



Published in final edited form as:

J Autism Dev Disord. 2022 May ; 52(5): 2274–2283. doi:10.1007/s10803-021-05118-1.

Still Left Behind: Fewer Black School-Aged Youth Receive ASD Diagnoses Compared to White Youth

Serene Habayeb^{1,2}, Lauren Kenworthy², Andrea De La Torre^{3,2}, Allison Ratto²

¹Department of Psychology and Behavioral Health, Children's National Hospital, 111 Michigan Ave NW, Washington, District of Columbia 20010, USA

²Center for Autism Spectrum Disorder, Children's National Hospital, 15245 Shady Grove Rd. Suite 350, Rockville, Maryland 20850, USA

³Department of Psychology, University of Baltimore, 1420 N Charles Street, Baltimore, Maryland 21201, USA

Abstract

Prior research suggests that Black children are at risk for delays in diagnosis of autism, but factors that influence diagnostic timing across races remain unclear. This study analyzed data from Black and White children who received a first-time autism diagnosis at a specialty clinic. Black youth were under-represented in the group who received a first diagnosis in middle/late childhood (i.e., after age six). Receiving a diagnosis later in childhood was related to higher IQ (trend level) and more internalizing problems for White children whereas it was related to lower IQ (trend level) and higher ASD symptom intensity for Black children. Findings suggest racial disparities in early identification of autism may be diminishing but persist among those diagnosed later in childhood..

Keywords

Autism Spectrum Disorder; First Diagnosis; Diagnosis Delay; Race

Introduction

Recent estimates suggest that 1 in 54 children in the United States have a diagnosis of Autism Spectrum Disorder (ASD; Maenner et al. 2020). Although autism can reliably be diagnosed by age two (Lord et al. 2006), up to half of children do not receive their first ASD diagnosis until after age six (Sheldrick et al. 2017). This is concerning given that early intervention in early childhood prior to school entry has been found to improve receptive and expressive language, cognitive functioning, and adaptive skills (Reichow et al. 2018). A number of variables have been identified as predictors of diagnostic timing. For example, higher socioeconomic status, and greater parental concern about initial symptoms are

[✉] Serene Habayeb, shabayeb@childrensnational.org.

Authors Contribution This study was conducted by SH under direct mentorship from AR. SH and AR contributed to the study conception, design, data analysis, and material preparation. AT performed a literature review and contributed to writing the introduction and discussion. LK serves as the clinic director and oversaw data collection and data management of this clinical dataset. The first draft of the manuscript was written by SH with support from AT and AR and all authors reviewed and commented on subsequent versions of the manuscript. All authors read and approved the final manuscript.

associated with earlier diagnoses (Daniels & Mandell 2014; Mandell et al. 2005). Intensity of some autistic traits (e.g., hand flapping, odd social play) also relate to earlier diagnoses (Mandell et al. 2005). Those who receive diagnoses later in life often have more subtle symptoms and higher cognitive functioning in early childhood, and therefore get missed by screening protocols that are often only in place until about age three (Mazurek et al. 2014; Ozonoff et al. 2018). The relationship between cognitive functioning and receiving an ASD diagnosis is mixed, particularly in those with an intellectual disability (ID). Although autistic children with co-occurring ID usually receive their first autism diagnosis earlier than those without ID (Maenner et al. 2020), having an ID may decrease the likelihood of healthcare professionals assessing for ASD once an ID has been determined, again, delaying an appropriate diagnosis (Mandell et al. 2009). Receiving a diagnosis later in life is often associated with greater emotional and behavioral problems, possibly due to not accessing preventative healthcare services but rather waiting until crises arise in order to seek care and receive a diagnosis (Liptak Stuart & Auinger 2006).

Historically, research has found that Black children on average receive later diagnoses than White children (Mandell et al. 2002). It is, therefore, promising that more recent research shows that autism prevalence rates for Black and White children, at ages four and eight, do not significantly differ (Maenner et al. 2020; Shaw et al. 2020). This may suggest that more Black children are appropriately receiving diagnoses earlier in life, perhaps due to increased public health initiatives to increase early developmental monitoring and screening for ASD in the first few years of life (Daniel et al. 2009; King et al. 2010). However, the disparity in diagnostic timing does not appear to be completely resolved, as there are continued findings that Black children, on average, receive their first diagnosis of ASD later than White children (Baio et al. 2018; Shaw et al. 2020). Black children are almost three times less likely to get an ASD diagnosis on their first visit to a specialty care clinic compared to White children (Mandell et al. 2007) and often require three or more visits until they receive an autism diagnosis (Constantino et al. 2020). Most Black children receive their first diagnosis after age five, coming on average three years after parents first voice developmental concerns (Constantino et al. 2020).

Racial disparities in differences of age of diagnosis may be mediated or moderated by a number of other factors. Overall, fewer children in low socioeconomic status (SES) communities receive appropriate diagnoses of ASD, and Black children are over-represented in low SES communities (Maenner et al. 2020). However socioeconomic disparities do not fully explain the different prevalence rates of ASD between racial groups; even within low SES communities, racial minority children are still less likely to have an ASD diagnosis (Durkin et al. 2017). Relatedly, poor access to appropriate healthcare may lead to a delay in receiving an appropriate diagnosis for some, although this is not consistent across racial groups. One study found that while White children with a consistent source of medical care received earlier ASD diagnoses, Black children with a consistent source of medical care were less likely to (Emerson et al. 2016). This may be related to cultural differences in parenting beliefs and attitudes for some Black families. For example, ethnic minority parents are less likely to contact a health care professional for ASD related concerns compared to White parents, and at times may wait years to receive a formal diagnosis (Zelege et al. 2019). One qualitative study seeking insight from Black parents of autistic children

reported that Black parents perceive racial disparities in receiving diagnoses due to 1) insufficient education and training on ASD for Black parents 2) health care professionals unresponsiveness to concerns of Black parents and 3) health care professionals poor knowledge of ASD diagnostic criteria (Pearson & Meadan 2018).

Timing of diagnosis and race also seems to be affected by the intensity of ASD symptoms and cognitive functioning. Black children are over-represented among youth with ASD and co-occurring ID and under-represented among youth with ASD without ID, who often are not diagnosed until middle childhood or adolescence (Constantino et al. 2020; Jo et al. 2015; Maenner et al. 2020). Among those with “mild/moderate” ASD, there is a higher proportion of White children receiving later diagnoses, compared to Black children.

