Title

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Journal

Journal of the American Academy of Child and Adolescent Psychiatry, 62(9)

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Publication Date

2023-09-01

DOI

10.1016/j.jaac.2023.05.005

Peer reviewed

Published in final edited form as:

J Am Acad Child Adolesc Psychiatry. 2023 September ; 62(9): 949–952. doi:10.1016/j.jaac.2023.05.005.

Prospects for Leveling the Playing Field for Black Children with Autism

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Keywords

Autism; Intellectual Disability; Intervention; Health Disparity; Minority

Among the many race-based health disparities that have persistently plagued the United States (U.S.) population,[1] the disproportionate burden of adverse neurodevelopmental outcome to Black children affected by autism spectrum disorder (ASD) is particularly devastating given its major lifelong consequences. Recently, in three successive reports from the Autism and Developmental Disabilities Monitoring (ADDM) program of the

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Disclosures Dr. John Constantino receives royalties from Western Psychological Services for the commercial distribution of the Social Responsiveness Scale (SRS-2). Dr. Ami Klin is inventor of technologies deployed by EarliTec Diagnostics, Inc., of which he is an equity holder. EarliTec is a company that develops medical devices for early diagnosis of ASD and gives a portion of its revenue to support treatment of children with ASD. There are no other author disclosures to report.

U.S. Centers for Disease Control (CDC) (birth cohort years 2014, 2016, and 2018), we and our collaborators reported that although the prevalence of community-diagnosed ASD had equalized for Black and Non-Hispanic White (NHW) children in the U.S., there has persisted a pronounced racial disparity in the proportion of ASD-affected children with comorbid intellectual disability (ID), on the order of 50% for Black children with ASD versus 20% for White children with ASD.[2] Here we provide data to support the following: much earlier diagnosis is possible; early diagnosis alone is not likely to close the ID comorbidity disparity; and judicious efforts over care-as-usual are necessary to ensure that Black children have access to timely implementation of developmental therapy, for which we observed promising associations with improved cognitive and adaptive outcomes in our sample.

Subsequently, our group reported that among Black children with ASD (i) there was, on average, a 42-month delay in diagnosis following parents' first expression of concern about their child's development to a health professional, and (ii) ID comorbidity was not predicted by familial factors associated with cognitive variation in the general population.[3] Timely diagnosis was impacted by excessive wait times to secure professional appointments, misdiagnosis, cost (despite most children being insured), scheduling conflicts, and transportation issues.

Collectively, these findings have documented structural factors influencing serious, race-based outcomes disparities in ASD,[4] highlight significant opportunity to expedite diagnosis, and secondarily to implement appropriate and timely developmental intervention to close the gap in ID comorbidity across race. This is a report of the results of an attempt to capitalize upon this opportunity in St. Louis (STL) and Atlanta (ATL).

Method

Black toddlers (N=209) ages 18-36 months diagnosed or referred with suspicion of ASD were enrolled in US NIH MH100027, *Autism Genetics Network, Phase II: Increasing the Representation of Human Diversity* across two data collection sites: Washington University in STL (n=52) and Emory University in ATL (n=157). All children received a diagnostic evaluation and were followed longitudinally for a period of at least 18 months to track service acquisition and to obtain standardized ratings of ASD symptom burden, cognitive outcome, and adaptive function during their fourth year of life.

For children at the STL site who were unable to access autism-specific services in the community, families were offered one of two philanthropically-subsidized clinical intervention tracts: 1) a center-based program delivering 10 hours per week of applied-behavior-analysis (ABA)-based developmental therapy (duration between 6 and 18 months determined by the timing of each child's aging out of Part C and reaching eligibility for special education through public schools); or 2) a home-based (in-person/telehealth) 12-week parent-training curriculum modeled after naturalistic developmental behavioral interventions (NDBI).[5] Families were able to select between the two at the outset of the course of intervention whenever slots in both tracts were available.

A principal goal of this analysis was to determine whether enhancing community awareness, establishing rapid referral pathways for primary care providers, and expanding clinical capacity for diagnostic assessment (see Supplement 1 and Figure S1, available online, for details)—resulted in earlier identification of a cohort representative of all Black children with ASD (i.e. not disproportionately severe in symptomatology). In addition, for the STL cohort, we examined outcomes of children as a function of access to autism-specific developmental therapy and in comparison to the ATL toddler sample and a sample of older-diagnosed Black children matched for severity in early childhood.

