Prevalence and Disparities in the Detection of Autism Without Intellectual Disability

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abstract

BACKGROUND: Intellectual ability predicts functional outcomes for children with autism spectrum disorder (ASD). It is essential to classify ASD children with and without intellectual disability (ID) to aid etiological research, provide services, and inform evidence-based educational and health planning.

METHODS: Using a cross-sectional study design, data from 2000 to 2016 active ASD surveillance among 8-year-olds residing in the New York-New Jersey Metropolitan Area were analyzed to determine ASD prevalence with and without ID. Multivariable Poisson regression models were used to identify trends for ASD with ID (ASD-I) and without ID (ASD-N).

RESULTS: Overall, 4661 8-year-olds were identified with ASD. Those that were ASI-I were 1505 (32.3%) and 2764 (59.3%) were ASD-N. Males were 3794 (81.4%), 946 (20.3%) were Non-Hispanic Black (Black), 1230 (26.4%) were Hispanic, and 2114 (45.4%) were Non-Hispanic white (white). We observed 2-fold and 5-fold increases in the prevalence of ASD-I and ASD-N, respectively, from 2000-2016. Black children were 30% less likely to be identified with ASD-N compared with white children. Children residing in affluent areas were 80% more likely to be identified with ASD-N compared with children in underserved areas. A greater proportion of children with ASD-I resided in vulnerable areas compared with children with ASD-N. Males had higher prevalence compared with females regardless of ID status; however, male-to-female ratios were slightly lower among ASD-I compared with ASD-N cases.

CONCLUSIONS: One-in-3 children with ASD had ID. Disparities in the identification of ASD without ID were observed among Black and Hispanic children as well as among children residing in underserved areas.

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WHAT IS KNOWN ON THIS SUBJECT: At present, intellectual ability remains the best predictor of functional outcomes and classification of degree of impairment among children with ASD. Studies have shown that many children with ASD do not have ID.

WHAT THIS STUDY ADDS: This study evaluated time trends in ASD with and without ID from 2000 to 2016 from a population-based study in a large and diverse metropolitan area and evaluated trends by sociodemographic factors. Health disparities were identified in ASD identification.

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Over the past decades, research has shown multifold increases in autism spectrum disorder (ASD) prevalence in the United States.[1–4] Although the Centers for Disease Control and Prevention (CDC) estimated ASD prevalence at 0.6% in 2000, by 2018, estimates rose to 2.3% (1-in-44 8-year-old children), surpassing intellectual disability (ID) as 1 of the most common neurodevelopmental disorders among US children.[4–6] Heterogeneity of ASD expression presents a challenge for research on etiology and interventions.[7,8] At present, intellectual ability represented by IQ remains the best predictor of functional outcomes and classification of ASD.[9,10] Before 2000, estimates suggested up to 75% of ASD children had ID. Recent studies report that 30% to 40% of ASD children have ID, indicating better identification of children with ASD without ID.[4,9–16]

Research shows varying ASD prevalence patterns when intellectual ability is considered. For example, although overall ASD prevalence estimates are considerably higher for males than females, among individuals with ASD and ID, the sex difference is less pronounced.[9,11,12,17] Similarly, recent findings indicate that the average lifetime costs to support individuals with ASD varies by intellectual level with estimated lifetime costs of $2.4 million for individuals with ASD and ID versus $1.4 million for ASD individuals without ID.[18] Consistent population-based surveillance is needed to track prevalence trends, describe changes in ASD expression, provide data for planning and allocation of resources and inform policy. Moreover, monitoring of ASD trends with consideration of intellectual disability can provide useful information on overall in population health and disparities as well as inform strategies for early ASD identification and intervention.

Few studies have examined trends in ASD prevalence and intellectual ability referencing demographic correlates, such as race and socioeconomic status (SES). Given the evidence demonstrating disparities in health and educational services, better understanding of the prevalence of ASD and ID by sociodemographic factors is needed.[19,20] For example, recent analysis showed that Non-Hispanic Black children with ASD were less likely to participate in Early Intervention Programs compared with Non-Hispanic white children.[21] Additionally, many studies have documented disparities in early ASD identification among Hispanic and Non-Hispanic Black children.[22,23] Similarly, SES is associated with early identification and services.[21,24,25] Understanding the associations of ASD and intellectual ability can inform recommendations and interventions provided by pediatricians and to guide future policy regarding ASD.