Given these complex inter-relationships, the goal of this study was 1) to understand whether age of receiving a first diagnosis differed between Black and White children presenting to a hospital clinic and 2) to identify factors that predict age of first diagnosis, focusing on the effects of parent-reported autism symptom intensity, behavioral/emotional functioning, and cognitive abilities. Since most historical research suggests a delay in diagnosis for Black children (Constantino et al. 2020; Daniels & Mandell 2014), we hypothesized that on average, Black children would receive a later first diagnosis compared to White children. We further hypothesized that children who received later diagnoses would show lower symptom intensity, but that this relationship would be moderated by race, such that Black children would require relatively more symptoms overall in order to receive a diagnosis of ASD. In terms of behavioral/emotional functioning, we hypothesized that higher behavioral/emotional difficulties would be associated with a later age of diagnosis, but that this relationship would also be moderated by race, such that Black children would require a higher level of behavioral/emotional dysfunction to receive a diagnosis of ASD from a clinic compared to White children. In terms of cognitive functioning, we hypothesized that lower cognitive abilities would be associated with an earlier age of diagnosis, and that this relationship would be stronger for White compared to Black children.

Methods

Participants

This project used archival data and was conducted in compliance with standards established by the institution’s Institutional Review Board (IRB), including procedures for informed consent. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Data was collected from a clinical sample of 5,697 youth seen between 2014 and 2020 for a diagnostic or neuropsychological clinical evaluation in an outpatient neuropsychology clinic which also housed an autism center. The clinic was affiliated with an academic medical center in the Washington, DC metropolitan area. From the total sample, a subsample of 514 participants was identified who met the following criteria: received a first-time diagnosis of ASD, identified as either Black/African American (N = 164) or White (N = 350), and

had complete data available on demographic variables and measures of interest (Table 1). Demographic variables (i.e., child race/ethnicity, gender) were gathered via parent report. Consistent with gender ratios in ASD (Maenner et al. 2020), there were more males ($N = 383$) than females ($N = 128$) in the sample, and three participants were transgender or gender nonconforming. The gender difference (males vs. females) was similarly distributed among in the Black and White group, $X^2(1, 511 = 0.056, p = 0.813)$. In this system, data about SES are not routinely gathered. The clinic serves families from a wide range of socioeconomic levels from the large urban area as well as neighboring suburbs and exurban areas. The clinic serves children with both private and public insurance. Previous research from our clinic found that SES did not significantly differ between parents of Black and White children with ASD without co-occurring ID (Ratto et al. 2016). This was a highly educated sample, as among both Black and White families, most parents had at least a college education.

Ages of first diagnosis ranged from 1.3 years to 20 years, and visits were conducted between 2014 and 2020. The distribution of age was moderately positively skewed (skewness statistic = 0.74) given that more children are seen for diagnostic evaluations earlier in life at our clinic. There were no outliers in our data (i.e., no points lying outside the outer fences of a boxplot). All participants were diagnosed with ASD by a clinician (child clinical psychologist or psychiatrist), with advanced training and expertise in ASD and related conditions, based on DSM-5 (American Psychiatric Association 2013) diagnostic criteria, utilizing both developmental history and direct observation and clinical assessment. Of these, 94.5% met criteria for an ASD based on the Autism Diagnostic Observation Schedule (ADOS; Lord et al. 2006) or its recent revision, the ADOS-2 (Lord et al. 2006). Diagnostic information was entered by the evaluating clinician directly into the database, and the clinician was prompted by the data entry system to indicate for each diagnosis entered whether this was a new/first-time diagnosis (i.e., the child did not have a diagnosis of ASD before the visit).

Measures

Parent-Reported Behavioral–Emotional Functioning

The Child Behavior Checklist (CBCL; Achenbach et al. 2001) was used to assess behavioral and emotional problems. Both the Preschool (1.5 – 5) and School-Age (6–18) versions of the measures were used. The CBCL is a parent-report questionnaire and identifies emotional and behavioral symptoms, generating T-scores across a number of domain scales. For the present study, domains of interest included: Internalizing Problems (CBCL-I) and Externalizing Problems (CBCL-E) in order to get an overall assessment of internalizing and externalizing symptomatology.

Parent-Reported Autism Symptom Intensity

The Social Responsiveness Scale (SRS; Constantino 2002) and the updated second edition of the measure (Constantino & Gruber 2014) are parent report questionnaires of the intensity of autistic traits. Both the Preschool and School-Age versions of the measures were used.

The measures yield T-scores across a number of domains. For the purposes of this study, the measure's total score was used as a measure of parent-reported autism symptom intensity.

Cognitive Ability

A full scale IQ (FSIQ) score (reported as a standard score), using the following measures, was used to assess cognitive ability: the Wechsler Intelligence Scale for Children-Fifth Edition (Wechsler 2014), Wechsler Intelligence Scale for Children-Fourth Edition (Wechsler 2003), the Wechsler Adult Intelligence Scale-Third Edition (Wechsler 1997), Wechsler Abbreviated Scale of Intelligence, Second Edition (Wechsler 2011), Wechsler Abbreviated Scale of Intelligence (Wechsler 1999) or the Differential Ability Scales-Second Edition (Elliott et al. 2007). Younger individuals rarely complete a cognitive assessment as part of an autism diagnostic evaluation at this particular clinic given that IQ scores in individuals with ASD are known to change throughout early childhood (Solomon et al. 2018). Given that FSIQ scores were systematically missing for many younger children FSIQ data was only included in the analyses for a sub-sample of children (N = 306; Black N = 82; White N = 224; Table 1). FSIQ data was available for 3 out of 126 children in the 0–3 year-old range, 99 out of 184 children in the 4–7 year-old range, and for all children in both the 8–11 year-old and 12 and older age ranges.

Analysis

Study data were managed using REDCap (Research Electronic Data Capture) electronic data capture tools hosted at Children's National Hospital (Harris et al. 2009). REDCap is a secure, web-based application designed to support data capture for research studies, providing 1) an intuitive interface for validated data entry; 2) audit trails for tracking data manipulation and export procedures; 3) automated export procedures for seamless data downloads to common statistical packages; and 4) procedures for importing data from external sources. IBM SPSS statistics software (version 26) was used for all analyses. Independent samples *t*-tests were used to assess whether age of first diagnosis differed between racial groups in our sample. For some analyses, the sample was dichotomized into those who received earlier diagnoses of ASD (i.e., under age 6 years; N = 247) and those who received diagnoses later in childhood (i.e., age 6–20 years; N = 267; Table 1; henceforth referenced as Earlier Diagnosis and Later Diagnosis groups). We selected the age cut-off at 6 as meaningful because it represents the timepoint during which children have aged out of early intervention programs and transitioned to school-age programming. Further, research estimates that up to half of children do not receive a first diagnosis of ASD until age 6 (Sheldrick et al. 2017). A Chi-squared analysis explored whether children of each race were over-represented in different groups (Earlier Diagnosis vs. Later Diagnosis). Regressions were conducted to examine relationships between independent variables (CBCL-E, CBCL-I, SRS, FSIQ) and the dependent variable, age of first diagnosis, and to explore if and how these relationships were moderated by race (moderation). See Fig. 1

Results

Descriptive data of the full sample are presented in Table 1. Mean scores and standard deviations for each variable of interest are presented overall and for each race subgroup.