Results

Timing of Diagnosis

The average age of diagnosis was 29.6 (+/−5.4) months for the STL cohort and 25.4 (+/− 4.2) for the ATL cohort. For 82.4% and 88.5% of the children (STL and ATL respectively) the research program established the child's initial ASD diagnosis, indicating that the research effort itself was responsible for the timing of diagnosis. To determine whether this early-diagnosed cohort of children had higher-than-average syndrome severity (i.e. a potential reason for earlier detection), we compared Vineland-3 mean Adaptive Behavior Composite (ABC) scores of the children in this sample to a large previously-acquired sample[3] of later-diagnosed Black children with ASD and found them to be highly comparable (STL 67.1 +/− 12.3 vs. 65.5 +/− 12.0).

Intervention Access and Impact.—Despite early diagnoses, the mean number of cumulative hours per week the children received, i.e. after an ASD diagnosis—and therefore service eligibility—was established, was 0.45 hours per week (+/− 1.13) in STL and 1.0 hours per week (+/−1.8) in ATL. When the STL families were subsequently provided opportunity to enroll in the supplemental clinical intervention tracts, 35 enrolled. This increased the mean number of cumulative hours per week of intervention to 4.67 (+/− 6.64) hours per week.

Data comparing i) the entire cohort of early-diagnosed Black ASD toddlers, ii) early-diagnosed Black ASD toddlers who completed a full 9 months of center-based intervention at an average of 6 hours per week (n=14 to date), iii) early-diagnosed Black ASD toddlers who received no supplemental developmental therapy, iv) a contrast sample of later-diagnosed Black children with ASD from the same parent study, and v) naturalistic follow-up data from early-diagnosed toddlers enrolled at Emory are presented in table 1. There were a number of notable findings. First, early diagnosis in and of itself did not translate to improved adaptive functioning among the children, i.e. when comparing the Vineland ABC scores for groups i and iv, which were matched on the basis of core ASD symptom severity. Second, when considering the impact of intervention, the Mullen Scale of Early Learning (MSEL) composite scores for the intervention group (ii) improved by an average of 7 points, whereas the score for the non-intervention group (iii) and the larger cohort (iv) did not significantly change over time. Third, the Vineland ABC scores for the non-intervention group (iii) deteriorated significantly over time, whereas those for the intervention group (ii) remained stable.

Discussion

These observations should inform a national approach to an urgent public health issue raised by the U.S. CDC's documentation of a gross disparity (O.R. > 2.0) in ID comorbidity among Black youth with ASD:

First, we have shown that much earlier diagnosis (in comparison to the national average) is possible for Black children with ASD, and in this program, a community-wide effort to identify and diagnose children with ASD expedited the average age of diagnosis by three years without any evidence of systematic bias in identifying more severely affected children.

Second, we observed that earlier diagnosis alone did not result in more favorable cognitive outcomes for Black children with ASD, when compared with later-diagnosed Black children from our own research program or when compared with previously published cohorts of predominately NHW early diagnosed children. Standardized scores for cognitive and adaptive functioning of our earlier diagnosed children were considerably lower (on the order of one standard deviation) than those of NHW children in previously published cohorts, despite relative equivalency in timing of motor milestones.[6] In our cohort, most Black early-diagnosed children were not receiving more than a modicum of developmental therapy, whether diagnosed before or after the onset of the COVID-19 pandemic.

Third, when offered the opportunity for more intensive intervention, a majority pursued the opportunity. Among those who received intervention of reasonable intensity and duration,[7] we observed an average 7-point gain in MSEL composite scores (a half standard deviation gain; standard score mean=100 and SD=15), while a small non-intervention contrast group made no such gain and deteriorated with respect to adaptive function. This naturalistic erosion has been observed in an independent longitudinal cohort.[8] A number of factors influenced enrollment in the supplemental intervention, including scheduling conflicts and the burden of parents' time required for the respective interventions. The intervention group was more impaired at baseline than the non-intervention group, which may have resulted from parents of more severely-affected children being more motivated to pursue intervention.

We conclude that earlier diagnosis is possible for black youth with ASD and would create opportunity for a much higher proportion of the children to receive timely autism-specific services under Part C and subsequently via early childhood special education. Early diagnosis without such intervention is unlikely to influence severe national race-based disparities in cognitive outcomes of children with ASD, especially given known racial and ethnic disparities in geographic access to ASD resources across the US.[9] It is therefore a matter of utmost priority to follow these preliminary findings with a definitive attempt to determine whether timely implementation of autism-specific developmental and educational services can close the prevalence gap for ID between Black and NHW children affected by ASD.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements

This work was made possible by NIH R01 MH100027 (Daniel H. Geschwind, P.I.), NIH P50 HD103525 (John N. Constantino, P.I.), and a gift from the Steward Family Foundation to Washington University. The authors gratefully acknowledge the commitment of the participating families to promote and ensure equity in the science and clinical treatment of autism spectrum disorders, and the support of clinical diagnostic and intervention planning efforts in St. Louis made possible through the Waitlist Reduction and Training Services program of the Missouri Department of Mental Health.

