers followed up to ages 5–9 years and where 73% were screened at 18 and/or 24 months, found that screen-positive children were 10 times more likely to be later diagnosed with ASD than unscreened children. Moreover, compared to unscreened or screen-negative children with autism, screen-positive autistic children were diagnosed significantly earlier (by 10 months and 12 months, respectively) [40]. A similar lowering of age at diagnosis was observed in Oman after implementation of the same universal screening as part of routine immunization visits at age 18 months [41]. In San Diego County (California), a more intense version of universal screening has been implemented for years that requires training and collaborative work with community pediatricians, use of digital tools, repeated screening at 12, 18 and 24 months, prompt referral of screened positives followed by rapid treatment engagement [42]. In an evaluation of the *Get SET Early*[9] program, 203 pediatricians administered 57,603 screens during well-baby visits in the second year of life over a 5-year period. Overall, the program resulted in toddlers being referred for evaluation and treatment at a median age of 19.9 months, and even earlier (15.7 months) for those first screened at 12 months, compared to the average age at diagnosis of 4 years in the US. As discussed above (and see Fig. 3), the impact on prevalence of the *Get SET Early* program was illustrated in the last 3 CDC surveys where the San Diego site consistently obtained the highest prevalence estimates. The Puerto Rico prevalence estimates from the last CDC survey [2] also speak to the effect on age at diagnosis and impact on prevalence of the implementation of a developmental monitoring and screening program. After the recent deployment of the LTSAE program that only benefited children under age 5, the prevalence of autism at age 4 (4.72%) was 1.8 times higher than that at age 8 (2.64%) suggesting a direct impact on prevalence of implementation of the LTSAE program.

Thus, over the last 3 decades, major progress has been accomplished for screening and diagnosing earlier autism. Health care providers have access to a range of specific autism screening tools that can be deployed at different levels of care. When systematically used in primary care settings, they allow for earlier age at referral and diagnosis which has contributed to the rise in autism prevalence.

Access to services

Evidence for the efficacy of early intensive behavioral interventions emerged in the 1990s. In 2001, experts in the field recommended 25 h/week of intervention [43]. Multiple autism diagnostic centers were also developed in tertiary care hospitals with adoption of evidence-based guidelines for the evaluation and management of autism at different ages. In earlier CDC surveys, prevalence in African American and Hispanic children lagged behind that in White children, reflecting social inequalities in accessing health care; likewise, prevalence was positively associated with higher social-economic status. In the first CDC surveys, it was therefore predictable that, assuming a gradual reduction of barriers to access to care over time, the overall CDC prevalence would increase. Indeed, the prevalence difference for racial/ethnic minorities disappeared in the 2018 survey and reversed in the last two. The association with higher SES has also attenuated. These trends suggest an improvement in accessing services by minority and underserved groups which has contributed to the prevalence increase. Finally, in the US, new mandates for public and private health insurances facilitated access to services for a larger population, removing barriers to access diagnosis and treatment [44].

Overdiagnosis

Given the long waiting lists and the lengthy and costly procedures used in comprehensive evaluations, initiatives to increase diagnostic capacity may have resulted in expedited and less reliable diagnostic evaluations. In research too, the prohibitive costs of comprehensive case or outcome assessment encouraged "light phenotyping" approaches where questionnaire measures (known to lack specificity) are used as proxy for a diagnosis. In schools and community settings, isolated use of a singular diagnostic tool (such as the Autism Diagnostic Observational Schedule (ADOS) to which numerous providers are now trained to administer) and mechanical interpretation of its scores tend to replace expert clinician-led evaluations. At older ages (school age children, adults), overlap between autism symptoms and symptoms of common psychiatric disorders require a more careful differential diagnosis and the clinical experience in psychopathology that many autism service providers lack [45]. The extent of overdiagnosis was documented in a recent study [46]; however, it is yet unknown if this factor has contributed to the rising prevalence.

These multiple changes in the social context have contributed to making ASD diagnosis more readily accessible. These changes have operated gradually over time, their timing has varied by region and country, and their effects are synergistic. Thus, it has not been possible to quantify the effect of these changes on survey results and prevalence estimation. Nevertheless, evidence of their impact exists, and their combination most certainly contributed to the upward prevalence trend.

[9] SET: S = Screen; E = Evaluate; T = Treat.

