Psychological Medicine

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Course of head growth from birth to adolescence across the psychosis spectrum

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Original Article

Cite this article: Ashley, S. A., Merritt, K., Solmi, F., Luque Laguna, P., Logeswaran, Y., Jones, D. K., Linden, D., David, A. S., & Reichenberg, A. (2026). Course of head growth from birth to adolescence across the psychosis spectrum. *Psychological Medicine*, **56**, e267, 1–8
https://doi.org/10.1017/S0033291726105418

Received: 22 October 2025 UTC
Revised: 22 April 2026 UTC
Accepted: 24 June 2026 UTC

Keywords:
head circumference; longitudinal; neurodevelopment; psychosis; psychotic symptoms; schizophrenia

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Abstract

Background. Charting developmental abnormalities in future psychotic disorder could help identify pathogenic processes. Head circumference (HC) is a proxy for brain size. Studies of HC and its developmental trajectories in future psychotic disorder cases and across the psychosis spectrum are inconclusive. It is important to determine whether deviations in HC are specific or reflect a general growth disorder.

Methods. We used data from the Avon Longitudinal Study of Parents and Children (ALSPAC) to chart HC trajectories from birth to 15 years in individuals with operationally-defined psychotic disorder (N = 62), psychotic experiences (PE; N = 339) and controls (N = 3,527). We extracted total intracranial volume (TIV) from MRI data on a subset (N = 347) scanned at age 20.

Results. HC at birth, 7 and 15 years was correlated with TIV at age 20 (r's = 0.45–0.77). The psychotic disorder group had consistently smaller HC from birth through to adolescence (B = −0.26, p = 0.02). Females with PE had smaller HC across childhood and adolescence (B = −0.27, p = 0.016), while males with PE had consistently larger HC (B = 0.35, p = 0.023). The effects did not change when controlling for maternal and pregnancy-related characteristics but attenuated after controlling for height, except in males with PE.

Conclusions. Deviations in head and hence brain size across the psychosis spectrum likely reflect a more general growth abnormality. Future studies should examine factors regulating growth during early life in relation to psychosis, including sex, to understand underlying mechanisms.

Introduction

According to the neurodevelopmental model of schizophrenia (Murray, Lewis, & Lecturer, 1987), subtle deviations in brain development should be already apparent in childhood, years before overt clinical symptoms of the adult disorder manifest. Volumetric brain abnormalities in schizophrenia are well documented in systematic reviews and meta-analyses (Haijma et al., 2013). These abnormalities include reduced intracranial and total brain volumes and are present in antipsychotic medication-naïve cases (Haijma et al., 2013), suggesting early neurodevelopmental origins. However, the existing literature does not provide adequate longitudinal evidence for this hypothesis.

Head circumference (HC), a standard measure of head size, is believed to accurately reflect brain volume. It is routinely collected around birth and therefore could offer valuable insights into early brain development (Bartholomeusz, Courchesne, & Karns, 2002; Hshieh et al., 2016). Indeed, supporting the neurodevelopmental origin of schizophrenia, small HC has been associated with increased risk of schizophrenia in several studies (Cantor-Graae, Ismail, & McNeil, 1998; Hultman et al., 1997; Robinson et al., 2023). Yet other studies found an effect of larger HC, no association, or relationships that were restricted to females (Cannon, Jones, & Murray, 2002b; Hultman et al., 1999; Kunugi et al., 1996; Nilsson et al., 2005; Wahlbeck et al., 2001; Welham et al., 2009). A meta-analysis did not find a statistically significant association between small HC (<32 cm) after birth and risk for schizophrenia, but there was evidence for publication bias (Davies et al., 2020).

It is unclear whether abnormal HC represents a brain-specific process, or a more generalized growth disorder. Results of individual studies examining the association between length at birth and schizophrenia are mixed, some reporting that shorter length is associated with higher risk (Burchard, 1916; Davies et al., 2020; Gunnell et al., 2005; Kemali, Polani, Polani, & Amati, 1976; Perrin et al., 2007; Zammit et al., 2007), while others find no association (Latham & Kirkpatrick, 2018). A meta-analysis suggested that small HC and short length at birth may both be related to later

development of a psychotic disorder (Davies et al., 2020). Determining if abnormal HC represents a brain-specific process has important implications for identifying potential underlying biological mechanisms.

In contrast to recent studies examining the neurodevelopmental hypothesis, which focus on developmental trajectories from birth through adulthood, including behavior and cognition (Cannon et al., 2002a; Mollon et al., 2018), studies of HC in schizophrenia have predominantly focused on the period surrounding birth and the early years of life. To the best of our knowledge, only two studies examined HC trajectories. One reported evidence of accelerated head growth from birth to 2 months in females but not in males who went on to develop schizophrenia (Brown et al., 2017). Another birth cohort study showed that young female but not male adults with non-affective psychosis had larger head circumferences at birth than controls but no differences at 5 years old (Welham et al., 2009). Longitudinal studies of HC from birth and through adolescence are lacking.

In the present study, we report data from a population-representative longitudinal study (the Avon Longitudinal Study of Parents and Children [ALSPAC]) that has followed children from birth to adulthood. HC was prospectively assessed at multiple time points from birth until age 15. We charted HC growth from birth through mid-adolescence (age 15) in individuals with operationally defined psychotic disorder (at age 18), the much larger group of individuals with psychotic experiences (PE), and controls drawn from the same cohort, and examined if group difference represents a brain-specific process, or a more generalized growth disorder.

Methods

Sample

Participants were drawn from ALSPAC, a UK-based population cohort study from the Southwest of England (Boyd et al., 2013; Fraser et al., 2013). Pregnant women with expected delivery dates between April 1, 1991 and December 31, 1992 were recruited, resulting in 14,062 live births (see Supplementary Table S1). Please note that the study website contains details of all the data that is available through a fully searchable data dictionary and variable search tool (http://www.bristol.ac.uk/alspac/researchers/our-data/). Ethical approval was granted by the ALSPAC Ethics and Law Committee (ALEC) and the Local Research Ethics Committees. Informed consent for the use of data collected via questionnaires and clinics was obtained from participants following the recommendations of the ALEC.

