

https://dspace.org/

Early predictors of sensory processing sensitivity in members of the Birth to Twenty Plus cohort

May, Andrew K.; Wessels, Stephan H.; Norris, Shane A.; Richter, Linda M.; Lombard, Zane ...

2023
Elsevier
https://hdl.handle.net/10539/49852
Andrew K. May, Stephan H. Wessels, Shane A. Norris, Linda M. Richter, Zané Lombard, Michael M. Pitman, Early predictors of sensory processing sensitivity in members of the Birth to Twenty Plus cohort, Journal of Research in Personality, Volume 104, 2023, 104370, ISSN 0092-6566, https://doi.org/10.1016/j.jrp.2023.104370.

Downloaded from a DSpace repository. https://dspace.org/

Journal of Research in Personality 104 (2023) 104370



Contents lists available at ScienceDirect

Journal of Research in Personality

journal homepage: www.elsevier.com/locate/jrp





Early predictors of sensory processing sensitivity in members of the Birth to Twenty Plus cohort

Andrew K. May [a,*], Stephan H. Wessels [a], Shane A. Norris [b,c], Linda M. Richter [c], Zané Lombard [a], Michael M. Pitman [d]

[a] Division of Human Genetics, School of Pathology, Faculty of Health Sciences, University of the Witwatersrand and the National Health Laboratory Service, Johannesburg, South Africa
[b] Developmental Pathways for Health Research Unit, Faculty of Health Sciences, University of the Witwatersrand, Johannesburg, South Africa
[c] DSI-NRF Centre of Excellence in Human Development, University of the Witwatersrand, Johannesburg, South Africa
[d] Department of Psychology, School of Human and Community Development, University of the Witwatersrand, Johannesburg, South Africa

ARTICLE INFO

Keywords:
Sensory processing sensitivity
Environmental sensitivity
Gestational age
Adversity
Longitudinal
Birth cohort

ABSTRACT

Sensory processing sensitivity (SPS) is a personality trait describing individuals who are more receptive to their environmental context. SPS has implications across the lifespan, but few longitudinal predictors of the trait are currently known. We examined potential predictors of adult SPS levels among 858 members of the Birth to Twenty Plus cohort. Available data included birth weight, gestational age, socioeconomic status, prenatal maternal stress (PMS), child behaviour scores, and genotypes for three loci (5-HTTLPR, *DRD4*, and *MAOA*). Highly Sensitive Person scale scores at ages 28–29 were regressed on these variables. Early gestational age ($p < .01$) and PMS ($p = .05$) were significantly associated with higher SPS. Our results tentatively support the hypothesis that prenatal adversity is associated with heightened sensitivity.

1. Introduction

Sensory processing sensitivity is a personality trait comprised of four key characteristics, including (i) deep cognitive processing of stimuli (in the sense of Craik & Lockhart, 1972), (ii) sensitivity to subtle environmental stimuli, (iii) strong emotional reactions, and (iv) a propensity to become easily overaroused in stimulation-rich settings (Aron & Aron, 1997; Aron, 2019; Greven et al., 2019). Individuals demonstrating these characteristics are referred to as highly sensitive persons (HSPs), estimated to constitute 15–20% of the general population (Aron & Aron, 1997; Aron et al., 2012; Aron & Aron, 2013). Since its initial proposal in 1997, SPS has garnered appreciable cross-disciplinary empirical support (Acevedo et al., 2018; Aron et al., 2012; Jagiellowicz et al., 2016), and is regarded as one of three core theories of human sensitivity, alongside differential susceptibility (Belsky & Pluess, 2009b; Belsky et al., 1991) and biological sensitivity to context (Boyce et al., 1995; Boyce & Ellis, 2005) theories. These three theories together form the Environmental Sensitivity metaframework (Pluess, 2015), which collates developmental, psychological, physiological, and genetic evidence to demonstrate the existence and importance of interindividual differences in the processing of environmental stimulation.