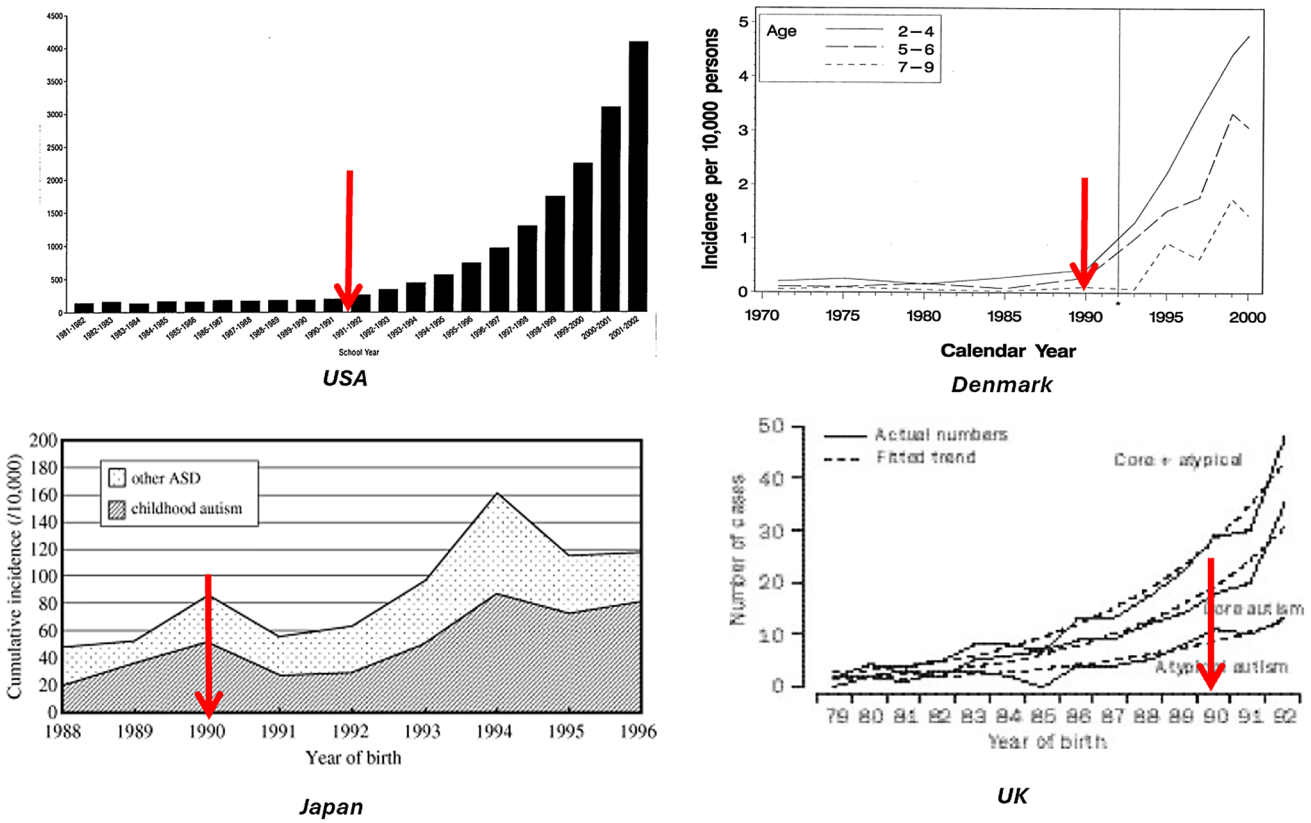

Fig. 5 Prevalence rose worldwide in the 1990's.
Sources: USA: [52]; Denmark : [53]; Japan : [54]; UK : [55]

"Genes do not cause an epidemic, we need an environmental toxin"

With a high sibling recurrence risk of about 20% [47] and twin studies showing much higher concordance in monozygotic same-sex twin pairs when compared to dizygotic ones, the heritability of ASD is one of the highest among psychiatric conditions with estimates of 83%–87% deriving from large-scale population studies [48]. A Swedish study of a 22,678 twin pairs born between 1982 and 2008 specifically examined if the respective contributions of genetic and environmental factors to autism clinically diagnosed had changed over time [49]. While the prevalence increased during the study period, the role of genetic factors remained very high and stable (heritability ranging from 88% to 97% across cohorts; aggregate: 93%) and that of environmental factors remained low and did *not* increase. The authors concluded appropriately that their results "do not suggest that environmental factors explain the increasing prevalence of ASD". The pace of discovery of rare and common genetic variants has led to the identification of a high number of variants etiologically associated to ASD [50]. Gene discoveries have begun to improve clinical care [51].

