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Different sensory dimensions in infancy are associated with separable etiological influences and with autistic traits in toddlerhood

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Background: Infants vary significantly in the way they process and respond to sensory stimuli, and altered sensory processing has been reported among infants later diagnosed with autism. Previous work with adolescents and adults suggests that variability in sensory processing may have a strong genetic basis. Yet, little is known about the etiological factors influencing sensory differences in infancy, when brain circuits supporting social and non-social cognition are sculpted and learning about the world via sensory input largely occurs in interaction with caregivers. **Methods:** We analysed data from a community sample of monozygotic (MZ) and dizygotic (DZ) 5-month-old same-sex twins (n = 285 pairs, n = 158 MZ pairs, n = 150 male pairs) from the BabyTwins Study in Sweden (BATSS) using exploratory factor analysis, generalised estimating equations and multivariate twin models to delineate the phenotypic and etiological structure of individual variability across different sensory processing dimensions, as measured by the Infant/Toddler Sensory Profile. Developmental links to later autistic traits were also assessed, as measured by total scores from the Quantitative Checklist for Autism in Toddlers at 36 months. **Results:** Results suggested separability between sensory processing dimensions (i.e. sensation seeking, sensation avoiding, sensory sensitivity and low registration) at a phenotypic and etiological level, with significant contributions from additive genetics and family environment that were unique to each sensory dimension and significant but smaller contributions from shared influences. Sensory domains also showed etiological separability, with unique genetic influences to each domain, while contributions from shared environment were in part shared across domains. A higher incidence of tactile-related behaviours and behaviours associated with sensory sensitivity, sensation avoiding, and low registration were significantly associated with higher levels of autistic traits in toddlerhood. **Conclusions:** This study provides a map of the phenotypic and etiological structure of sensory processing in infancy, which will be informative for studies of both typical and atypical development. **Keywords:** Sensory processing; infancy; autistic traits; etiological structure; multivariate; twin study.

Introduction

Sensory processing refers to the ability to organise incoming sensory information from the surrounding environment and generate behavioural responses accordingly. Understanding a child's sensory processing profile in a critical developmental stage as early in infancy could be fundamental to understand developmental cascades associated with neurodevelopmental outcome. According to an influential framework for sensory processing (Dunn, 1997), a child's ability to adapt to environmental demands depends on the interaction between two orthogonal dimensions: neurological thresholds and self-regulatory behaviour. Neurological thresholds correspond to perceptual sensitivity and indicate the amount or intensity of stimulation required to detect and discriminate between stimuli. Self-regulatory behaviour corresponds to behavioural reactivity to sensory stimuli in relation to sensory-related needs. Children who actively seek to adjust sensory stimulation according to their neurological threshold are considered active respondents, while others are considered passive respondents. The Infant/Toddler Sensory Profile (ITSP; Dunn, 2002) is a standardised parent/caregiver questionnaire measuring a child's sensory reactivity in daily life according to this framework. Despite being largely used in research and clinical settings (Muhlenhaupt, 2005), there is surprisingly scarce independent empirical testing on the psychometric properties of the ITSP instrument. This holds in particular for infants younger than 6 months of age, who were excluded from original assessments of test–retest reliability (reported as 0.74 after 2-to-3 weeks for infants older than 7 months of age) and internal structure validation (Dunn & Daniels, 2002).

Individual variability in sensory processing is present across the neurodevelopmental spectrum and can already be observed in infancy (DeGangi, 2017). Altered sensory processing is currently included among diagnostic criteria for Autism Spectrum Disorder (hereafter referred to as autism), a complex neurodevelopmental condition characterised by two core symptom domains: socio-communicative difficulties and narrow and

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focused patterns of interests or behaviours (the latter including altered reactions to the sensory environment; *Diagnostic and Statistical Manual of Mental Disorders*, 2013). Differences in sensory processing can vary widely across autistic individuals and across the lifespan, yet our understanding of the mechanisms underlying this variability and developmental impacts remains limited.

Although there is relatively little behavioural genetics work on sensory processing, available studies indicate a strong genetic basis, with up to 47% heritability estimates for sensory sensitivity in adolescence (Assary, Zavos, Krapohl, Keers, & Pluess, 2021; Oniszczenko et al., 2003), and genetic factors contributing to the stability of these traits across the lifespan (Kandler, Riemann, & Angleitner, 2013; Schmidt et al., 2013). Similarly, evidence for genetic influence on sensory sensitivity (Goldsmith, Buss, & Lemery, 1997) and sensory defensiveness (Goldsmith, Van Hulle, Arneson, Schreiber, & Gernsbacher, 2006) was found in toddlerhood. Evidence for shared genetic influences between sensory reactivity and autistic traits was also found in 9-to-12-year-old twins (i.e. 60% genetic correlation; Taylor et al., 2018). Yet, no previous work has investigated etiological influences on variability in sensory processing early in infancy. More knowledge about the etiological influences on the association between infants' sensory processing and later autistic traits could provide new leads on the developmental cascades underlying the development of autism.

The main aim of this study was to investigate the structure of genetic and environmental influences on variability across different sensory dimensions early in infancy, and to what extent this variability and its etiological influences link to later autistic traits. For that, we used a two-step approach to validate the structure of infant sensory dimensions (step 1) before testing etiological influences through twin modeling (step 2). In particular, this study investigated individual variability in sensory processing early in infancy by: (1) testing the hypothesized 4-dimensional structure of sensory processing in infancy; (2) investigating genetic and environmental influences on individual variability in early sensory processing; (3) testing to what extent these influences are shared or unique to different aspects of sensory processing; (4) investigating phenotypic and etiological associations between sensory processing in infancy and autistic traits in toddlerhood.

Methods

Participants

Participants in the study were recruited for the BabyTwins Study Sweden (BATSS; Falck-Ytter et al., 2021) in the greater Stockholm area from the Swedish population registry, for a total of 622 same-sex twins (311 pairs). Zygosity was estimated based on DNA sampled from saliva (see Falck-Ytter et al., 2021).

General inclusion criteria for the study ensured that the infants (i) were part of same-sex twin pairs, lived together, (ii) were aged between 5 and 6 months, (iii) had at least one parent speaking the testing language at home, (iv) provided detailed information about delivery, medical and psychiatric history, and basic demographic information from both biological parents, (v) lived with at least one biological parent. General exclusion criteria were: (i) diagnosis of epilepsy or history of fits/convulsions, (ii) known presence of a genetic syndrome related to autism, (iii) known presence of significant uncorrected vision or hearing impairment, (iv) premature birth prior to week 34, (v) presence of a developmental or medical condition likely to affect brain development (e.g. Cerebral Palsy), (vi) presence of twin-to-twin transfusion syndrome, (vii) birth weight below 1.5 kg.

Twenty-eight infants were excluded from the study sample based on general exclusion criteria, and thirty-four infants were excluded because data were deemed not valid due to excessive unanswered items (i.e. above 95th percentile). The final sample consisted of 560 infants (275 complete pairs, 153 monozygotic, and 10 incomplete pairs, 5 monozygotic; see Table 1 for demographics).

Measures of sensory processing

Sensory processing was assessed at 5 months of age through the ITSP (Dunn, 2002, version 1 adapted to Swedish). The parent/caregiver rates the frequency of the infant's behaviour on 36 items according to a 5-point Likert-type scale going from 1 (*almost always*) to 5 (*almost never*). Scoring was then reverse-coded in line with the Sensory Profile 2 (Licciardi & Brown, 2023), and items rated by parents as 'Not Applicable' were scored as 0.

The 0-to-6 months version of the ITSP tests the infant's reactivity within the following sensory domains (i.e. ITSP Sections): auditory, visual, tactile, oral, vestibular and general sensory processing. Items from the different Sections are then grouped into sensory dimensions (i.e. ITSP Quadrants; Dunn, 2002): low registration (i.e. high sensory threshold with passive responsive strategies); sensation seeking (i.e. high sensory threshold with active responsive strategies); sensory sensitivity (i.e. low sensory threshold with passive responsive strategies); sensation avoiding (i.e. low sensory threshold with active responsive strategies).

