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Pica, Autism, and Other Disabilities

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Abstract

BACKGROUND AND OBJECTIVES: Pica, the repeated ingestion of nonfood items, can be life-threatening. Although case reports describe pica in children with autism spectrum disorder (ASD) or intellectual disability (ID), there has been little systematic study of pica prevalence. We assessed pica in children 30 to 68 months of age (median = 55.4 months) with and without ASD.

METHODS: Our sample from the Study to Explore Early Development, a multisite case-control study, included children with ASD (n = 1426), children with other developmental disabilities (DDs) (n = 1735), and general population-based controls (POPs) (n = 1578). We subdivided the ASD group according to whether children had ID and the DD group according to whether they had ID and/or some ASD characteristics. Standardized developmental assessments and/or questionnaires were used to define final study groups, subgroups, and pica. We examined pica prevalence in each group and compared ASD and DD groups and subgroups to the POP group using prevalence ratios adjusted for sociodemographic factors.

RESULTS: Compared with the prevalence of pica among POPs (3.5%), pica was higher in children with ASD (23.2%) and DD (8.4%), and in the following subgroups: ASD with ID (28.1%), ASD without ID (14.0%), DD with ID (9.7%), DD with ASD characteristics (12.0%),

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and DD with both ID and ASD characteristics (26.3%); however, pica prevalence was not elevated in children with DD with neither ID nor ASD characteristics (3.2%). Between-group differences remained after adjustment (adjusted prevalence ratio range 1.9–8.0, all $P < .05$).

CONCLUSIONS: Pica may be common in young children with ASD, ASD characteristics, and ID. These findings inform the specialized health care needs of these children.

Pica, the repeated ingestion of nonfood items lacking nutritional value,[1,2] can result in gastrointestinal parasites, lead toxicity, nutritional deficiencies, choking, poisoning, sepsis, and intestinal obstruction or perforation.[3–6] These complications are associated with substantial morbidity and have led to fatalities in some patients.[2,7,8]

Pica is considered a self-injurious behavior, defined as self-inflicted, harmful behavior that occurs without apparent intent of willful self-harm.[5,7,9–11] Individuals with autism spectrum disorder (ASD) and/or intellectual disability (ID) have higher rates of self-injurious behavior (all types) than the general population,[12,13] and pica specifically has been implicated as a problem for these populations.

However, studies of pica in individuals with ASD and other developmental disabilities (DDs) are limited. Available information is primarily from published case series and reports.[2,3,14–21] In few studies has pica prevalence in individuals with ASD been systematically assessed. In their prospective population-based cohort study, Emond et al[22] reported that children who were eventually diagnosed with ASD were more likely to have increased pica behavior at 38 and 54 months (12.3% and 12.5%, respectively) than controls (2.3% and 0.7%). Neumeyer et al[23] assessed children with ASD who were treated at 15 Autism Treatment Network sites; they reported pica prevalence was 3.0% in children <6 years old and 2.4% in children >6 years old. In a literature review conducted by Matson et al,[1] pica prevalence estimates in children or adults with ASD and/or ID ranged from 4% to 26%; the highest estimates were found in populations that were institutionalized because of their disabilities. Because most studies in this review were limited to severe cases of pica resulting in intervention. the total prevalence of pica is likely higher than reported in these studies.

Previous studies were limited by small sample sizes, nonstandardized classification of ASD or DD, nonrepresentative (clinical) samples, and the lack of a general population comparison group. There is a need for more comprehensive epidemiological assessments of pica prevalence in children with ASD and other DDs, such that health care providers and parents are aware of the potential risks and can implement appropriate prevention measures and, if needed, initiate behavioral treatments early.

We evaluated pica in preschool-aged children with and without ASD using data from the Study to Explore Early Development (SEED), a large multisite case-control study. SEED included a diverse sample of children with ASD and other DDs, a general population-based control (POP) group, and a collection of detailed information about children's development to systematically classify case status and case subtypes.

METHODS

Study Design

The SEED methodology has been previously described.[24] Briefly, SEED data were collected in 2 phases (2007–2012 and 2013–2016) from 6 US study sites (located in CA, CO, GA, MD, NC, and PA). At each site, children with ASD and other DDs were recruited from multiple special education and clinical sources that provide services to children with disabilities. Children recruited from each source were those with select special education or International Classification of Disease codes indicative of ASD or other DDs often seen as precursors or cooccurring diagnoses in children eventually diagnosed with ASD. POP group children were recruited on the basis of randomly selected birth records; they were sampled from the same birth years and study sites but were not otherwise matched to the case groups. This study was approved by Institutional Review Boards at the Centers for Disease Control and Prevention and each site.