Research Question 1: Do Black and White Children differ in Terms of Receiving Earlier Versus Later First ASD Diagnoses?

In the full sample, an independent samples *t*-test indicated that White children, on average, received their first diagnosis at an older age compared to Black children, $t(512) = -2.33$, $p = 0.02$ (Table 1). The sample was then dichotomized at age 6 as described above. Chi-squared analysis indicated that distribution of race differed significantly between the Earlier Diagnosis and Later Diagnosis groups, with more than expected White children and less than expected Black children in the older group, $X^2(1, 514) = 5.33$, $p = 0.02$ (Fig. 2), indicating that Black children were less likely to receive a first-time diagnosis of ASD in later childhood.

Research Question 2: Do Parent-Reported Autism Symptom Intensity, and Behavioral, Emotional, and Cognitive Functioning Predict Age of First Diagnosis and are Predictions Moderated by Race?

T-tests were conducted to assess racial group differences in our independent variables. In the full sample White children showed greater internalizing and externalizing symptoms than Black children (Table 1). No group differences were found in the intensity of parent-reported autism symptoms. In the subsample of children with complete FSIQ data, scores were higher in the White group ($M = 99.92$, $SD = 18.79$) than the Black group ($M = 87.88$, $SD = 16.67$), $t(304) = -12.04$, $p < 0.001$.

Given that FSIQ scores were systematically missing for many younger children, FSIQ was not included as a predictor variable in the first set of analyses which assessed predictors of behavioral and emotional functioning and autism symptom intensity. In order to assess whether independent variables were appropriate to include in regression models, bivariate correlations were run to explore relationships between and among independent and dependent variables, as well as to assess for collinearity. Based on the results of bivariate correlations, collinearity was not present among any variables. CBCL-E scores were not found to be related to age at diagnosis and therefore was not included as an independent variable in the regression model (Table 2). Following these preliminary analyses, a regression was run to analyze the relationship between the independent variables, intensity of parent-reported autism symptoms (i.e., SRS Total Score) and internalizing symptoms (i.e., CBCL-I), and the dependent variable, age at first diagnosis. In step 1, race (included in order to interact with SRS and CBCL-I in step 2), SRS, and CBCL-I were included in the model to analyze their relationships to age of first diagnosis. In step 2, interaction variables (i.e., interactions of SRS with race and CBCL-I with race) were added to the model in order to conduct a moderation analysis.

A regression indicated that SRS and CBCL-I scores together significantly predicted age of diagnosis (Table 2). The relationship between SRS scores and age of first diagnosis was

moderated by race, such that higher SRS scores predicted a later age of diagnosis to a stronger degree for Black children compared to White children. The relationship between CBCL-I and age of first diagnosis was also moderated by race, such that higher CBCL-I scores predicted later ages of diagnosis in White children but earlier ages of diagnosis in Black children. See Fig. 3 for a visual representation of the moderation analysis with SRS and CBCL-I score plotted against age of diagnosis predicted by the multivariate model.

Another regression was run, only including participants with complete FSIQ data. When including FSIQ in the model, FSIQ independently predicted age of diagnosis, net of the variance explained by other variables in the model ($\beta = 0.125$, $p = 0.039$). The relationship between FSIQ and age of diagnosis appeared to be moderated by race at a trend level ($\beta = -0.548$, $p = 0.075$); such that among Black children lower IQ was related to later age of diagnosis, while among White children higher IQ was related to later age of diagnosis (Fig. 4).

Discussion

Our study found that Black children were less likely to receive a first-time diagnosis of ASD later in childhood (i.e., older than age six) compared to White children. In our sample of children who received their first diagnosis of ASD at a specialized autism center in a large children's hospital, White children were, on average, older age than Black children. Receiving a diagnosis of ASD later in childhood was related to higher levels of parent-reported autism symptom intensity; particularly for Black children compared to White children. For White children, having higher levels of emotional problems was related to receiving a diagnosis of ASD later in childhood, however this finding was opposite in Black children. Finally, our study found a trend level pattern indicating that higher cognitive abilities related to a later age of diagnosis in White children while lower cognitive abilities related to a later age of diagnosis in Black children. This suggests that when getting a diagnosis later in life, Black children show relatively more autistic traits and lower cognitive functioning, and White children show relatively more emotional problems and higher cognitive abilities. This pattern may indicate that, compared to older White children, older Black children, particularly those with higher IQs, less intense parent-reported autism symptoms, and more emotional problems, are less readily recognized as having ASD and/or have greater difficulty accessing a specialty clinic. This concept is supported by our initial finding that among older children receiving a first-time ASD diagnosis, Black youth were under-represented and White youth were over-represented.

Our findings may suggest a few important factors. First, the fact that the distribution of diagnoses by race widened after age 6 suggests that many Black children are appropriately receiving diagnoses earlier in life, contrary to older research suggesting a delay in early diagnosis of ASD for racial minority children (Mandell et al. 2002). This is potentially a byproduct of increased public health initiatives to increase early developmental monitoring and screening (Daniel et al. 2009; King et al. 2010). This finding is also in line with recent national prevalence data which found that rates of ASD are similar among Black and White four and eight year old children (Maenner et al. 2020; Shaw et al. 2020).

