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Head Growth Trajectories During the First Year of Life and Risk of Autism Spectrum Disorder

Rewaa Balaum¹ | Leena Elbedour¹ | Einav Alhozyel¹ | Gal Meiri^{2,3} | Dikla Zigdon^{2,3} | Analya Michaelovski^{3,4} | Orly Kerub⁵ | Idan Menashe^{1,3} 

¹Department of Epidemiology, Biostatistics and Community Health Sciences, Faculty of Health Sciences, Ben-Gurion University of the Negev, Beer-Sheva, Israel | ²Preschool Psychiatric Unit, Soroka University Medical Center, Beer-Sheva, Israel | ³Azrieli National Center for Autism and Neurodevelopment Research, Ben-Gurion University of the Negev, Beer-Sheva, Israel | ⁴Child Development Center, Soroka University Medical Center, Beer-Sheva, Israel | ⁵Ministry of Health, Jerusalem, Israel

Correspondence: Idan Menashe (idanmen@bgu.ac.il)

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ABSTRACT

Atypical infant head circumference (HC)—including increased rates of macrocephaly and microcephaly—has been linked to autism spectrum disorder (ASD). However, specific head growth trajectories associated with ASD remain poorly defined. This retrospective case–control study aimed to delineate these trajectories and examine their relationship to height. The study sample included 262 children diagnosed with ASD and 560 matched controls. Growth measures at 1, 2, 4, 6, 9, and 12 months of age were obtained from health clinics in southern Israel. The sample was classified into seven clusters based on HC patterns across these six time points, and associations with ASD were estimated using conditional logistic regression. Results demonstrated significant correlations between HC and height throughout the study period (Pearson correlation $r=0.44$ – 0.55 , $p<0.001$), with stronger correlations in ASD ($r=0.50$ – 0.67) compared to controls ($r=0.32$ – 0.50). Children with consistently small or large HC exhibited the highest ASD likelihood (adjusted odds ratio [aOR]=2.95, 95% CI=1.88–4.94; and aOR=3.17, 95% CI=1.92–5.01, respectively), with the most extreme percentiles (0–5th and 95th–100th) showing the strongest associations (aOR=9.53, 95% CI=2.49–35.26; aOR=6.51, 95% CI=2.91–15.35, respectively). These associations were primarily driven by children with similar height trajectories (aOR=7.71, 95% CI=3.23–15.43; and aOR=6.89, 95% CI=2.99–13.26, respectively), indicating that atypical HC growth in ASD during infancy may reflect broader physiological growth dysregulation.

1 | Introduction

Anthropometric measures, particularly head circumference (HC), play a critical role in monitoring early childhood development. HC, along with height and weight, is utilized by clinicians to evaluate health and nutritional well-being in infants and children (US Centers for Disease Control and Prevention 2024). Deviations from standard growth benchmarks have also been shown to be associated with neurodevelopmental and

cognitive impairment in childhood (Heinonen et al. 2008; Hilaire et al. 2021; Pongcharoen et al. 2012; Sanefuji et al. 2021; Welling et al. 2020), emphasizing the critical role of growth monitoring in detecting at-risk children.

Abnormal head growth patterns in infants have been associated with a subsequent diagnosis of autism spectrum disorder (ASD) in some children. Several studies have reported that a subset of children with ASD exhibit smaller HC at

Rewaa Balaum and Leena Elbedour contributed equally to this work.

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Lay Summary

This study explored the link between head size and autism spectrum disorder (ASD) in infants. Head and height growth of 262 children with ASD and 560 children without ASD were examined, using data collected from health clinics in southern Israel. We grouped infants based on their growth patterns during the first year of life and found that children with head sizes at the low or high end of the growth chart were more likely to have ASD, especially when their height followed the same pattern. These results suggest that unusual head growth may be part of an overall growth difference in some infants with ASD.

birth, followed by a period of accelerated growth during infancy (Courchesne et al. 2003; Crucitti et al. 2020; Dinstein et al. 2017; Mraz et al. 2007). Additional studies using prenatal ultrasound scans have suggested that such head growth abnormalities may begin in utero (Aydin et al. 2024; Bonnet-Brilhault et al. 2018; Regev et al. 2021). Furthermore, several MRI studies have suggested that such abnormal head growth associated with ASD involves an excess of cerebrospinal fluid volume, and enlargement of brain regions associated with language, social, and communication functioning (Courchesne et al. 2003, 2011; Hazlett et al. 2017; Sacco et al. 2015; Shen et al. 2018).