SPS has been particularly useful in quantifying individual differences in sensitivity because it can be assessed, among both adults and children, using a validated self-report instrument (as well as parent-report and observational instruments; Lionetti et al., 2019). The Highly Sensitive Person (or Child) scale (Aron & Aron, 1997; Pluess et al., 2018) has been reliably used across a variety of heterogeneous samples (May et al., 2020; Sengül-Inal & Sümer, 2017; Smith et al., 2019), helping to meaningfully distinguish HSPs from less sensitive individuals. This distinction has facilitated novel insights into how HS persons are highly plastic in the face of environmental contingencies, for better and for worse, thriving or deteriorating in direct proportion to the quality of their surrounds (Aron et al., 2012; Ellis et al., 2011; Lionetti et al., 2018). Current evidence suggests SPS moderates several critical outcomes for individuals (e.g. depression, life satisfaction, child behaviour), across multiple stages of the lifespan (Aron et al., 2010; Booth et al., 2015; de Villiers et al., 2018; Lionetti et al., 2019; Yano et al., 2020; Wu et al., 2021). Furthermore, some studies have noted that HSPs are particularly amenable to positive intervention. For example, Pluess and Boniwell (2015) found that highly sensitive school girls benefitted significantly

* Corresponding author at: Sydney Brenner Institute for Molecular Bioscience (SBIMB), Faculty of Health Sciences, University of the Witwatersrand, Johannesburg, South Africa.
E-mail address: andrew.may@wits.ac.za (A.K. May).

https://doi.org/10.1016/j.jrp.2023.104370
Received 31 August 2022; Received in revised form 23 February 2023; Accepted 21 March 2023
Available online 24 March 2023
0092-6566/© 2023 Elsevier Inc. All rights reserved.

from a depression prevention program, maintaining significantly lower depression scores up to 12 months post-intervention, compared to less sensitive girls who appeared not to have benefitted from the intervention. Similarly, Nocentini et al. (2018) conducted a large randomised control trial to examine the efficacy of an anti-bullying intervention amongst Grade 4–6 students. Across the entire sample, the intervention appeared to significantly lower bullying and victimisation. However, when subdividing participants in the intervention condition by sensitivity levels, the children high in SPS (especially boys) primarily accounted for this finding. Low sensitivity children demonstrated no significant reduction in bullying or victimisation. For both studies, it is speculated that highly sensitive individuals were able to deeply process, and thus better internalise, the content of the intervention, explaining the more pronounced effects. Consequently, there has been a growing research focus on identifying accurate strategies to earmark HSPs (particularly strategies that do not rely on self-report instrumentation, such as biological markers; Greven et al., 2019) so that they might be offered tailored interventions to maximise their potential and mitigate their vulnerability to harmful or excessive stimulation.

Preventive efforts are likely to yield greater success when administered early in life, thus there is substantial value in elucidating longitudinal predictors of sensitivity. Current speculation is that sensitivity differences between individuals begin calibration from in utero; (Barker, 1998; Bateson et al., 2004; Boyce & Ellis, 2005; Pluess & Belsky, 2011), that is, there is prenatal programming of postnatal plasticity (see Pluess & Belsky, 2011). From the fetal stage, the body attempts to 'forecast' (Bateson et al., 2004) future environmental conditions so that it can plot a developmental trajectory (in this case, calibrate a level of sensitivity) that will be most beneficial for the long term fitness of the individual (the fetal programming hypothesis, which is central to the Developmental Origins of Health and Disease literature; Barker, 1998; Heindel & Vandenberg, 2015; O'Donnell & Meaney, 2017). Consistently nurturant or adverse experiences are assumed to promote higher levels of sensitivity, whilst irregular conditions that shift unpredictably between supportive and detrimental are thought to promote less sensitive individuals who are more capable of handling these vicissitudes (Belsky et al., 1991; Pluess & Belsky, 2011). Notably, however, these calibrations are only possible to the extent that they are supported by relevant genetic mechanisms (Belsky et al., 2009; Pluess & Belsky, 2011). If these suppositions are true, there might exist a reliable set of innate and contextual cues, from conception and pregnancy, which can be leveraged to predict later sensitivity.

