Published in final edited form as:
Am J Public Health. 2009 March ; 99(3): 493–498. doi:10.2105/AJPH.2007.131243.

Racial/Ethnic Disparities in the Identification of Children With Autism Spectrum Disorders

David S. Mandell, ScD, **Lisa D. Wiggins, MS**, **Laura Arnstein Carpenter, PhD, BCBA**, **Julie Daniels, PhD**, **Carolyn DiGuiseppi, MD, PhD**, **Maureen S. Durkin, PhD, DrPH**, **Ellen Giarelli, EdD**, **Michael J. Morrier, MA, BCBA**, **Joyce S. Nicholas, PhD**, **Jennifer A. Pinto-Martin, PhD**, **Paul T. Shattuck, PhD**, **Kathleen C. Thomas, PhD**, **Marshalyn Yeargin-Allsopp, MD**, and **Russell S. Kirby, PhD**

David S. Mandell is with the Departments of Psychiatry and Pediatrics, University of Pennsylvania School of Medicine, Philadelphia. Lisa D. Wiggins and Marshalyn Yeargin-Allsopp are with the National Center on Birth Defects and Developmental Disabilities, Centers for Disease Control and Prevention, Atlanta, GA. Laura Arnstein Carpenter is with the Division of Genetics and Developmental Pediatrics, Medical University of South Carolina, Charleston. Julie Daniels and Kathleen C. Thomas are with the Cecil G. Sheps Center for Health Services Research, Chapel Hill, NC. Carolyn DiGuiseppi is with the Department of Epidemiology, Colorado School of Public Health, University of Colorado, Denver. Maureen S. Durkin is with the Departments of Population Health Sciences and Pediatrics, University of Wisconsin School of Medicine and Public Health, Madison. Ellen Giarelli and Jennifer A. Pinto-Martin are with the University of Pennsylvania School of Nursing, Philadelphia. Michael J. Morrier is with the Emory Autism Center, Emory University School of Medicine, Atlanta, GA. Joyce S. Nicholas is with the Departments of Biostatistics, Bioinformatics, and Epidemiology and Neurosciences, Medical University of South Carolina, Charleston. Paul T. Shattuck is with the George Warren Brown School of Social Work, Washington University, St Louis, MO. Russell S. Kirby is with the Department of Community and Family Health, University of South Florida, Tampa.

Abstract

Objectives—We sought to examine racial and ethnic disparities in the recognition of autism spectrum disorders (ASDs).

Methods—Within a multisite network, 2568 children aged 8 years were identified as meeting surveillance criteria for ASD through abstraction of evaluation records from multiple sources. Through logistic regression with random effects for site, we estimated the association between race/ethnicity and documented ASD, adjusting for gender, IQ, birthweight, and maternal education.

Results—Fifty-eight percent of children had a documented autism spectrum disorder. In adjusted analyses, children who were Black (odds ratio [OR] = 0.79; 95% confidence interval [CI] = 0.64, 0.96), Hispanic (OR = 0.76; CI = 0.56, 0.99), or of other race/ethnicity (OR = 0.65; CI = 0.43, 0.97)

Requests for reprints should be sent to David S. Mandell, University of Pennsylvania School of Medicine, Center for Mental Health Policy and Services Research, 3535 Market Street, 3rd Floor, Philadelphia, PA 19104 (mandelld@mail.med.upenn.edu.).

Contributors

D. S. Mandell conceptualized the study, conducted the statistical analyses, and wrote the first draft of the article. L. D. Wiggins assisted with the statistical analyses. P.T. Shattuck and R. S. Kirby provided statistical advising. All other authors made substantial contributions to conceptualizing the study, interpreting findings, and drafting the article.

Note. The findings and conclusions in this report are those of the authors and do not necessarily represent the views of the Centers for Disease Control and Prevention.

Human Participant Protection

This study was approved by the institutional review boards of the University of Pennsylvania School of Medicine and the Centers for Disease Control and Prevention.

were less likely than were White children to have a documented ASD. This disparity persisted for Black children, regardless of IQ, and was concentrated for children of other ethnicities when IQ was lower than 70.

Conclusions—Significant racial/ethnic dispatrities exist in the recognition of ASD. For some children in some racial/ethnic groups, the presence of intellectual disability may affect professionals' further assessment of developmental delay. Our findings suggest the need for continued professional education related to the heterogeneity of the presentation of ASD.