Participants and Study Group Classification

Children eligible for SEED enrollment were those who lived in the respective site's study area both at birth and at enrollment and lived with a caregiver since at least 6 months of age who could provide legal consent and communicate in English (all sites) or Spanish (2 sites). The child was required to live with the biological mother in the second phase of SEED data collection but not the first; nonetheless, the respondent for 98% of children in the first phase was the biological mother. Therefore, in this report, we refer to the respondent as the mother. Children were 30 to 68 months of age (median = 55.4 months) at the time of their SEED developmental assessment.

This analysis included children from all 3 study groups (ASD, DD, and POP) who were not missing data on pica. Although children were initially identified as eligible for 1 of the 3 study groups, final study classification was determined by standardized research developmental assessment results.[25] After enrollment, mothers of all children were administered the Social Communication Questionnaire (SCQ)[26] to screen for ASD symptoms in their child. Children with scores ≥11 were considered potential ASD cases regardless of their initial classification. Additionally, children who had a previous ASD diagnosis or special education classification were considered potential ASD cases regardless of their SCQ scores. All children were administered an in-person general developmental assessment, the Mullen Scales of Early Learning.[27] Children in the potential ASD group were additionally administered the Autism Diagnostic Observation Schedule,[28] and their caregivers were administered the Autism Diagnostic Interview-Revised.[29] Children who met the study criteria for ASD on the basis of their Autism Diagnostic Observation Schedule and Autism Diagnostic Interview-Revised assessments were classified as ASD cases[25]; children who had been classified as potential ASD cases on the basis of symptoms or past diagnoses but did not meet the study ASD case criteria were classified as DD with ASD characteristics. Children selected for the DD group who had an SCQ score <11 and no previous ASD educational classification or diagnosis were classified as DD without ASD characteristics. A small number of children recruited to the POP group screened positive on the SCQ and met study criteria for ASD; they received a final ASD study classification.

We further divided children with ASD, DD with ASD characteristics, and DD without ASD characteristics according to whether they had cooccurring ID. ID was defined as a Mullen Scales of Early Learning developmental score (which is considered comparable to IQ) <70.[27] Thus, we examined 6 subgroups of children: (1) ASD with ID, (2) ASD without ID, (3) DD with ID and ASD characteristics, (4) DD with ID but without ASD characteristics, (S) DD with ASD characteristics but without ID, and (6) DD with neither ID nor ASD characteristics.

Pica Data

Mothers completed structured telephone interviews and self-administered forms. Data collected included sociodemographic characteristics and child health conditions and behaviors. The Child Behavior Checklist,[30] a standardized self-administered form, asked respondents to indicate whether their child had exhibited problem behaviors within the previous 2 months. Response options for each item were “not true,” “somewhat or sometimes true,” and “very true or often true.” Pica was ascertained via the item, “child eats or drinks things that are not food-not including sweets.” If this item was answered affirmatively, the respondent was asked to describe the consumed substance. We considered a child to have pica if the mother responded, “somewhat or sometimes true” or “very true or often true.” We reviewed free text responses on item(s) the child had eaten to assess the accuracy of responses to the pica question. Although free text data were collected in both phases of SEED, only data from the second phase of SEED had been processed and made available for this assessment; nonetheless, we have no reason to believe responses differed between the 2 study phases. Our review indicated the Child Behavior Checklist pica question was accurately capturing pica; >95% of respondents specified their child was consuming 1 or more nonfood items, only 2% incorrectly specified a food item, and a few responses were too vague to classify. Examples of nonfood items reported are presented in Supplemental Table 3.

Data Analysis

We compared distributions of sociodemographic factors according to whether the child had pica and by main study group (ASD versus POP and DD versus POP) using χ^2 tests. We assessed the prevalence of children who engaged in pica by main study group and for each of the aforementioned subgroups. We calculated unadjusted prevalence ratios (PRs) and adjusted prevalence ratios (aPRs) and 95% confidence intervals, in which the prevalence of pica for each ASD and DD case group and subgroup was compared with the prevalence for the POP group. aPRs were calculated by using modified Poisson regression[31]; in addition to ASD or DD cases status, models included child sex, child and maternal age at enrollment, maternal race or ethnicity, maternal education, and household income.

We conducted supplemental analyses to examine (1) whether associations observed in our main analysis varied within various demographic strata (same factors as included in adjustment models) and (2) whether associations were similar using a more restrictive pica definition (by which only “very true or often true” counted as pica). For stratified analyses, we calculated stratum-specific PRs and used the Mantel-Haenszel method[32] to estimate pooled aPRs.