Autistic traits

Level of autistic traits was assessed at 36 months of age through the Quantitative Checklist for Autism in Toddlers (Q-CHAT; Allison et al., 2008), which consists of 25 parent-rated items coding the frequency of autism-related behaviours on a 5-point Likert-type scale going from *always* to *never*, with half the items being reverse-scored based on assessed behaviour. The total score from the Q-CHAT provides a quantitative measure of autistic traits, with higher scores indicating more autistic traits.

Statistical analyses

The analysis plan was pre-registered in the Open Science Framework (see https://osf.io/wx5fz) after data collection but prior to data analysis.

ITSP structure validation. Internal structure of the ITSP was tested through exploratory factor analysis (EFA) on item-level scores (Watkins, 2018) as a first validation step before moving forward with twin modeling. Analyses were

Table 1 Sample description

Zygosity		DZ (n = 249)	MZ (n = 311)	p
Age (days)	Mean (*SD*)	167.7 (9.0)	167.5 (8.6)	.806
Sex (n)	Female	123	145	.570
	Male	126	166	
Gestation age (days)	Mean (*SD*)	260.8 (7.8)	257.9 (7.8)	<.001
Mean parental education	Primary school	7 (2.8)	7 (2.3)	.480
	Secondary school	22 (8.8)	40 (12.9)	
	Tertiary – Undergraduate	122 (49.0)	143 (46.0)	
	Tertiary – Postgraduate	98 (39.4)	121 (38.9)	
Family income	<20,000 SEK	2 (0.8)	4 (1.3)	.176
	>100,000 SEK	44 (17.7)	36 (11.6)	
	20–30,000 SEK	6 (2.4)	10 (3.2)	
	30–40,000 SEK	18 (7.2)	16 (5.1)	
	40–50,000 SEK	23 (9.2)	43 (13.8)	
	50–60,000 SEK	26 (10.4)	30 (9.6)	
	60–70,000 SEK	36 (14.5)	43 (13.8)	
	70–80,000 SEK	33 (13.3)	38 (12.2)	
	80–90,000 SEK	28 (11.2)	57 (18.3)	
	90–100,000 SEK	24 (9.6)	26 (8.4)	
	Unknown	1 (0.4)	4 (1.3)	
	(Missing)	8 (3.2)	4 (1.3)	
Mean parental age (years)	Mean (*SD*)	35.4 (5.0)	35.1 (4.8)	.459
ITSP Auditory Section	Mean (*SD*)	21.2 (3.5)	20.7 (3.3)	.065
ITSP Visual Section	Mean (*SD*)	17.4 (3.7)	17.2 (3.3)	.390
ITSP Tactile Section	Mean (*SD*)	11.4 (3.4)	12.0 (3.3)	.047
ITSP Vestibular Section	Mean (*SD*)	19.3 (3.0)	19.3 (3.4)	.953
ITSP General Section	Mean (*SD*)	15.0 (2.9)	15.2 (2.9)	.399
ITSP Low Registration	Mean (*SD*)	27.4 (5.4)	27.5 (5.2)	.819
ITSP Sensation Seeking	Mean (*SD*)	25.6 (3.4)	25.1 (3.7)	.108
ITSP Sensation Avoiding	Mean (*SD*)	8.5 (2.9)	8.9 (2.9)	.124
ITSP Sensory Sensitivity	Mean (*SD*)	22.8 (5.9)	22.8 (5.4)	.945
QCHAT Total Score	Mean (*SD*)	21.3 (7.6)	22.1 (7.6)	.176

Continuous data are reported as *mean (standard deviation)* and uncorrected p-value on differences across the sample stratified by zygosity. DZ, dizygotic twin; ITSP, Infant/Toddler Sensory Profile; MZ, monozygotic twin; QCHAT, Quantitative Checklist for Autism in Toddlerhood.

performed selecting 1 twin from each pair to avoid cluster-level dependency in the data. Item scores were treated as approximately continuous given the 6 scoring levels and given that the assumption of equal distance between two neighboring levels is most likely to hold for monotonic ordinal values on a Likert scale.

Data factorability was tested through the Kaiser-Meyer-Olkin factor adequacy score (KMO) computed on the correlation matrix and through Bartlett's test for sphericity (Bartlett, 1954; Dziuban & Shirkey, 1974). Items were then selected for inclusion in the EFA model based on the criterion KMO >0.6 (Kaiser, 1974). The number of factors was determined largely from visual inspection of a scree plot and with support from parallel analysis conducted through principal axis factoring with n = 100 data simulations and the conventional Kaiser criterion (eigenvalue = 1) as reference criterion (Cattell, 1966). However, meaningfulness and interpretability of factors were used as more conservative criteria to select the number of factors (n = 4). Next, factors were extracted and rotated with oblique rotation (i.e. *oblimin*; Jennrich & Sampson, 1966) to limit bias in subsequent multivariate analyses that may be induced by factor orthogonality. Factors were validated based on internal consistency, assessed through McDonald's omega reliability scores (McDonald, 1999), item-level factor loadings, and correlation with Quadrant scores (Gorsuch, 2013). Fitting of the identified model solution was further tested through confirmatory factor analysis on the sample obtained from the other twin (Johnston, 2014) and implemented in *R* using the package *lavaan* (Rosseel, 2012).

Etiological structure. Etiological structure was tested through twin modelling for ITSP Quadrant and Section scores, and the estimated latent *factors*. Here, we report the general approach used on the three different sets of scores separately (now referred to as *sensory processing scores*).

First, we tested for significant associations between sensory processing scores and potentially relevant covariates, that is, age, sex, parental age, parental education, family income and gestational age. This was done to account for potential confounding effects induced by demographic, parental characteristics and socio-economic differences known to be relevant for twin analyses. We used the robust sandwich estimator in generalised estimating equations (GEE) to account for correlations between twins in a pair (Carlin et al., 2005). Sensory scores were residualized by the covariates found to be significant after Bonferroni correction for multiple comparisons (based on six covariates tested: $\alpha = .05/6 = .008$).

Next, we used univariate twin models to test the validity of assumptions of equality of means and variances across zygosity and twin order for each sensory processing score, transformed to ensure skewness lower than 0.3, then residualized by significant covariates (see 'Results' for details) and scaled. Based on the observation of twin correlations, we selected an ACE model to decompose covariance among scales into additive genetics (A), shared environment (C) and unique environment factors (E). Multivariate twin models were used to decompose the observed variance/covariance matrix into genetic and environmental latent factors, allowing for a more

articulated understanding of the underlying etiological structure across traits. Of note, the General Section score (assessing more overarching behavioural patterns, like reaction to change in routines) was not included in our multivariate analysis to avoid bias in the estimated structural model due to the cross-domain nature of general processing behaviours.

Different structural solutions were tested by comparing three different multivariate models in order of decreasing complexity: the correlated factors model, the independent pathway model, and the common pathway model (Rijsdijk, 2002). In the correlated factors model, an ACE model is fitted to each measure, and then estimates are computed for the degree to which A, C and E correlate between the different traits. In the independent pathway model, A-C-E influences are separated between common influences (shared across different traits) and unique influences; yet, the measures are still treated as distinct phenotypes. In the common pathway model, a latent factor is modelled to explain shared variance across traits. Next, A-C-E influences are separated between common influences acting through the latent factor and unique influences on the remaining unexplained variance for each trait. Model fit was compared on Bayesian Information Criterion (BIC) for selection of model solution based on the lowest BIC value and non-significant difference from the saturated model, indexed by the χ^2 distribution. Data analysis was performed in *R 4.1.2* (R Core Team, 2010), and twin model fitting was performed through maximum likelihood optimization with the *R* package *OpenMx*, version 2.19.8 (Neale et al., 2016).

Association with autistic traits. We used GEE to test the phenotypic associations between ITSP Section, Quadrant or *factor* scores at 5 months and Q-CHAT total scores at 36 months. Of note, we had 34% missing data for the Q-CHAT in the selected sample, largely due to attrition. Missing data were imputed through multiple imputation based on a regression tree algorithm as implemented in the *R* package *mice* (Buuren & Groothuis-Oudshoorn, 2011) and detailed in (Bussu & Falck-Ytter, 2023). Here we summarise the imputation process and refer to the Table S39, Figures S5 and S6 as well as (Bussu & Falck-Ytter, 2023) for further details.