There is increasing evidence that autism spectrum disorders (ASDs) are often diagnosed several years after the onset of symptoms or are misdiagnosed as other disorders[1–3] even though an experienced clinician can accurately diagnose ASD in children as young as 2 years.[4–7] Delay in ASD diagnosis may be because of inadequate screening practices,[8–10] slow response to parental concerns,[11,12] or a lack of awareness of symptoms that manifest early in life.[13,14] Conversely, misdiagnosis is likely because of the similarity of certain features of ASD with other conditions that manifest in childhood.[15–17] For example, symptoms frequently observed in children with ASD, such as hyperactivity and behavioral difficulties, can lead clinicians to diagnose attention deficit/hyperactivity disorder instead of ASD.[18,19] Thepresenceof repetitive behaviors may lead to a diagnosis of obsessive–compulsive disorder, and noncompliance related to resistance to change may lead to a diagnosis of oppositional-defiant disorder.[20,21] Early diagnosis of ASD also is complicated by intellectual disability, which occurs in 50% to 60% of cases of children with more-severe symptom presentation[22] and can result in a primary diagnosis of developmental delay or intellectual disability. We have used "intellectually disabled" to describe children with IQs lower than 70.

This pattern of delayed and missed ASD diagnosis may be exacerbated among medically underserved ethnic and racial minorities. A large body of research documents ethnic and racial disparities in the diagnosis and treatment of many health conditions[23]; evidence has been inconclusive, however, for disparities in the diagnosis of ASD. Recent epidemiological studies have found conflicting results on ethnic or racial differences in the community prevalence of ASD.[3,22,24] Studies that have examined racial and ethnic differences in the age and accuracy of identification of ASD have also produced mixed results. For example, Mandell et al. studied Medicaid claims data from 1993 to 1999 and found that Black children were identified as having ASD later[25] and weremorelikely to be diagnosed with conduct disorder or adjustment disorder[26] than were White children. A survey of a large convenience sample of parents of children with ASD in the same geographic area, however, found no ethnic differences in age of diagnosis.[1] A report from the Centers for Disease Control and Prevention found no difference between the proportions of non-Hispanic White and non-Hispanic Black parents responding positively to the question, "Has a health care professional ever told you that your child has autism?" but this study was underpowered to test differences between the reported prevalence of ASD in White and Black children. Hispanic parents, however, were less likely than were non-Hispanic parents to say that their child had been diagnosed with ASD.[27]

The major limitation of these studies of disparities in the identification of ASD is that only children with a documented diagnosis of ASD were included. This strategy excludes children who meet diagnostic criteria but have not been identified by the health care or education systems as having ASD. Therefore, it is not possible to tell whether observed ethnic differences are because of true group differences in prevalence or of disparities in diagnostic practices.

Estimates of ethnic and racial disparities in the identification of ASD can inform the development of interventions to ameliorate them. For instance, if professionals consistently miss the diagnosis of ASD in certain groups, programs can be designed to provide better access to screening, referral, and education about developmental milestones. We examined ethnic and racial disparities in the identification of ASD. Data were collected by the Centers for Disease

Control and Prevention–sponsored Autism and Developmental Disabilities Monitoring (ADDM) network, which consists of multiple sites throughout the United States that collect population-based data on children who are at risk for ASD. Because case identification relies on record review rather than previous ascertainment by the health care or education systems, this dataset is an ideal way to examine whether the identification of ASD is dependent on ethnicity or race. On the basis of previous studies, we hypothesized that Black and Hispanic children meeting diagnostic criteria for ASD would be less likely than White children to be identified as such in their health and education records.

METHODS

Study Design

Our study was a cross-sectional study among 8-year-old children with ASD identified by population surveillance. The sample includes children who were born in 1994, reside in 1 of the 14 targeted study areas across the United States, and meet the ASD case definition for the study. Children were identified through abstraction of their records available at multiple educational and clinical sources. Clinician reviewers scored abstracted data to determine whether the ASD case definition was met. The outcome of interest was whether children meeting surveillance criteria for an ASD had any documentation of this diagnosis in their health care or education records.