RESULTS

Overall, 4739 children were included in this analysis: 1426 with ASD, 1735 with other DDs, and 1578 POPs (Fig 1)

In all 3 study groups, most mothers were >35 years of age, were non-Hispanic white (NHW), had a bachelor's degree or higher, and had a household income >$50 000 (Table 1). Nonetheless, we observed study group differences; mothers of children with ASD and DD were significantly less likely to be NHW, have a bachelor's degree, or have a high household income than mothers of POP group children. There were markedly more males in the ASD and DD groups than the POP group. Mothers of children with pica were significantly younger and less likely to be NHW, have a bachelor's degree, or have a high household income than mothers of children without pica. Children with pica were more likely to be male. More than 70% of children in all study and pica groups were 48 to 68 months of age with slight differences observed between study groups.

Pica was reported in 23.2%, 8.4%, and 3.5% of children in the ASD, DD, and POP groups, respectively (Fig 2, Table 2). Within the ASD group, pica was reported in 28.1% of children with ID and 14.0% of children without ID. Within the DD group, pica was reported in 26.3% of children with both ID and some ASD characteristics, 12.0% of children with ASD characteristics but without ID, 9.7% of children with ID but without ASD characteristics, and 3.2% of children with neither ID nor ASD characteristics.

Unadjusted PRs indicated associations between pica and all ASD groups and subgroups and between pica and DD overall and 3 of the 4 DD subgroups. There was no association between pica and DD with neither ID nor ASD characteristics (Table 2). Despite some variability due to small sample sizes, the findings from stratified analyses were generally comparable to the unadjusted analyses (Supplemental Tables 4 and 5).

Multivariable aPRs were also similar to the unadjusted PRs (Table 2). Pica remained strongly associated with ASD overall, ASD with ID, ASD without ID, and DD with both ID and some ASD characteristics (aPRs 4.4–8.0]. Pica was moderately associated with DD overall, DD with ASD characteristics without ID, and DD with ID but not ASD characteristics (aPRs 1.9–2.5). Pica was not associated with DD with neither ID nor ASD characteristics.

The differences between ASD and DD case groups and subgroups and the POP group were even more pronounced when we used a more restrictive pica classification, (ie limited to a maternal response of "very true or often true" to the pica question) (Supplemental Table 6).

DISCUSSION

Our findings suggest that pica is more common in preschool-aged children with ASD, whether or not ID was present, and with other types of DD in which the child had some ASD characteristics, ID, or both. In comparison with the POP group, pica was strongly associated with ASD and with other DDs characterized by both ASD symptoms and ID and moderately associated with DD with ASD characteristics only or ID only. Notably, pica was

not elevated in children with DDs that did not include ASD characteristics or ID. Thus, our findings suggest that not all children with DDs are at increased risk for pica. Using a more conservative definition of pica did not change the relative findings.

Our pica prevalence estimate in children with ASD is higher than the prevalence reported by Emond et al[22] in their cohort study of children with ASD (12.3% and 12.5%), and markedly higher than the prevalence reported by Neumeyer et al[23] in their study of children in an autism treatment network (3.0% and 2.4%). Likewise, although our estimates fall within the range of estimates from studies presented by Matson et al[1] in their review of pica in children or adults with ASD and/or ID (4% to 26%) it is notably higher than several estimates from that review. Our findings expand on these reports in several ways. Whereas many previous studies were limited by nonstandardized classification of ASD or ID, we collected objective developmental data as part of the SEED protocol to systematically classify ASD and ID. Most past studies examined a limited subset of pica, such as pica that came to medical attention and/or required treatment; in contrast, with SEED we examined pica behavior ascertained from a standardized developmental questionnaire[30] regardless of whether the child had developed adverse consequences from their pica. Another strength of this study in comparison with others is the inclusion of a general population comparison group, which was actively recruited from the same study areas as the ASD and DD case groups. Finally, SEED'S large sample size and detailed developmental data collection allowed us to obtain stable estimates of pica among children in various disability subgroups and thus hone in more clearly than past studies on types of disability that were and were not associated with pica.

Nonetheless, we were not able to explore associations between pica and specific DDs. The SEED DD group, identified from diagnostic and special education codes, is heterogeneous, including children with many different (and sometimes multiple] disabilities. We were unable to assess specific non-ASD DD diagnoses separately because of sample size limitations and complexities in categorizing the various diagnostic and education disability codes into meaningful subgroups. However, our subdivisions of the DD group according to the presence of ASD characteristics objectively measured on a standardized instrument and on whether the child's developmental test scores were in the ID range allowed us to evaluate important subgroups of children with disability defined by rigorous standardized assessments.