The current literature on the topic of ASD and ID has many limitations: some provide cross-sectional reports at single time points,[12] whereas others rely on administrative or clinical data that could lead to underestimation and/or bias,[9,26–29] others lack recent data,[14,30,31] or use homogeneous samples that are not generalizable to diverse populations.[32,33] To address these limitations, we used data from a population-based, active surveillance system to examine intellectual ability among 8-year-old children with ASD in a diverse US metropolitan region, from 2000 to 2016. We describe intellectual ability among children with ASD by sex, race and ethnicity, and SES and characterize temporal trends over this period.

METHODS

Cross-sectional data from the New Jersey Autism Study, which is part of the CDC - Autism and Developmental Disabilities Monitoring (ADDM) Network, were analyzed. ADDM is a population-based active surveillance tracking ASD.[34,35] The biannual ADDM surveillance provides ASD prevalence estimates for birth cohorts, at age 8. New Jersey data for 2000, 2002, 2006, 2010, 2012, 2014 and 2016 (representing birth cohorts 1992, 1994, 1998, 2002, 2004, 2006 and 2008, respectively) were included in this analysis.[5,13,36–40] Data from 2018 surveillance cycle were excluded here because changes in the 2018 ADDM methodology.[41]

ADDM ascertainment method is a 2-phase process.[35] In phase I, educational and clinical records of children satisfying birth year and residency criteria were reviewed. Records for children showing at least 1 specific, predetermined, ASD indicator were abstracted.[35] In phase II, using standard procedures, expert clinician reviewers identified and characterized ASD cases. ASD case definition was satisfied if behaviors documented in professional evaluations reflected the Diagnostic and Statistical Manual of Mental Disorders (DSM)-IV-TR ASD criteria (Supplemental Fig 3). Beginning with the 2014 ASD surveillance cycle, the ADDM Network also included a case definition based on DSM-5 criteria in addition to the case definition based on DSM-IV-TR criteria used from 2000 to 2012. Since ADDM network identified minimal differences in ASD prevalence using DSM-IV-TR and DSM-5 criteria,[38] to remain consistent across the study period, the DSM-IV-TR surveillance

case definition was used in this analysis.

Across all surveillance cycles, the methodology, ASD case definition and surveillance region remained constant. The study was approved by the Institutional Review Board of Rutgers University – New Jersey Medical School.

Population and Setting

The surveillance region included 4 counties (Essex, Hudson, Ocean, and Union) representing approximately 25% of the total New Jersey (8-year-old) population. The region is within the New York-New Jersey Metropolitan Area, the most populous metropolis and encompasses a diverse population (approximately, 40% to 42% Non-Hispanic white, 22% to 27% Non-Hispanic Black and 25% to 32% Hispanic). Each surveillance cycle included approximately 30 000 children. Population denominators were obtained from the National Center for Health Statistics vintage 2019 bridged-race postcensal data[42] (Supplemental Table 4).

Outcome Variable

The study included 2 case definitions: ASD-I and ASD-N. ASD-I included children with ID as defined by an intelligence quotient (IQ) score ≤ 70. ASD-N was defined as IQ score >70 and included children with borderline, average, and above average IQ. By convention and for research purposes, ID is traditionally defined according to IQ test scores.[4,15,26,38] Case definitions reflected the most recent IQ score in each child's record. Across all cycles, 4661 children were identified with ASD; 81% ($n = 3762$) had documented IQ scores.

Records of children with missing IQ test data ($n = 899$) were reviewed to classify children as ASD-I or ASD-N. When IQ test findings were unavailable, ASD-I was determined based on: (1) documented cognitive delay, deficit, or impairment by a professional ($n = 181$), (2) attempted administration of an IQ test that was discontinued because of nontestable status ($n = 117$), or (3) a special education classification of cognitive impairment ($n = 5$) at school. Conversely, ASD-N was determined for children without IQ scores based on: (1) special education classification of speech-only deficit ($n = 9$), (2) documentation of age-appropriate cognitive skills and/or average or higher academic skills by standardized performance tests ($n = 95$), or (3) ASD cases classified by the surveillance with mild impairment and no indication of special education services ($n = 91$). Following this enhanced classification, 398 ASD cases (9%) had undetermined intellectual ability and were excluded from further analysis.