We used data from all individuals who had undergone diagnostic interviewing at age 18 (N = 4,675, N with PE = 428). From this sample, 4,588 had at least one measurement of HC, and 3,866 also had complete data on all confounders. Therefore, our analytic sample comprised 3,866 individuals (2,177 females). To improve statistical power and minimize the influence of small sample sizes, only timepoints for HC measurements with at least ten participants in both the PE and control groups were included in the analyses.

We also used data from a subset of individuals (N = 347, 207 females) from ALSPAC who have structural MRI scans acquired at age 20 (for details on sample characteristics and image acquisition see Supplementary Material and references; Drakesmith et al., 2016a, 2016b).

Head circumference

HC was measured in all ALSPAC cohort members at birth to 1 year, and at years 7 and 15. Additional measurements between the ages of 4 and 61 months (22 time points overall) were also collected from a subsample of this cohort (see Supplementary Table S1 and Supplementary Figure S1 for available sample size at each measurement time point). HC was collected at 22 time points; the maximum measurement time was 19 (n = 1) and the minimum 1 (n = 26). All 22 time points were included in the longitudinal modeling. For visualization purposes, plots display simplified integer labels.

HC measurements were collected by the ALSPAC team and supplemented by routine measurements by local health services. Outliers at each measurement point were identified using cut-offs from the World Health Organization (WHO) Child Growth Standards and removed (De Onis, 2006; Shi, Korsiak, & Roth, 2018).

Psychotic experiences and psychotic disorder

PE were assessed using the Psychosis-Like Symptom Interview (PLIKSi) (Niarchou, Zammit, & Lewis, 2015; Sullivan et al., 2020; Zammit et al., 2013), a semi-structured interview comprising 12 items relating to hallucinations (visual and auditory), delusions (spied on, persecution, grandiosity, thoughts read, reference, control, and other), and thought interference (broadcasting, insertion, and withdrawal). The interview was delivered by trained psychologists when the participant was aged 17/18 years. Participants were asked if they had ever had that experience since the age of 12, responding with either yes, no, or maybe. The interviewer followed up to determine if the experience met criteria for being psychotic, following definitions and guidelines from the Schedules for Clinical Assessment in Neuropsychiatry (Sullivan et al., 2020; WHO, 1994). Interviewers rated PE as either 'absent', 'suspected', or 'definitely present'. Unclear responses were 'rated down.'

Psychotic disorder was defined as having a 'definite psychotic experience' occurring at least once per month over the previous 6 months, which also causes severe distress, impacts social or occupational functioning, or led to help-seeking (Zammit et al., 2013).

Confounders

We included participant age, maternal body mass index (BMI), gestational age, birth weight, and maternal highest-level-of-education as confounders.

Data analysis

HC trajectories were modeled using linear mixed-effects regression, incorporating a random intercept on child and a random slope on time. We began by assessing the trajectory of HC over time by running two unconditional models; the first using mean-centered age in months (unconditional model 1) and the second using mean-centered age and age squared (unconditional model 2). If there was evidence of a non-linear association with age, we retained it in the final model. We also ran model fit parameters, Akaike Information Criterion and Bayesian Information Criterion (see also Supplementary Tables S3, S3A, and S3B).

Next, in the primary analysis, we applied a univariable model (model 1) incorporating our clinical variable of interest (psychotic disorder/control or PE/control), and the two age indicators (mean-centered age and age squared) while progressively adjusting for

https://doi.org/10.1017/S0033291726105418

pre-selected confounders. These included maternal education (model 2), maternal BMI (model 3), and gestational age and birth weight (model 4).

We explored the following interactions with group in three separate models: age, age and age squared, and sex. If there was a statistically significant interaction between group and sex, we presented findings stratified by sex.

Sensitivity analyses

To examine if findings are driven by interpolating across time gaps between measurements, or by participants with insufficient longitudinal data, we compared HC at birth, 7 years, and 15 years – time points with the highest number of participants – between case groups and controls.

Secondary analyses

Associations between HC and MRI-derived total intracranial volume (TIV) were examined using Pearson correlations. To test whether atypical HC trajectories may reflect general growth differences, we added height as a time-varying covariate in model 4. (See Supplementary Material, Methods section).

All analyses were conducted in Stata 17 (StataCorp, 2021).

Results

Of the 3,866 individuals in the analytic sample, 62 (1.6%) had a psychotic disorder (49 females), and 339 (8.8%) had PE (218 females). Demographic characteristics of the analytic sample in this study and the full ALSPAC cohort are presented in Supplementary Table S1.

Using a subsample with volumetric MRI measurements at age 20 we show that across a total of 347 participants (114 PE (78 females), of which 30 (23 female) were classified as psychotic disorder, and 233 controls (129 females)) who had neuroimaging data, there was a moderate correlation between TIV at age 20 years and HC at birth (N = 270; r = 0.45, p < 0.0001), and strong correlations with HC at ages 7 (N = 329; r = 0.77, p < 0.0001) and 15 years (N = 265; r = 0.77, p < 0.0001; Figure 1). Across the same age groups, the correlations between TIV and HC remained positive and statistically significant for both males (r = 0.55–0.79, p < 0.0001) and females (r = 0.41–0.74, p < 0.0001).

HC trajectories through infancy, childhood and adolescence and psychotic disorder

Table 1 shows the demographics and developmental characteristics of the psychotic disorder and PE samples and controls. Figure 2 illustrates measures of HC at birth and at ages 1, 2, 3, 5, 7 and 15 years in cohort members with psychotic disorder and controls, with associated model statistics listed in Table 2 and Supplementary Table S2.