Study Sample

The sample included all 2568 children born in 1994 who met ASD case definition as defined by the ADDM network in the 2002 study year. The ADDM network is a population-based surveillance program established by the Centers for Disease Control and Prevention in 2000. Fourteen sites estimated the prevalence of ASD in the 2002 study year.[24,28] The 14 sites included select study areas in 14 states: Alabama, Arizona, Arkansas, Colorado, Georgia, Maryland, Missouri, New Jersey, North Carolina, Pennsylvania, South Carolina, Utah, Wisconsin, and West Virginia. ADDM data were linked to birth certificate data to obtain measures of maternal education and birthweight.[29]

Case Definition

Case definition and methods for case ascertainment have been described in detail previously.[24,28,30] Briefly, 8-year-old children were classified by experienced clinician reviewers as having an ASD if they (1) had a documented previous ASD clinical diagnosis by a qualified examiner or a documented qualification for autism-related special education services during 1994 through 2002 or (2) did not have a documented ASD classification but had an evaluation record from an educational or clinical source indicating social and communication behaviors consistent with an ASD. To qualify as a documented clinical diagnosis, the clinician or other examiner had to have specifically stated in his or her evaluation that the child met diagnostic criteria for autistic disorder, pervasive developmental disorder not otherwise specified, or Asperger's syndrome. Previous studies have found documented ASD diagnoses in the community to have 98% specificity for a surveillance diagnosis of ASD.[3,31]

Case Ascertainment

To find ASD cases, we screened records of children born in 1994 who had been evaluated at a health or an educational source. Potential cases seen at health sources were sought through a range of discharge diagnoses, billing codes, or reasons for referral (e.g., ASD, intellectual disability, obsessive–compulsive disorder). Potential cases seen at educational sources included children who had been evaluated for developmental or behavioral concerns or who had received special education services.

Abstractors screened records of potential cases for a previously documented ASD classification or any of several ASD behavioral triggers specified in ADDM abstraction guidelines.[28] Behavioral triggers are symptoms that may indicate ASD, such as reduced eye contact or a lack of interest in other children. Suspected cases had their records abstracted, including developmental histories, psychometric test results, evaluation summaries, andincases suspectedbecause of behavioral triggers, verbatim transcriptions of behaviors associated with an ASD. Data collection is describedindetailelsewhere.[28,30]

Trained clinicians reviewed information abstracted from suspected ASD case records. The clinician reviewers applied a standardized coding scheme based on the 4th edition of the Diagnostic and Statistical Manual of Mental Disorders (*DSM-IV*).[32] This review process consisted of systematic classification of behaviors and final determination of case status (i.e., ASD or non-ASD) by qualified clinician reviewers.[30]

Variables

Documented ASD classification was the outcome variable of interest and was coded as present if the child's record documented a clinical diagnosis of ASD or qualification for special education services under an autism eligibility category.

Race/ethnicity was the primary independent variable of interest. We used 8 race/ethnicity categories: American Indian or Alaska Native; Asian; Black or African American; Hispanic, regardless of race; Native Hawaiian or Pacific Islander; White; other race or multiracial; and race or ethnicity not stated. Because children identified as Black are not all African American, we have used the term *Black*. Because of the small sample size associated with some groups, 4 categories—American Indian or Alaska Native; Native Hawaiian or Pacific Islander; other race or multiracial; and race or ethnicity not stated—were collapsed into 1 group.

Other child-level variables of interest included gender, cognitive impairment (measured as IQ<70), birthweight (>2500 g or ≤ 2500g), maternal education, and whether the child was from a site with access to children's education records. Gender was included because ASD affects predominantly males and therefore may be associated with practitioners' recognition of ASD.[22] The variables of cognitive impairment, birthweight, and education were included because of previous research associating them with either developmental monitoring of children or ethnicity.[23,29] The dummy variable indicating access to education records was added because IQ test results were more likely to be available in sites with such access.

Analyses

Analyses took place in 3 stages. First, percentages or means and standard deviations were calculated, as appropriate, for all variables of interest as a function of children's ethnicity. We used the χ^2 test to examine statistical differences in the distribution of each variable by ethnicity. If a statistically significant result was observed, pairwise tests were conducted with the Bonferroni method to correct for multiple testing. Second, we estimated the adjusted odds of having a documented diagnosis of ASD as a function of ethnicity with logistic regression with random effects; this model accounted for potential clustering by including the site as a random effect. Third, because of documented ethnic differences in intellectual disability[33] and its potentially important role in the recognition of ASD,[34] we estimated odds of having a documented diagnosis of ASD as a function of ethnicity within each cognitive impairment status category. We used the GLIMMIX macro in SAS 9.1.3 (SAS Institute Inc, Cary, NC) to implement the random effects models.[35]

RESULTS

Table 1 provides information on the sample as a function of ethnicity. Overall, 58% of children meeting ASD case definition had documentation of this classification on their clinical or education records; this proportion was not statistically significantly different by ethnicity. The prevalence of intellectual disability differed by ethnicity ($P<.001$); Black children were most likely and children of "other" race/ethnicity were least likely to have an IQ lower than 70 documented in their records. Black children also were most likely to have a birthweight of less than 2500 g ($P<.001$). Maternal education also differed by ethnicity, with White mothers most likely and Hispanic mothers least likely to have some college education ($P<.001$). There was no statistically significant difference between the percentages of White and Black children who came from sites with access to children's education records. Hispanic and Asian children were more likely to come from these sites, and children of other ethnicities were less likely.