While the implication of improved diagnosis among young Black children should be commended, our research suggests that race-based health disparities continue to be evident among older children; fewer Black children are receiving their first diagnoses later in life compared to White children. Our findings are in line with the fact that White parents are more likely to seek out health care professionals with concerns for ASD than Black parents are (Zelege et al. 2019). This could be related to cultural factors, such as Black parents being more likely to attribute subtle symptoms (e.g., social difficulties with peers, overly formal language) to a child's temperament rather than as clinically symptomatic and indicative of a possible disorder, and therefore do not choose to seek professional services (Mandell & Novak 2005). Alternatively, findings may suggest issues of access. After aging out of early intervention services at age 3 or 5, it is often challenging for Black families, particularly those of lower SES, to access specialty ASD care (Pearson & Meadan 2018). Our findings may also reflect race-based biases on behalf of healthcare professionals leading to not providing a referral to a specialty ASD clinic (Pearson & Meadan 2018) only doing so once functional difficulties worsen. Such biases may include healthcare professionals misinterpreting minority children's behavior, misunderstanding cultural norms, or being unresponsive to concerns of Black parents unless they are more severe (Ennis-Cole et al. 2013). In turn, Black children are often misdiagnosed with other developmental or mental health disorders and are less likely than White children to receive a diagnosis of ASD at their first visit with a healthcare professional (Mandell et al. 2007).

Our findings suggest that when Black children do receive a first diagnosis of ASD later in childhood, they present with a relatively greater intensity of parent-reported autism symptoms compared to their White peers. Our findings are consistent with recent research suggesting that children with less intense parent-reported autism symptoms who receive their first diagnosis later in childhood, are more likely to be White than Black (Jo et al. 2015). Our findings, however, are inconsistent with research that suggests that intensity of autistic traits usually relates to earlier diagnosis (Jo et al. 2015; Mandell et al. 2005). It is possible that our sample, seen in a specialty ASD clinic, reflect Black children who missed a critical window early in childhood to receive a diagnosis, and in order for them to present to a specialty clinic, they must present with more intense parent-reported autism symptoms later in life. Further, this finding may reflect levels of parent concern or "certainty" of ASD. As previously mentioned, Black parents may be less likely to interpret behaviors as clinically symptomatic compared to White parents (Mandell & Novak 2005). Therefore, while White parents may seek services even when their child presents with less clear or impairing symptoms of ASD, Black parents may only seek diagnostic services later in life if they are more certain that their child has ASD, or if their child's symptoms are having a more significant impact on their daily functioning.

We also found that greater levels of internalizing symptoms related to a later age of diagnosis for White children, while lower levels of internalizing symptoms related to a later age of diagnosis for Black children. For White children, along with less intense symptoms of ASD, this may reflect that either internalizing symptoms masked ASD-specific weaknesses earlier in life, or that internalizing problems emerged secondary to growing up without an ASD diagnosis and therefore inadequate supports. Black children with ASD, however, are more likely to be misdiagnosed with another mental health disorder (Pearson & Meadan

2018). Therefore, it is likely that there are misdiagnosed autistic Black children with higher internalizing problems that did not seek an ASD diagnosis, and were therefore not reflected in our sample.

Finally, our data support a trend level finding suggesting that for White children higher cognitive functioning related to later ages of diagnosis, and for Black children lower cognitive functioning predicted later diagnoses. For White children, this may suggest that intelligent young children do not present as impaired and therefore do not receive diagnoses until later in life. However, our findings in the Black subsample align with the fact that Black children with ASD and cognitive impairments are overrepresented compared to White children with ASD, and often get their diagnoses later (Maenner et al. 2020). Taken together, our data suggests that compared to White children, Black children must show greater cognitive weaknesses and must have more intense parent-reported autism symptoms in order to appropriately receive an ASD diagnosis later in childhood.

A number of limitations must be considered in interpreting our data. First, given that this data was collected as part of standard clinical evaluations, data on SES was not available. This is an important limitation given that ASD prevalence is higher in higher SES communities (Maenner et al. 2020). SES is likely an important covariate when exploring the role of race. Second, cognitive data was only available for a subsample of children and was systematically missing for younger children. We recognize this as a limitation, reflective of the clinic's procedure and effort to maximize the number of children at-risk for ASD seen in early childhood, to support the initiation of early intervention services. Third, we do not have information about prior non-autism diagnoses, or previous points of contact for children who received diagnoses later in life. Therefore, the mechanisms which led to later diagnoses for some children cannot be inferred based on this data. In turn, this data was cross-sectional, and although we interpreted the data with age of diagnosis as the outcome, it is possible that the relationship is not truly causal. We also did not have data on cooccurring ID diagnoses. We recognize that cooccurring diagnoses of ID and ASD may interact with race in predicting timing of ASD diagnoses and future research should consider this factor. Finally, data on autism symptom severity and internalizing behaviors were based on parent report. Although measures were standardized, cultural factors may affect the ways in which Black and White parents complete the questionnaires.

Despite these limitations, our study highlighted a clear racial health disparity, wherein older Black children are not equally accessing the ASD diagnostic services that White children are. This is concerning given that without a formal diagnosis, children are likely not receiving appropriate services and supports. It is vital for future research to further explore the mechanisms through which Black children are limited in receiving such clinical services later in life. In turn, equitable initiatives to support Black children with ASD across childhood is vital.

Funding

This work was supported by the District of Columbia Intellectual and Developmental Disabilities Research Center (DC-IDDRC) Award Grant No. U54HD090257 by NICHD (PI: V. Gallo) and Clinical and Translational Science Institute at Children's National (Award Grant No. UL1TR001876).