Macrocephaly and microcephaly have also been shown to present in some children with ASD. Macrocephaly, defined as an HC above the 97th percentile for sex and age (National Institutes of Health 2024a), has been widely reported to be more prevalent among children with ASD than typically developing children (Ben-Itzhak et al. 2013; Ciriugliaro et al. 2024; Courchesne et al. 2003; Crucitti et al. 2020; Grandgeorge et al. 2013; Lainhart et al. 2006; Sacco et al. 2015; van der Net et al. 2024). Conversely, microcephaly, characterized by an HC below the 3rd percentile for sex and age (NIH 2024b), has received less empirical attention but has been observed among a subset of autistic individuals during specific developmental windows, including early infancy (Ben-Itzhak et al. 2013; Cederlund 2021; Crucitti et al. 2020). Here too, recent data have demonstrated that some of these children are already born with such congenital head abnormalities (Courchesne et al. 2003), which in certain cases can be detected through standard prenatal ultrasound screenings (Regev et al. 2022). However, it is important to note that these patterns are not universal, and many children with ASD do not demonstrate such atypical growth trajectories (Dinstein et al. 2017)

Altogether, these findings highlight the complex relationship between abnormal head growth during infancy and ASD. In addition, it is unclear whether this relationship is restricted to the head or is part of a generalized physiological growth abnormality, as suggested by several studies (Campbell et al. 2014; Chawarska et al. 2011; Dissanayake et al. 2006). These studies were constrained by relatively small sample sizes, restricted age ranges, or sparse longitudinal data, thus limiting definitive conclusions regarding whether HC differences reflect unique neurodevelopmental deviation or broader physiological growth. Our study addresses these gaps by analyzing a larger retrospective

cohort with anthropometric data collected prospectively across multiple time points during the first year of life. Using clustering methods to characterize specific HC and height trajectories, as well as any overlaps, we aim to investigate these early growth dynamics in ASD.

2 | Methods

2.1 | Study Setting

This retrospective case–control study was conducted in southern Israel, the Negev. This area is defined by a population of approximately 1 million residents, and 16,000 live births recorded annually, which are divided nearly equally between the two primary ethnicities, Jews and Bedouin Arabs (Central Bureau of Statistics of Israel 2021). All newborns undergo routine monitoring at 47 government-funded mother–child health clinics (MCHCs) around the region up until the age of 6 years, where trained clinicians assess the child’s physiological growth and developmental wellness during each checkup (Bin Nun et al. 2010; Israel Ministry of Health 2023). These assessments rely on direct observation, standardized testing of the child, and concurrent parent reports. If any concerns arise during these sessions regarding the child’s attainment of proper growth or developmental standards, a more comprehensive evaluation is conducted if needed, during which the child may then be referred to a Child Developmental Center (Israel Ministry of Health 2023).

2.2 | Case–Control Ascertainment

Both cases and controls were drawn from the population of children born in southern Israel between 2014 and 2017 whose development and nutrition were monitored at the MCHCs operated by the Ministry of Health. Cases were randomly ascertained from all children with ASD who are registered in the database of the Azrieli National Center for Autism and Neurodevelopment Research (ANCAN) and were diagnosed according to DSM-5 criteria (American Psychiatric Association 2013) by a child developmental psychologist and either a child psychiatrist or pediatric neurologist at the Child Development Center of Soroka University Medical Center (SUMC) as described previously (Dinstein et al. 2020; Meiri et al. 2017). These cases did not significantly differ in their basic sociodemographic and clinical characteristics from other ANCAN’s cases that were diagnosed at SUMC (Table S1). Controls were children not diagnosed with any developmental disorder, including ASD, and frequency matched to cases by date of birth (± 3 months), sex (male/female), and ethnicity (Jewish/Arab-Bedouin). To reduce the possibility of potential confounding, we did not include children born preterm (< 37 weeks of pregnancy) and children lost to follow-up (missing > 2 checkups). We also excluded three children who had been referred for further assessment due to possible developmental delay, but who had not been diagnosed with ASD at the time of data collection to create a clear case–control comparison specifically focusing on ASD. Ultimately, the study sample included a total of 262 cases and 560 controls (Figure S1).