The psychotic disorders group showed consistently smaller HC starting at birth and throughout infancy, childhood, and up to adolescence (B = −0.26, 95% CI: −0.47 to −0.04, p = 0.02). There was no statistically significant group by age interaction (p > 0.5), and no statistically significant interaction of group by sex (p = 0.2). Group differences were not attenuated in models that included adjustment for maternal education (B = −0.23, 95% CI: −0.45 to

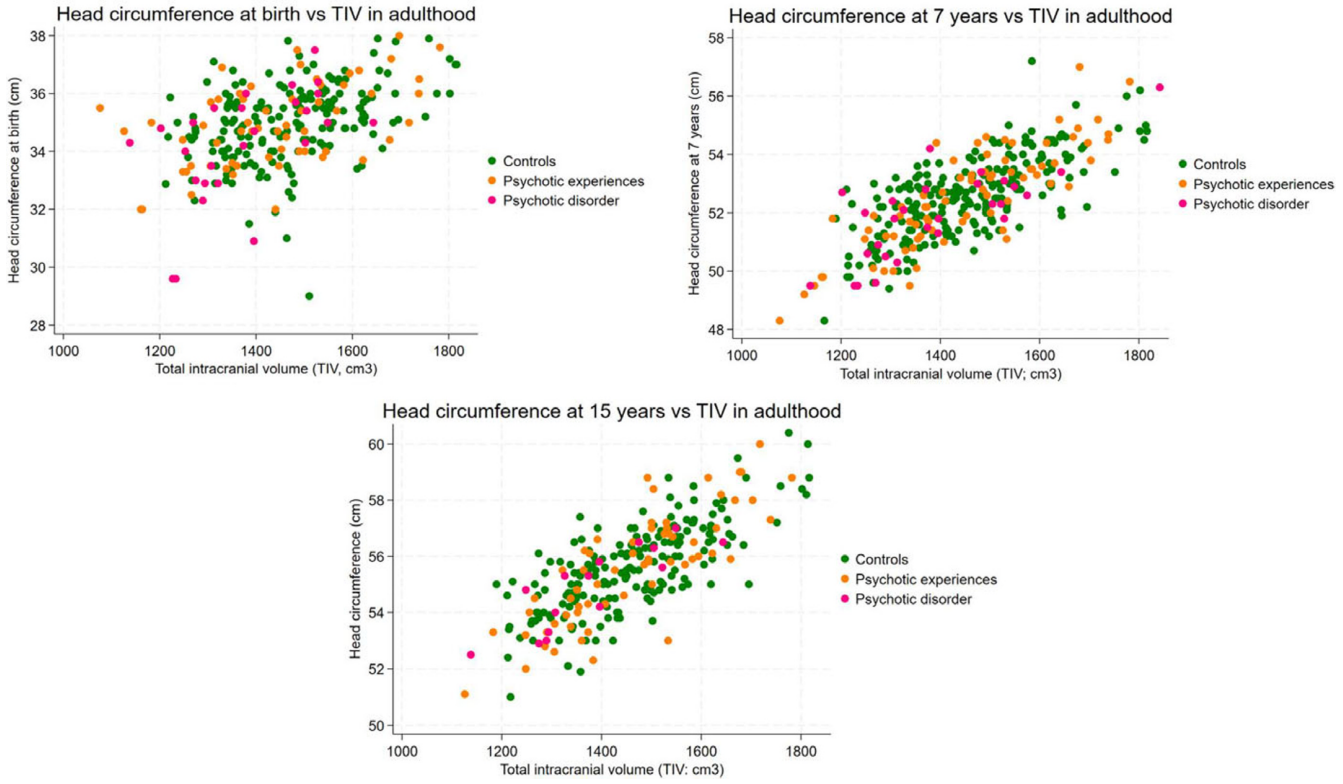

Figure 1. Scatterplot showing total intracranial volume (TIV, cm^3) data collected at age 20 years and its relationship with head circumference (cm) measured at birth (N = 270), 7 years (N = 329), 15 years (N = 265).

https://doi.org/10.1017/S0033291726105418

Table 1. Characteristics of the analytical sample, overall and by case/control status for psychotic experiences (PE) and psychotic disorder in females and males

	Female analytic sample	Controls	Psychotic experiences	Psychotic disorder	Male analytic sample	Controls	Psychotic experiences	Psychotic disorder
	N	*n* (%)	*n* (%)	*n* (%)	*N*	*n* (%)	*n* (%)	*n* (%)
Total	2,177	1,959 (89.99%)	218 (10.01%)	49 (2.25%)	1,689	1,568 (92.84%)	121 (7.16%)	13 (0.77%)
Ethnicity								
White	2,057	1,857	158	42	1,596	1,487	109	13
[a]*Non-white (plus missing)*	120	102	14	7	93	81	8	0
Maternal highest educational attainment								
Compulsory	1,189	1,037	113	39	831	767	56	8
Non-compulsory	988	922	56	6	858	801	52	5
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
Gestational age (weeks)	39.60 (1.65)	39.61 (1.66)	39.57 (1.59)	39.65 (2.18)	39.37 (1.86)	39.37 (1.87)	39.38 (1.74)	38.69 (1.44)
Birth weight (grams)	3393.34 (483.52)	3392.79 (478.30)	3398.35 (529.27)	3356.14 (683.61)	3484.18 (572.31)	3488.74 (576.18)	3425.18 (518.17)	3383.85 (450.47)
Length at birth (cm)	50.51 (2.24)	50.51 (2.19)	50.51 (2.66)	50.00 (2.87)	51.20 (2.51)	51.21 (2.51)	51.05 (2.58)	51.43 (2.43)
Height at 15.5 years (cm)	164.73 (6.02)	164.77 (5.97)	164.33 (6.46)	163.18 (7.62)	174.43 (7.54)	174.44 (7.48)	174.24 (8.29)	175.77 (9.43)
Maternal pre-pregnancy BMI	22.78 (3.68)	22.76 (3.67)	22.97 (3.84)	23.09 (4.12)	22.90 (3.76)	22.88 (3.73)	23.18 (4.21)	23.03 (3.34)
Average number of head circumference measurements	8.05 (4.12)	8.07 (4.12)	7.46 (3.83)	7.41 (4.00)	8.37 (4.24)	8.37 (4.24)	8.87 (4.55)	8.68 (5.05)

Note: PE, psychotic experiences at age 17/18.
[a]Non-white ethnicity and missing values were combined to avoid cells with <5 individuals as a requirement from ALSPAC to avoid potential breaches of anonymity. We collapsed maternal highest educational attainment into a binary variable to avoid breaches of anonymity, analyses used the non-collapsed version consisting of five levels of educational attainment (Compulsory i.e. CSE School leaving exam, Vocational, O level – National high school exam age 15/16; Non-compulsory i.e. A level – National high school exam age 17/18, University Degree).