The search for environmental risk factors has been less successful. No space or time clusters exist that could point to candidate exposures, and there has been no adoption study of autism. It is also noteworthy that the rise in prevalence occurred worldwide at the same time, around the 1990 s, when diagnostic definitions and practices were harmonized and progressively but profoundly transformed (Fig. 5 [52–55]); the simultaneous rise in prevalence around the globe is not suggestive of an underlying environmentally mediated mechanism. False claims of links between autism and measles-mumps-rubella and thimerosal-containing vaccines have been debunked although their negative impact on public health and vaccination uptake have been long-lasting. In light of growing evidence of atypical development occurring in the first months of life [56], environmental risk research has shifted towards prenatal or periconceptional exposures.

With few exceptions (advanced parental age, prenatal exposure to valproic acid), progress has been limited by a lack of hypothesis-driven investigations. In announcing a search for "the environmental toxin", HHSS sated that

they would "look at everything", raising concerns about the value of new fishing expeditions. In a growing number of observational studies, several associations between autism risk and a myriad of prenatal exposures have been reported. Yet, replication is rarely obtained, and most associations remain confounded. Recent examples illustrate the risk of overinterpreting associations as signaling a causal link. For example, associations reported in observational studies of prenatal exposure to antidepressants (selective serotonin inhibitors) or antalgics (acetaminophen) or a range of maternal health conditions attenuated or disappeared when investigators could adjust on unmeasured familial and genetic confounders, using sibling comparison designs or negative controls [57–59]. Associations between prenatal exposures and offspring autism risk cannot disentangle genetic and environmental effects unless genetically sensitive designs are employed.

The strong genetic underpinnings are widely accepted in the autism academic and clinical community. Contrary to the HHSS statement, "the study of genetic causes has…" *not* "been a dead end". Further research into the additional contribution to ASD risk of environmental exposures is legitimate (and this needs not to hypothesize they are responsible for an epidemic) but requires very large population-based cohorts of pregnant women that remain scarce[10]. In addition, caution is necessary in evaluating observed associations as a causality assessment necessitates considering measured and unmeasured/unknown confounders including genetic factors as well as careful triangulation of the evidence. This requires planning, expertise and transparency. A frenetic search for an hypothetical "environmental toxin" appears futile.

In conclusion

The increase in prevalence in the last 30 years has been observed everywhere in the world and occurred when the definition of autism was broadened and new diagnostic practices were implemented. When the impact of one single methodological change on prevalence estimation could be quantified, research has established that they strongly contributed to the upward trend in prevalence. As briefly reviewed above, studies show that changes in the concepts and diagnostic criteria of ASD, diagnostic substitution, improved detection in the community and case ascertainment in surveys, the recognition of less severe forms of autism not associated with intellectual disability, have been demonstrated to individually account for non-trivial proportions of the prevalence increase. The precise quantification of each of these factors is difficult to obtain and does not immediately generalize across datasets, time periods, surveys and populations.

Nevertheless, these effects appear in isolation to be powerful enough such as the 5-fold difference in prevalence due to ascertainment differences across CDC sites, the 25%−66% increase in prevalence ascribed to diagnostic substitution or the 2.2-fold increase due to applying different diagnostic criteria to the same body of data to name a few. No study has been able to examine the joint effects of these factors and determine if they add up or overlap, and if so in which sequences over time and across populations. It is likely that the effects of each methodological change are time-varying. For example, diagnostic substitution may have been a strong driver of the increase in the 1990 s whereas changes in insurance mandates have operated much more recently. Furthermore, multiple contextual changes occurred in social policy, awareness, advocacy, de-stigmatization, service organization, access to services, availability of evidence-based interventions, research funding and so on whose effect has just not been quantified alone or in combination. Given the magnitude of the impact of some factors and the additional unquantified impact of other changes, it is certain that most, if not all, the upward trend is accounted for by those methodological changes or artefacts. By the same token, there is a fairly remote possibility that some small part of the trend could be attributable to unknown environmental risk although there is evidence that the role of environmental factors in the etiology of autism has not increased over time [49].