2.3 | Data Collection

Growth measure data were prospectively collected by MCHC clinicians and recorded in the MCHC database. Participants' ID numbers were used to retrieve their MCHC records, which included their socio-demographic, birth, and clinical information, as well as HC and height measures at 1, 2, 4, 6, 9, and 12 months for the purposes of the study. All study participants had ≥ 5 growth measures during their first year of life.

2.4 | Data Analysis

We modeled individual HC growth using HC measures from all six time points during the first year of life (1, 2, 4, 6, 9 and 12 months) using a latent growth curve (LGC) framework to classify "constant" versus "increasing/decreasing" clusters. Time scores were set to these months (centered) and a linear slope factor was estimated with a random intercept. The slope factor (β) and its 95% confidence interval were determined for each child. We used these values to determine whether a child's HC was increasing (95% CI of $\beta > 0$), decreasing (95% CI of $\beta < 0$), or constant (95% CI of β includes 0). Then, we further classified these groups based on their HC percentiles using the age- and sex-standardized infant growth charts (Israel Ministry of Health 2024) as follows: Within the "constant" group, we calculated the mean of their HC percentiles throughout the first year of life and distinguished between children with consistently small (CS), consistently medium (CM) and consistently large (CL) HC (mean[HC] $< 25\%$, mean[HC] = 25% – 75% , and Mean[HC] $> 75\%$ respectively). Within the "increasing" and "decreasing" groups, we calculated the mean percentile of the first two measure points (at 1 and 2 months of age) and the mean percentile of the last two measure points (at 9 and 12 months of age). Then, we used these values to distinguish between children with increasing HC from $< 25\%$ to 25% – 75% (INC-S2M), increasing HC from 25% – 75% to $> 75\%$ (INC-M2L), decreasing HC from $> 75\%$ to 25% – 75% (DEC-L2M), and decreasing HC from 25% – 75% to $< 25\%$ (DEC-M2S). Consequently, seven mutually exclusive growth trajectories were determined (Figure 1). The same analysis was also conducted for height measures (Figure S2).

Demographic and birth characteristics were compared between cases and controls using standard univariate statistics (e.g., *t*-test, chi-square test). Conditional logistic regression models were then employed to assess the independent associations of HC and height trajectories with risk of ASD while adjusting for demographic and birth variables that differed between cases and controls.

3 | Results

The study sample characteristics are presented in Table 1. Of the 822 children, 78.2% were male, and 76.8% were Jewish. Children with ASD, compared to children without ASD, were significantly more likely to come from families of lower socioeconomic status ($p < 0.001$). In addition, children with ASD were born with a significantly lower weight (3.24 ± 0.45 kg vs. 3.32 ± 0.39 kg, $p = 0.024$) and with a smaller HC (34.18 ± 2.32 cm vs. 34.88 ± 1.54 cm, $p < 0.001$).

The seven distinct HC growth trajectories identified in the study sample are depicted in Figure 1. Atypical HC was observed in approximately 71.0% of children with ASD. Most children ($n = 238$; 29.0%) exhibited an HC measure within the interquartile range throughout the first year of life (CM trajectory). Corresponding to quartile distributions, approximately 25.0% of the Constant group were assigned to the CS and CL trajectories, respectively, making up 29.2% of the total sample. Finally, the HC percentile of 22.1% of the children increased during the first year, while the HC percentile of 19.7% of the children decreased.

Next, the association of the different HC trajectories with a subsequent diagnosis of ASD was examined. The results are presented in Table 2. Consistently small or large HC throughout the first year of life was significantly associated with approximately threefold higher odds of a subsequent ASD diagnosis compared with consistently normal HC (aOR = 2.95, 95% CI = 1.88–4.94 and aOR = 3.17, 95% CI = 1.92–5.01 for CS and CL compared to CM trajectories, respectively). Children whose HC increased during the first year of life also had higher odds of ASD (aOR = 2.03, 95% CI = 1.21–3.17 and aOR = 2.45, 95% CI = 1.57–4.15 for INC-S2M and INC-M2L trajectories, respectively), while those whose HC decreased exhibited milder, non-significant elevation (aOR = 1.49, 95% CI = 0.88–2.52 and aOR = 1.06, 95% CI = 0.51–2.16 for DEC-M2S and DEC-L2M trajectories, respectively).