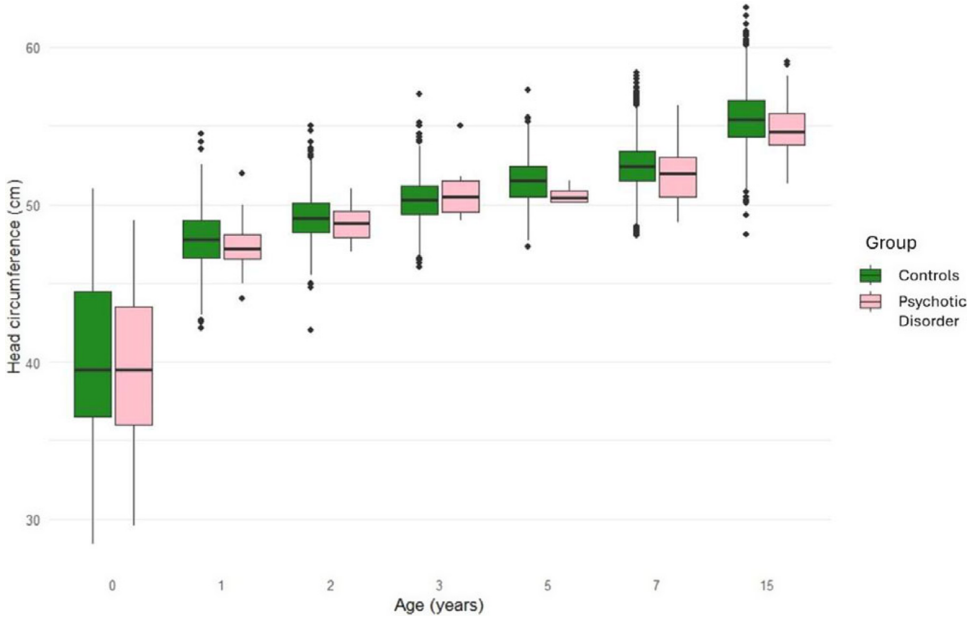

Figure 2. Head circumference trajectories (cm) in participants with and without psychotic disorder by age. The panel charts head circumference data using boxplots, and 95% confidence intervals.

https://doi.org/10.1017/S0033291726105418

Table 2. Univariable and multivariable linear mixed regressions assessing head circumference in psychotic disorder versus controls (Analytic sample *N* = 3,589, Psychotic Disorder *N* = 62, Controls *N* = 3,527)

	Beta coefficient	95% CI	*p*-value
Univariable model 1	−0.26	−0.47 to −0.04	0.02
Model 2: model 1 + maternal education	−0.23	−0.45 to −0.02	0.035
Model 3: model 2 + maternal BMI	−0.23	−0.44 to −0.02	0.035
Model 4: model 3 + gestational age and birth weight	−0.20	−0.41 to −0.003	0.054

Note: Unconditional Models 1 (mean-centered age) and 2 (model 1 + age squared) are presented in Supplementary Material. For univariable model 1 onwards, we incorporated random effects of participant id and mean-centered age. Univariable model 1 consisted of group status and time variables. Interactions between group with age ($p > 0.5$) and age squared ($p > 0.5$) were not statistically significant and therefore the group coefficient was not included in this table.

−0.02, $p = 0.035$), BMI ($B = -0.23$, 95% CI −0.44 to −0.02, $p = 0.035$), and gestational age and birth weight ($B = -0.20$, 95% CI: −0.41 to 0.003, $p = 0.054$).

Sensitivity analyses supported the main analyses. The psychotic disorders group had smaller HC at birth, and at 7 years, and 15 years of age, and the differences were statistically significant at the $p < 0.05$ level at ages 7 and 15 (Supplementary Table S4).

HC trajectories through infancy, childhood and adolescence and psychotic experiences

Because there was a statistically significant group by HC by sex interaction ($p = 0.02$), we conducted analyses stratified by sex. Figure 3 illustrate measures of HC at birth and at ages 1, 2, 3, 5, 7 and 15 years in cohort members with PE and controls, for males and females separately. Associated model statistics are listed in Table 3 and Supplementary Tables S3A and S3B. Females with PE had statistically significantly smaller HC compared to female controls across childhood and adolescence ($B = -0.27$, 95% CI: −0.49 to −0.05, $p = 0.016$). Group differences were not attenuated in models that included adjustment for maternal education ($B = -0.23$, 95% CI: −0.45 to −0.006, $p = 0.044$), BMI ($B = -0.22$, 95% CI −0.44 to −0.004, $p = 0.046$), and gestational age and birth weight ($B = -0.25$, 95% CI: −0.46 to 0.04, $p = 0.022$).

In contrast, males with PE had consistently larger HC across childhood and adolescence compared to male controls ($B = 0.35$, 95% CI: 0.05 to 0.65, $p = 0.023$). Group differences were not attenuated in models that included adjustment for maternal education ($B = 0.34$, 95% CI: 0.04 to 0.65 $p = 0.025$), BMI ($B = 0.33$, 95% CI: 0.04 to 0.64 $p = 0.029$), and gestational age and birth weight ($B = 0.40$, 95% CI: 0.12 to 0.69 $p = 0.006$).

In females, results of the sensitivity analyses were consistent with those of the main analyses (Supplementary Table S5). In males, however, there was smaller HC at birth, but larger HC at ages 7 and 15 (Supplementary Table S5).

HC and general growth trajectories

Adjustment for height, as a time-varying covariate, led to attenuation of group differences in HC trajectories between individuals with psychotic disorder and controls (−0.05, 95% CI: −0.21 to 0.11, $p = 0.52$), and in group differences in HC trajectories between females with and without PE ($B = -0.12$, 95% CI: −0.29 to 0.05, $p = 0.16$). In contrast, group differences remained statistically significant in males with PE ($B = 0.36$, 95% CI: 0.13 to 0.58, $p = 0.002$).