In presenting the last CDC report, the HHSS presented an interpretation of the data that was both ill-informed and biased and directly contradicted expert assessment of the CDC team itself. Similar misinterpretations of this upward trend in autism prevalence fueled anti-vaccine conspiracies 25 years ago that careful research rapidly refuted but had profound and lasting negative effects on the autism community and the public. The field does not need another crackpot theory about elusive environmental toxins. This pursuit, together with the systematic disregard for experts, the defunding of ongoing cutting-edge autism research and the channeling of resources towards operators and projects of dubious scientific merit, are raising concerns and act as a warning for what may come out from future HHSS directed initiatives. The autism scientific community stands on alert [60].

Acknowledgements Dr. Fombonne was an expert witness in trials on childhood vaccines and autism risk on behalf of the US Department of Justice and Department of Human & Health Services and of vaccine manufacturers (2003–2009). He is consulting on behalf of defendants in litigation relating to heavy metals in baby food and in litigation

[10] The Norwegian Mother, Father and Child Cohort (MoBa) and the UK Avon Longitudinal Study of Parents and Children (ALSPAC) cohorts are good examples.

related to acetaminophen. His research has been funded by INSERM, MRC, CIHR, Autism Speaks, Simons Foundation, and NIMH. None of his research was funded by industry.

Funding The author declares that no funds, grants, or other support were received during the preparation of this manuscript.

Declarations

Competing interests The author has no relevant financial or non-financial interests to disclose.