Table 2 presents the results of the logistic regression with random effects predicting the presence of a documented ASD classification. Black, Hispanic, and "other" race/ethnicity children had lower odds of having a documented ASD classification than did White children. Boys were more likely to have a documented ASD than were girls. Children with an unknown IQ and those with an IQ lower than 70 were more likely to have a documented diagnosis of ASD than were children with an IQ of 70 or higher. Children of mothers with at least some college education were more likely than were children of mothers with less than a high school diploma to have documentation of ASD. The intraclass correlation was 0.037 among sites. We estimated goodness of fit with the Bayesian information criterion,[36] which was 10204.5.

Table 3 presents the results of the random effects logistic regression predicting ASD documentation, stratified by IQ status. Among children with IQs lower than 70, Black, Hispanic, and Asian children were less likely than were White children to have a documented ASD. Boys were more likely than were girls to have a documented ASD. Among children with IQs of 70 and higher, Black children were less likely than were White children to have a documented ASD, and children of mothers who had at least some college education were more likely to have a documented ASD. Among children for whom IQ was unknown, only greater maternal education increased the odds of a documented ASD.

DISCUSSION

We have found that among children meeting the case definition of ASD, only 58% had documentation of an ASD classification in their health or education records. Unadjusted analysis found no statistically significant difference in this documentation by ethnicity. In adjusted analyses, however, children who were Black, Hispanic, or of other, non-White ethnicities were less likely than were White children to have documentation of an ASD in their records. For Black children, this disparity persisted regardless of their known IQ; for Hispanic and Asian children, the disparity was concentrated among those with intellectual disability. These findings are in line with previous research showing that Black children with ASD are diagnosed at older ages than are White children[25] and that Hispanic children are less likely than are White children to be diagnosed with ASD at all.[27] Our results have considerably strengthened evidence on this disparity because the study included a community sample of children who met surveillance criteria for an ASD rather than relying solely on children already classified as ASD. Our results also suggest the potentially confounding role of other demographic characteristics (gender, maternal education) and clinical characteristics (birthweight, IQ) in the study of disparities.

Limitations

Several study limitations should be mentioned. First, the case identification strategy relied solely on secondary data. Especially for cases in which community professionals did not suspect ASD, children's records may not have included information that would have led to children meeting the study definition for ASD. Conversely, children with a putative ASD diagnosis were included without further examination. Previous studies have found this strategy to include few false positives,[3,31] and we did not test this assumption. Another limitation is that data abstracted from clinical and education records were not validated against direct observations. Third, we did not include information on specific symptoms, which can vary greatly across individuals[37]; if symptoms manifested differently among different ethnic groups, they may have confounded observed associations. Finally, data on some variables were missing for a portion of the sample. Although we included variables that might have explained some of the patterns of missing data, the nonrandom nature of missing data may have influenced the results.

Implications

Despite these limitations, our findings have important implications. The results suggest significant racial and ethnic disparities in the identification of children with ASD. For Hispanic and Asian children, this disparity was concentrated among children with co-occurring intellectual disability, who made up two fifths of participants and as much as 60% of children with autistic disorder in the general US population.[34] The presence of significant global intellectual disability can complicate the diagnosis of ASD.[38,39] Our results suggested that when clinicians observe cognitive impairment, they may be less likely to further assess some traditionally underserved minorities.