Demographic Variables

Sex and race and ethnicity data were obtained from individual records and supplemented from birth certificates. Race and ethnicity was categorized for Non-Hispanic white (white), Non-Hispanic Black (Black) and Hispanic. Other race and ethnicity categories were excluded from race and ethnicity analysis because of small sample sizes, and less than 1% ($n = 29$) of the study population had no race and ethnicity information and were also excluded from race and ethnicity analysis. SES was based on median household income (MHI) at the census tract level and categorized as a 3-level variable based on MHI tertiles for all census tracts in New Jersey, namely: low-SES (MHI $\leq$ \$57 933), mid-SES (MHI = \$57 934–\$87 313) and high-SES (MHI $>$ \$87 313).

Additional SES indicators were considered, including the social vulnerability index (SVI) and the poverty rate, to assess whether different aspects of wealth were also associated with ASD-I and ASD-N. SVI is a CDC-developed multifactorial index representing 15 demographic factors grouped into 4 themes. The overall index (scaled 0–1, with 1 representing highest vulnerability) combines all 4 themes: (1) traditional SES factors; (2) household composition and disability; (3) minority status and language; (4) housing type and transportation. SVI was categorized as a 3-level variable based on all census tracts in New Jersey. Geographic areas with 20% or greater poverty rates were classified as "poverty areas" and areas with less than 20% poverty rates as "nonpoverty areas" consistent with US Census classification.[43]

Data Analysis

ASD prevalence was estimated overall and by intellectual ability status (ASD-I and ASD-N) in each of the 7 cycles (2000–2016). Wilson score method was used to compute 95% confidence intervals (CI). ASD prevalence was evaluated by sex, race and ethnicity, and SES. Descriptive statistics and Pearson χ^2 tests were used to characterize differences between children with ASD-I and ASD-N. Prevalence ratio (PR) and 95% CI were calculated to compare prevalence estimates overall and by sex and race and ethnicity from 2000 to 2016. Analyses examining SES were restricted to surveillance cycles 2010 through 2016 as comparable SES data from 2000 to 2006 were not available. Multivariable Poisson regression was used to analyze trends in ASD rates over time allowing for overdispersion; the log of the population was treated as an offset. Models were stratified by ASD type (ASD-I or ASD-N) and were adjusted for sex, race and ethnicity, SES, and birth year across

2010 to 2016 surveillance cycles. In sensitivity analyses, we refit models employing the stricter definition of ASD-I and ASD-N, based on IQ scores only. Statistical analyses were performed using SAS 9.4.

RESULTS

From 2000 to 2016, 4661 children satisfied the ASD case definition. ASD prevalence estimates increased 3-fold from 9.6 per 1000 (95%CI: 8.5–10.7) in 2000 to 31.8 per 1000 (95%CI: 30.0–33.8) in 2016. During the same period, ASD-I increased 2-fold, from 2.9 per 1000 (95%CI: 3.6–5.0) to 7.3 per 1000 (95%CI: 7.6–9.6), whereas ASD-N increased approximately 5-fold, from 3.8 per 1000 (95%CI: 3.3–4.7) in 2000 to 18.9 per 1000 (95% CI: 19.2–22.3) in 2016 (Fig 1 and Table 1).

The majority of ASD cases (n = 2764; 59%) satisfied the definition of ASD-N, whereas 32% (n = 1505) met the definition of ASD-I. Among ASD-N cases with IQ scores, 33% (n = 879) had borderline IQ (71–84). ASD-N represented 57% of ASD cases in 2000 as compared with 72% in 2016.

Comparison by Sociodemographic Factors

Males had higher overall ASD prevalence estimates compared with females across all birth cohorts, ranging from 3.6 to 4.1 male-to-female ratio across the 16-year period. Prevalence was higher among males regardless of ID status (Figs 2, A and D); however, the male-to-female ratio was higher among ASD-N, compared with ASD-I cases. ASD-N prevalence estimates for males increased 5.2-fold (PR = 5.2; 95% CI: 4.2–6.4), whereas estimates for ASD-I males increased 2.1-fold (PR = 2.1; 95% CI: 1.6–2.5). Similar increases were observed among females with ASD-N (PR = 5.8; 95% CI: 3.5–9.3) and ASD-I (PR = 1.8; 95% CI: 1.2–2.8).

ASD-I and ASD-N estimates varied by race and ethnicity. Increases in ASD-I and ASD-N were evident across all races and ethnicities between 2000 and 2016, with the greatest increase observed among Black (PR = 5.0; 95% CI: 3.1–8.1) and Hispanic (PR = 9.3; 95% CI: 5.6–15.4) children with ASD-N. By 2016, across all races and ethnicities, ASD-N estimates were higher than ASD-I estimates (Figs 2, B and E).