References

1. https://www.cdc.gov/autism/addm-network/index.html. Accessed 14 September 2025.
2. Shaw KA, Williams S, Patrick ME, et al. Prevalence and early identification of autism spectrum disorder among children aged 4 and 8 years - Autism and developmental disabilities monitoring network, 16 sites, United States, 2022. MMWR Surveill Summ. 2025;74(2):1–22. https://doi.org/10.15585/mmwr.ss7402a1.
3. Fombonne E, MacFarlane H. Epidemiology of autism spectrum disorders. In: Volkmar FR, Pelphrey KA, Vivanti G, editors. Handbook of autism and pervasive developmental disorders. 5th ed. Hoboken: Wiley; 2025.
4. Fombonne E, Editorial. The rising prevalence of autism. J Child Psychol Psychiatry. 2018;59(7):717–20. https://doi.org/10.1111/jcpp.12941.
5. Treffert DA. Epidemiology of infantile autism. Arch Gen Psychiatry. 1970;22(5):431–8. https://doi.org/10.1001/archpsyc.1970.01740290047006.
6. Burd L, Fisher W, Kerbeshian J. A prevalence study of pervasive developmental disorders in North Dakota. J Am Acad Child Adolesc Psychiatry. 1987;26(5):700–3. https://doi.org/10.1097/00004583-198709000-00014.
7. Fombonne E, MacFarlane H, Salem A. Epidemiological surveys of autism spectrum disorder: advances and remaining challenges. J Autism Dev Disord. 2021;51:4271–90.
8. Fombonne E. Epidemiology of pervasive developmental disorders. Pediatr Res. 2009;65(6):591–8.
9. Rosen NE, Lord C, Volkmar FR. The diagnosis of autism: from Kanner to DSM-III to DSM-5 and beyond. J Autism Dev Disord. 2021;51(12):4253–70. https://doi.org/10.1007/s10803-021-04904-1.
10. Kielinen M, Linna SL, Moilanen I. Autism in Northern Finland. Eur Child Adolesc Psychiatry. 2000;9(3):162–7.
11. Hansen SN, Schendel DE, Parner ET. Explaining the increase in the prevalence of autism spectrum disorders: the proportion attributable to changes in reporting practices. JAMA Pediatr. 2015;169(1):56–62. https://doi.org/10.1001/jamapediatrics.2014.1893.
12. Kim YS, Fombonne E, Koh YJ, Kim SJ, Cheon KA, Leventhal BL. A comparison of DSM-IV pervasive developmental disorder and DSM-5 autism spectrum disorder prevalence in an epidemiologic sample. J Am Acad Child Adolesc Psychiatry. 2014;53(5):500–8. https://doi.org/10.1016/j.jaac.2013.12.021.
13. Maenner MJ, Rice CE, Arneson CL, Cunniff C, Schieve LA, Carpenter LA, et al. Potential impact of DSM-5 criteria on autism spectrum disorder prevalence estimates. JAMA Psychiatr. 2014;71(3):292–300. https://doi.org/10.1001/jamapsychiatry.2013.3893.
14. Croen LA, Grether JK, Hoogstrate J, Selvin S. The changing prevalence of autism in California. J Autism Dev Disord. 2002;32(3):207–15.
15. King M, Bearman P. Diagnostic change and the increased prevalence of autism. Int J Epidemiol. 2009;38(5):1224–34. https://doi.org/10.1093/ije/dyp261.
16. Shattuck PT. The contribution of diagnostic substitution to the growing administrative prevalence of autism in US special education. Pediatrics. 2006;117(4):1028–37. https://doi.org/10.1542/peds.2005-1516.
17. Bishop DV, Whitehouse AJ, Watt HJ, Line EA. Autism and diagnostic substitution: evidence from a study of adults with a history of developmental language disorder. Dev Med Child Neurol. 2008;50(5):341–5. https://doi.org/10.1111/j.1469-8749.2008.02057.x.
18. Coo H, Ouellette-Kuntz H, Lloyd JE, Kasmara L, Holden JJ, Lewis ME. Trends in autism prevalence: diagnostic substitution revisited. J Autism Dev Disord. 2008;38(6):1036–46. https://doi.org/10.1007/s10803-007-0478-x.
19. Mandell DS, Ittenbach RF, Levy SE, Pinto-Martin JA. Disparities in diagnoses received prior to a diagnosis of autism spectrum disorder. J Autism Dev Disord. 2007;37(9):1795–802. https://doi.org/10.1007/s10803-006-0314-8.
20. Hertz-Picciotto I, Delwiche L. The rise in autism and the role of age at diagnosis. Epidemiology. 2009;20(1):84–90. https://doi.org/10.1097/EDE.0b013e3181902d15.
21. Rubenstein E, Tewolde S, Michals A, Fox M, Wang N. Prevalence of autism among Medicaid-enrolled adults. JAMA Psychiatr. 2023;80(12):1284–7. https://doi.org/10.1001/jamapsychiatry.2023.3708.
22. Hughes MM, Shaw KA, DiRienzo M, Durkin MS, Esler A, Hall-Lande J, et al. The prevalence and characteristics of children with profound autism, 15 sites, United States, 2000–2016. Public Health Rep. 2023;138(6):971–80. https://doi.org/10.1177/00333549231163551.
23. Scragg P, Shah A. Prevalence of Asperger's syndrome in a secure hospital. Br J Psychiatry. 1994;165(5):679–82. https://doi.org/10.1192/bjp.165.5.679.
24. Shah A, Holmes N, Wing L. Prevalence of autism and related conditions in adults in a mental handicap hospital. Appl Res Ment Retard. 1982;3(3):303–17. https://doi.org/10.1016/0270-3092(82)90022-4.
25. Mandell DS, Lawer LJ, Branch K, Brodkin ES, Healey K, Witalec R, Johnson DN, Gur RE. Prevalence and correlates of autism in a state psychiatric hospital. Autism. 2012;16(6):557–67. https://doi.org/10.1177/1362361311412058.
26. O'Nions E, Lewer D, Petersen I, Brown J, Buckman JEJ, Charlton R, Cooper C, El Baou C, Happé F, Manthorpe J, McKechnie DGJ, Richards M, Saunders R, Zanker C, Mandy W, Stott J. Estimating life expectancy and years of life lost for autistic people in the UK: a matched cohort study. Lancet Reg Health Eur. 2023;36:100776. https://doi.org/10.1016/j.lanepe.2023.100776.
27. Brugha TS, McManus S, Bankart J, Scott F, Purdon S, Smith J, Bebbington P, Jenkins R, Meltzer H. Epidemiology of autism spectrum disorders in adults in the community in England. Arch Gen Psychiatry. 2011;68(5):459–65.
28. Brugha TS, Spiers N, Bankart J, Cooper SA, McManus S, Scott FJ, et al. Epidemiology of autism in adults across age groups and ability levels. Br J Psychiatry. 2016;209(6):498–503.
29. Grosvenor LP, Croen LA, Lynch FL, Marafino BJ, Maye M, Penfold RB, Simon GE, Ames JL. Autism diagnosis among US children and Adults, 2011–2022. JAMA Netw Open. 2024;7(10):e2442218. https://doi.org/10.1001/jamanetworkopen.2024.42218.
30. Sun X, Allison C, Matthews FE, Sharp SJ, Auyeung B, Baron-Cohen S, et al. Prevalence of autism in Mainland China, Hong