The effects of maternal education were stronger when children had an unknown IQ or an IQ of 70 or higher. Given the frequent co-occurrence of intellectual disability with ASD, children without severe cognitive impairment may be less typical in their presentation or their deficits less pronounced. In these cases, maternal education may be a proxy for greater knowledge of developmental milestones or greater ability to advocate with clinicians and educators for proper diagnosis. Mothers with more education also may be more aware of the extent to which an ASD diagnosis can result in additional services.[40,41]

There are many conceptual lenses through which one might interpret our findings. The Institute of Medicine report *Unequal Treatment* suggests that racial differences in diagnostic patterns may be attributable to institutional factors such as access to health care, general prejudices held by the clinician, clinicians' and families' interpretation of symptoms, and clinicians' application of rational (if erroneous) algorithms on the likelihood of a child having ASD.[23] The application of these faulty algorithms, sometimes referred to as statistical discrimination, can occur when clinicians have different expectations about the probability of autism occurring in children of different ethnicities.[42] For example, clinicians may correctly believe that ASD is more common among males.[22] They then may erroneously apply this statistic and diagnose girls less often than boys, even when the same symptoms are apparent. The fact that boys with ASD were more likely than are girls with ASD to be identified in our study lends some credence to this hypothesis. In the same vein, given a similar set of symptoms, practitioners may be more likely to diagnose autism in White children and intellectual disability in non-White children, given that research suggests lesser prevalence of intellectual disability in Whites.[33] The analysis stratified by cognitive status offers some support for this hypothesis. The disparity in the presence of a documented ASD was most apparent among Hispanic and Asian children with IQs lower than 70. When clinicians observe intellectual impairment in these groups, they may be less likely to continue evaluation because their unspoken hypotheses are confirmed.

The statistical discrimination model indicated a critical need to train health care and education professionals, especially those working in underserved communities, about the prevalence and presentation of ASD and its frequent co-occurrence with severe cognitive disability. The finding of the association of maternal education with a documented ASD indicated a need for further educating families about the signs of ASD and, perhaps more critical, the importance of timely and accurate diagnosis.

Kilbourne et al. suggested a model of health disparities research with 3 components: identification of disparities, discovery of malleable factors that mediate differential health outcomes, and strategies for reducing these disparities.[43] We have provided robust findings suggesting the presence of significant disparities in the identification of children with ASD. Factors associated with disparities in our study, combined with results from other studies, suggest that clinician and family beliefs, knowledge, and behavior contribute to this disparity.[26,44–47] Other studies, not specific to ASD, have suggested that the source of the disparity may lie in the interaction between parents and professionals.[48,49] Clearly, the next step in this line of research is to identify and test strategies that may lead to the early identification of children with ASD who currently are overlooked. ■

References

1. Mandell DS, Novak MM, Zubritsky CD. Factors associated with the age of diagnosis among children with autism spectrum disorders. Pediatrics 2005;116(6):1480–1486. [PubMed: 16322174]
2. Wiggins LD, Baio J, Rice C. Examination of the time between first evaluation and first autism spectrum diagnosis in a population-based sample. J Dev Behav Pediatr 2006;27(suppl 2):S79–S87. [PubMed: 16685189]
3. Yeargin-Allsopp M, Rice C, Karapurkar T, Doernberg N, Boyle C, Murphy C. Prevalence of autism in a US metropolitan area. JAMA 2003;289(1):49–55. [PubMed: 12503976]
4. Baird G, Charman T, Cox A, et al. Screening and surveillance for autism and pervasive developmental disorders. Arch Dis Child 2001;84(6):468–475. [PubMed: 11369559]
5. Bryson SE, Rogers SJ, Fombonne E. Autism spectrum disorders: early detection, intervention, education, and psychopharmacological management. Can J Psychiatry 2003;48(8):506–516. [PubMed: 14574826]
6. Charman T, Taylor E, Drew A, Cockerill H, Brown J, Baird G. Outcome at 7 years of children diagnosed with autism at age 2: predictive validity of assessments conducted at 2 and 3 years of age and pattern of symptom change over time. J Child Psychol Psychiatry 2005;46(5):500–513. [PubMed: 15845130]
7. Lord C, Risi S, DiLavore P, Shulman C, Thurm A, Pickles A. Autism from 2 to 9 years of age. Arch Gen Psychiatry 2006;63(6):694–701. [PubMed: 16754843]
8. Dearlove J, Kearney D. How good is general practice developmental screening? BMJ 1990;300:1177–1180. [PubMed: 1693300]
9. Dobos AE Jr, Dworkin P, Bernstein B. Pediatricians' approaches to developmental problems: has the gap been narrowed? J Dev Behav Pediatr 1994;15:34–39. [PubMed: 7515076]
10. Sices L, Feudtner C, McLaughlin J, Drotar D, Williams M. How do primary care physicians identify young children with developmental delays? A national survey. J Dev Behav Pediatr 2003;24(6):409–417. [PubMed: 14671474]
11. Glascoe FP. Parents' concerns about children's development: prescreening technique or screening test? Pediatrics 1997;99(4):522–528. [PubMed: 9093291]
12. Shevell MI, Majnemer A, Rosenbaum P, Abrahamowicz M. Profile of referrals for early childhood developmental delay to ambulatory subspecialty clinics. J Child Neurol 2001;16(9):645–650. [PubMed: 11575603]
13. Shah K. What do medical students know about autism? Autism 2001;5(2):127–133. [PubMed: 11706861]
14. Heidgerken AD, Geffken G, Modi A, Frakey L. A survey of autism knowledge in a health care setting. J Autism Dev Disord 2005;35:323–330. [PubMed: 16119473]