Further stratification of children in the HC CS trajectory group (Table S2) revealed that those in the lowest 5th percentile (i.e., 0%–5%, including microcephalic children) exhibited the highest odds of ASD compared with children with typical HC development (aOR = 9.53, 95% CI = 2.49–35.26), followed by those in the 5th–10th percentile (aOR = 5.78, 95% CI = 2.12–12.42). Children with HC in the 10th–15th percentiles also demonstrated a significant, albeit weaker, association (aOR = 2.62, 95% CI = 1.64–4.46). A similar dose–response effect was observed among children in the consistently large (CL) HC trajectory (Table S3). Compared with the CM trajectory, consistent HC in the ≥ 95 th percentile (95%–100%) was associated with 6.51-fold higher odds of ASD (aOR = 6.51, 95% CI = 2.91–15.35) and HC in the 90th–95th percentiles was associated with 4.49 times the odds (aOR = 4.49, 95% CI = 1.93–9.10). More modest but significant elevations were seen at the 85th–90th (aOR = 2.27, 95% CI = 1.17–5.91) and 80th–85th (aOR = 2.02, 95% CI = 1.03–4.25) strata, whereas the 75th–80th percentile was not associated with ASD (aOR = 1.45, 95% CI = 0.31–4.21).

As expected, HC measures were positively correlated with the height measures in these children across the six checkup points during the first year of life (Figure 2). Stronger correlations were observed in early infancy compared to late infancy ($r = 0.54$, $r = 0.55$, $r = 0.54$, $r = 0.49$, $r = 0.46$ and $r = 0.44$ for 1, 2, 4, 6, 9, and 12 months of age, respectively; $p < 0.001$ for all) and in cases compared to controls ($r = 0.67$ – 0.50 for cases, and $r = 0.50$ – 0.32 for controls). Given the strength of these correlations, it was not surprising that the same height trajectories were identified (Figure S2) and showed comparable associations with ASD (Table S4). Here too, the highest odds ratios for ASD were observed with the CS and CL height trajectories (aOR = 2.49, 95% CI = 1.59–3.83; and aOR = 2.22, 95% CI = 1.51–3.76, respectively).

Finally, to assess the contribution of overall growth to the association between HC abnormalities and ASD, we divided the