References

- Achenbach TM, Rescorla LA, McConaughy SH, Pecora PJ, Wetherbee KM, & Ruffle TM (2001). *Child Behavior Checklist for Ages 6–18*. Burlington, VT: Achenbach System of Empirically Based Assessment
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC
- Baio J, Wiggins L, Christensen DL, Maenner MJ, Daniels J, Warren Z, & Dowling NF (2018). Prevalence of autism spectrum disorders in a total population sample-autism and developmental disabilities monitoring network, 11 sites, United States, 2014. *MMWR Surveillance Summaries*, 67(6), 1–25. 10.15585/mmwr.ss6706a1
- Constantino JN (2002). *Social Responsiveness Scale*. Western Psychological Services: Los Angeles.
- Constantino J, Abbacchi A, Saulnier C, Klaiman C, Mandell D, Zhang Y, & Geschwind D (2020). Timing of the diagnosis of autism in african american children. *Pediatrics*, 146(3), e20193629. 10.1542/peds.2019-3629 [PubMed: 32839243]
- Constantino JN, & Gruber. (2014). *Social Responsiveness scale-second edition (SRS-2)*. Journal of Psychoeducational Assessment. 10.1111/j.1571-9979.2008.00196.x
- Daniel KL, Prue C, Taylor MK, Thomas J, & Scales M (2009). 'Learn the signs. act early': a campaign to help every child reach his or her full potential. *Public Health*, 123, e11–e16. 10.1016/j.puhe.2009.06.002
- Daniels AM, & Mandell DS (2014). Explaining differences in age at autism spectrum disorder diagnosis: a critical review. *Autism*, 18(5), 583–597. 10.1177/1362361313480277 [PubMed: 23787411]
- Durkin MS, Maenner MJ, Baio J, Christensen D, Daniels J, Fitzgerald R, & Yeargin-Allsopp M (2017). Autism spectrum disorder among US children (2002–2010): socioeconomic, racial, and ethnic disparities. *American Journal of Public Health*, 107(11), 1818–1826. 10.2105/AJPH.2017.304032 [PubMed: 28933930]
- Elliott CD, Salerno JD, Dumont R, & Willis JO (2007). *Differential ability scales* (2nd ed.). Theories, Tests, and Issues. San Antonio, TX.
- Emerson ND, Morrell HER, & Neece C (2016). Predictors of age of diagnosis for children with autism spectrum disorder: the role of a consistent source of medical care, race, and condition severity. *Journal of Autism and Developmental Disorders*, 46(1), 127–138. 10.1007/s10803-015-2555-x [PubMed: 26280401]
- Ennis-Cole D, Durodoye BA, & Harris HL (2013). The impact of culture on autism diagnosis and treatment: considerations for counselors and other professionals. *The Family Journal*. 10.1177/1066480713476834
- Harris PA, Taylor R, Thielke R, Payne J, Gonzalez N, & Conde JG (2009). Research electronic data capture (REDCap)-A meta-data-driven methodology and workflow process for providing translational research informatics support. *Journal of Biomedical Informatics*. 10.1016/j.jbi.2008.08.010
- Jo H, Schieve LA, Rice CE, Yeargin-Allsopp M, Tian LH, Blumberg SJ, & Boyle CA (2015). Age at autism spectrum disorder (asd) diagnosis by race, ethnicity, and primary household language among children with special health care needs, United States, 2009–2010. *Maternal and Child Health Journal*, 19(8), 1687–1697. 10.1007/s10995-015-1683-4 [PubMed: 25701197]
- King TM, Tandon SD, Macias MM, Healy JA, Duncan PM, Swigonski NL, & Lipkin PH (2010). Implementing developmental screening and referrals: lessons learned from a national project. *Pediatrics*, 125(2), 350–360. 10.1542/peds.2009-0388 [PubMed: 20100754]
- Liptak GS, Stuart T, & Auinger P (2006). Health care utilization and expenditures for children with autism: Data from US national samples. *Journal of Autism and Developmental Disorders*, 36(7), 871–879. 10.1007/s10803-006-0119-9 [PubMed: 16855879]
- Lord C, Risi S, DiLavore PS, Shulman C, Thurm A, & Pickles A (2006). Autism from 2 to 9 years of age. *Archives of General Psychiatry*, 63(6), 694. 10.1001/archpsyc.63.6.694 [PubMed: 16754843]
- Maenner MJ, Shaw KA, Baio J, Washington A, Mary P, DiRienzo M, & Dietz PM (2020). Prevalence of autism spectrum disorder among children aged 8 years-autism and developmental disabilities

monitoring network, 11 sites, United States, 2016 centers for disease control and prevention mmwr editorial and production staff (serials). MMWR Morbidity and Mortality Weekly Report Surveillance Summaries, 69, 1–12. 10.15585/mmwr.ss6904a1

- Mandell DS, Ittenbach RF, Levy SE, & Pinto-Martin JA (2007). Disparities in diagnoses received prior to a diagnosis of autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 37(9), 1795–1802. 10.1007/s10803-006-0314-8 [PubMed: 17160456]
- Mandell DS, Listerud J, Levy SE, & Pinto-Martin JA (2002). Race differences in the age at diagnosis among medicaid-eligible children with autism. *Journal of the American Academy of Child and Adolescent Psychiatry*, 41(12), 1447–1453. 10.1097/00004583-200212000-00016 [PubMed: 12447031]
- Mandell DS, & Novak M (2005). The role of culture in families' treatment decisions for children with autism spectrum disorders. *Mental Retardation and Developmental Disabilities Research Reviews*, 11(2), 110–115. 10.1002/mrdd.20061 [PubMed: 15977313]
- Mandell DS, Novak M, & Zubritsky CD (2005). Factors associated with age of diagnosis among children with autism spectrum disorders. *Pediatrics*, 116(6), 1480–1486. 10.1542/peds.2005-0185 [PubMed: 16322174]
- Mandell DS, Wiggins LD, Carpenter LA, Daniels J, DiGuseppi C, Durkin MS, & Kirby RS (2009). Racial/ethnic disparities in the identification of children with autism spectrum disorders. *American Journal of Public Health*, 99(3), 493–498. 10.2105/AJPH.2007.131243 [PubMed: 19106426]
- Mazurek MO, Handen BL, Wodka EL, Nowinski L, Butter E, & Engelhardt CR (2014). Age at first autism spectrum disorder diagnosis: the role of birth cohort, demographic factors, and clinical features. *Journal of Developmental and Behavioral Pediatrics*, 35(9), 561–569. 10.1097/DBP.0000000000000097 [PubMed: 25211371]
- Ozonoff S, Young GS, Brian J, Charman T, Shephard E, Solish A, & Zwaigenbaum L (2018). Diagnosis of autism spectrum disorder after age 5 in children evaluated longitudinally since infancy. *Journal of the American Academy of Child and Adolescent Psychiatry*, 57(11), 849–857.e2. 10.1016/j.jaac.2018.06.022 [PubMed: 30392626]
- Pearson JN, & Meadan H (2018). African American parents' perceptions of diagnosis and services for children with autism. *Education and Training in Autism and Developmental Disabilities*, 53(1), 17–32.
- Ratto AB, Anthony BJ, Kenworthy L, Armour AC, Dudley K, & Anthony LG (2016). Are non-intellectually disabled black youth with asd less impaired on parent report than their white peers? *Journal of Autism and Developmental Disorders*, 46(3), 773–781. 10.1007/s10803-015-2614-3 [PubMed: 26439481]
- Reichow B, Hume K, Barton EE, & Boyd BA (2018). Early intensive behavioral intervention (EIBI) for young children with autism spectrum disorders (ASD). *Cochrane Database of Systematic Reviews*. 10.1002/14651858.CD009260.pub3
- Shaw KA, Maenner MJ, Baio J, Washington A, Christensen DL, Wiggins LD, & Dietz PM (2020). Early identification of autism spectrum disorder among children aged 4 years — early autism and developmental disabilities monitoring network, six sites. United States. MMWR Surveillance Summaries, 69(3), 1–11. 10.15585/mmwr.ss6903a1
- Sheldrick RC, Maye MP, & Carter AS (2017). Age at first identification of autism spectrum disorder: an analysis of two us surveys. *Journal of the American Academy of Child & Adolescent Psychiatry*, 56(4), 313–320. 10.1016/j.jaac.2017.01.012 [PubMed: 28335875]
- Solomon M, Iosif AM, Reinhardt VP, Libero LE, Nordahl CW, Ozonoff S, & Amaral DG (2018). What will my child's future hold? phenotypes of intellectual development in 2–8-year-olds with autism spectrum disorder. *Autism Research*. 10.1002/aur.1884
- Wechsler D (1997). Wechsler Adult Intelligence Scale - (3rd ed.). Harcourt Assessment.
- Wechsler D (1999). Wechsler Abbreviated Scale of Intelligence. Harcourt Assessment: San Antonio.
- Wechsler D (2003). Wechsler Intelligence Scale for Children - (4th ed.). Harcourt Assessment.
- Wechsler D (2011). Wechsler Abbreviated Scale of Intelligence. San Antonio: Second Edition.
- Wechsler D (2014). Wechsler Intelligence Scale for Children. NCS Pearson: Fifth Edition.