ASD-I and ASD-N estimates varied by SES. ASD-N prevalence estimates were lower among children residing in low-SES compared with high-SES areas (Fig 2F). From 2010 to 2016, estimates for ASD-N were stable for children residing in High-SES areas but increased 1.9-fold (95% CI: 1.5–2.3) and 1.6-fold (95% CI: 1.3–1.7) among children residing in low and mid-SES areas, respectively. Estimates for ASD-I also increased significantly for children residing in low-SES areas (Table 1). Results using alternative SES metrics were similar. Among ASD-I children, 31% resided in poverty areas, compared with 15% of ASD-N children. Similarly, 68% of ASD-I children resided in SVI-designated highly-vulnerable areas, compared with 38% of ASD-N children (Table 2).

Multivariable Regression

In multivariable regression analyses, ASD-N was twice as prevalent as ASD-I (adjusted rate ratio [ARR] = 2.1, 95% CI:1.8–2.5). The male-to-female ratio (MF-ARR) was higher among individuals with ASD-N (MF-ARR = 4.4, 95% CI:3.8–5.3) than ASD-I cases (MF-ARR = 3.9, 95% CI:3.3–4.7). Differences by race and ethnicity were evident. Whereas Black (ARR = 2.1, 95% CI:1.7–2.5) and Hispanic (ARR = 1.7, 95% CI:1.4–2.1) children were more likely to be identified with ASD-I, compared with white children, Black children were 30% less likely to be identified with ASD-N compared with white children. Among ASD-I cases there were no differences by SES; however, children residing in high-SES (ARR = 1.8, 95% CI:1.5–2.2) and mid-SES (ARR = 2.0,

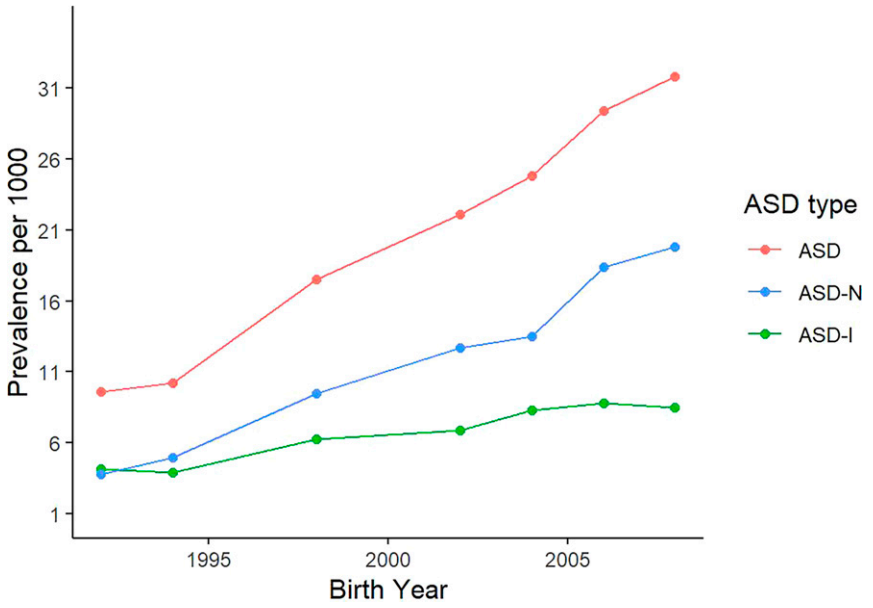

FIGURE 1
ASD prevalence estimates per 1000 8-year-old children in New Jersey overall and by intellectual ability.

TABLE 1 ASD With ID (ASD-I) and ASD Without ID (ASD-N) Prevalence Ratio in New Jersey Overall and by Sex, Race and Ethnicity, 2000 to 2016 and SES 2010 to 2016

	Prevalence Ratio (95% CI)	
	ASD-I, 2016–2000	ASD-N, 2016–2000
Overall	2.0 (1.6–2.5)	5.3 (4.4–6.4)
Sex		
Male	2.1 (1.6–2.7)	5.2 (4.2–6.4)
Female	1.8 (1.2–2.8)	5.8 (3.5–9.3)
Race and ethnicity		
Non-Hispanic white	1.5 (1.0–2.2)	4.2 (3.3–5.4)
Non-Hispanic Black	2.0 (1.4–2.8)	5.0 (3.1–8.1)
Hispanic	2.8 (1.8–4.3)	9.3 (5.6–15.4)
SES (MHI)[a]		
Low SES	1.4 (1.1–1.7)	1.9 (1.5–2.3)
Mid SES	1.4 (1.0–2.0)	1.6 (1.3–2.0)
High SES	0.7 (0.5–1.2)	1.0 (0.8–1.3)

ASD-I, ASD and intellectual disability; ASD-N, ASD without intellectual disability; CI, confidence interval; ID, intellectual disability; MHI, median household income; SES, socioeconomic status.