The observed missing data pattern was not missing completely at random, with observed dependencies in data missingness in relation to gestational age and family income (Bussu & Falck-Ytter, 2023), and as expected for complex longitudinal datasets due to attrition. Nevertheless, it has been shown that iterative multiple imputation strategies can provide unbiased estimates also in the case of a 'missing-at-random' data pattern (Jakobsen, Gluud, Wetterslev, & Winkel, 2017). Furthermore, recent work has investigated bias and efficiency of multiple imputation strategies in the presence of up to 95% missing data (Austin & van Buuren, 2022; Madley-Dowd, Hughes, Tilling, & Heron, 2019), suggesting that multiple imputation can be used to handle missing data under multiple scenarios with a high prevalence of missing data.

The infants were tested at the age of 5, 14, 24 and 36 months through questionnaires and/or lab visits that included a range of behavioural and brain-based methods (see Falck-Ytter, 2021 for an overview). Lab-based observations of emerging cognitive abilities at age 5 months and questionnaire data available from all time-points were used as input in the sequential imputation process. Multiple imputation was performed using a Round-Robin regression approach with 30 maximum iterations to generate 30 imputed datasets (selected to approximate the proportion of missing data). Given the structure introduced in the dataset by the longitudinal nature of the data, the order of imputation was adapted accordingly, and the level of autistic traits at 36 months of age (i.e. Q-CHAT Total Score) was imputed last using all available measures as predictors (see Table S39 for a detailed list). Convergence of the imputation process was evaluated by visual inspection of the imputed values across iterations to ensure that there were no evident trends in variability within and between chains across iterations (see Figure S5). Similarly, the distribution of imputed as compared to observed data was visually inspected to ensure that imputed data were realistic and had similar distributions (see Figure S6). Oscillations were approximately stable, with no larger variability between imputation chains (separate lines) than within chains. Similarly, the data distribution after imputation was in line with the observed data distribution, supporting the validity of the imputation results.

Imputed Q-CHAT score was used as a dependent variable in GEE association models. Before being used as input for the GEE analysis, Q-CHAT scores were residualized on the same covariates the sensory processing scores were previously residualized on to account for confounding. All dependent and independent variables were scaled so that the resulting *Beta* values were standardized. This procedure yielded 12 separate models tested in total, with Bonferroni correction used to correct for multiple comparisons adjusted for separate score sets (i.e. 0.05/4).

Etiological associations between sensory processing scores and later autistic traits were tested for the sensory scores that showed a significant association with QCHAT scores. Bivariate twin models based on a Cholesky decomposition method (Neale & Cardon, 1992) were used to model latent genetic and environmental influences on the trait variance and covariance. The Cholesky model allows introducing time-order information in the model, providing estimates for A-C-E components that were unique for sensory scores at 5 months and autistic traits at 36 months, respectively; as well as components that were shared between sensory scores and Q-CHAT. The model solution was identified based on the lowest BIC value and non-significant difference from the saturated model.

Results

Sample demographics and descriptive statistics are shown in Table 1.

GEE analyses showed a significant negative effect of average parental age on sensation avoiding ($b = -.12$, $SE = .05$, $p = .004$) and visual scores ($b = -.17$, $SE = .06$, $p = .006$), and a negative effect of average parental education on sensation avoiding ($b = -.18$, $SE = .06$, $p = .003$). Therefore, sensory scores and Q-CHAT total scores were residualized by these covariates in subsequent analyses.

ITSP structure validation

Twenty-eight items were selected for inclusion in the EFA model (see Table S1 for item description), for a total KMO score of 0.75 and a significant Barlett's test ($\chi^2(378) = 1{,}464$, $p < .001$). Scree plot and parallel analysis indicated the presence of four different factors (see Figure S1). The item-level pattern for the extracted factors is reported in Table S2. Examination of the items loading onto each factor provided information on the interpretation of the factors extracted, which, despite some overlap (allowed by the oblique factor rotation), mapped out onto the ITSP quadrants. Specifically, *Factor 1* loaded more onto items from the low registration quadrant, with $r = .59$ correlation with

the corresponding ITSP Quadrant score. *Factor 2* loaded more onto items from Sensation Avoiding (r = .91). *Factor 3* loaded more onto items from sensory sensitivity (r = .70). *Factor 4* loaded more onto items from sensation seeking (r = .24), but showed an overlap with Sensory Sensitivity (r = .31; see Table S4).

Internal consistency for the extracted factors was examined through McDonald's omega scores, showing reasonable levels for Factor 1 (ω = .72), Factor 2 (ω = .67), and Factor 3 (ω = .63), and satisfactory level for Factor 4 (ω = .56), still higher than the original report for the 0-to-6 months age range (reporting Cronbach's alpha reliability levels between .17 and .83; Dunn & Daniels, 2002).

Confirmatory factor analysis showed a mediocre fit to the 4-factor model, with root mean square error of approximation, RMSEA = 0.046 (confidence interval, CI = [0.039; 0.053]); standardised root mean square residual, SRMR = 0.057; Tucker-Lewis Index, TLI = 0.769; and Comparative Fit Index from the baseline model, CFI = 0.799. An alternative 3-factor model solution was tested through CFA as a sensitivity check (see Supplemental Analysis in the Supplementary Material). However, the model fit was nominally poorer, and so was the interpretability of the factors identified.

Given the clear mapping of the factors extracted in this first validation step onto the ITSP *Quadrants*, we decided to move forward with the twin modelling on the standardised ITSP scores (i.e. domain and quadrant scores), while we refer to the Supplementary Material for twin modelling findings on the factors extracted here.

Etiological structure of sensory domains: ITSP Section scores

Twin correlations were significantly higher for MZ than for DZ twins across all Section scores (based on non-overlapping confidence intervals), suggesting genetic influences on those measures. Yet, DZ correlations were also high (above or equal to .75), highlighting the contribution from a shared environment (see Figure 1A). All twin modeling assumptions were met (see Tables S4–S7 for details), and univariate ACE models confirmed influences from both additive genetics and the shared environment across all domains. In particular, we observed a moderate influence from additive genetics, with the largest influence observed for the tactile domain (A = 0.38; CI = [0.26; 0.54]), and a large influence from the shared environment, particularly for the visual domain (C = 0.70; CI = [0.58; 0.78]), while the unique environment showed minor contributions (ACE model solutions; see Figure 1B and Tables S8–S11 for details).

Phenotypic correlations were significant (i.e. confidence intervals not including zero) and ranged between .14 (i.e. auditory vs. visual sections) and .32 (i.e. tactile vs. vestibular sections). In particular, we observed moderate levels of phenotypic correlation between tactile, vestibular and auditory domains, while correlations with the visual domain were weaker (see Figure 1C). Cross-twin cross-trait correlations suggested possible familial influences on the covariance between tactile versus vestibular, tactile versus auditory and tactile versus visual domains, while the covariance between other domains was likely to show negligible familial effects based on comparable correlations between MZ and DZ twins (see Figure 1C).

The common pathway model was identified as a multivariate model solution for ITSP Sections based on model fitting compared to the other solutions, being the most parsimonious and not significantly different from the fully saturated model (see Table S12 for details on model fit statistics). Hence, infants' reactive behaviours across different sensory domains were structured hierarchically, with a single latent factor accounting for shared variance among the different domains and largely influenced by the shared environment (C = 0.82, CI = [0.59; 0.99]), while common influences from additive genetics were mild (A = 0.17, CI = [0.004; 0.40],) and unique environment showed no significant influence (E = 0.004, CI = [0; 0.03]; see Figure 2 for a schematic representation of the model solution). Vestibular and tactile domains shared the largest proportion of variance (respectively .36 and .30), followed by the auditory (i.e. .19) and visual domains (i.e. .10). Additive genetics explained a moderate proportion of unique variability across all scales, particularly the tactile domain, in line with univariate findings. While approximately 50% of influences from the shared environment on tactile and vestibular behaviours were shared across different sensory domains, influences from the shared environment were largely unique for visual and auditory behaviours (respectively 88% and 75%).