Discussion

Using a population-based cohort followed prospectively from birth, this study charts the course of HC from birth through to age 15 years in individuals with psychotic disorder and PE, and examines if differences in course represent a brain-specific process, or a more generalized growth disorder. Our findings advance knowledge in three important ways.

Individuals who grew up to have operationally defined psychotic disorder showed smaller HC starting at birth and throughout childhood and adolescence. HC is recorded in populations throughout the world, often from birth, and is generally assumed to be a proxy measure of brain size. Crucially, we show that HC correlates strongly and significantly with MRI measures of brain volume in early adulthood. In adulthood, schizophrenia is associated with reduced total brain volume (Haijma et al., 2013). Thus, we provide evidence for the first time in a population-based longitudinal study that neurodevelopmental brain abnormalities associated with psychotic disorders manifest already around birth and remain static and persistent throughout development.

We also compared trajectories of HC between individuals with the broader phenotype of PE versus controls. In our sample, females and males with PE showed diverging trajectories of HC. Females who had PE had smaller HC compared to female controls starting in childhood and through adolescence. In contrast, compared to male controls, males with PE had larger HC.

HC correlates with height (Martins & Lyons Jones, 1994; Perrin et al., 2007). Importantly, when controlling for height across development, the association between smaller HC and psychotic disorder, and in females, but not males, with PE, was attenuated, suggesting that general growth restriction, rather than brain growth specifically, may be dysregulated in psychosis, with potentially sex-specific modulation. The neurodevelopmental manifestations of the liability to the psychosis spectrum, as reflected in early-life head growth, may be qualitatively different in males and females with psychotic symptoms. Future studies with larger samples should verify this finding.

The mechanism linking growth and schizophrenia remains to be elucidated. However, the pattern of smaller height and general abnormal growth, coupled with evidence for female sex skewness of the association, suggests a possible role for insulin-like growth factor-I (IGF-I). This plays a major role in the regulation of both prenatal and postnatal growth (Le Roith, Scavo, & Butler, 2001) and shows patterns of sexual dimorphism with higher concentrations in females (Geary et al., 2003). Perinatal insults such as maternal infection, complications during childbirth, and prenatal and postnatal nutritional deficiencies are all linked to increased risk of psychotic disorders and reduced HC and growth, and may contribute to the observed associations (Davies et al., 2020; Hultman et al., 1999).

The study has several limitations. As with most longitudinal studies spanning infancy through adulthood, varying numbers of individuals were available at different ages. Therefore, while our sample was drawn from a well-characterized, population-based birth cohort, the results require replication in independent samples.

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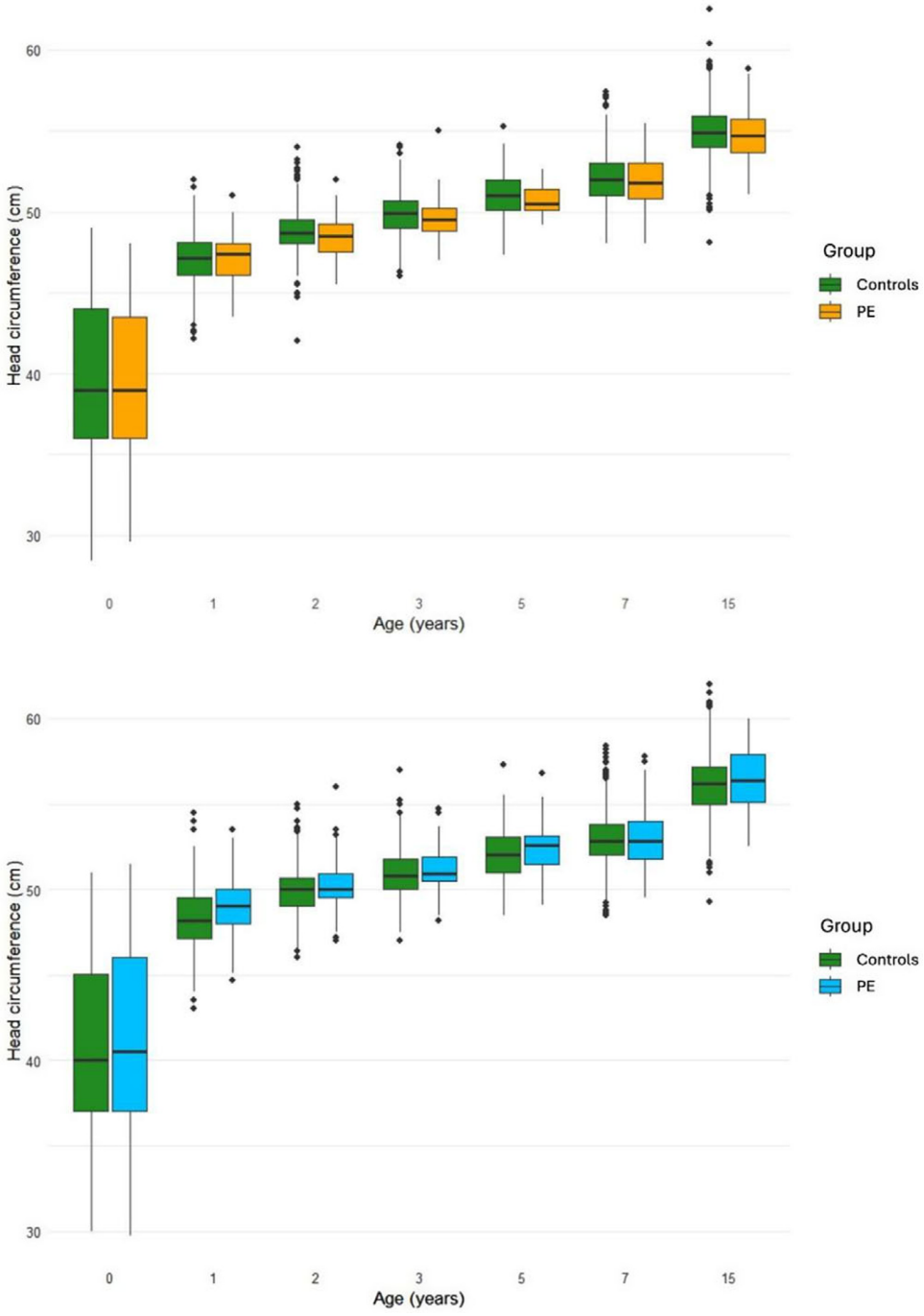

Figure 3. Head circumference trajectories (cm) in females, *N* = 2,177 (Top) and males, *N* = 1,689 (Bottom) with and without psychotic experiences (PE). Head circumference is modeled with 95% confidence intervals. We present age in years using integer values.