Given the young age of the participants in this study sample, it is possible that pica prevalence in this study will not reflect pica prevalence at older ages. Nonetheless, in stratified analyses, we assessed whether the associations varied within the narrow range of ages included in our study and found that all associations observed in the main analysis were observed among the oldest stratum of children, those 48 to 68 months of age. Although we cannot assess whether affected children will continue to manifest pica as they age, our data nonetheless indicate it is a serious health concern in preschool-aged children. Notably, all children in this study were much older than 18 months, the age at which ingestion of foreign objects is deemed inappropriate.[33]

Although we were unable to examine underlying reasons for the high pica prevalence we observed in children with ASD, ASD characteristics, and ID, Mayes and Zickgraf[34] found that pica in children with autism was one component of a larger pattern of atypical eating behaviors, such as limited food preferences and hypersensitivity to food textures. Furthermore, sensory processing difficulties are commonly reported in children with ASD and may result in both atypical eating and pica behavior.[35] Additionally, some children with ASD and/or ID may not understand the difference between edible and inedible objects.[33,35] Treatments for pica that have empirical support include applied behavior analysis[1] and functional analysis, which helps identify triggers for pica and determine if pica is secondary to sensory-seeking behavior, automatic reinforcement, or social factors such as seeking attention from caregivers.[7,33] Parent prevention measures include closely monitoring children, keeping items out of reach, using childproof locks, finding activities that occupy children's attention, and informing other caregivers of concerns.[33]

Despite our study's strengths, we also note several limitations. SEED developmental data are cross-sectional, limiting causal inference. Although we defined pica on the basis of a standardized developmental assessment questionnaire,[30] pica was a single behavioral item on this instrument; while our internal assessment of available text responses about nonfood items was reassuring about the validity of the pica responses, we lacked data on clinical diagnoses and sequelae of pica and the typical quantity of nonfood items ingested. We also lacked data on some established biological risk factors for pica such as nutritional deficiencies and lead toxicity. It is possible that ascertainment of pica varied for cases and controls because caregivers might more closely monitor children with ASD and ID than children without these conditions. However, the impact of differential monitoring on pica prevalence estimates could have been in either direction; if children with ASD or ID were more closely monitored, caretakers had not only increased opportunity to observe pica, but also increased opportunity to prevent pica. Moreover, the difference between the level of observation for case and control children was likely not large, given that all children were young and thus likely to have been monitored fairly closely. Because SEED sought to enroll a diverse population of children recruited from a wide range of education and clinical sources, numerous families targeted for potential recruitment could not be located or contacted; however, a previous study in one SEED site indicated that a large proportion of these families were likely no longer eligible for SEED because they moved out of the study area; additionally, that study documented that although maternal age and education were associated with differential participation between ASD cases and POP controls, several other demographic and health factors were not associated with participation, and findings from most SEED analyses were likely robust[36] We adjusted for both maternal age and education in this analysis. This study takes place in 6 different population-based US sites; however, results may not be generalizable throughout the United States.

CONCLUSIONS

We found that preschool-aged children with ASD, ASD characteristics, and ID more commonly engage in pica and therefore could benefit from close monitoring, strategies to restrict access to dangerous items and possibly behavioral therapy.[1,5,7,37] These findings can help promote awareness about pica among pediatricians and the disability community.

Given the current paucity of research, more studies of the prevalence, correlates, and clinical outcomes of pica in children with ASD and other DDs are needed.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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ABBREVIATIONS

aPR	adjusted prevalence ratio
ASD	autism spectrum disorder
DD	developmental disability
ID	intellectual disability
NHW	non-Hispanic white
POP	general population-based control
PR	prevalence ratio
SCQ	Social Communication Questionnaire
SEED	Study to Explore Early Development

REFERENCES

1. Matson JL, Hattier MA, Belva B, Matson ML. Pica in persons with developmental disabilities: approaches to treatment. Res Dev Disabil 2013;34(9):2564–2571 [PubMed: 23747942]
2. Ashworth M, Hirdes JP, Martin L. The social and recreational characteristics of adults with intellectual disability and pica living in institutions. Res Dev Disabil 2009;30(3):512–520 [PubMed: 18789647]
3. Rashid F, Davies L, Iftikhar SY. Magnetised intragastric foreign body collection and autism: an advice for carers and literature review. Autism 2010;14(2):139–145 [PubMed: 20395283]
4. Rose EA, Porcerelli JH, Neale AV. Pica: common but commonly missed. J Am Board Fam Pract 2000;13(5):353–358 [PubMed: 11001006]