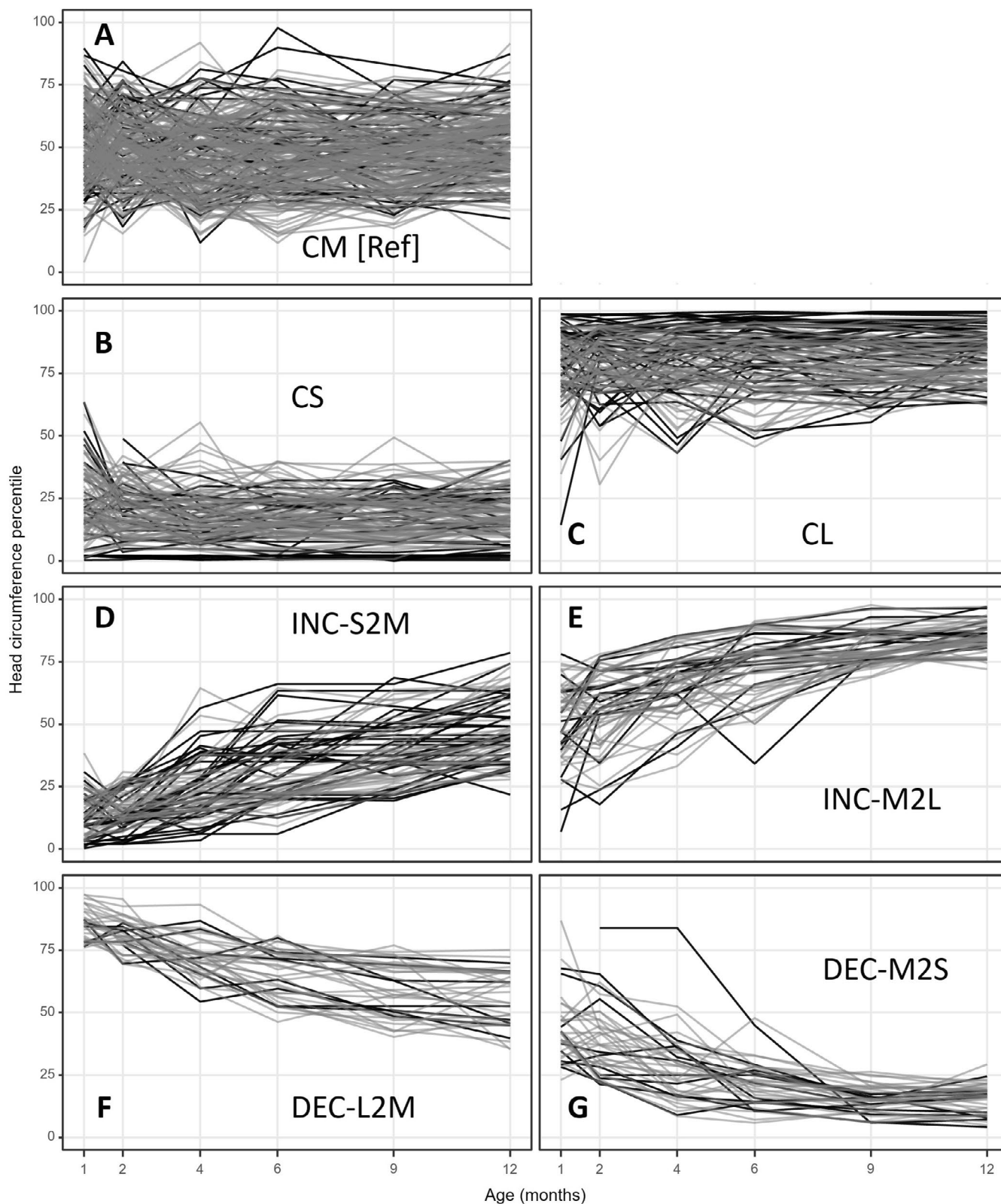


FIGURE 1 | Head circumference (HC) trajectories: seven different clusters of head circumference (HC) trajectories during infancy are depicted: (A) CM—normal HC within the interquartile range (25th–75th percentile) throughout infancy; (B) CS—consistently small (0–25th percentile); (C) CL—consistently large (75th–100th percentile); (D) INC-S2M—increase from small (0–25th percentile) to medium (25th–75th percentile); (E) INC-M2L—increase from medium (25th–75th percentile) to large (75th–100th percentile); (F) DEC-L2M—decrease from large (75th–100th percentile) to medium (25th–75th percentile); (G) DEC-M2S—decrease from medium (25th–75th percentile) to small (0–25th percentile). Black lines are children with ASD and gray lines are children without ASD.

CS and CL HC groups into those that overlapped with their corresponding height groups and those that did not. The association of each of the resulting groups with ASD was then tested (Table 3). This analysis revealed that the association between

abnormal HC and ASD was primarily seen in infants with generalized growth abnormalities, rather than in those with isolated HC growth abnormalities (aOR = 7.71, 95% CI = 3.23–15.43 compared to aOR = 1.62, 95% CI = 0.98–3.09 for the overlapping and non-overlapping CS groups, respectively; and aOR = 6.89, 95% CI = 2.99–13.26 compared to aOR = 2.61, 95% CI = 1.57–4.21 for the overlapping and non-overlapping CL groups, respectively).

TABLE 1 | Baseline characteristics of the study sample.

Variable	ASD (N = 262)	Non-ASD (N = 560)	p
Sex (male), N (%)	205 (78.2%)	438 (78.2%)	1
Ethnicity (Jewish), N (%)	201 (76.8%)	430 (76.8%)	1
Socio-economic status, N (%)			
High	9 (3.3%)	124 (22.1%)	<0.001
Middle	163 (62.3%)	254 (45.4%)	
Low	90 (34.4%)	182 (32.5%)	
Cesarean section, N (%)	70 (26.7%)	127 (22.7%)	0.211
Apgar ≤ 7 at 1 min, N (%)	17 (6.6%)	25 (4.5%)	0.483
Apgar ≤ 7 at 5 min, N (%)	5 (1.9%)	7 (1.2%)	0.486
Birth weight, kg, mean (SD)	3.24 (0.45)	3.32 (0.39)	0.024
Head circumference at birth, cm, mean (SD)	34.18 (2.32)	34.88 (1.54)	<0.001