15. Cuccaro ML, Wright HH, Rownd CV, Abramson RK, Waller J, Fender D. Professional perceptions of children with developmental difficulties: the influence of race and socioeconomic status. J Autism Dev Disord 1996;26(4):461–469. [PubMed: 8863096]
16. Noterdaeme M, Amorosa H, Mildenberger K, Sitter S, Minow F. Evaluation of attention problems in children with autism and children with a specific language disorder. Eur Child Adolesc Psychiatry 2001;10(1):58–66. [PubMed: 11315537]
17. Ohta M, Nagai Y, Hara H, Sasaki M. Parental perception of behavioral symptoms in Japanese autistic children. J Autism Dev Disord 1987;17(4):549–563. [PubMed: 3680155]
18. Aman MG, Langworthy KS. Pharmacotherapy for hyperactivity in children with autism and other pervasive developmental disorders. J Autism Dev Disord 2000;30(5):451–459. [PubMed: 11098883]
19. Goldstein S, Schwebach A. The comorbidity of pervasive developmental disorder and attention deficit hyperactivity disorder: results of a retrospective chart review. J Autism Dev Disord 2004;34(3):329–339. [PubMed: 15264500]
20. Hatton DD, Hooper SR, Bailey DB, Skinner ML, Sullivan KM, Wheeler A. Problem behavior in boys with fragile X syndrome. Am J Med Genet 2002;108(2):105–116. [PubMed: 11857559]
21. Remington G, Sloman L, Konstantareas M, Parker K, Gow R. Clomipramine versus haloperidol in the treatment of autistic disorder: a double-blind, placebo-controlled, crossover study. J Clin Psychopharmacol 2001;21(4):440–444. [PubMed: 11476129]
22. Fombonne E. Epidemiology of autistic disorder and other pervasive developmental disorders. J Clin Psychiatry 2005;66(suppl 10):3–8. [PubMed: 16401144]
23. Institute of Medcine. Unequal Treatment. National Academies Press; Washington, DC: 2002.
24. Centers for Disease Control and Prevention. Surveillance summaries. MMWR Morb Mortal Wkly Rep 2007;56(SS-1):1–28. [PubMed: 17218934]
25. Mandell DS, Listerud J, Levy SE, Pinto-Martin JA. Race differences in the age at diagnosis among Medicaid-eligible children with autism. J Am Acad Child Adolesc Psychiatry 2002;41(12):1447–1453. [PubMed: 12447031]
26. 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):1795–1802. [PubMed: 17160456]
27. Centers for Disease Control and Prevention. Mental health in the United States: parental report of diagnosed autism in children aged 4–17 years—United States, 2003–2004. MMWR Morb Mortal Wkly Rep 2006;55(17):481–486. [PubMed: 16675944]
28. Rice CE, Baio J, Van Naarden Braun K, et al. A public health collaboration for the surveillance of autism spectrum disorders. Paediatr Perinat Epidemiol 2007;21(2):179–190. [PubMed: 17302648]
29. Cooper LA, Beach MC, Johnson RL, Inui TS. Delving below the surface. Understanding how race and ethnicity influence relationships in health care. J Gen Intern Med 2006;21(Suppl 1):S21–S27. [PubMed: 16405705]
30. Van Naarden Braun K, Pettygrove S, Daniels J, et al. Evaluation of a methodology for a collaborative multiple source surveillance network for autism spectrum disorder—Autism and Developmental Disabilities Monitoring Network, 14 Sites, United States, 2002. MMWR Morb Mortal Wkly Rep 2007;56(SS-1):29–40. [PubMed: 17230143]
31. Fombonne E, Heavey L, Smeeth L, et al. Validation of the diagnosis of autism in general practitioner records. BMC Public Health 2004;4:5. [PubMed: 15113435]
32. Diagnostic and Statistical Manual of Mental Disorders. Fourth Edition. American Psychiatric Association; Washington, DC: 1994.
33. Leonard H, Wen X. The epidemiology of mental retardation: challenges and opportunities in the new millennium. Ment Retard Dev Disabil Res Rev 2002;8(3):117–134. [PubMed: 12216056]
34. Gillberg C, Billstedt E. Autism and Asperger syndrome: coexistence with other clinical disorders. Acta Psychiatr Scand 2000;102(5):321–333. [PubMed: 11098802]
35. Fitzmaurice, G.; Laird, N.; Ware, J. Applied Longitudinal Analysis. Wiley; New York, NY: 2004.
36. Schwarz G. Estimating the dimension of a model. Ann Statistics 1978;6:461–464.
37. Volkmar FR, Lord C, Bailey A, Schultz RT, Klin A. Autism and pervasive developmental disorders. J Child Psychol Psychiatry 2004;45(1):135–155. [PubMed: 14959806]