Kong and taiwan: a systematic review and meta-analysis. Mol Autism. 2013;4:7.
31. Zhou H, Xu X, Yan W, Zou X, Wu L, Luo X, et al. Prevalence of autism spectrum disorder in China: a nationwide multicenter population-based study among children aged 6 to 12 years. Neurosci Bull. 2020;36(9):961–71.
32. Fombonne E, Du Mazaubrun C, Cans C, Grandjean H. Autism and associated medical disorders in a French epidemiological survey. J Am Acad Child Adolesc Psychiatry. 1997;36(11):1561–9. https://doi.org/10.1016/S0890-8567(09)66566-7.
33. Delobel-Ayoub M, Saemundsen E, Gissler M, Ego A, Moilanen I, Ebeling H, Rafnsson V, Klapouszczak D, Thorsteinsson E, Arnaldsdóttir KM, Roge B, Arnaud C, Schendel D. Prevalence of autism spectrum disorder in 7-9-Year-Old children in Denmark, Finland, France and iceland: A Population-Based registries approach within the ASDEU project. J Autism Dev Disord. 2020;50(3):949–59. https://doi.org/10.1007/s10803-019-04328-y.
34. Kim YS, Leventhal BL, Koh YJ, Fombonne E, Laska E, Lim EC, et al. Prevalence of autism spectrum disorder in a total population sample. Am J Psychiatry. 2011;168(9):904–12.
35. Roman-Urrestarazu A, Tyson A, Gatica-Bahamonde G, van Kessel R, Yang J, Mansilla C, et al. Bayesian prevalence of autism and unmet special education needs in Chile in a sample of three million school-age children. Autism. 2025;29(10):13623613251342310. https://doi.org/10.1177/13623613251342310.
36. Fombonne E, Marcin C, Manero AC, Bruno R, Diaz C, Villalobos M, Ramsay K, Nealy B. Prevalence of autism spectrum disorders in Guanajuato, mexico: the Leon survey. J Autism Dev Disord. 2016;46(5):1669–85. https://doi.org/10.1007/s10803-016-2696-6.
37. https://www.cdc.gov/act-early/index.html. Accessed 14 September 2025.
38. Hyman SL, Levy SE, Myers SM, COUNCIL ON CHILDREN WITH DISABILITIES, SECTION ON DEVELOPMENTAL AND BEHAVIORAL PEDIATRICS. Identification, Evaluation, and management of children with autism spectrum disorder. Pediatrics. 2020;145(1):e20193447. https://doi.org/10.1542/peds.2019-3447.
39. National Institute for Health and Care Excellence (NICE). Autism: recognition, referral and diagnosis of children and young people on the autism spectrum (NICE clinical guideline 128). London: National Collaborating Centre for Women's and Childrens Health. 2011. [cited 1/12/2013]. Available from url: http://www.nice.org.uk/guidance/CG128
40. Carbone PS, Campbell K, Wilkes J, Stoddard GJ, Huynh K, Young PC, Gabrielsen TP. Primary care autism screening and later autism diagnosis. Pediatrics. 2020;146(2):e20192314. https://doi.org/10.1542/peds.2019-2314.
41. Al-Mamari W, Idris AB, Al Aamri H, Al Khatri H, Al Sinani A, Al Kharusi R, Mirghani M, Al-Jabri M, Jalees S, Al-Hadhrami S, Islam M, Fombonne E. Could a national screening program reduce the age of diagnosis of autism spectrum disorder? J Autism Dev Disord. 2025. https://doi.org/10.1007/s10803-025-06770-7.
42. Pierce K, Gazestani V, Bacon E, Courchesne E, Cheng A, Barnes CC, et al. Get SET early to identify and treatment refer autism spectrum disorder at 1 year and discover factors that influence early diagnosis. J Pediatr. 2021;236:179–88. https://doi.org/10.1016/j.jpeds.2021.04.041.
43. National Research Council. Educating children with autism. Washington, DC: National Academies; 2001. https://doi.org/10.17226/10017.
44. Barry CL, Kennedy-Hendricks A, Mandell D, Epstein AJ, Candon M, Eisenberg M. State mandate laws for autism coverage and High-Deductible health plans. Pediatrics. 2019;143(6):e20182391. https://doi.org/10.1542/peds.2018-2391.