4 | Discussion

The findings of the present study offer several insights into the relationship between physiological growth parameters during infancy and a subsequent diagnosis of ASD. The results demonstrate that both exceptionally large and small HC measures during infancy are significantly associated with a subsequent diagnosis of ASD. These findings align with previous reports examining the association between macro- and microcephaly in early life and ASD (Ben-Itzhak et al. 2013; Cederlund 2021; Cirnigliaro et al. 2024; Dementieva et al. 2005; Fombonne et al. 1999; Lainhart et al. 2006; Sacco et al. 2015). Nevertheless, while prior research has generally corroborated the presence of macro- or microcephaly in ASD based on a limited number of HC measures potentially leading to an inflation of the estimated rates of macro- and microcephaly in ASD, the present study underscores the importance of consistent and continuous surveillance of HC, allowing for a more refined classification of HC growth trajectories at infancy and their variable association with ASD.

Various previous studies have reported that children with ASD are more likely to present with relatively small HC at birth, followed by rapid overgrowth during the first year (Courchesne et al. 2003; Crucitti et al. 2020; Dinstejn et al. 2017; Mraz et al. 2007; Redcay and Courchesne 2005). In our data, 11.4%

TABLE 2 | Association between ASD and head circumference (HC) growth trajectories across infancy (1–12 months).

HC cluster	ASD cases (N = 262)	Controls (N = 560)	Crude odds ratio	95% CI	Adjusted odds ratio ^a	95% CI
CM (N = 238)	48 (18.3%)	190 (33.9%)	[Ref]		[Ref]	
CS (N = 120)	53 (20.2%)	67 (12.0%)	3.13	1.94–5.06	2.95	1.88–4.94
CL (N = 120)	55 (21.0%)	65 (11.6%)	3.35	2.08–5.41	3.17	1.92–5.01
INC-S2M (N = 92)	30 (11.4%)	62 (11.1%)	1.92	1.12–3.28	2.03	1.21–3.17
INC-M2L (N = 90)	34 (13.0%)	56 (10.0%)	2.40	1.41–4.09	2.45	1.57–4.15
DEC-M2S (N = 99)	28 (10.7%)	71 (12.7%)	1.56	0.91–2.68	1.49	0.88–2.52
DEC-L2M (N = 63)	14 (5.3%)	49 (8.8%)	1.13	0.58–2.22	1.06	0.51–2.16

Abbreviations: CM [Ref], medium (typical) HC within the interquartile range (25th–75th percentile) across infancy; CS, consistently small (0–25th percentile); CL, consistently large (75th–100th percentile); INC-S2M, increase from small (0–25th percentile) initially to medium (25th–75th percentile); INC-M2L, increase from medium (25th–75th percentile) initially to large (75th–100th percentile); DEC-M2S, decrease from medium (25th–75th percentile) initially to small (0–25th percentile); DEC-L2M, decrease from large (75th–100th percentile) initially to medium (25th–75th percentile).

^aAdjusted for sex, ethnicity, socioeconomic status, birth delivery mode, 1- and 5-min Apgar scores, and birth weight.