Etiological structure of sensory quadrants

Twin correlations were significantly higher for MZ than DZ twins across all quadrant scores (based on non-overlapping confidence intervals), suggesting the presence of genetic influences. Yet, DZ correlations were also high (above .74), highlighting the contribution from shared environment (see Figure 3A). All twin modeling assumptions were met (see Tables S13–S16 for details), and univariate ACE models confirmed influences from both additive genetics and shared environment across all quadrants. Influences from additive genetics were moderate, with the largest influence on sensory sensitivity (A = 0.44; CI = [0.32; 0.61]) followed by sensation avoiding (A = 0.38; CI = [0.27; 0.54]), with comparable genetic influences on low registration (A = 0.29; CI = [0.18; 0.44]) and sensation seeking (A = 0.30; CI = [0.20; 0.43]). There was a large

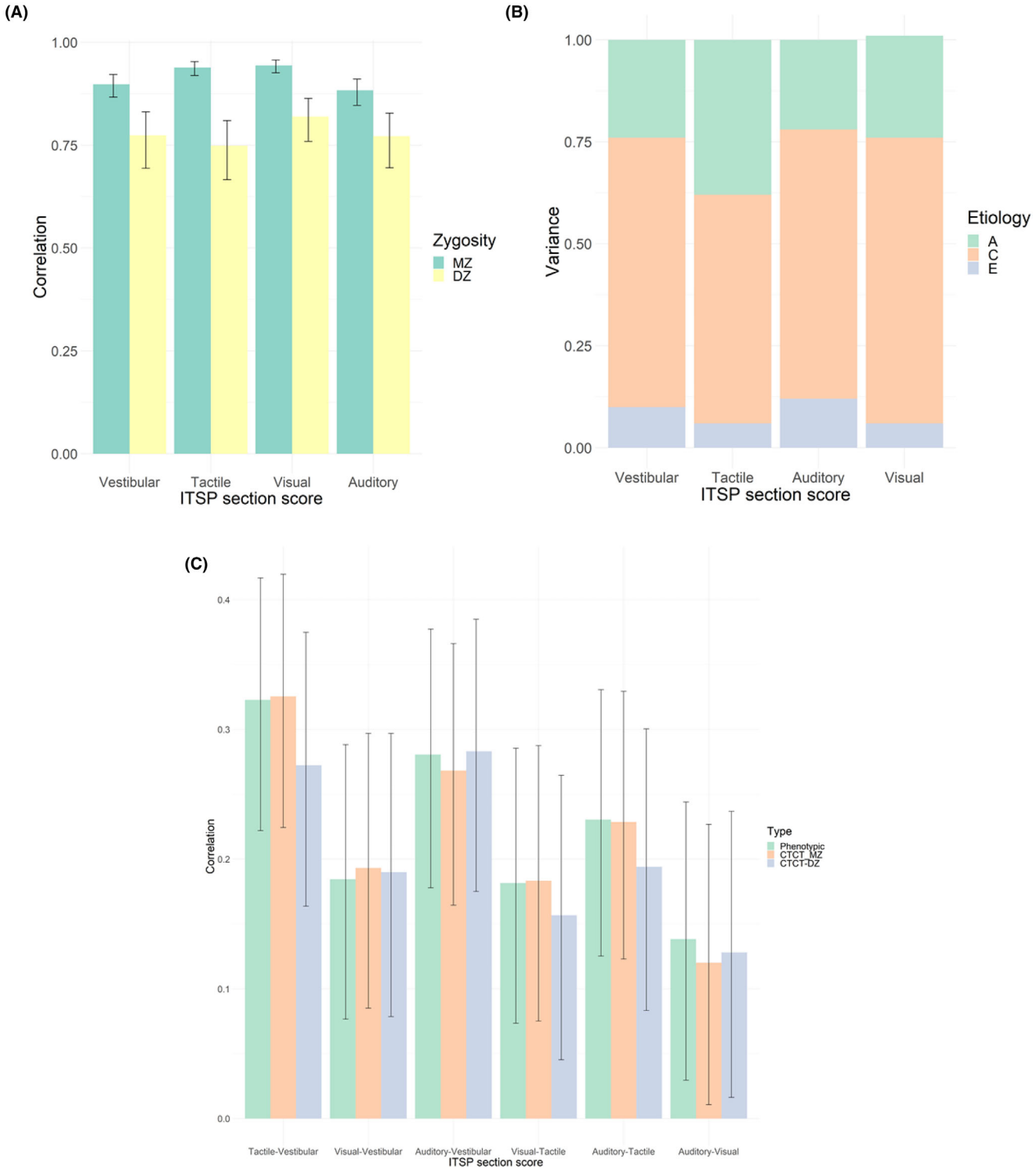

Figure 1 (A) Twin correlations by zygosity for each sensory domain, as measured by the ITSP Section scores. (B) Summary of findings from univariate twin models. (C) Phenotypic correlations for the sensory domains (in green) and cross-twin cross-trait correlations split between monozygotic (orange) and dizygotic twins (purple). A, % variance explained by additive genetics (heritability); C, % variance explained by shared environmental influences; DZ, dizygotic twins; E, % variance explained by unique environmental influences; ITSP, Infant/Toddler Sensory Profile; MZ, monozygotic twins. Error bars are showing 95% confidence intervals

influence from shared environment across the different Quadrants, ranging from 0.52 for Sensory sensitivity (C = 0.52; CI = [0.35; 0.64]) to 0.65 for Sensation Seeking (C = 0.65; CI = [0.51; 0.74]), while unique environment showed minor contributions (see Figure 3B and Tables S17–S20 for details).

Phenotypic correlations were all significant (based on confidence interval not including zero) except for the correlation between sensation seeking and sensation avoiding or sensory sensitivity, which can be interpreted in light of the opposite characterization of those behaviours (see Figure 3C). Correlation was moderate and positive