[a] Prevalence ratio compares prevalence estimates between 2016 to 2000 except for SES category comparing 2016 to 2010 for ASD-I and ASD-N cases.

95% CI:1.7–2.3) areas were more likely to have ASD-N compared with children from low-SES areas. An increase in identification of ASD-N by birth cohort was observed. Children born in 2008 were 80% more likely to be identified with ASD-N compared with children born in 2002. Similarly, there was a 40% increase in ASD-I identification over time. Sensitivity analyses using a narrower case definition of ASD-I and ASD-N, based solely on IQ scores, yielded similar results (Table 3).

DISCUSSION

From 2000 to 2016, we observed a 500% increase in the prevalence of ASD without ID and a 200% increase in the prevalence of ASD with ID using consistent, population-based active surveillance in a diverse and populous region. ASD prevalence increased across all sex, race and ethnicity, and SES subgroups and the greatest increases were seen among children without intellectual impairment. These findings are consistent with prior studies.[4,12–14] Although earlier studies reported that a large proportion of children with ASD had ID, more recent findings suggest the reverse, namely that most children with ASD have intellectual ability in the nondisabled range. In this study, 57% of ASD cases did not have ID in 2000 versus 72% in 2016, a trend likely explained by better recognition of ASD among children with average intellectual ability. Adjusting for various factors and time trends, for every child identified with ASD-I, 2 children with ASD-N were identified.

Consistent with previous findings, the male-to-female ratio was 3.9 among children with ASD-I; slightly lower than the male-to-female ratio

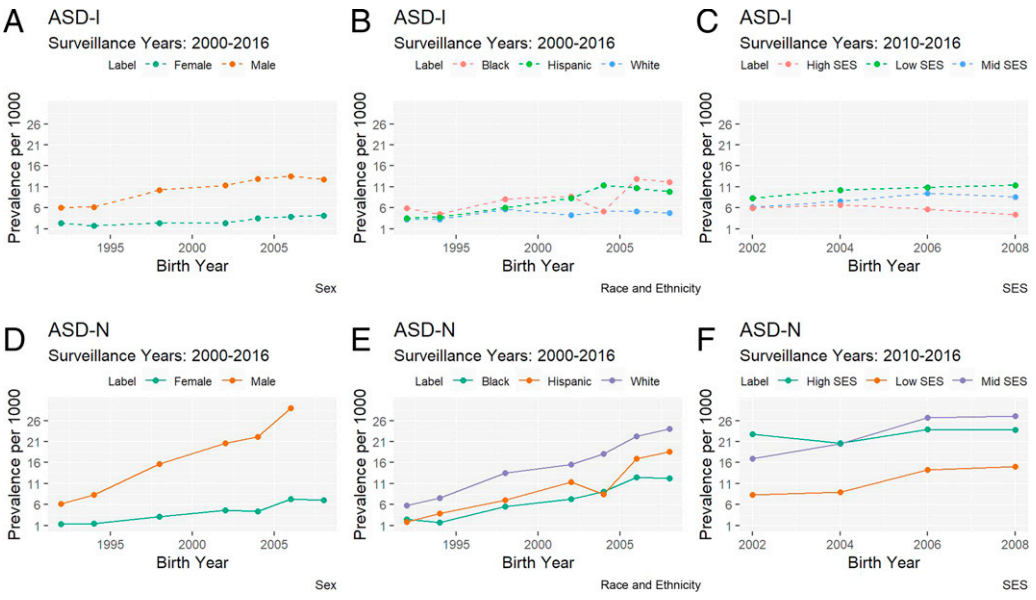

FIGURE 2

ASD-I and ASD-N prevalence per 1000. (A) ASD-I 2000–2016 prevalence by sex. (B) ASD-I 2000–2016 prevalence by race and ethnicity. (C) ASD-I 2010–2016 prevalence by socioeconomic status. (D) ASD-N 2000–2016 prevalence by sex. (E) ASD-N 2000–2016 prevalence by race and ethnicity. (F) ASD-N 2010–2016 prevalence by socioeconomic status.