5. Williams DE, McAdam D. Assessment, behavioral treatment, and prevention of pica: clinical guidelines and recommendations for practitioners. Res Dev Disabil 2012;33(6):2050–2057 [PubMed: 22750361]
6. Bell KE, Stein DM. Behavioral treatments for pica: a review of empirical studies. Int J Eat Disord 1992;11 (41):377–389
7. Kodak T, Piazza CC. Assessment and behavioral treatment of feeding and sleeping disorders in children with autism spectrum disorders. Child Adolesc Psychiatr Clin N Am 2008; 17 (4): 887–905, x-xi [PubMed: 18775376]
8. McAdam DB, Sherman JA, Sheldon JB, Napolitano DA. Behavioral interventions to reduce the pica of persons with developmental disabilities. BehavModif 2004;28(1):45–72
9. Dempsey J, Dempsey AG, Guffey D, Minard CG, Goin-Kochel RP. Brief report: further examination of self-injurious behaviors in children and adolescents with autism spectrum disorders. J Autism Dev Disord 2016;46(5): 1872–1879 [PubMed: 26781113]
10. Fee V, Matson J. Definition, Classification, and Taxonomy. In: Luiselli J, Matson J, Singh N, eds. Self-Injurious Behavior New York, NY: Springer; 1992: 3–20
11. Poppes P, van der Putten AJJ, Vlaskamp C. Frequency and severity of challenging behaviour in people with profound intellectual and multiple disabilities. Res Dev Disabil 2010;31(6): 1269–1275 [PubMed: 20728304]
12. Soke GN, Rosenberg SA, Hamman RF, et al. Brief report: prevalence of self-injurious behaviors among children with autism spectrum disorder-a population-based study. J Autism Dev Disord 2016;46(11):3607–3614 [PubMed: 27565654]
13. McClintock K, Hall S, Oliver C. Risk markers associated with challenging behaviours in people with intellectual disabilities: a meta-analytic study. J Intellect Disabil Res 2003;47(pt 6): 405–416 [PubMed: 12919191]
14. Ali Z Pica in people with intellectual disability: a literature review of aetiology, epidemiology and complications. J Intellect Dev Disabil 2001;26(3):205–215
15. Danford DE, Huber AM. Pica among mentally retarded adults. Am J Ment Defic 1982;87(2) 141–146 [PubMed: 7124824]
16. Hardan A, Sahl R. Psychopathology in children and adolescents with developmental disorders. Res Dev Disabil 1997;18(5):369–382 [PubMed: 9292930]
17. Swift I, Paquette D, Davison K, Saeed H. Pica and trace metal deficiencies in adults with developmental disabilities, Br J Dev Disabil 1999;45 (89): 111–117
18. Mitteer DR, Romani PW, Greer BD, Fisher WW. Assessment and treatment of pica and destruction of holiday decorations. J Appl Behav Anal 2015; 48(4):912–917 [PubMed: 26380947]
19. Hergüner A, Hergüner S. Pica in an adolescent with autism spectrum disorder responsive to aripiprazole. J Child Adolesc Psychopharmacol 2016; 26(1):80–81 [PubMed: 26780755]
20. Serour F, Witzling M, Frenkel-Laufer D, Gorenstein A. Intestinal obstruction in an autistic adolescent. Pediatr Emerg Care 2008;24(10):688–690 [PubMed: 19240672]
21. Wijetilleke A, Sakran M, Kamat-Nerikar R. Vomiting in a girl with autism. Clin Pediatr (Phila) 2009;48 (2):224–227 [PubMed: 18832543]
22. Emond A, Emmett P, Steer C, Golding J. Feeding symptoms, dietary patterns, and growth in young children with autism spectrum disorders. Pediatrics 2010;126(2). Available at: www.pediatrics.org/cgi/content/full/126/2/e337
23. Neumeyer AM, Anixt J, Chan J, et al. Identifying associations among co-occurring medical conditions in children with autism spectrum disorders. Acad Pediatr 2019;19(3): 300–306 [PubMed: 30053632]
24. Schendel DE, Diguiseppi C, Croen LA, et al. The Study to Explore Early Development (SEED): a multisite epidemiologic study of autism by the Centers for Autism and Developmental Disabilities Research and Epidemiology (CADDRE) network. J Autism Dev Disord 2012;42(10):2121–2140 [PubMed: 22350336]
25. Wiggins LD, Reynolds A, Rice CE, et al. Using standardized diagnostic instruments to classify children with autism in the study to explore early development. J Autism Dev Disord 2015;45 (5) 1271–1280 [PubMed: 25348175]