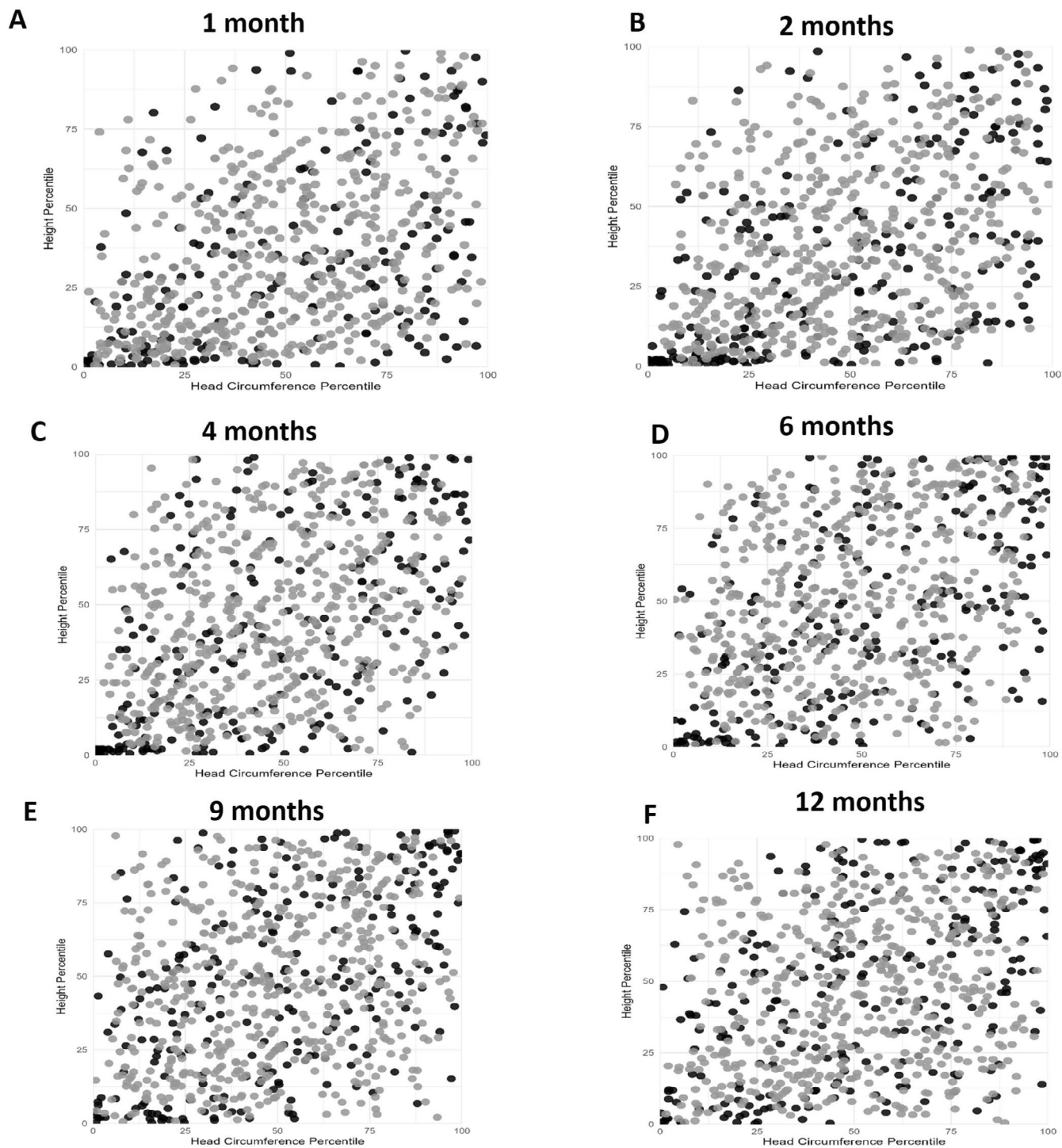


FIGURE 2 | Scatter plots depicting the correlation between head circumference (HC, x-axis) and height percentiles (y-axis) at 1, 2, 4, 6, 9, and 12 months of age (A–F). Black circles are children with ASD and gray circles are children without ASD.

of children with ASD and 11.1% of children without ASD demonstrated such HC growth (INC-S2M trajectory), which was associated with a significant odds ratio (OR) of 2.03 for ASD. Alternatively, children who demonstrated decreasing HC measures were not shown to have increased ASD odds. Similar findings of HC overgrowth in normocephalic children at birth subsequently diagnosed with ASD were also observed in other studies (Chawarska et al. 2011; Ciriigliaro et al. 2024; Courchesne et al. 2001; Dawson et al. 2007; Dementieva

et al. 2005; Fukumoto et al. 2011; Hazlett et al. 2017; Libero et al. 2016; Nordahl et al. 2011).

An important finding from the present study suggests that most of the risk of ASD associated with HC abnormalities during infancy may be attributed to overall growth abnormalities and not a restricted characteristic of head growth. Various studies have also pointed to such a relationship between head and body growth abnormalities associated with ASD risk (Chawarska

TABLE 3 | Association of ASD and overlapping height and head circumference (HC) clusters.

Group	ASD cases (N=156)	Crude odds ratio	95% CI	Adjusted odds ratio ^a	95% CI
HC CM (N=238)	48	[Ref]		[Ref]	
HC CS (N=120)					
Height CS (N=37)	25	8.25	3.87–17.59	7.71	3.23–15.43
Height non-CS (N=83)	28	2.02	1.16–3.51	1.62	0.98–3.09
HC CL (N=120)					
Height CL (N=41)	27	7.63	3.72–15.67	6.89	2.99–13.26
Height non-CL (N=79)	28	2.17	1.24–3.80	2.61	1.57–4.21

Abbreviations: CM [Ref], normal HC within the interquartile range (25th–75th percentile) across infancy; CS, consistently small (0–25th percentile); CL, consistently large (75th–100th percentile).