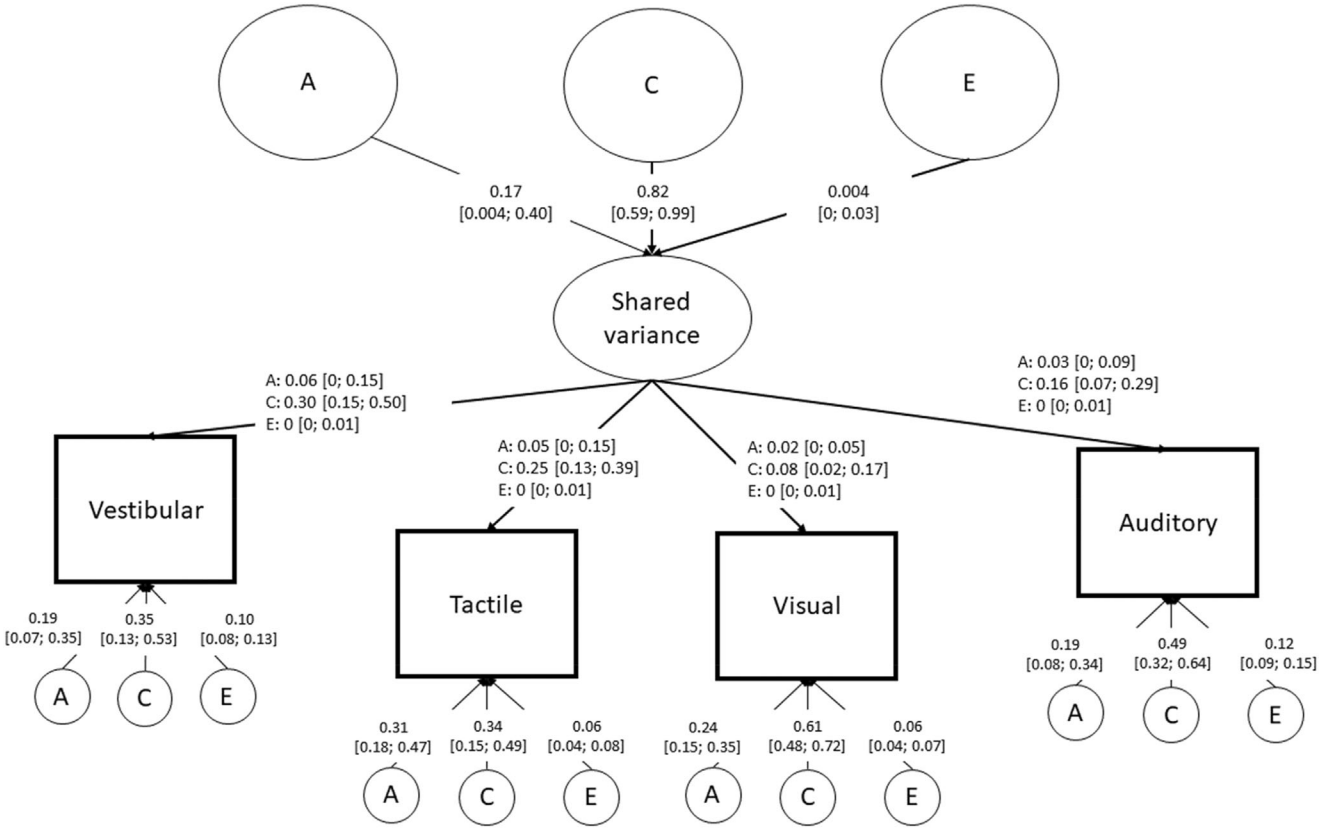

Figure 2 Common pathway model solution to the multivariate model of sensory domain scores, as measured by Sections from the Infant/Toddler Sensory Profile. Observed measures are represented by squares, and latent factors by circles. Variance partitions (with 95% confidence intervals) are reported on the edges

between sensory sensitivity and sensation avoiding (r = .47), which highlights potential overlaps in behavioral observations, with sensory sensitivity being assessed based on avoidant reactivity. In line with the quadrant definition, there was a moderate negative correlation between sensation seeking and low registration (−.26), yet there was also a positive but weak correlation between sensory sensitivity and low registration (r = .16) and between low registration and sensation avoiding (r = .18), highlighting a lack of complete orthogonality across quadrants (see Figure 3C). Cross-twin cross-trait correlations suggested possible familial influences on the covariance between Sensory Sensitivity and Sensation Avoiding, as well as Sensation Seeking and Low Registration, while the covariance between other Quadrants was likely to show negligible familial effects based on comparable correlations between MZ and DZ twins (see Figure 3C).

The independent pathways solution was identified as a multivariate twin model solution for Quadrant scores (see Table S21 for details). Hence, sensory dimensions were partially overlapping but still etiologically separable, as shown by the presence of both shared and unique contributions to the different Quadrant scores (see Figure 4 for a schematic representation of the model solution).

In line with univariate estimates, variance across the different quadrants was largely explained by additive genetics and shared environment. Sensory sensitivity showed the largest genetic contribution, of which approximately 61% was indeed unique to sensory sensitivity. Similarly, genetic influences across the other sensory quadrants were moderate and largely unique (with a 28% overlap for sensation avoiding, 36% overlap for sensation seeking, and 48% overlap for Low Registration; see Figure 4). Influences from shared environment were substantial, with the largest contribution on sensation seeking and largely unique (15% of it being common to the different Quadrants). Influences from shared environment on sensory sensitivity, sensation avoiding, and low registration were instead largely common to all quadrants (respectively 69%, 48% and 39%).

Given the clear mapping of *factors* identified from EFA onto quadrants and similarity of findings, results from twin analyses on factor scores are reported in the Supplementary Material (see Tables S22–S30 and Figures S2–S4).

Links to autistic traits in toddlerhood

For Section scores, phenotypic associations (corrected for multiple comparisons, alpha = .0125)

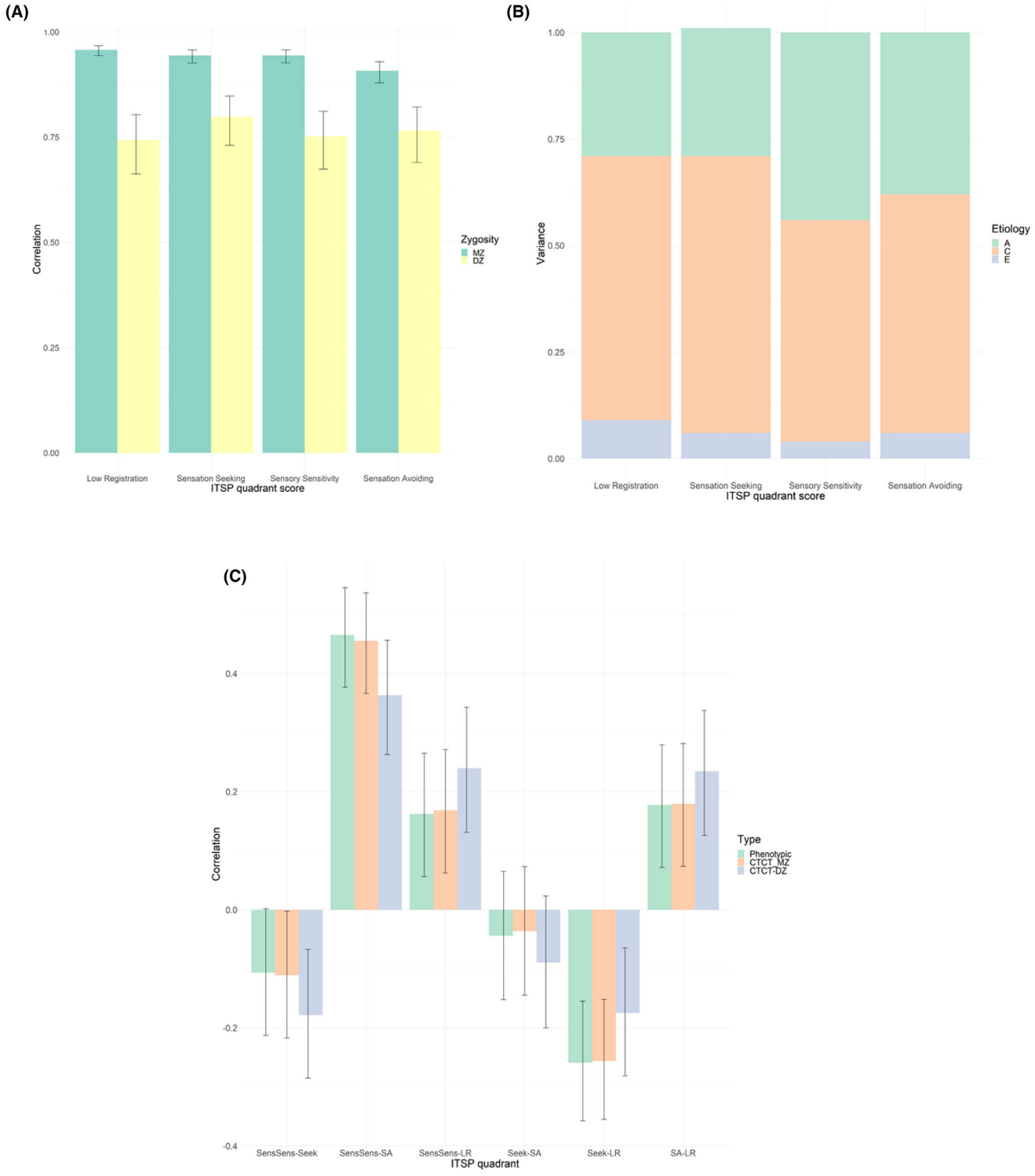

Figure 3 (A) Twin correlations by zygosity for each sensory Quadrant. (B) Summary of findings from univariate twin models. (C) Phenotypic correlations for the sensory Quadrants (in green) and cross-twin cross-trait correlations split between monozygotic (orange) and dizygotic twins (purple). A, % variance explained by additive genetics (heritability); C, % variance explained by shared environment influences; CTCT, cross-twin cross-trait; DZ, dizygotic twins; E, % variance explained by unique environment influences; ITSP, Infant/Toddler Sensory Profile; MZ, monozygotic twins. Error bars are showing 95% confidence intervals

were positive and significant between tactile scores at 5 months and Q-CHAT scores at 36 months (b = .14, SE = .05, p = .011), while associations with other sections were not significant (vestibular: b = .003, error = .05, p = .94; auditory: b = .13, error = .05, p = .014; visual: b = .09, error = .05, p = .09).