26. Rutter M, Bailey A, Lord C. SCQ: Social Communication Questionnaire Torrance, CA: Western Psychological Services; 2003
27. Mullen E Mullen Scales of Early Learning Circle Pines, MN: American Guidance Services, Inc.; 1995
28. Lord C, Risi S, Lambrecht L, et al. The autism diagnostic observation schedule-generic: a standard measure of social and communication deficits associated with the spectrum of autism. J Autism Dev Disord 2000;30(3): 205–223 [PubMed: 11055457]
29. Lord C, Rutter M, Le Couteur A. Autism Diagnostic Interview-Revised: a revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. J Autism Dev Disord 1994;24(5):659–685 [PubMed: 7814313]
30. Achenback T Child Behavior Checklist Burlington, VT: Achenbach System of Empirically Based Assessment; 2013
31. Zou G A modified poisson regression approach to prospective studies with binary data. Am J Epidemiol 2004; 159 (7):702–706 [PubMed: 15033648]
32. Mantel N, Haenszel W. Statistical aspects of the analysis of data from retrospective studies of disease. J Natl Cancer Inst 1959;22(4):719–748 [PubMed: 13655060]
33. Autism Speaks Canada. Guides to pica for parents and professionals 2020. Available at: https://www.autismspeaks.ca/science-services-resources/resources/tool-kits/guides-to-pica-for-parents-and-professionals/. Accessed December 9, 2020
34. Mayes SD, Zickgraf H. Atypical eating behaviors in children and adolescents with autism, ADHD, other disorders, and typical development. Res Autism Spectr Disord 2019;64:76–83
35. Provost B, Crowe TK, Osbourn PL, McClain C, Skipper BJ. Mealtime behaviors of preschool children: comparison of children with autism spectrum disorder and children with typical development. Phys Occup Ther Pediatr 2010;30(3):220–233 [PubMed: 20608859]
36. Schieve LA, Harris S, Maenner MJ, Alexander A, Dowling NF. Assessment of demographic and perinatal predictors of non-response and impact of non-response on measures of association in a population-based case control study: findings from the Georgia Study to Explore Early Development. Emerg Themes Epidemiol 2018;15:12 [PubMed: 30147744]
37. Call NA, Simmons CA, Mevers JE, Alvarez JP. Clinical outcomes of behavioral treatments for pica in children with developmental disabilities. J Autism Dev Disord 2015;45(7):2105–2114 [PubMed: 25636679]

WHAT'S KNOWN ON THIS SUBJECT:

Previous pica prevalence estimates in children or adults with autism spectrum disorder (ASD) and/or intellectual disability are reported to range from 2% to 26%. However, there are few epidemiological studies on pica prevalence in individuals with ASD and other developmental disabilities.

WHAT THIS STUDY ADDS:

Results from a large case-control study suggest that pica is common in preschool-aged children with ASD, ASD characteristics, and/or intellectual disability. Pediatricians and parents should be aware of the potential for pica among this population of children.