TABLE 2 Sociodemographic Characteristics of Children With ASD With ID (ASD-I) and ASD Without ID (ASD-N)

	ASD-I		ASD-N		*P*
	n	%	*n*	%	
Overall	2560		1196		
Sex					.001
Male	944	79	2120	83	
Female	242	21	440	17	
Race and ethnicity					<.001
Non-Hispanic white	329	28	1367	53	
Non-Hispanic Black	387	32	364	14	
Hispanic	373	31	639	25	
SES (MHI)[a]					<.001
Low SES	770	64	956	37	
Mid SES	274	23	875	34	
High SES	152	13	729	29	
SES (poverty)[b]					<.001
Poverty area	325	31	331	15	
Non-poverty area	732	69	1861	85	
SES (SVI)[c]					
High vulnerability	720	68	835	38	
Mid vulnerability	191	18	678	31	
Low vulnerability	147	14	680	31	

ASD, autism spectrum disorder; ASD-I, ASD and intellectual disability; ASD-N, ASD without intellectual disability; ID, intellectual disability; MHI, median household income; SES, socioeconomic status; SVI, Social Vulnerability Scale.
[a] Socioeconomic status based on median household income at the census tract level.
[b] Socioeconomic status based on poverty rate at the census tract level.
[c] Socioeconomic status based on social vulnerability index.

of 4.4 among children with ASD-N. We anticipated a considerably lower sex ratio in the ASD-I group based on recent research.[44] The high sex ratio has been consistently observed in ASD populations and has been attributed to possible clinical differences between males and females and under identification and/or ascertainment bias in identifying females with ASD. This analysis shows possible under-identification of females with ASD regardless of ID status as the sex ratio among ASD-I children is 4:1.

By 2016, ASD-N was higher than ASD-I among all races, which suggests better identification and increased identification of children with ASD-N. However, our findings underscore the persistent sociodemographic disparities in the identification of ASD-N. Black children had lower estimates of ASD-N compared with white children, suggesting likely under-identification or misdiagnosis of Black children. Multiple studies have reported racial disparities in ASD identification,[23,45–48] disparities that may be driven by under-identification or misdiagnosis of children with ASD-N.[49] Under-identification may

TABLE 3 Multivariable Poisson Regression Model Stratified by ASD Type (ASD-I or ASD-N) and Adjusted for Sociodemographic Factors, 2010 to 2016 and Sensitivity Analysis Using the Narrower ASD Type Case Definition Based on IQ Scores

	Based on Enhanced ASD-I and ASD-N Study Case Definition (n = 4263)		Based on IQ Scores ASD-I and ASD-N Study Case Definition (n = 3762)	
	Model 1[a]	Model 2[b]	Model 3[c]	Model 4[d]
ASD type	ASD-I	ASD-N	ASD-I	ASD-N
Sex				
Female	Reference	Reference	Reference	Reference
Male	3.9 (3.3–4.7)	4.4 (3.8–5.3)	3.8 (3.2–4.6)	4.5 (3.8–5.4)
Race and ethnicity				
White	Reference	Reference	Reference	Reference
Black	2.1 (1.7–2.5)	0.7 (0.5–0.8)	2.3 (1.9–2.9)	0.7 (0.6–0.9)
Hispanic	1.7 (1.4–2.1)	0.8 (0.7–1.0)	1.8 (1.5–2.2)	0.9 (0.8–1.1)
SES				
Low	Reference	Reference	Reference	Reference
Mid	1.0 (0.9–1.2)	2.0 (1.7–2.3)	1.0 (0.9–1.2)	2.0 (1.7–2.3)
High	0.8 (0.6–1.0)	1.8 (1.5–2.2)	0.8 (0.6–1.0)	1.9 (1.6–2.3)
Birth year				
2002	Reference	Reference	Reference	Reference
2004	1.3 (1.0–1.5)	1.2 (1.0–1.5)	1.1 (0.9–1.4)	1.1 (0.9–1.4)
2006	1.5 (1.2–1.8)	1.6 (1.4–2.0)	1.6 (1.3–2.0)	1.6 (1.3–1.9)
2008	1.4 (1.1v1.7)	1.8 (1.5–2.2)	1.5 (1.2–1.9)	1.7 (1.4–2.1)