To date, few studies have directly examined sensitivity in longitudinal cohorts, limiting knowledge of potential early life predictors of sensitivity. Based on proxy variables and outcomes, early adversity appears to be the only suggested predictor of later high sensitivity. For example, maternal prenatal distress has been robustly correlated to childhood cortisol levels (Gutteling et al., 2005; Huizink et al., 2008; O'Connor et al., 2005), negative affectivity (Huizink et al., 2002), and difficult temperament (Pesonen et al., 2005). Similarly, low birth weight (a marker of poor prenatal quality) predicts difficult infant temperament (Pluess & Belsky, 2011). These outcomes are also considered as potentially indicative of a low threshold nervous system, thereby indirectly implying that prenatal distress (and whichever contextual factors induce such distress) promotes a more sensitive disposition in children. However, no correlation between prenatal quality and later, directly measured sensitivity (whether during childhood, adolescence, or adulthood) has been investigated. Arguments have been advanced that carriers of certain genetic variants are more predisposed to high sensitivity (Belsky et al., 2009). Chief amongst these are the 5-HTTLPR short (14 repeat allele; Conley et al., 2013; Homberg et al., 2016; Licht et al., 2011; Licht et al., 2020), long alleles (7 repeats or longer) of the *DRD4* exon III variable number tandem repeat (VNTR) and the low activity variant of the *MAOA* upstream VNTR (Belsky et al., 2009). A hypothesis-free genome-wide association study has identified six loci with suggestive significance ($p < 1\times10^{-5}$) in conferring heightened sensitivity, and efforts have been made to construct and examine two different polygenic scores (Keers & Pluess, 2017; Keers et al., 2016; Pluess et al., 2022). However, studies of genetic influences on sensitivity remain in their infancy, and are restricted to European-ancestry individuals.

Motivated by the paucity of current knowledge, we attempted to identify early predictors of SPS in the longitudinal Birth to Twenty Plus cohort (Barbarin & Richter, 2001b; Richter et al., 2007). Specifically, we measured SPS among adult participants (all aged 28–29 years old) and regressed these measurements onto a composite of environmental and genetic variables, pre-, neo- and post-natal, such as prenatal maternal stress, birth weight, gestational age, socioeconomic status, parent-reported ratings of internalising and externalising behaviour, and genetic loci. As a primarily low SES cohort, we hypothesised that early adversity (indexed through prenatal maternal stress, birth weight, gestational age, and/or socioeconomic status) would emerge as a predictor of later sensitivity. We did not anticipate any main effect associations between sensitivity and genetic variation, given the complex interplay between genes and the environment.

2. Material and methods

2.1. Study participants

Participants for this study were all members of the Birth To Twenty Plus (Bt20+) cohort ($n = 858$ out of 3273 total members). Briefly, the Bt20+ study has aimed to comprehensively investigate the developmental trajectories of a group of South African youths from birth to 20 years of age and beyond (Richter et al., 2007). Following the collapse of the Apartheid regime in South Africa, previously restricted city spaces were rapidly populated, mainly through the establishment and expansion of unplanned townships. Such was the speed of this urbanisation process that researchers became interested in the potential effects on health and development (both positive and negative) for the first generation of children raised within these townships. Although these children would be in closer proximity to high quality healthcare and educational facilities, they would be reared in densely populated areas struggling through traditional urban stressors, a high disease burden, and an unparalleled socio-cultural transition. Cohort members were recruited by inviting participation from mothers who gave birth to singleton babies in public hospitals and clinics in the Soweto-Johannesburg metropolitan region, during a 7-week period from April 23 to June 8, 1990. Annual efforts were made to follow up the initial cohort of neonates ($n = 3273$) and track variables pertinent to their developmental stage, beginning with growth, nutrition and rearing environment, but steadily shifting to behaviour, cognitive ability, psychosocial adjustment, puberty, sexual experimentation, and drug use, as cohort members matured. On balance, the cohort is characterised by predominantly Black African members (78.5%), reared in dense family households with low socioeconomic status. High levels of poverty, nutritional deficiency, teenage pregnancy, sexually transmitted infections, and violence exposure have been previously noted. Further details on the cohort are available elsewhere (Barbarin & Richter, 2001b; Richter et al., 2004, 2007).

Importantly, given the various social, infrastructural and political challenges in South Africa during the 1990s, and the high mobility of cohort members, sample attrition limited the comprehensiveness and completeness of data collection (Richter et al., 2007).

2.2. Procedure

The present study gathered together several predictor variables collected during preschool years (0–7) of the Bt20+ study, in order to assess their potential association to adult levels of SPS.