38. Filipek PA, Accardo PJ, Ashwal S, et al. Practice parameter: screening and diagnosis of autism. Neurology 2000;55:468–479. [PubMed: 10953176]
39. Volkmar F, Cook E, Pomeroy J, Realmuto G, Tanguay P. Practice parameters for the assessment and treatment of children, adolescents and adults with autism and other pervasive developmental disorders. J Am Acad Child Adolesc Psychiatry 1999;38(12 Suppl):32S–54S. [PubMed: 10624084]
40. Stahmer AC, Mandell DS. State infant/toddler program policies for eligibility and services provision for young children with autism. Adm Policy Ment Health 2007;34(1):29–37. [PubMed: 16758329]
41. Yell ML, Drasgow E. Litigating a free appropriate public education: the Lovaas hearings and cases. J Spec Educ 2000;33(4):205–214.
42. Balsa AI, McGuire TG. Statistical discrimination in health care. J Health Econ 2001;20:881–907. [PubMed: 11758051]
43. Kilbourne AM, Switzer G, Hyman K, Crowley-Matoka M, Fine MJ. Advancing health disparities research within the health care system: a conceptual framework. Am J Public Health 2006;96(12): 2113–2121. [PubMed: 17077411]
44. Mandell DS, Novak M. The role of culture in families' treatment decisions for children with autism spectrum disorders. Ment Retard Dev Disabil Res Rev 2005;11:110–115. [PubMed: 15977313]
45. Coonrod E, Stone W. Early concerns of parents of children with autistic and nonautistic disorders. Infants Young Child 2004;17(3):258–269.
46. Daley TC. From symptom recognition to diagnosis: children with autism in urban India. Soc Sci Med 2004;58:1323–1335. [PubMed: 14759679]
47. Glascoe FP. Parents' evaluation of developmental status: how well do parents' concerns identify children with behavioral and emotional problems? Clin Pediatr (Phila) 2003;42(2):133–138. [PubMed: 12659386]
48. Wissow LS, Larson SM, Roter D. Longitudinal care improves disclosure of psychosocial information. Arch Pediatr Adolesc Med 2003;157:419–425. [PubMed: 12742876]
49. Wissow LS, Larson S, Anderson J, Hadjiisky E. Pediatric residents' responses that discourage discussion of psychosocial problems in primary care. Pediatrics 2005;115(6):1569–1578. [PubMed: 15930218]

TABLE 1

Characteristics of Children Born in 1994 and Aged 8 Years, by Race/Ethnicity: United States, 2002

	Total (N = 2568)	White (n = 1620)	Black (n = 589)	Hispanic (n = 194)	Asian (n = 57)	Other (n = 108)	
Documented autism spectrum disorder (n = 1497), %	58	60	56	52	60	51	.065
Boys (n = 2077), %	81	80	82	80	79	84	.783
IQ,[a,b,c,d,e] %							<.001
≤ 70 (n = 1063)	41	47	25	39	35	47	
< 70 (n = 856)	33	28	49	39	37	21	
Unknown (n = 649)	25	25	26	22	28	32	
Birthweight,[a,c,d,e,f,g] %							<.001
≤ 2500 g	7	6	13	5	3	3	
> 2500 g	63	67	61	62	44	34	
Missing	30	27	26	33	53	63	
Maternal education[a,b,c,d,e,f,g,h,i]							<.001
Less than high school graduate	11	7	15	30	9	8	
High school graduate or equivalent	24	25	27	20	5	11	
Some college	35	40	30	14	30	18	
Missing	30	28	28	36	56	63	
From site with access to children's education data[a,b,c,e,f]	76	75	76	91	84	68	<.001

Note. Percentages may not total 100% because of rounding.