ASD, autism spectrum disorder; ASD-I, ASD and intellectual disability; ASD-N, ASD and no intellectual disability; IQ, intelligence quotient; SES, socioeconomic status.
[a] Model 1 evaluated rate ratio for ASD-I (enhanced study case definition) adjusted for sociodemographic factors, from 2010 to 2016 surveillance cycles.
[b] Model 2 evaluated rate ratio for ASD-N (enhanced study case definition) adjusted for sociodemographic factors, from 2010 to 2016 surveillance cycles.
[c] Model 3 (sensitivity analysis) evaluated rate ratio for ASD-I (IQ scores only) adjusted for sociodemographic factors, from 2010 to 2016 surveillance cycles.
[d] Model 4 (sensitivity analysis) evaluated rate ratio for ASD-N (IQ scores only) adjusted for sociodemographic factors, from 2010 to 2016 surveillance cycles.

result in loss of access to services. Universal ASD screening at routine pediatric visits is needed to better identify children with moderate to mild forms of ASD. Pediatricians are in an ideal position to address diagnostic inequalities. By 36-months, a child has typically seen a pediatrician at multiple well-child visits, and use of effective screeners to monitor child development can lead to earlier identification and the initiation of appropriate services at earlier ages. Cultural barriers may also impact ASD identification and targeted, culturally-sensitive parent-focused education may increase ASD knowledge and awareness as well as community acceptance, potentially reducing disparities in the utilization of services.[50–52]

Our findings demonstrate significant health disparities in ASD, particularly in relation to SES. From 2010 to 2016, ASD-N prevalence was higher in affluent areas compared with disadvantaged areas, indicating probable under-identification of children with ASD-N in disadvantaged areas. Many US studies have shown a positive SES gradient in relation to ASD prevalence. This SES relationship remains when intellectual ability is considered. Additionally, our findings indicate that a greater proportion of children with ASD-I reside in underserved, highly vulnerable communities, where they likely have diminished access to care and services. The SES-based differential among ASD-I children shows the need for additional effort to improve the early detection and linkage to services, especially in socially disadvantaged communities.

As identification of ASD-N improves, particularly among Black and Hispanic children, continued increases in ASD prevalence are likely. With up to 72% of the ASD population having borderline or average intellectual ability, emphasis should be placed on early screening, early identification, and early intervention to promote optimal functional outcomes. Studies have shown improved outcomes with early intense interventions[53–56] and this may be particularly true in ASD children without ID.[57] Moreover, research has shown gains in intellectual and adaptive functioning with intense intervention at younger ages in general.[53]

Strengths and Limitations

This was a population-based study using a standard ASD case definition, in a well-defined, diverse region that used an active case-finding methodology, independent of ASD diagnosis. ASD case definition, region, and methodology were consistent across all surveillance cycles. IQ data were available for 81% of cases and upon further review we determined intellectual ability for 91% of cases. Importantly, the study population was highly diverse and included large numbers of Hispanic and Black children, allowing us to generate accurate estimates for these understudied groups. Although many studies have reported on ASD and ID at a single time point, we examined patterns in ASD prevalence over a 16-year period. In addition, New Jersey is an autism epicenter and may be an indicator of future ASD trends in the United States.

Several limitations are acknowledged, including the broad categorization of ASD into 2 types, ASD-I and ASD-N. A substantial proportion of ASD-N cases had borderline IQ and these children likely experience significant challenges. Additionally, the age of IQ testing was not included in the analysis and ID was defined based on IQ scores without consideration to adaptive scores. Although the surveillance region included 4 urban and suburban counties encompassing a large diverse metropolitan population, the observed findings may not be representative of ASD prevalence across the United States. The ADDM surveillance method uses information from health and educational records, and ascertainment bias cannot be ruled out as some children lacked documented IQ scores.

CONCLUSIONS

We observed that 2-in-3 children with ASD do not have cooccurring ID, indicating increased identification of ASD without ID among all demographic subgroups from 2000 to 2016. However, our findings underscore the likely presence of health disparities in ASD without ID identification, especially among disadvantaged children. ASD is a major public health concern and prevalence estimates are likely to continue to rise as disparities are reduced and ASD identification is improved. Since ASD is a complex heterogeneous disorder, it is important to further study ASD in relation to intellectual ability to understand etiology and to inform effective interventions and appropriate services as well as aid in educational and health planning at the community level, as the needs for children with ASD and ID differ from the needs of children with ASD without ID. Furthermore, tracking ASD trends from diverse populations can identify health disparities and provide vital information on shifts in community health over time. Future work should focus on addressing

health disparities in the identification of ASD through the expansion of screening programs and improved linkage to care.