Zelege WA, Hughes TL, & Drozda N (2019). Disparities in diagnosis and service access for minority children with ASD in the United States. *Journal of Autism and Developmental Disorders*, 49(10), 4320–4331. 10.1007/s10803-019-04131-9 [PubMed: 31342443]

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

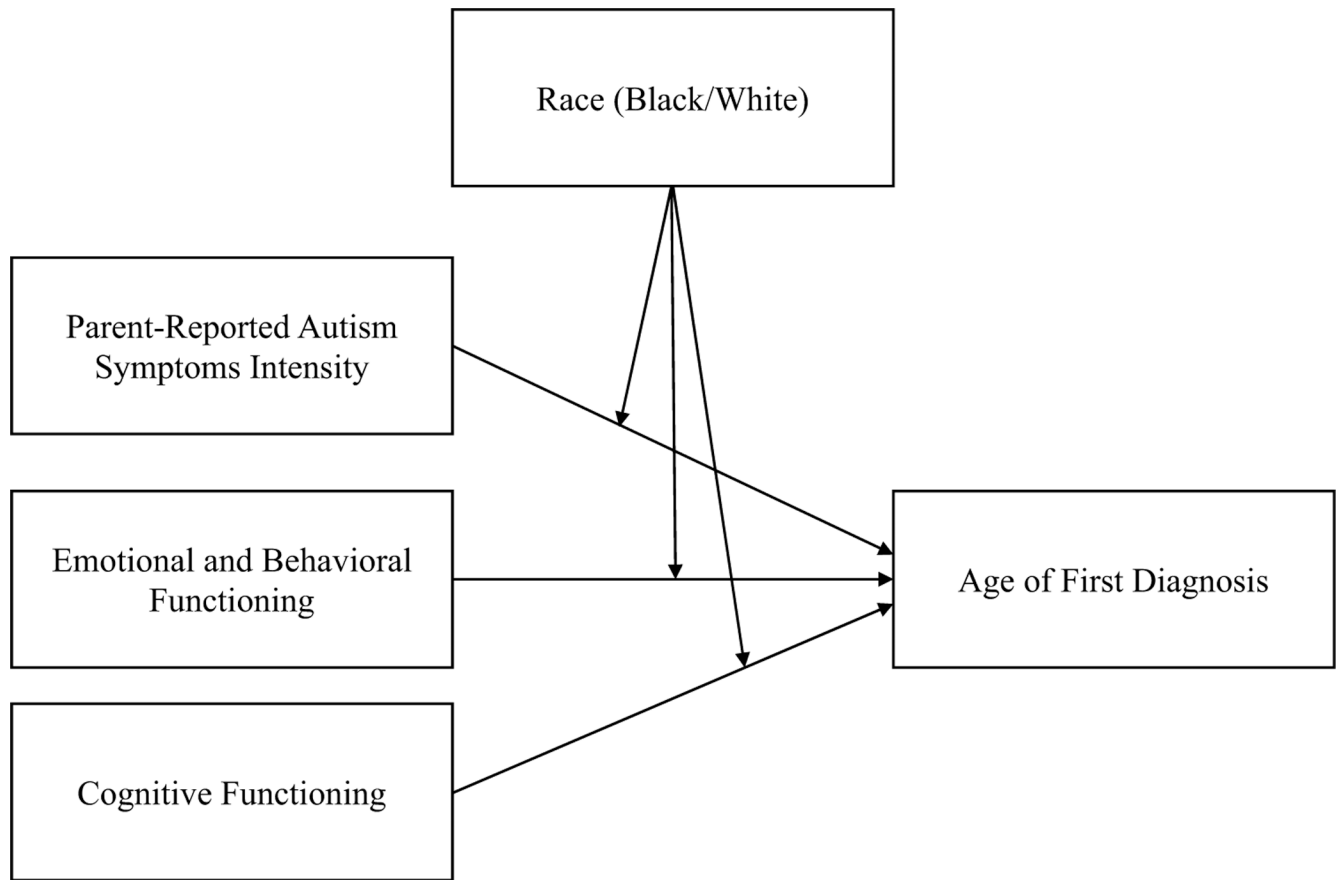


Fig. 1.