The number of individuals meeting our operational case definition of psychotic disorder was small and did not entail independent diagnosis by a healthcare provider. There were few males with psychotic disorder in this sample (n = 13), so the findings based on these individuals must be interpreted with caution. In ALSPAC, both PE and psychotic disorder were more prevalent in females. While large epidemiological studies report higher prevalence of PE in males during childhood (Karcher et al., 2022), in adolescence and adulthood this shifts, and females have up to 50% higher prevalence of PE (Johns et al., 2004; McGrath et al., 2015). We adjusted for birth weight rather than a cumulative measure of obstetric complications in our analyses. This was done because birth weight is an overall robust indicator of intrauterine health, development, and infant survival. It is related to maternal lifestyle factors and cardiometabolic health, as well as captures familial factors related to intrauterine development. In addition, information about birth weight in ALSPAC is almost complete. In contrast, there is a high (~30%) rate of missing data for information on individual obstetric complications (Merritt et al., 2023).

In sum, this study provides novel evidence of a deviation in HC trajectories that persists from birth to adolescence in young people

https://doi.org/10.1017/S0033291726105418

Table 3. Head circumference trajectories in females and males with psychotic experiences (PE) versus Controls

	Females			Males		
	Beta coefficient	95% CI	*p*-value	Beta coefficient	95% CI	*p*-value
Univariable model 1	−0.27	−0.49 to −0.05	0.016	0.35	0.05 to 0.65	0.023
Model 2: model 1 + maternal education	−0.23	−0.45 to −0.006	0.044	0.34	0.04 to 0.65	0.025
Model 3: model 2 + maternal BMI	−0.22	−0.44 to −0.004	0.046	0.33	0.03 to 0.64	0.029
Model 4: model 3 + gestational age and birth weight	−0.25	−0.46 to −0.04	0.022	0.40	0.12 to 0.69	0.006

Note: Unconditional Model 1 (age) and Model 2 (mean-centered age and age squared) are presented in Supplementary Material. For univariable model 1 onwards, we incorporated random effects of participant id and mean-centered age. Univariable model 1 consisted of PE and time variables. Stratified interactions between PE and age were non-significant (all *p* values >0.5) and therefore the group coefficient was not included in this table. Presented are results of linear mixed regressions assessing head circumference in PE versus controls, stratified by sex (Analytic samples: Females: *N* = 2,177, PE = 218, Controls = 1,959; Males: *N* = 1689, PE = 121, Controls = 1,568).

with psychotic disorders and more broadly, PE. This deviation in HC is potentially part of a more general abnormality in growth across the psychosis spectrum, supporting a neurodevelopmental origin of schizophrenia. Future studies should examine factors regulating growth during early life, including sex, to understand the molecular mechanisms relating to abnormal development and psychosis.

Supplementary material. The supplementary material for this article can be found at http://doi.org/10.1017/S0033291726105418.

Acknowledgments. The United Kingdom Medical Research Council, the Wellcome Trust, and the University of Bristol provide core support for the Avon Longitudinal Study of Parents and Children (ALSPAC). This research was specifically funded by Medical Research Council grant MR/S003436/1. Dr Ashley was supported by the Beatrice and Samuel A Seaver Foundation PhD Studentship.

Author contribution. We are grateful to all the families who took part in this study, the midwives for their help in recruiting them, and the whole ALSPAC team, which includes interviewers, computer and laboratory technicians, clerical workers, research scientists, volunteers, managers, receptionists, and nurses.

Funding statement. The funding sources had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; preparation, review, or approval of the manuscript; and decision to submit the manuscript for publication.

Competing interests. The authors report no conflicts of interests.

References

Bartholomeusz, H. H., Courchesne, E., & Karns, C. M. (2002). Relationship between head circumference and brain volume in healthy normal toddlers, children, and adults. *Neuropediatrics*, **33**(5), 239–241. https://doi.org/10.1055/s-2002-36735

Boyd, A., Golding, J., Macleod, J., Lawlor, D. A., Fraser, A., Henderson, J., Molloy, L., Ness, A., Ring, S., & Smith, G. D. (2013). Cohort profile: The 'children of the 90s'-the index offspring of the Avon longitudinal study of parents and children. *International Journal of Epidemiology*, **42**(1), 111–127. https://doi.org/10.1093/ije/dys064

Brown, A. S., Gyllenberg, D., Hinkka-Yli-Salomäki, S., Sourander, A., & McKeague, I. W. (2017). Altered growth trajectory of head circumference during infancy and schizophrenia in a National Birth Cohort. *Schizophrenia Research*, **182**, 115–119. https://doi.org/10.1016/j.schres.2016.10.032

Burchard, E. (1916). *Physique and psychosis: An analysis of the postulated relationship between bodily constitution and mental disease syndrome*. John Hopkins Press. Comprehensive Psychological Monographs. Vol. 73. (Ref Type: Serial, Book, Monograph).