[a] Difference between Whites and Blacks significant at $P < .05$ in posthoc pairwise comparison.

[b] Difference between Whites and Hispanics significant at $P < .05$ in posthoc pairwise comparison.

[c] Difference between Blacks and Hispanics significant at $P < .05$ in posthoc pairwise comparison.

[d] Difference between Blacks and others significant at $P < .05$ in posthoc pairwise comparison.

[e] Difference between Hispanics and others significant at $P < .05$ in posthoc pairwise comparison.

[f] Difference between Whites and Asians significant at $P < .05$ in posthoc pairwise comparison.

[g] Difference between Whites and others significant at $P < .05$ in posthoc pairwise comparison.

[h] Difference between Blacks and Asians significant at $P < .05$ in posthoc pairwise comparison.

[i] Difference between Hispanics and Asians significant at $P < .05$ in posthoc pairwise comparison.

TABLE 2

Logistic Regression Predicting Presence of Previously Documented Autism Spectrum Disorders Among Children Born in 1994 and Aged 8 Years: United States, 2002

	OR (95% CI)
Race/ethnicity	
White (Ref)	1.00
Black	0.79* (0.64, 0.96)
Hispanic	0.76* (0.56, 0.99)
Asian	0.91 (0.52, 1.56)
Other	0.65* (0.43, 0.97)
Gender	
Girl (Ref)	1.00
Boy	1.35* (1.10, 1.64)
IQ	
≥ 70 (Ref)	1.00
< 70	1.67* (1.38, 2.03)
Unknown	1.70* (1.38, 2.09)
Birthweight	
> 2500 g (Ref)	1.00
≤ 2500 g	1.07 (0.78, 1.47)
Missing	0.81 (0.41, 1.59)
Maternal education	
Less than high school graduate (Ref)	1.00
High school graduate	1.31 (0.98, 1.76)
Some college	1.44* (1.08, 1.91)
Missing	0.80 (0.39, 1.58)
From site with access to children's education data	1.09 (0.90, 1.32)

Note. OR = odds ratio; CI = confidence interval.

* *P* <.05.

TABLE 3

Results of Logistic Regression Predicting Presence of a Clinical or Educational Record of Autism, by IQ

	IQ < 70 (n = 856), OR (95% CI)	IQ ≥ 70 (n = 1063), OR (95% CI)	IQ Missing (n = 649), OR (95% CI)
Race/ethnicity			
White (Ref)	1.00	1.00	1.00
Black	0.67* (0.49, 0.92)	0.68* (0.47, 0.97)	1.12 (0.75, 1.69)
Hispanic	0.53* (0.32, 0.87)	0.88 (0.54, 1.43)	1.13 (0.55, 2.31)
Asian	0.38* (0.15, 0.92)	1.22 (0.49, 3.03)	2.52 (0.69, 9.17)
Other	0.52 (0.22, 1.18)	0.65 (0.36, 1.16)	0.82 (0.39, 1.71)
Gender			
Girl (Ref)	1.00	1.00	1.00
Boy	1.49* (1.06, 2.10)	1.31 (0.91, 1.85)	1.29 (0.88, 1.87)
Birthweight			
> 2500 g (Ref)	1.00	1.00	1.00
≤ 2500 g	0.98 (0.58, 1.63)	1.31 (0.79, 2.19)	0.89 (0.47, 1.70)
Missing	0.53* (0.19, 1.51)	1.04 (0.25, 4.30)	1.20 (0.35, 4.14)
Maternal education			
Less than high school graduate (Ref)	1.00	1.00	1.00
High school graduate	1.20 (0.76, 1.91)	1.20 (0.72, 2.00)	1.91* (1.05, 3.47)
Some college	1.19 (0.75, 1.90)	1.58* (1.03, 2.51)	1.99* (1.10, 3.62)
Missing	1.27 (0.44, 3.69)	0.64 (0.15, 2.78)	0.41 (0.12, 1.47)
From site with access to children's education data	1.18 (0.82, 1.71)	1.26 (0.92, 1.73)	0.87 (0.62, 1.22)

*$P < .05$.