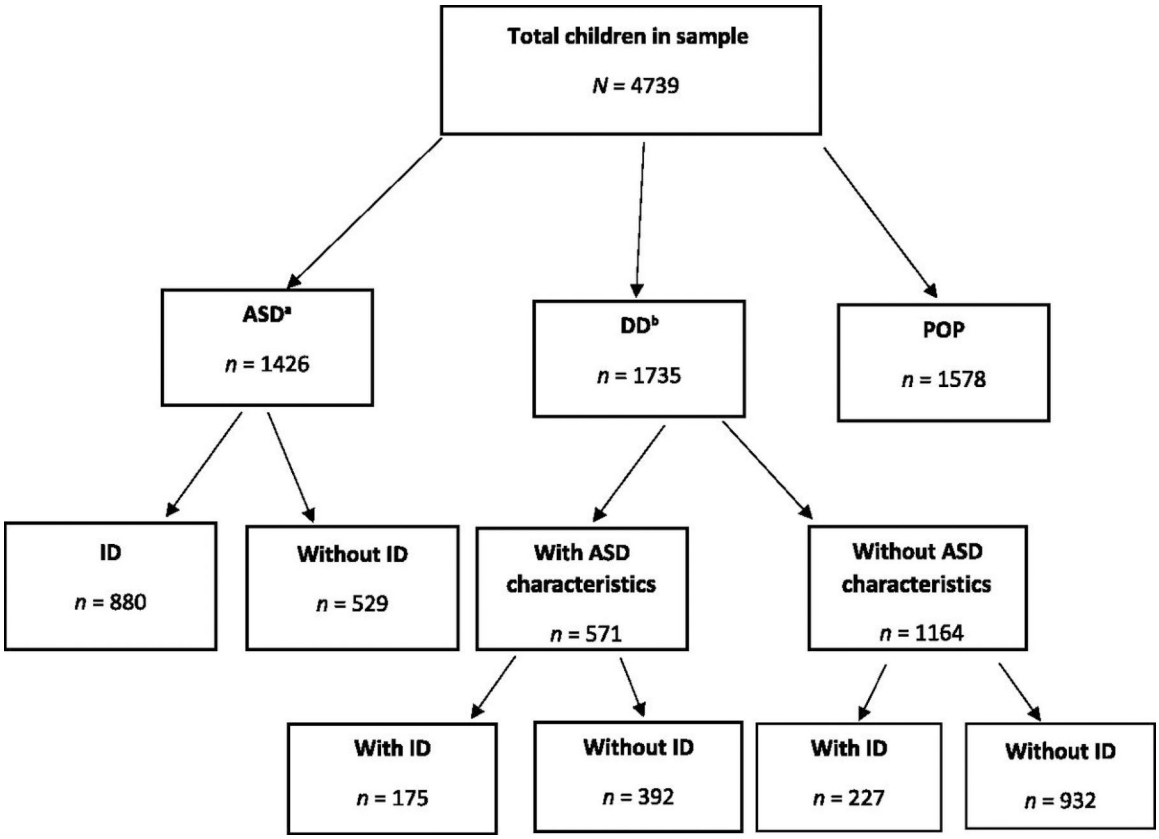

FIGURE 1.

The flowchart reveals the way in which the sample of children was divided into different groups and subgroups. The main study groups are ASD, DD, and POP. The ASD and DD groups were subdivided into children with and without ID. The DD group was further subdivided into children with and without ASD characteristics. [a] Seventeen children with ASD were missing data on IQ. [b] Nine children with DD were missing data on IQ.

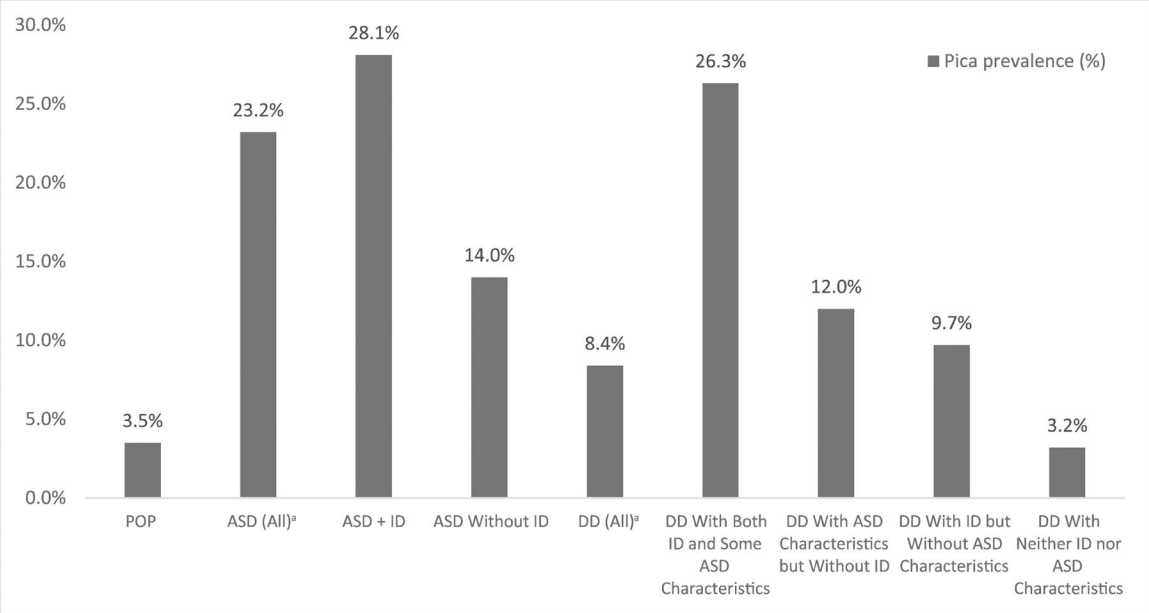

FIGURE 2.

Pica prevalence in SEED study groups and subgroups. [a]This includes children missing data on IQ, as noted in Fig 1.