Historical data on member birth weight ($n = 3267$), gestational age ($n = 3171$), maternal education ($n = 2932$), socioeconomic status ($n = 3273$), and behaviour ratings at age 7 ($n = 1436$) were available at the

outset of the study. Prenatal maternal stress (PMS) data were also available for 1586 mothers of cohort members. To supplement these existing data, genotypes were determined at three loci (5-HTTLPR, *DRD4*, and *MAOA*; $n = 1219$). As part of a larger battery of tests and questionnaires assessing the lives and cognitive performance of Bt20+ cohort members at ages 28–29 (2018–2019), SPS was measured using the Highly Sensitive Person Scale ($n = 1402$) during one-on-one interviews at the Developmental Pathways to Health Research Unit at Chris Hani Baragwanath Academic Hospital, starting from June 2018. Interviewers briefly explained the instrument and its response scale and then proceeded to read out each item individually, giving the interviewee a chance to respond on the 7-point Likert scale. Interviewers captured responses using electronic tablet devices and data were stored in a confidential electronic database. The supplemental study procedures were covered under ethical clearance from the University of Witwatersrand Human Research Ethics Committee (Medical; clearance certificate number M160232).

2.3. Measures

2.3.1. The Highly Sensitive Person Scale

Sensory processing sensitivity was assessed using the Highly Sensitive Person Scale (HSPS) - a 27-item instrument that taps both general sensitivity to stimulation (e.g "Do you tend to be more sensitive to pain?", "Do you notice and enjoy delicate or fine scents, tastes, sounds, works of art?"), as well as a propensity to become easily overwhelmed in situations giving rise to substantial stress to the nervous system (e.g. "Are you easily overwhelmed by strong sensory input?", "Do you become unpleasantly aroused when a lot is going on around you?"). During its development, the instrument attained Cronbach's alphas ranging from 0.64 to 0.75 across six different, age-diverse samples drawn from the US population (Aron & Aron, 1997). Subsequent research has consistently attained alphas greater than 0.80, suggesting the instrument has a high level of internal consistency reliability (Aron & Aron, 2013). Regarding convergent validity, the HSPS correlated well ($r = 0.64$) with Mehrabian's measure of sensory screening (Mehrabian, 1977) and the Behavioural Inhibition System scale (Carver & White, 1994; Smolewska et al., 2006), which are the measures most closely aligned to SPS (Aron & Aron, 1997). Lower correlations have been observed with items measuring social introversion (e.g. Eysenck and Eysenck's EPI measure; extraversion items from the five-factor personality model) and neuroticism, which suggests good discriminant validity, given arguments that sensitivity is at least partially independent from both these traits (Aron & Aron, 1997; Gerstenberg, 2012; Lionetti et al., 2018).

2.3.2. The South African Child Assessment Schedule

The South African Child Assessment Schedule (SACAS) is a structured, caregiver-report questionnaire aimed at assessing developmental milestones in the maturation and social demands of children aged 5–10 years (Barbarin & Richter, 2001a). Based on several previous measures, the instrument was adapted to capture information on four key issues: 1) the child's overall adjustment to the demands of school; 2) the child's ability to communicate and express feelings and thoughts; 3) the child's relationship with peers; and 4) the child's mental health and general social competence.

Items were adapted from several previously validated scales, including the Achenbach-Connors-Quay (Achenbach et al., 1991), the Achenbach Child Behaviour Checklist (Achenbach & Ruffle, 2000), the Problem Behaviour Index (Peterson & Zill, 1986), the Health Resources Inventory (Gesten, 1976), and the National Center for Education Statistics' Kindergarten Teacher Survey on Student Readiness (National Center for Education Statistics, 1993). Items drawn from these various scales were supplemented with items that tapped the diagnostic criteria for mental disorders based on the international statistical classification of diseases and related health problems (ICD-10; for the latest version, see World Health Organisation, 2016). All items were then commented on by a panel of South African mothers, who offered suggestions on improved wording and additional items they felt were necessary to capture problematic child behaviour. The final item set comprised 86 items, scored on a three-point scale, from 0 to 2 (selected items were reverse scored; Barbarin & Richter, 2001a; Barbarin & Richter, 2001b).