For quadrant scores, we found a significant positive association between Q-CHAT and sensory sensitivity (b = .16, SE = .05, p = .002), sensation

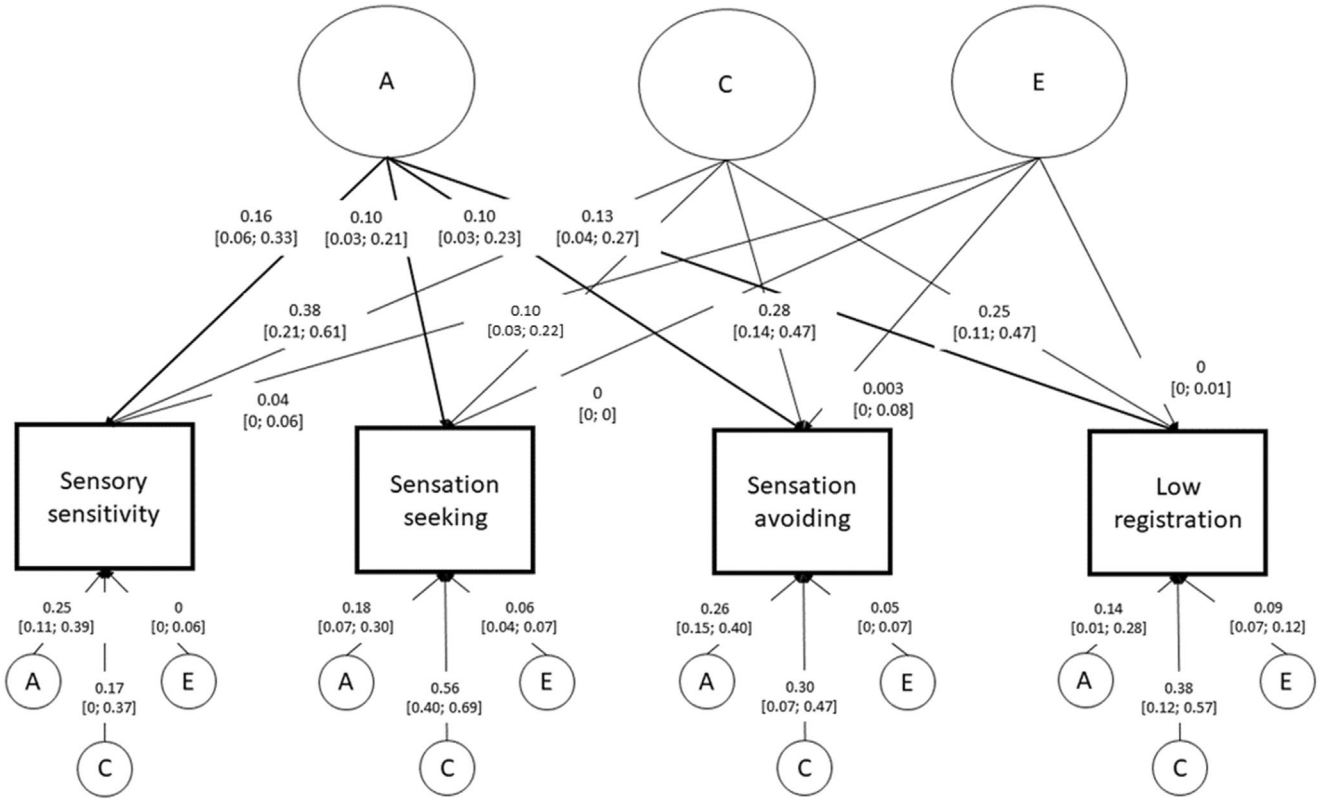

Figure 4 Independent pathways model solution to the multivariate model of sensory quadrants, as measured by the Infant/Toddler Sensory Profile. Observed measures are represented by squares, and latent factors by circles. Variance partitions (with 95% confidence intervals) are reported on the edges

avoiding (b = .13, SE = .05, p = .011) and low registration (b = .14, SE = .06, p = .0119), while the association with sensation seeking was not significant (b = −.06, error = .05, p = .23). Results were analogous for *factor* scores (see Table S35).

Follow-up etiological bivariate twin analyses on significant phenotypic associations indicated the ACE model as the most-fitting solution (see Tables S31–S34), and were in line with twin estimates presented above for sensory scores (see Figure 5). For Q-CHAT, we found significant estimates for additive genetics (approximately A = 0.35) and unique environment (approximately E = 0.33), while influences from shared environment were not significant (see Figure 5 for detailed metrics). The models indicated no significant influences shared between sensory and QCHAT scores, likely due to limited statistical power for decomposition of low phenotypic correlations (i.e. $r_{ph}^{Tactile}$ = .14; $r_{ph}^{LowRegistration}$ = .13; $r_{ph}^{SensorySensitivity}$ = .16; $r_{ph}^{SensationAvoiding}$ = .14). However, common influences from shared environment accounted for a large portion of the phenotypic correlations: $r_C^{SensorySensitivity}$ = .16 (calculated as proportion of shared C over the sum of shared and unique C on the Q-CHAT score: 0.05/(0.05 + 0.27); see Figure 5A), $r_C^{SensationAvoiding}$ = .15 (see Figure 5B), and $r_C^{Tactile}$ = .11 (see Figure 5D), while it was negligible for low registration ($r_C^{LowRegistration}$ = .03; see Figure 5C).

We refer to Tables S35–S38 for results from analogous analyses on sensory *factors*.

Discussion

Individual differences in young infants' way of responding to external stimulation appear to have implications for developmental trajectories across the neurodevelopmental spectrum (Falck-Ytter & Bussu, 2023). This study showed separability for individual differences across sensory processing dimensions (i.e. Quadrants) and identified unique associations to autistic traits in toddlerhood.

The exploratory factor analysis indicated a 4-factor solution as most fitting, providing support to Dunn's sensory processing framework (Dunn & Daniels, 2002) by showing an extension to the infant period of the quadrant structure. However, a note of caution is due given the mediocre fit of the 4-factor solution obtained from confirmatory factor analysis. This was likely due to the remaining overlap between different factors, which was also evident in the partial overlap of etiological influences across *quadrants*. While the ITSP has been validated by independent research, previous work on the construct validity of the ITSP using factor analysis is very limited and performed by the original authors of the