^aAdjusted for sex, ethnicity, socioeconomic status, birth delivery mode, 1- and 5-min Apgar scores, and birth weight.

et al. 2011; Dissanayake et al. 2006; Mraz et al. 2007; de Vinck-Baroody et al. 2015), while other studies have reported that HC growth is independent of other physiological growth measures in ASD (Courchesne et al. 2003; Lainhart et al. 2006; Grandgeorge et al. 2013). While the reason for this generalized overgrowth in ASD is yet unknown, hormonal (Raghavan et al. 2018), immune (Sacco et al. 2007), and genetic (Fu et al. 2023) hypotheses have been proposed. With further analytic and clinical validation, early HC and height trajectories may serve as combined screening markers for identifying children inferred to have a higher risk of developing certain subsets of ASD, ultimately facilitating earlier diagnosis and treatment and improving prognosis.

A notable strength of the present study lies in its use of trajectory measurements of HC, moving beyond the reliance on single or few point measurements often employed in the literature. This approach provides a more nuanced understanding of specific trajectories that may distinguish subsets of ASD cases. Additionally, the incorporation of prospectively collected growth measurements within the existing Israeli MCHC system is another strength of the study.

The study has also several limitations. First, the restriction of the observation period to the first year of life, as previous research has identified aberrant growth patterns beyond this period (Courchesne et al. 2011; Redcay and Courchesne 2005). Nevertheless, this focus is particularly relevant given that current standardized screening methods can identify possible ASD as early as 12 months of age but typically not earlier (Hyman et al. 2020). Thus, the findings provide valuable insight into whether HC growth trajectories during infancy may serve as a prospective screening tool for earlier identification of subtypes of ASD. Additionally, this study did not include sex-specific analyses, despite emerging evidence suggesting possible discrepancies in growth trajectories between males and females with ASD (Crucitti et al. 2020; Fukumoto et al. 2011; Surén et al. 2013). Importantly, the use of standardized age-and-sex growth charts to determine the growth percentile of each participant may account for some of these reported sex differences. In addition, genetic testing was not available for our sample, impeding classification of syndromic versus idiopathic cases of

ASD. Finally, the exclusion of participants with developmental concerns other than ASD limited the assessment of the specificity of growth patterns in ASD. This exclusion, nonetheless, was intended to strengthen the case-control comparison focused specifically on ASD.

5 | Conclusions

Our findings suggest that the reported associations between atypical head growth during infancy and ASD may be attributed to broader physical growth anomalies. This conclusion highlights the importance of a multifaceted, longitudinal examination of such anthropometric measures in studies of child development.

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Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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- percentile); (C) CL—consistently large (75th–100th percentile); (D) INC-S2M—increase from small (0–25th percentile) to medium (25th–75th percentile); (E) INC-M2L—increase from medium (25th–75th percentile) to large (75th–100th percentile); (F) DEC-L2M—decrease from large (75th–100th percentile) to medium (25th–75th percentile); (G) DEC-M2S—decrease from medium (25th–75th percentile) to small (0–25th percentile). Black lines are children with ASD and gray lines are children without ASD. **Table S1:** Comparison of sociodemographic and clinical characteristics the study ASD cases vs. other children with ASD in the ANCAN database that were diagnosed at SUMC. **Table S2:** Association between ASD and subgroups children with constantly small (CS) head circumference (HC) across infancy (1–12 months). **Table S3:** Association between ASD and subgroups children with constantly large (CL) head circumference (HC) across infancy (1–12 months). **Table S4:** Association between ASD and height trajectories across infancy (1–12 months).

Supporting Information

Additional supporting information can be found online in the Supporting Information section. **Figure S1:** A flowchart depicting the ascertainment of cases and controls to the study. **Figure S2:** Height trajectories: seven different clusters of height trajectories during infancy are depicted: (A) CM—normal HC within the interquartile range (25th–75th percentile) throughout infancy; (B) CS—consistently small (0–25th