Cannon, M., Caspi, A., Moffitt, T. E., Harrington, H., Taylor, A., Murray, R. M., & Poulton, R. (2002a). Evidence for early-childhood, pan-developmental impairment specific to schizophreniform disorder: Results from a longitudinal birth cohort. *Archives of General Psychiatry*, **59**(5), 449–456. https://doi.org/10.1001/ARCHPSYC.59.5.449

Cannon, M., Jones, P. B., & Murray, R. M. (2002b). Obstetric complications and schizophrenia: Historical and meta-analytic review. *American Journal of Psychiatry*, **159**(7), 1080–1092. https://doi.org/10.1176/appi.ajp.159.7.1080

Cantor-Graae, E., Ismail, B., & McNeil, T. F. (1998). Neonatal head circumference and related indices of disturbed fetal development in schizophrenic patients. *Schizophrenia Research*, **32**(3), 191–199. https://doi.org/10.1016/S0920-9964(98)00060-7

Davies, C., Segre, G., Estradé, A., Radua, J., De Micheli, A., Provenzani, U., Oliver, D., de Pablo, G., Ramella-Cravaro, V., Besozzi, M., Dazzan, P., Miele, M., Caputo, G., Spallarossa, C., Crossland, G., Ilyas, A., Spada, G., Politi, P., Murray, R. M., … Fusar-Poli, P. (2020). Prenatal and perinatal risk and protective factors for psychosis: A systematic review and meta-analysis. *The Lancet Psychiatry*, **7**(5), 399–410. https://doi.org/10.1016/S2215-0366(20)30057-2

De Onis, M. (2006). WHO child growth standards based on length/height, weight and age. *Acta Paediatrica*, **95**(Suppl 450), 76–85. https://doi.org/10.1111/j.1651-2227.2006.tb02378.x

Drakesmith, M., Dutt, A., Fonville, L., Zammit, S., Reichenberg, A., Evans, C. J., Lewis, G., Jones, D. K., & David, A. S. (2016a). Mediation of developmental risk factors for psychosis by white matter microstructure in young adults with psychotic experiences. *JAMA Psychiatry*, **73**(4), 396–406. https://doi.org/10.1001/jamapsychiatry.2015.3375

Drakesmith, M., Dutt, A., Fonville, L., Zammit, S., Reichenberg, A., Evans, C. J., McGuire, P., Lewis, G., Jones, D. K., & David, A. S. (2016b). Volumetric, relaxometric and diffusometric correlates of psychotic experiences in a non-clinical sample of young adults. *NeuroImage: Clinical*, **12**, 550–558. https://doi.org/10.1016/j.nicl.2016.09.002

Fraser, A., Macdonald-wallis, C., Tilling, K., Boyd, A., Golding, J., Davey smith, G., Henderson, J., Macleod, J., Molloy, L., Ness, A., Ring, S., Nelson, S. M., & Lawlor, D. A. (2013). Cohort profile: The Avon longitudinal study of parents and children: ALSPAC Mothers cohort. *International Journal of Epidemiology*, **42**(1), 97–110. https://doi.org/10.1093/IJE/DYS066

Geary, M. P. P., Pringle, P. J., Rodeck, C. H., Kingdom, J. C. P., & Hindmarsh, P. C. (2003). Sexual dimorphism in the growth hormone and insulin-like growth factor axis at birth. *Journal of Clinical Endocrinology and Metabolism*, **88**(8), 3708–3714. https://doi.org/10.1210/JC.2002-022006

Gunnell, D., Harrison, G., Whitley, E., Lewis, G., Tynelius, P., & Rasmussen, F. (2005). The association of fetal and childhood growth with risk of schizophrenia. Cohort study of 720,000 Swedish men and women. *Schizophrenia Research*, **79**(2–3), 315–322. https://doi.org/10.1016/j.schres.2005.07.022

Haijma, S. V., Haren, N. V., Cahn, W., Cédric, P., Koolschijn, M. P., Hulshoff Pol, H. E., & Kahn, R. S. (2013). Brain volumes in schizophrenia: A meta-analysis in over 18 000 subjects. *Schizophrenia Bulletin*, **39**(5), 1129–1138. https://doi.org/10.1093/schbul/sbs118

Hshieh, T. T., Fox, M. L., Kosar, C. M., Cavallari, M., Guttmann, C. R. G., Alsop, D., Marcantonio, E. R., Schmitt, E. M., Jones, R. N., & Inouye, S. K. (2016). Head circumference as a useful surrogate for intracranial volume in older adults. *International Psychogeriatrics*, **28**(1), 157–162. https://doi.org/10.1017/S104161021500037X

Hultman, C. M., Ohman, A., Cnattingius, S., Wieselgren, I. M., & Lindstrom, L. H. (1997). Prenatal and neonatal risk factors for schizophrenia. *British Journal of Psychiatry*, **170**(2), 128–133. https://doi.org/10.1192/BJP.170.2.128

Hultman, C. M., Sparén, P., Takei, N., Murray, R. M., & Cnattingius, S. (1999). Prenatal and perinatal risk factors for schizophrenia, affective psychosis, and reactive psychosis of early onset: Case-control study. *BMJ*, **318**(7181), 421–426. https://doi.org/10.1136/bmj.318.7181.421

Johns, L. C., Cannon, M., Singleton, N., Murray, R. M., Farrell, M., Brugha, T., Bebbington, P., Jenkins, R., & Meltzer, H. (2004). Prevalence and correlates of self-reported psychotic symptoms in the British population. *British Journal of Psychiatry*, **185**(4), 298–305. https://doi.org/10.1192/BJP.185.4.298

Karcher, N. R., Paul, S. E., Johnson, E. C., Hatoum, A. S., Baranger, D. A. A., Agrawal, A., Thompson, W. K., Barch, D. M., & Bogdan, R. (2022). Psychotic-like experiences and polygenic liability in the adolescent brain cognitive development study. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, **7**(1), 45–55. https://doi.org/10.1016/j.bpsc.2021.06.012

Kemali, D., Polani, N., Polani, P. E., & Amati, A. (1976). A dermatoglyphic study of 219 Italian schizophrenic males. *Clinical Genetics*, **9**(1), 51–60. https://doi.org/10.1111/J.1399-0004.1976.TB01549.X

Kunugi, H., Takei, N., Murray, R. M., Saito, K., & Nanko, S. (1996). Small head circumference at birth in schizophrenia. *Schizophrenia Research*, **20**(1–2), 165–170. https://doi.org/10.1016/0920-9964(96)00007-2

Latham, K., & Kirkpatrick, B. (2018). Meta-analysis of adult height and birth length in schizophrenia. *Schizophrenia Research*, **195**, 110–114. https://doi.org/10.1016/J.SCHRES.2017.09.002

Le Roith, D., Scavo, L., & Butler, A. (2001). What is the role of circulating IGF-I? *Trends in Endocrinology and Metabolism*, **12**(2), 48–52. https://doi.org/10.1016/s1043-2760(00)00349-0