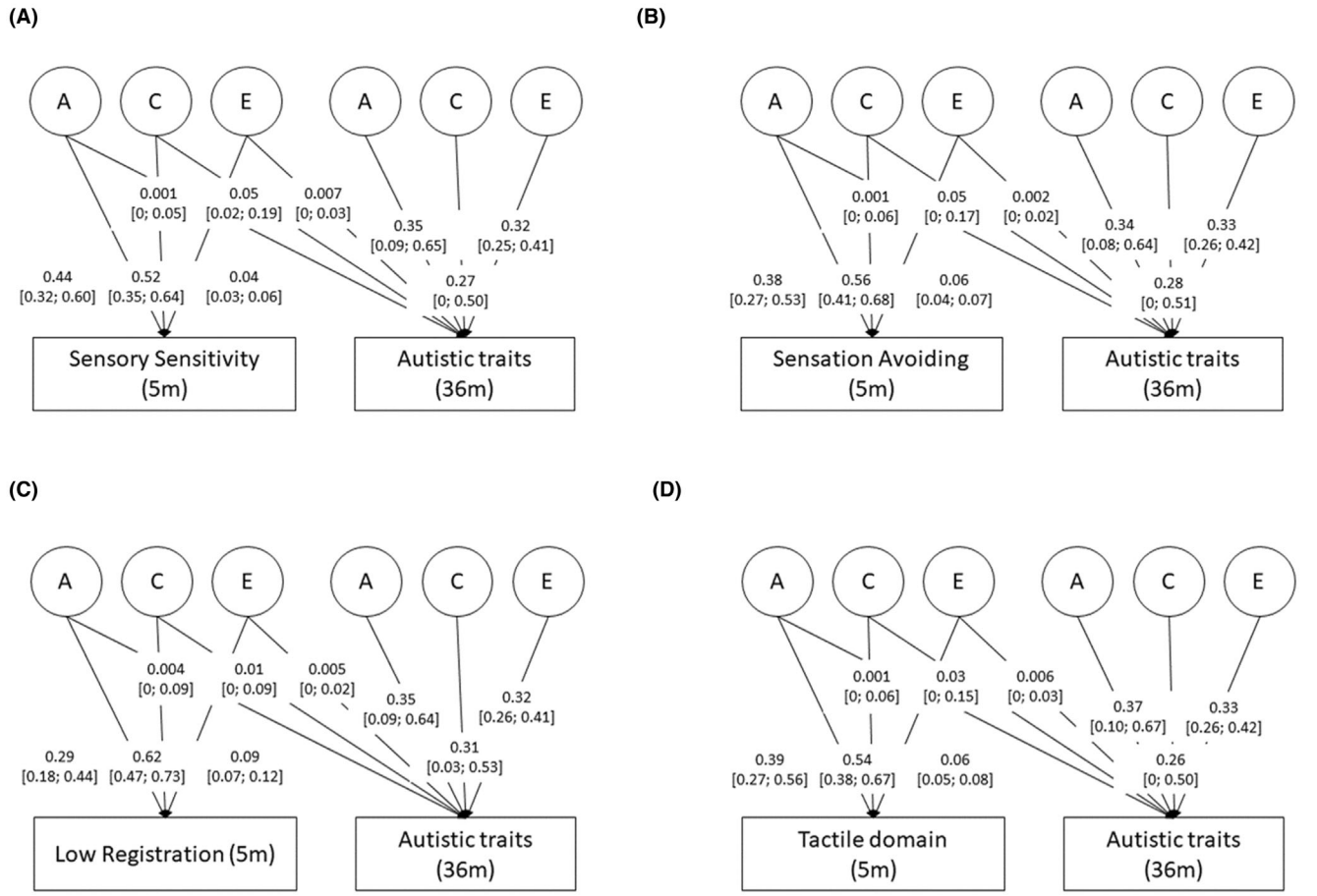

Figure 5 Bivariate ACE Cholesky models on the association between sensory scores from the Infant/Toddler Sensory Profile measured at 5 months (5 m) and autistic traits measured by the Quantitative Autism Checklist for Toddlers at 36 months of age (36 m). The different panels show specific model solutions for: (A) sensory sensitivity; (B) sensation avoiding; (C) low registration; (D) tactile domain. Observed measures are represented by squares, and latent factors by circles. Variance partitions (with 95% confidence intervals) are reported on the edges

instrument (Dunn & Daniels, 2002). To the best of our knowledge, only a handful of studies have investigated the construct validity of the ITSP in infant samples (below 6 months of age) using measures of internal consistency (Abu-Dahab, Malkawi, Nadar, Al Momani, & Holm, 2014; Yang, Tseng, Cermak, Lu, & Shieh, 2020) and supporting the original work from the test authors (Dunn & Daniels, 2002), also in line with our findings. However, previous work using factor analysis has largely focused on different instruments than the one used in this study (e.g. Sensory Profile instead of the ITSP) and older age groups, reporting mixed findings in support of the factorability of sensory-related behaviours into distinct sensory domains (e.g. visual, olfactory; Su & Parham, 2014) or distinct sensory dimensions (e.g. sensation seeking, sensory sensitivity; Dunn & Brown, 1997). Further work should investigate replication and generalizability of findings on the internal structure validity of the ITSP through factor analysis in independent infant samples. Nevertheless, the Quadrant structure finds additional support from the multivariate twin analyses, showing etiological separability between sensory dimensions in both influences from genetics and family environment.

Genetic influences were overall moderate (from .29 to .44), extending previous findings on the heritability of sensory sensitivity (A = 0.47; Assary et al., 2021) to infancy and to different domains and dimensions of sensory processing. Tactile behaviours were the most heritable among sensory domains (A = 0.38), in line with previous work on tactile acuity (A = 0.27) and sensitivity (A = 0.52) in adolescents and adults (Frenzel et al., 2012), and on tactile (A = 0.76) compared to auditory (A = 0.42) defensiveness in toddlerhood (Goldsmith et al., 2006). The higher heritability observed in older samples might be explained by gene–environment correlations, whereby genetic predisposition influences environmental exposure by actively shaping the surrounding environment, enhancing the observed influences from additive genetics (Plomin & Bergeman, 1991). In infancy, the shared environment, in the form of parent-mediated exposure to sensory stimuli, may play a more relevant role than later in development. Here, the shared environment showed indeed the largest contribution

to variability across the different sensory scores, ranging between .52 (sensory sensitivity) and .65 (sensation seeking) for sensory dimensions, and between .56 (tactile) and .70 (visual) for sensory domains. This extends previous work in older samples showing lower influences from the shared environment on tactile (C = 0) compared to auditory behaviours (C = 0.30; Goldsmith et al., 2006).

Distinct genetic factors were identified for different dimensions of sensory processing, with stronger effects on sensory sensitivity and sensation avoiding. Separability across different sensory dimensions points towards a multi-dimensional biological model for sensory processing. Hence, behavioral patterns associated with *hyper-* and *hypo*-reactivity can be observed in the same person (Ben-Sasson et al., 2009) based on the specific combination of etiological influences on each sensory dimension, contributing to the observed inter-individual variability or even subgroups in sensory processing. Future work should investigate more specific sensory phenotypes to identify the underlying neurobiological mechanisms, which may link to the unique etiological influences observed here.

Our findings extend previous work on infant siblings at elevated likelihood for autism (Feldman et al., 2022; Sacrey et al., 2015) by showing a mild but significant positive association between low registration, sensation avoiding, and sensory sensitivity in infancy and later autistic traits in a general population sample, where autistic traits are lower and the association may be less evident. While the overlap with behaviours rated in the ITSP is very limited, particularly given the age difference, it is worth highlighting that the Q-CHAT total score also includes items rating sensory-related behaviours. Therefore, the association found in this study may capture the link between early sensory reactivity and later sensory traits characteristic of autism, particularly in relation to higher sensory sensitivity or under-responsivity. However, we found no significant association between sensation seeking in infancy and later autistic traits. While this needs further replication, sensation seeking may serve a different purpose at this young infant age, increasing opportunities for learning. There was a significant positive association between tactile behaviours and autistic traits in toddlerhood, supporting the relevance of early touch behaviour for the development of social skills (Thye, Bednarz, Herringshaw, Sartin, & Kana, 2018) and autism more in general (Mammen et al., 2015).

While previous work reported a strong genetic correlation between sensory reactivity and autistic traits in adolescence (Neufeld et al., 2021; Taylor et al., 2018), our findings showed no significant influences shared between sensory reactivity in infancy and autistic traits in toddlerhood. The highly selected nature of the discordant twin study (Neufeld et al., 2021) and age differences between samples may explain the discrepancy. Younger infants may hold a more passive behaviour in relation to environmental exposure, in line with the observed influences from the shared environment. Our findings contribute to the cascading effect theory linking early sensory processing and later autistic traits (Cascio, Woynaroski, Baranek, & Wallace, 2016). The early caregiving environment is likely to mediate cascading effects of altered sensory processing in infancy, as shown by parent-mediated interventions on infant siblings (Baranek et al., 2015; Grzadzinski et al., 2021). Different aspects of parent responsiveness, directiveness, and scaffolding may differentially influence a child's sensory experience in everyday settings and, in turn, influence what they learn from the world. Future work should follow up on these findings in a clinical sample to understand how the complex, dynamic interplay between infant and caregiver may influence more specific aspects of sensory processing and, in turn, development across the neurodevelopmental spectrum.