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ABBREVIATIONS

ADDM: Autism and Developmental Disabilities Monitoring Network
ASD: Autism Spectrum Disorder
CDC: Centers for Disease Control and Prevention
CI: confidence interval
ID: intellectual disability
MHI: median household income
PR: prevalence ratio
SES: socioeconomic status

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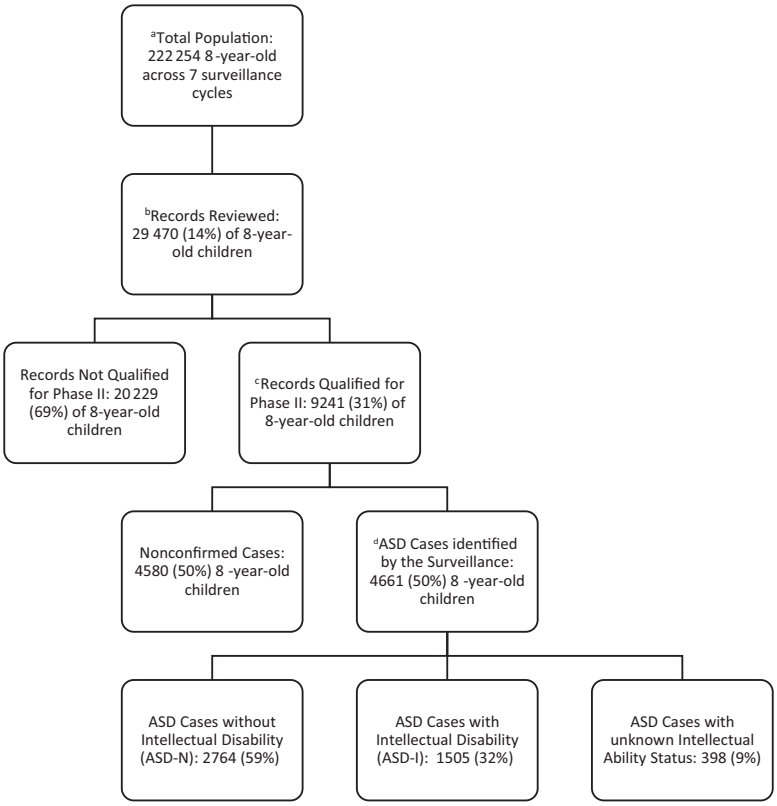
Supplemental Information



SUPPLEMENTAL FIGURE 3
New Jersey autism study surveillance process, 2000–2016. ASD, autism spectrum disorder; ASD-I, ASD with intellectual disability; ASD-N, ASD without intellectual disability.
[a] Population denominators were obtained from the National Center for Health Statistics (NCHS).
[b] Approximately 14% of the population qualify for phase I of the study based on residency, birth year, receipt of services through special education services in the surveillance year and/or having 1 or more surveillance specific International Classification of Diseases, Ninth Revision (ICD-9) codes.
[c] Records qualifying for phase II, had at least 1 surveillance indicator of ASD.
[d] ASD cases are confirmed based on active surveillance standard case definition based on DSM-IV-TR criteria.

SUPPLEMENTAL TABLE 4 Population Denominators for 8-Year-Olds Residing in the Surveillance Area During the Surveillance Year (by cycle)

	2000		2002		2006		2010		2012		2014		2016	
Surveillance Cycle	*N*	%	*N*	%	*N*	%	*N*	%	*N*	%	*N*	%	*N*	%
Overall	30 851		30 988		30 475		31 559		32 433		32 803		33 145	
Sex														
Male	15 797	51	15 941	51	15 471	51	16 034	51	16 614	51	16 721	51	16 903	51
Female	15 054	49	15 047	49	15 004	49	15 525	49	15 819	49	16 082	49	16 242	49
Race and ethnicity														
White, Non-Hispanic	13 059	42	13 246	43	13 167	41	13 577	43	13 706	42	13 579	41	13 269	40
Black, Non-Hispanic	8421	27	8228	27	7933	22	7387	23	7120	22	7132	22	7186	22
Hispanic	7750	25	7880	25	7780	31	8965	28	9746	30	10 173	31	10 619	32
SES (MHI tertiles)														
Low SES	—	—	—	—	—	—	16196	51	16493	51	16335	50	14719	44
Mid SES	—	—	—	—	—	—	7700	24	7333	23	7767	24	9249	28
High SES	—	—	—	—	—	—	5933	19	6745	21	6782	21	7740	23

Population denominators obtained from National Health Center for Statistics vintage 2019 bridged-race postcensal data. SES, socioeconomic status; —, not available.