45. Fombonne E. Is autism overdiagnosed? J Child Psychol Psychiatry. 2023;64(5):711–4. https://doi.org/10.1111/jcpp.13806.
46. Duvall SW, Greene RK, Phelps R, Rutter TM, Markwardt S, Grieser Painter J, Cordova M, Calame B, Doyle O, Nigg JT, Fombonne E, Fair D. Factors associated with confirmed and unconfirmed autism spectrum disorder diagnosis in children volunteering for research. J Autism Dev Disord. 2025;55(5):1660–72. https://doi.org/10.1007/s10803-024-06329-y.
47. Ozonoff S, Young GS, Bradshaw J, Charman T, Chawarska K, Iverson JM, et al. Familial recurrence of autism: updates from the baby siblings research consortium. Pediatrics. 2024;e2023065297. https://doi.org/10.1542/peds.2023-065297.
48. Sandin S, Lichtenstein P, Kuja-Halkola R, Hultman C, Larsson H, Reichenberg A. The heritability of autism spectrum disorder. JAMA. 2017;318(12):1182–4. https://doi.org/10.1001/jama.2017.12141.
49. Taylor MJ, Rosenqvist MA, Larsson H, Gillberg C, D'Onofrio BM, Lichtenstein P, Lundström S. Etiology of autism spectrum disorders and autistic traits over time. JAMA Psychiatry. 2020;77(9):936–43. https://doi.org/10.1001/jamapsychiatry.2020.0680.
50. https://gene.sfari.org/. Accessed 14 September 2025.
51. Klitzman R, Bezborodko E, Chung WK, Appelbaum PS. Receiving de Novo genetic diagnoses for autism with intellectual disability: parents' views of impacts on families' reproductive decisions. J Community Genet. 2024;15(1):85–96. https://doi.org/10.1007/s12687-023-00679-3.
52. Gurney JG, Fritz MS, Ness KK, Sievers P, Newschaffer CJ, Shapiro EG. Analysis of prevalence trends of autism spectrum disorder in Minnesota. Arch Pediatr Adolesc Med. 2003;157(7):622–7. https://doi.org/10.1001/archpedi.157.7.622.
53. Madsen KM, Lauritsen MB, Pedersen CB, et al. Thimerosal and the occurrence of autism: negative ecological evidence from Danish population-based data. Pediatrics. 2003;112(3):604–6. https://doi.org/10.1542/peds.112.3.604.
54. Honda H, Shimizu Y, Rutter M. No effect of MMR withdrawal on the incidence of autism: a total population study. J Child Psychol Psychiatry. 2005;46(6):572–9. https://doi.org/10.1111/j.1469-7610.2005.01425.x.
55. Taylor B, Miller E, Farrington CP, Petropoulos MC, Favot-Mayaud I, Li J, et al. Autism and measles, mumps, and rubella vaccine: no epidemiological evidence for a causal association. Lancet. 1999;353(9169):2026–9. https://doi.org/10.1016/s0140-6736(99)01239-8.
56. Dawson G, Rieder AD, Johnson MH. Prediction of autism in infants: progress and challenges. Lancet Neurol. 2023;22(3):244–54. https://doi.org/10.1016/S1474-4422(22)00407-0.
57. Ames JL, Ladd-Acosta C, Fallin MD, Qian Y, Schieve LA, DiGuiseppi C, et al. Maternal psychiatric conditions, treatment with selective serotonin reuptake inhibitors, and neurodevelopmental disorders. Biol Psychiatry. 2021;90(4):253–62. https://doi.org/10.1016/j.biopsych.2021.04.002.
58. Ahlqvist VH, Sjöqvist H, Dalman C, Karlsson H, Stephansson O, Johansson S, Magnusson C, Gardner RM, Lee BK. Acetaminophen use during pregnancy and children's risk of Autism, ADHD, and intellectual disability. JAMA. 2024;331(14):1205–14. https://doi.org/10.1001/jama.2024.3172.
59. Khachadourian V, Arildskov ES, Grove J, O'Reilly PF, Buxbaum JD, Reichenberg A, Sandin S, Croen LA, Schendel D, Hansen SN, Janecka M. Familial confounding in the associations between maternal health and autism. Nat Med. 2025;31(3):996–1007. https://doi.org/10.1038/s41591-024-03479-5.
60. https://www.coalitionofautismscientists.org/. Accessed 14 September 2025.