Moderation Analysis Conceptual Model

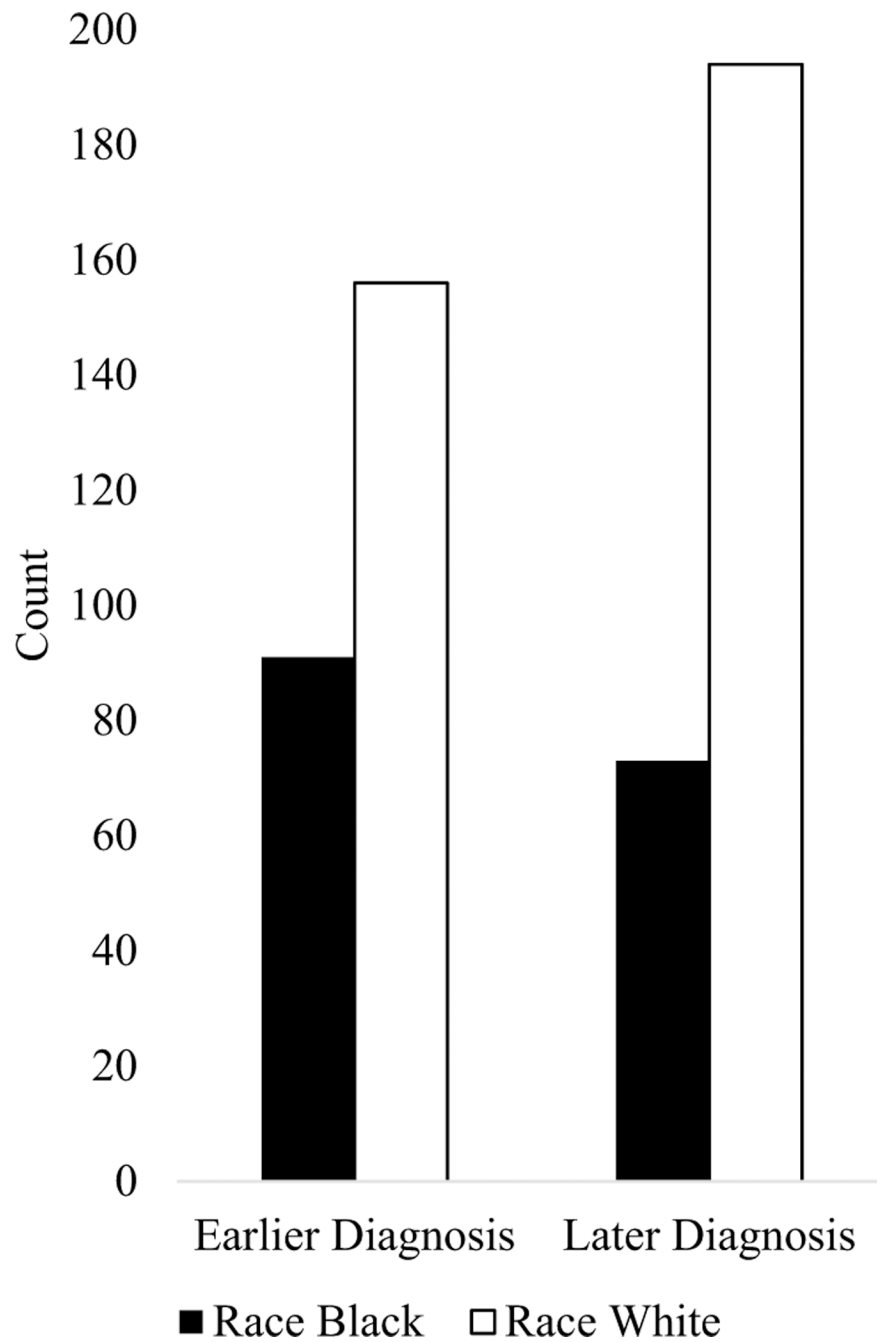


Fig. 2.
Count of Black and White Children Receiving Earlier and Later Diagnoses

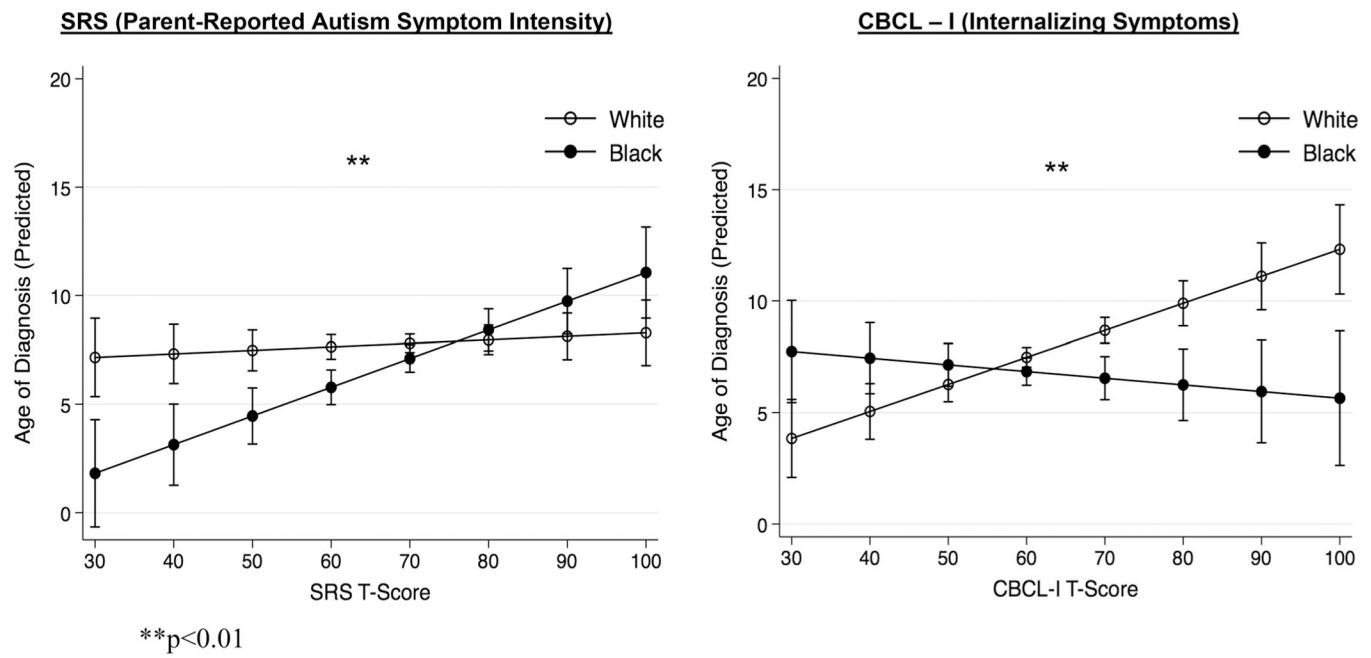


Fig. 3.
Moderation Analyses: SRS and CBCL-I Scores and Predicted Age of Diagnosis (Residuals)
with 95% Confidence Interval Margins Presented by Race