This study has limitations in the sample size, in relation to the higher statistical power required for the decomposition of small phenotypic correlations, and the use of a parent-report questionnaire to test sensory processing. Parents rating behaviours for both twins may induce a bias towards stronger influences from the shared environment. Higher twin concordance may emerge across zygosity groups due to a rater bias, and specifically, from parents rating their children more similarly or not being able to fully appreciate subtle differences in sensory-related behaviours early in infancy. Future research should collect data from different raters to assess this potential bias (Neale & Cardon, 1992). Yet, we would expect the bias to be uniform across the different scores, leading to a hierarchical etiological structure with a unitary latent factor for shared environmental influences likely representing rater bias (as observed for sensory domains). Sensory dimensions showed instead influences from the shared environment that were unique to each quadrant and are therefore unlikely to be explained by rater bias. Another limitation is the lack of differentiation between sensitivity and reactivity from parent-report data, masking alterations across different levels of sensory processing (He et al., 2023). Future work should extend these findings to a multi-method approach.

Conclusion

This study shows separability across different sensory dimensions early in infancy, which likely contributes to a rich spectrum of sensory-related behaviours expressed within and between individuals (Falck-Ytter & Bussu, 2023). We report considerable heritability for sensory sensitivity (.44) and tactile behaviours (.38), which also showed overall large contributions from shared environment (respectively .52 and .56) and stronger association to

later autistic traits. This highlights the need to further investigate the interaction between genetic background and environmental exposure on the early development of autism in relation to sensory processing. By investigating etiological influences on early sensory behaviours in relation to later autistic traits, our study provides a new perspective on the developmental cascade process resulting in a highly variable manifestation of autism. Based on our findings, we speculate that variability across the spectrum of behavioural and symptom manifestations observed in autism is the result of the interaction across different streams underlying different dimensions of sensory processing early in life and environmental influences as scaffolded by early sensory experiences. Given the observed etiological separability, our work highlights the need to clarify at a more detailed level the unique factors influencing different sensory dimensions, with potentially relevant implications for the development of more tailored support programs for individuals with sensory processing difficulties or a history of neurodevelopmental conditions.

Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article:

Figure S1. Number of factors estimation.

Table S1. ITSP items included in factor analysis.

Table S2. Factor loadings.

Table S3. Correlation between extracted factors and standardized quadrants.

Table S4. Assumption testing for univariate model tested on the Vestibular ITSP section score, compared to the fully saturated model.

Table S5. Assumption testing for univariate model tested on the Tactile ITSP section score, compared to the fully saturated model.

Table S6. Assumption testing for univariate model tested on the Auditory ITSP section score, compared to the fully saturated model.

Table S7. Assumption testing for univariate model tested on the Visual ITSP section score, compared to the fully saturated model.

Table S8. Univariate ACE model fitting on the Vestibular ITSP section score, with sub-models.

Table S9. Univariate ACE model fitting on the Tactile ITSP section score, with sub-models.

Table S10. Univariate ACE model fitting on the Auditory ITSP section score, with sub-models.

Table S11. Univariate ACE model fitting on the Visual ITSP section score, with sub-models.

Table S12. Multivariate twin model-fitting statistics on the ITSP section scores against the fully saturated model.

Table S13. Assumption testing for univariate model tested on the Low Registration ITSP quadrant, compared to the fully saturated model.

Table S14. Assumption testing for univariate model tested on the Sensation Seeking ITSP quadrant, compared to the fully saturated model.

Table S15. Assumption testing for univariate model tested on the Sensory Sensitivity ITSP quadrant, compared to the fully saturated model.

Table S16. Assumption testing for univariate model tested on the Sensation Avoiding ITSP quadrant, compared to the fully saturated model.

Table S17. Univariate ACE model fitting on the Low Registration ITSP quadrant, with sub-models.

Table S18. Univariate ACE model fitting on the Sensation Seeking ITSP quadrant, with sub-models.

Table S19. Univariate ACE model fitting on the Sensory Sensitivity ITSP quadrant, with sub-models.

Table S20. Univariate ACE model fitting on the Sensation Avoiding ITSP quadrant, with sub-models.

Table S21. Multivariate twin model-fitting statistics on the ITSP quadrant scores against the fully saturated model.

Table S22. Assumption testing for univariate model tested on the ITSP Factor 1 scores, compared to the fully saturated model.

Table S23. Assumption testing for univariate model tested on the ITSP Factor 2 scores, compared to the fully saturated model.

Table S24. Assumption testing for univariate model tested on the ITSP Factor 3 scores, compared to the fully saturated model.

Table S25. Assumption testing for univariate model tested on the ITSP Factor 4 scores, compared to the fully saturated model.

Table S26. Univariate ACE model fitting on the ITSP Factor 1 scores, with sub-models.

Table S27. Univariate ACE model fitting on the ITSP Factor 2 scores, with sub-models.

Table S28. Univariate ACE model fitting on the ITSP Factor 3 scores, with sub-models.

Table S29. Univariate ACE model fitting on the ITSP Factor 4 scores, with sub-models.

Table S30. Multivariate twin model-fitting statistics on the ITSP factors against the fully saturated model.

Figure S2. (A) Twin correlations by zygosity for each sensory factor. Error bars are showing 95% confidence intervals. (B) Summary of findings from univariate twin models.

Figure S3. Phenotypic correlations for the sensory factors (in green) and cross-twin cross-trait correlations split between monozygotic (MZ; in orange) and dizygotic twins (DZ; in purple).

Figure S4. Independent pathways model solution to the multivariate model of sensory factors, as extracted from the item-level analysis on the Infant/Toddler Sensory Profile.

Table S31. Bivariate Cholesky ACE model fitting on the Tactile ITSP section score at 5 months of age together with the QCHAT Total score at 36 months, with sub-models.

Table S32. Bivariate Cholesky ACE model fitting on the Sensory Sensitivity ITSP quadrant score at 5 months of age together with the QCHAT Total score at 36 months, with sub-models.

Table S33. Bivariate Cholesky ACE model fitting on the Sensation Avoiding ITSP quadrant score at 5 months of age together with the QCHAT Total score at 36 months, with sub-models.
Table S34. Bivariate Cholesky ACE model fitting on the Low Registration ITSP quadrant score at 5 months of age together with the QCHAT Total score at 36 months, with sub-models.
Table S35. Summary statistics of the association analysis between infants' sensory factor scores and later autistic traits.
Table S36. Bivariate Cholesky ACE model fitting on sensory Factor 1 average scores at 5 months of age together with the QCHAT Total score at 36 months, with sub-models.
Table S37. Bivariate Cholesky ACE model fitting on sensory Factor 2 average scores at 5 months of age together with the QCHAT Total score at 36 months, with sub-models.
Table S38. Bivariate Cholesky ACE model fitting on sensory Factor 3 average scores at 5 months of age together with the QCHAT Total score at 36 months, with sub-models.
Table S39. Proportion of missing data.
Figure S5. Imputation model convergence.
Figure S6. Distribution of imputed versus observed data.
Table S40. Factor loadings: 3-factor solution.

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The authors have declared that they have no competing or potential conflicts of interest.

Author contributions
This study was conceptualised by G.B. and T.F.Y., with contributions from A.M.P. Data were analysed by G.B., with material contributions from A.M.P. G.B. drafted the manuscript, which was refined and approved by all authors before submission.

Data availability statement
Data will be made available upon reasonable request to the corresponding author. Note that sharing of pseudonymized personal data will require a data-sharing agreement, according to Swedish and EU law. Data-analysis scripts are publicly available at https://github.com/brainhabit/BT-ITSP.

Ethical considerations
This study received ethical approval by the local authority named 'Ethical Review Board in Stockholm', and all procedures were performed in accordance with the 1964 Helsinki declaration and its later amendments. Informed consent was obtained from parents of all individual participants included in the study.

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Key points
- Individuals vary significantly in sensory processing, and this variability may have a strong genetic basis. Yet, little is known about the etiological factors influencing sensory differences in infancy.
- Results showed structural and etiological separability between sensory processing dimensions in infancy and unique associations with the level of autistic traits in toddlerhood.
- Results highlight the influence of shared environment on individual variability in sensory processing in infancy, despite considerable heritability for sensory sensitivity (.44) and tactile behaviours (.38).
- This study has implications for sensory-oriented interventions (e.g. for autism), which may benefit from narrowly targeting each sensory domain and dimension.

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