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J Am Acad Child Adolesc Psychiatry. Author manuscript; available in PMC 2024 March 19.

Table 1.

Selected Characteristics of the St. Louis Study Sample at Baseline (I), at Follow-up in the Fourth Year of Life (II and III, as a Function of Supplemental Intervention), and in Comparison to Two Contrast Samples (IV and V)

	I. ASD Toddlers, WUSTL[a]						II. WUSTL Toddlers Supplemental Intervention[b]						III. WUSTL Toddlers Care as Usual[c]						IV. Later-diagnosed Contrast Sample[d]			V. Toddlers Contrast Sample[e]					
	Baseline			Follow up			Baseline			Follow up			Baseline			Follow up			Follow up			Baseline			Follow up		
	N	Mean	SD	N	Mean	SD	N	Mean	SD	N	Mean	SD	N	Mean	SD	N	Mean	SD	N	Mean	SD	N	Mean	SD	N	Mean	SD
Age at Diagnosis (months)	52	29.6	5.4																42	87.4	51.8	157	25.4	4.2			
VABS-3 Age	50	28.9	5.2	20	48.1	6.2	14	29.4	5.5	14	48.6	5.7	6	28.0	6.0	6	46.7	7.5	41	109.8	50.9	157	25.9	5.1	27	49.6	18.5
VABS-3 Composite	50	65.6	12.0	20	65.0	13.2	14	65.4	14.8	14	65.6	12.9	6	71.2	7.8	6	63.3	15.1	41	71.3	9.1	135	62.4	12.4	27	65.6	11.6
VABS-3 Communication	50	57.0	18.7	20	60.3	21.8	14	54.8	21.9	14	59.3	21.8	6	71.2	7.0	6	62.5	23.5	41	72.3	12.3	135	61.4	13.5	27	62.0	17.0
VABS-3 Daily Living	50	73.5	15.8	20	71.9	13.2	14	74.1	17.6	14	72.6	11.3	6	77.3	11.5	6	70.3	18.0				135	53.9	21.8	27	67.1	16.9
VABS-3 Socialization	50	66.7	12.0	20	63.5	16.3	14	67.9	13.3	14	65.8	13.4	6	66.5	15.5	6	58.0	22.2				135	67.8	15.7	27	67.6	12.0
VABS-3 Motor	50	82.3	12.3	20	77.5	17.9	14	81.9	16.4	14	82.0	12.3	6	82.0	9.4	6	67.0	25.3				132	68.6	14.0	27	72.4	10.5
MSEL Age	41	30.8	6.3	20	49.9	7.6	13	29.5	4	12	49.6	7.5	6	26.5	6.3	6	49.5	9.2				131	27.9	5.1	28	49.6	5.4
MSEL Composite	40	57.2	14.3	19	61.1	15.2	13	53.6	8.4	12	60.7	14.0	6	61.0	9.7	6	64.0	18.9				131	57.9	11.4	28	57.8	16.4
Raven				5	84.0	12.1													16	86.3	23.5						
IQ Proxy[f]																			41	77.4	20.3						
ADIR Age				16	49.0	5.2				12	48.9	4.5				4	49.3	7.6							16	46.9	3.4
ADIR Social				16	16.3	7.1				12	17.8	6.1				4	11.8	9.1							16	13.8	5.2
ADIR Communication				16	11.8	3.1				12	11.3	2.2				4	13.3	5.2							15	17.1	5.6
ADIR Repetitive Behavior				16	4.2	1.0				12	4.5	2.3				4	3.0	2.2							15	11.3	3.2

Note: Emory participants were not assessed using the Raven's Progressive Matrices at follow-up. ADIR=Autism Diagnostic Interview; ASD=autism spectrum disorder; ATL= Atlanta; DAS=Differential Abilities Scale; IQ=Intelligence Quotient; MSEL=Mullen Scales of Early Learning; PPVT=Peabody Picture Vocabulary Test-4th Edition; STL=St. Louis; VABS=Vineland Adaptive Behavior Scale; WUSTL=Washington University in St. Louis.

[a] I. All ASD Toddlers for whom the respective data elements were available: STL site

[b] II. Toddlers who received supplemental intervention and whose cognitive status was evaluated at >18 months follow-up

[c] III Toddlers who completed follow-up cognitive assessments but did not receive ASD-specific developmental intervention services: STL site

[d] IV. Contrast sample of later-diagnosed Black children with ASD from the parent study (MH 100027) 3:1 matched to the toddlers in group II by core early childhood ASD symptoms ascertained by ADIR

[e] V. Contrast sample of toddlers enrolled in MH 100027 at Emory University in ATL

[f] IQ Proxy was derived from a cognitive score on the DAS, Raven, MSEL, or PPVT