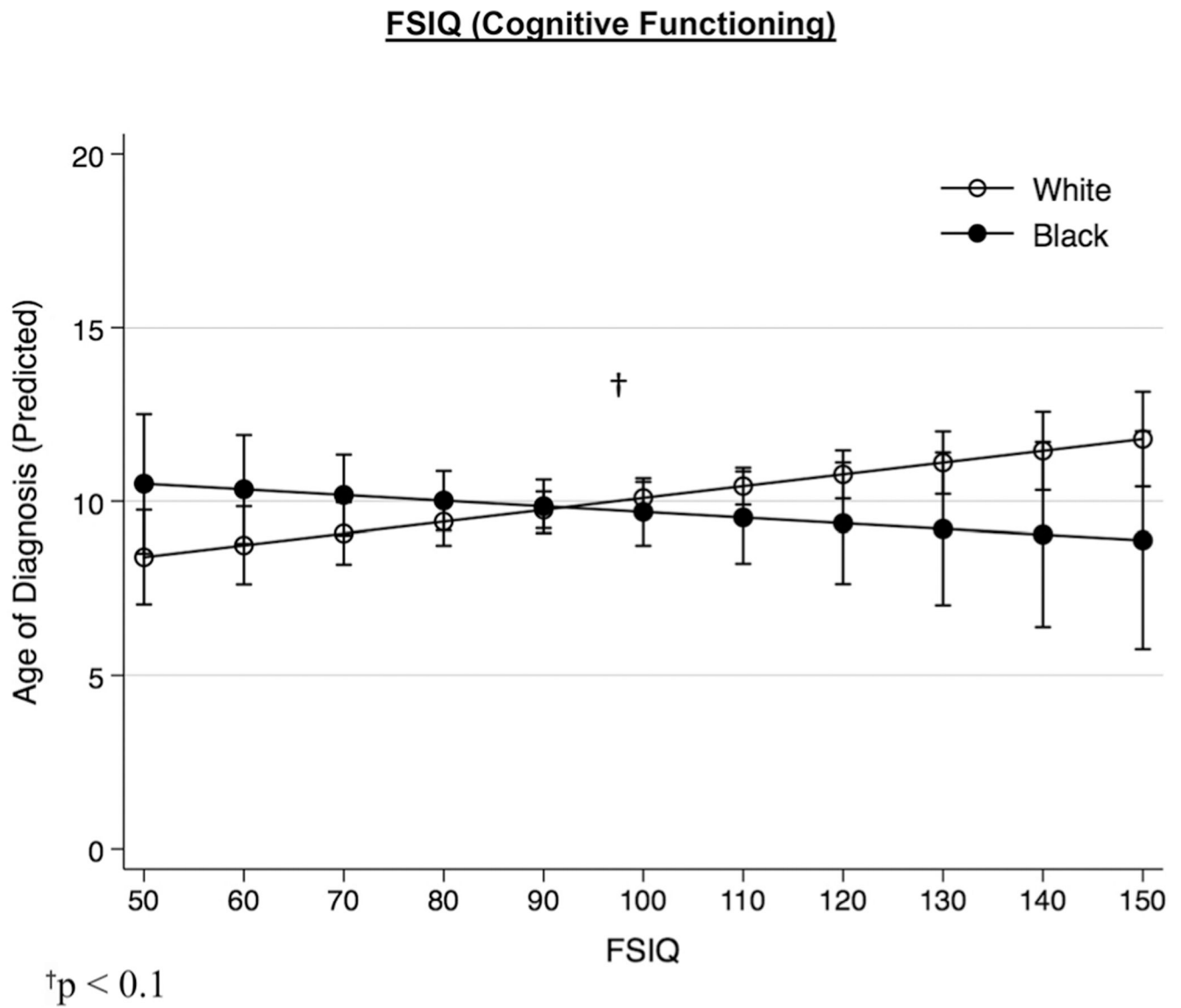


Fig. 4.
Moderation Analysis: FSIQ Scores and Predicted Age of Diagnosis (Residuals) with 95% Confidence Interval Margins

Table 1

Descriptive Data for Full Sample, Earlier Diagnosis Group, and Later Diagnosis Group

Variable	N	Age at Diagnosis M (SD)	SRS T-Score M (SD)	CBCL-I T-Score M (SD)	CBCL-E T-Score M (SD)	N	FSIQ M (SD)
Full sample							
Black	164	6.84 (3.99)	68.07 (12.44)	59.94 (10.62)	57.23 (10.63)	82	87.88 (16.67)
White	350	7.77 (4.32)	68.35 (11.36)	62.47 (9.91)	59.92 (10.85)	224	99.92 (18.79)
<i>t-test</i>		- 2.33 *	- 0.25	- 2.63 *	- 2.64 *		- 12.04 ***
Total	514	7.47 (4.24)	68.26 (11.70)	61.66 (10.20)	59.06 (10.84)	306	96.69 (18.99)
Earlier Diagnosis (< 6)							
Black	91	3.90 (1.01)	64.63 (11.66)	59.31 (10.01)	57.49 (10.79)	9	88.44 (18.33)
White	156	3.92 (1.05)	64.85 (11.52)	59.30 (9.94)	58.08 (11.15)	30	94.33 (23.68)
<i>t-test</i>		- 0.18	0.15	<0.01	- 0.41		- 0.69
Total	247	3.92 (1.03)	64.77 (11.55)	59.30 (9.95)	57.87 (11.00)	39	92.97 (22.47)
Later Diagnosis (> 6)							
Black	73	10.51 (3.20)	72.37 (12.10)	60.73 (11.35)	56.89 (10.50)	73	87.81 (16.59)
White	194	10.86 (3.36)	71.16 (10.42)	65.01 (9.15)	61.40 (10.40)	194	100.78 (17.84)
<i>t-test</i>		- 0.77	0.75	- 2.89 *	- 3.15 *		- 5.40 ***
Total	267	10.77 (3.31)	71.49 (10.90)	63.84 (9.96)	60.17 (10.60)	267	97.23 (18.41)

Note: FSIQ was systematically missing for many younger children FSIQ data was only included in the analyses for a sub-sample of children

*
p < 0.05**
p < 0.01***
p < 0.001

Table 2

Summary of Regression Analyses for Variables Predicting Age of Diagnosis

Step and Variable	B	SE B	β	ΔR^2
Step 1				.093
Race	-.73	.39	-.08 [†]	
SRS	.06	.02	.15 ^{**}	
CBCL-I	.07	.02	.17 ^{**}	
Step 2				.021
Race	.54	2.47	.06	
SRS	.01	.02	.05	
CBCL-I	.12	.03	.29 ^{***}	
SRS/Race Interaction	.12	.04	.89 ^{**}	
CBCL-I/Race Interaction	-.15	.05	-1.02 ^{**}	

[†]
p < 0.1^{*}
p < 0.05^{**}
p < 0.01^{***}
p < 0.001