TABLE 1

Descriptive Characteristics of the Study Population According to Case-Control Group and Engagement in Pica

Characteristics	ASD (All) (*n* = 1426), *n* (%)	DD (All) (*n* = 1735), *n* (%)	POP (*n* = 1578), *n* (%)	Pica = Yes (*n* = 531), *n* (%)	Pica = No (*n* = 4208) *n* (%)
Child sex					
Male	1163 (81.6)[a]	1156 (66.6)[a]	838 (53.1)	378 (71.2)[a]	2779 (66.0)
Female	263 (18.4)[a]	579 (33.4)[a]	740 (46.9)	153 (28.8)[a]	1429 (34.0)
Child age, mo					
30–48	314 (22.0)[a]	402 (23.2)[a]	441 (27.9)	144 (27.1)	1013 (24.1)
48–68	1112 (78.0)[a]	1333 (76.8)[a]	1137 (72.1)	387 (72.9)	3195 (75.9)
Maternal age, y					
<30	205 (14.4)	295 (17.0)[a]	191 (12.1)	103 (19.4)[a]	588 (14.0)
30–34	385 (27.0)	418 (24.1)[a]	401 (25.4)	139 (26.2)[a]	1065 (25.3)
≥35	835 (58.6)	1022 (58.9)[a]	986 (62.5)	289 (54.4)[a]	2554 (60.7)
Maternal race or ethnicity					
NHW	715 (50.9)[a]	937 (54.5)[a]	1075 (68.9)	240 (45.6)[a]	2487 (59.8)
NHB	324 (23.1)[a]	369 (21.5)[a]	214 (13.7)	139 (26.4)[a]	768 (18.5)
Hispanic	201 (14.3)[a]	268 (15.6)[a]	136 (8.7)	96 (18.3)[a]	509 (12.2)
Other or multiracial	165 (11.7)[a]	144 (8.4)[a]	135 (8.7)	51 (9.7)[a]	393 (9.5)
Maternal education					
<Bachelor’s degree	653 (46.1)[a]	794 (46.0)[a]	469 (29.9)	307 (57.9)[a]	1609 (38.5)
Bachelor’s degree or higher	765 (53.9)[a]	933 (54.0)[a]	1099 (70.1)	223 (42.1)[a]	2574 (61.5)
Household income, $					
<50000	514 (37.1)[a]	672 (40.3)[a]	373 (24.4)	255 (50.1)[a]	1304 (32.0)
50–89999	390 (28.2)[a]	419 (25.1)[a]	371 (24.2)	112 (22.0)[a]	1068 (26.2)
90000+	481 (34.7)[a]	576 (34.6)[a]	787 (51.4)	142 (27.9)[a]	1702 (41.8)

NHB, non-Hispanic Black.

[a] $P < .05$ derived from a χ^2 test in which the ASD study group was compared with the POP group, the DD study group was compared with the POP group, and pica = yes was compared with pica = no.

Table 2

Prevalence of Pica and Associations Between Study Case Classification and Pica

Study Case Classification	Total, *n*	Pica = Yes, *n* (%)	PR[a] (95% CI)	aPR[b] (95% CI)
POP	1578	55 (3.5)	Reference	Reference
ASD (all)[c]	1426	331 (23.2)	6.7 (5.1–8.8)	6.8 (5.0–9.1)
ASD + ID	880	247 (28.1)	8.1 (6.1–10.7)	8.0 (5.9–11.0)
ASD without ID	529	74 (14.0)	4.0 (2.9–5.6)	4.4 (3.0–6.5)
DD (all)[c]	1735	145 (8.4)	2.4 (1.8–3.2)	1.9 (1.4–2.7)
DD with both ID and some ASD characteristics	175	46 (26.3)	7.5 (5.3–10.8)	5.4 (3.4–8.5)
DD with ASD characteristics but without ID	392	47 (12.0)	3.4 (2.4–5.0)	2.5 (1.6–3.8)
DD with ID but without ASD characteristics	227	22 (9.7)	2.8 (1.7–4.5)	2.4 (1.4–4.1)
DD with neither ID nor ASD characteristics	932	30 (3.2)	0.9 (0.6–1.4)	0.9 (0.6–1.4)

Cl, confidence interval.

[a]PRs derived in which the prevalence of pica for each ASD and DD group and subgroup was compared with the prevalence for the POP group.

[b]aPRs derived from modified Poisson regression models in which ASD and DD groups and subgroups (8 models) were compared with the POP group. All models included adjustment for child sex, child age, maternal age, maternal race or ethnicity, maternal education, and household income.

[c]This includes children missing data on IQ, as noted in Fig 1.