Martins, A., & Lyons Jones, K. (1994). Correlation of occipitofrontal circumference and crown-rump length from birth to 15 months. *Clinical Dysmorpology*, **2**, 157–159. https://pubmed.ncbi.nlm.nih.gov/8055136/

McGrath, J. J., Saha, S., Al-Hamzawi, A., Alonso, J., Bromet, E. J., Bruffaerts, R., Caldas-De-Almeida, J. M., Chiu, W. T., De Jonge, P., Fayyad, J., Florescu, S., Gureje, O., Haro, J. M., Hu, C., Kovess-Masfety, V., Lepine, J. P., Lim, C. C. W., Mora, M. E. M., Navarro-Mateu, F., … Kessler, R. C. (2015). Psychotic experiences in the general population: A cross-National Analysis Based on 31,261 respondents from 18 countries. *JAMA Psychiatry*, **72**(7), 697–705. https://doi.org/10.1001/JAMAPSYCHIATRY.2015.0575

Merritt, K., Luque Laguna, P., Sethi, A., Drakesmith, M., Ashley, S. A., Bloomfield, M., Fonville, L., Perry, G., Lancaster, T., Dimitriadis, S. I., Zammit, S., Evans, C. J., Lewis, G., Kempton, M. J., Linden, D. E. J., Reichenberg, A., Jones, D. K., & David, A. S. (2023). The impact of cumulative obstetric complications and childhood trauma on brain volume in young people with psychotic experiences. *Molecular Psychiatry*, **28**(9), 3688–3697. https://doi.org/10.1038/s41380-023-02295-6

Mollon, J., David, A. S., Zammit, S., Lewis, G., & Reichenberg, A. (2018). Course of cognitive development from infancy to early adulthood in the psychosis Spectrum. *JAMA Psychiatry*, **75**(3), 270–279. https://doi.org/10.1001/JAMAPSYCHIATRY.2017.4327

Murray, R. M., Lewis, S. W., & Lecturer, L. (1987). Is schizophrenia a neurodevelopmental disorder? *British Medical Journal (Clinical Research Ed.)*, **295**(6600), 681–682. https://doi.org/10.1136/BMJ.295.6600.681

Niarchou, M., Zammit, S., & Lewis, G. (2015). The Avon longitudinal study of parents and children (ALSPAC) birth cohort as a resource for studying psychopathology in childhood and adolescence: A summary of findings for depression and psychosis. *Social Psychiatry and Psychiatric Epidemiology*, **50**(7), 1017–1027. https://doi.org/10.1007/S00127-015-1072-8

Nilsson, E., Stålberg, G., Lichtenstein, P., Cnattingius, S., Olausson, P. O., & Hultman, C. M. (2005). Fetal growth restriction and schizophrenia: A Swedish twin study. *Twin Research and Human Genetics*, **8**(4), 402–408. https://doi.org/10.1375/twin.8.4.402

Perrin, M. A., Chen, H., Sandberg, D. E., Malaspina, D., & Brown, A. S. (2007). Growth trajectory during early life and risk of adult schizophrenia. *The British Journal of Psychiatry*, **191**(6), 512–520. https://doi.org/10.1192/BJP.BP.106.034694

Robinson, N., Ploner, A., Leone, M., Lichtenstein, P., Kendler, K. S., & Bergen, S. E. (2023). Impact of early-life factors on risk for schizophrenia and bipolar disorder. *Schizophrenia Bulletin*, **49**(3), 768–777. https://doi.org/10.1093/SCHBUL/SBAC205

Shi, J., Korsiak, J., & Roth, D. E. (2018). New approach for the identification of implausible values and outliers in longitudinal childhood anthropometric data. *Annals of Epidemiology*, **28**(3), 204–211. https://doi.org/10.1016/j.annepidem.2018.01.007

StataCorp. (2021). *Stata statistical software: Release 17*. StataCorp LLC.

Sullivan, S. A., Kounali, D., Cannon, M., David, A. S., Fletcher, P. C., Holmans, P., Jones, H., Jones, P. B., Linden, D. E. J., Lewis, G., Owen, M. J., O'Donovan, M., Rammos, A., Thompson, A., Wolke, D., Heron, J., & Zammit, S. (2020). A population-based cohort study examining the incidence and impact of psychotic experiences from childhood to adulthood, and prediction of psychotic disorder. *American Journal of Psychiatry*, **177**(4), 308–317. https://doi.org/10.1176/appi.ajp.2019.19060654

Wahlbeck, K., Forsén, T., Osmond, C., Barker, D. J. P., & Eriksson, J. G. (2001). Association of schizophrenia with low maternal body mass index, small size at birth, and thinness during childhood. *Archives of General Psychiatry*, **58**(1), 48–52. https://doi.org/10.1001/ARCHPSYC.58.1.48

Welham, J., Scott, J., Williams, G., Najman, J., O'Callaghan, M., & McGrath, J. (2009). Growth in young adults who screen positive for non-affective psychosis: Birth cohort study. *Australian & New Zealand Journal of Psychiatry*, **43**(1), 61–67. https://doi.org/10.1080/00048670802534333

World Health Organization (WHO). (1994). *Schedules for Clinical Assessment in Neuropsychiatry*. American Psychiatric Press (on behalf of WHO).

Zammit, S., Kounali, D., Cannon, M., David, A. S., Gunnell, D., Heron, J., Jones, P. B., Lewis, S., Sullivan, S., Wolke, D., & Lewis, G. (2013). Psychotic experiences and psychotic disorders at age 18 in relation to psychotic experiences at age 12 in a longitudinal population-based cohort study. *American Journal of Psychiatry*, **170**(7), 742–750. https://doi.org/10.1176/appi.ajp.2013.12060768

Zammit, S., Rasmussen, F., Farahmand, B., Gunnell, D., Lewis, G., Tynelius, P., & Brobert, G. P. (2007). Height and body mass index in young adulthood and risk of schizophrenia: A longitudinal study of 1 347 520 Swedish men. *Acta Psychiatrica Scandinavica*, **116**(5), 378–385. https://doi.org/10.1111/J.1600-0447.2007.01063.X