Psychometrically, the SACAS has demonstrated acceptable levels of reliability ($\alpha = 0.6$–0.8, depending on how the instrument is sub-scaled) and sufficient validity (Barbarin & Richter, 1999, 2001a; Verkuijl et al., 2014). Validity was supported through a principal components factor analysis, along with high levels of convergence with other well validated and widely administrated measures of child health (Barbarin & Richter, 2001a). Furthermore, scores were higher for a clinical sample in comparison to a non-clinical sample (Barbarin & Richter, 2001a; Verkuijl et al., 2014).

Due to the wealth of items, the SACAS can be sub-scaled in a variety of ways, but we focused on the internalising and externalising subscales, which have good reliability estimates ($\alpha > 0.70$). For both the internalising subscale (32 items, e.g. "Is (CHILD) sad or depressed?", "Is (CHILD) too fearful or anxious?") and externalising subscale (34 items, e.g. "Does (CHILD) start fights?", "Does (CHILD) deliberately destroy things that belong to others?") higher scores indicate more problematic behaviour (Barbarin & Richter, 2001a; Verkuijl et al., 2014). Caregivers completed the SACAS when children were aged 5, 7 and 10 years of age. The number of responses collected at each age were 625, 1826, and 1248 respectively. Due to the substantially larger number of responses, we chose to use data from age 7.

2.3.3. Prenatal maternal stress

PMS was assessed using a 16-item instrument. The instrument measures common stressors in four broad categories, including marital stress ("During the last six months have you had a break-up with your husband or partner?"), family stress ("During the last six months have you given help to close family or friends in need?"), economic stress ("During the last six months, have you or your close family ever had too little money for basics, such as food, rent, clothes?") and societal stress ("During the last six months did you witness a violent crime?"). Items were administered via interview with mothers, who provided yes/no responses. All items were coded using 1 point for "yes" and 2 points for "no", except for a single item coded 1–4, representing different options to the question "During the last six months have you or a member of your close family been in real danger of being killed, in one of the following ways?" where 1 = by criminals, 2 = by police, army or other officials, 3 = during political activities, and 4 = this has not happened at all. Scores were tallied across all items (range: 16–34) with higher scores indicating *less* stressful pregnancies. The instrument has previously attained reasonable internal consistency reliability ($\alpha = 0.64$; Ramchandani et al., 2010).

2.3.4. Birth weight and gestational age

Birth weight and gestational age were obtained from official hospital records. These data were missing for three (birth weight) and 12 (gestational age) individuals in our study sub-sample. The sample included one extremely preterm individual (< 28 weeks), eleven very preterm individuals (< 32 weeks) and 106 preterm individuals (< 37 weeks). Regarding birth weight, ten individuals were born very low birth weight (< 1.5 kg, but not lower than 1 kg) and 92 were born low birth weight (< 2.5 kg).

2.3.5. Maternal education

Maternal education was recorded on a six-point scale based on the highest grade passed at school. A score of 1 reflected no formal schooling, whilst a score of 6 indicated the mother had some level of post-high school education. This simple metric was used solely as an indicator of educational background with no implications, inferences, or assumptions regarding levels of crystalised intelligence or cognitive

ability.

2.3.6. Socioeconomic status

Over a two year data collection period from infant birth to two years of age (1990–1992), mothers were asked to provide information on their household amenities and appliances. Specifically, mothers indicated if their household had electricity, a landline telephone, a fridge, a microwave, a washing machine, a television, a video recorder and/or a car (Barbarin & Khomo, 1997; Feeley et al., 2013). These household durables were scored in a binary manner (present = 1; absent = 0) and tallied together to create a metric for socioeconomic status (Filmer & Pritchett, 1999). Scores were z-standardised prior to further analyses. This initial metric of socioeconomic status (SES) correlated well with subsequent measures of SES during years 0–7 (which were unfortunately less comprehensive due to cohort attrition).

2.4. DRD4, MAOA, and 5-HTTLPR genotyping

2.4.1. DNA extraction and quantification

Bt20+ cohort members were invited to provide blood samples for DNA extraction in their early adolescence. DNA from these samples was previously extracted using the salting out method (Miller et al., 1988). Extracted DNA has since been stored in Tris-EDTA (TE) buffer at 4 °C. At the outset of this study, DNA stock concentrations were quantified using the Nanodrop Spectrophotometer ND-1000 (Thermo Fisher Scientific, USA). A standardised 50 ng/μL aliquot for each sample was prepared using TE buffer as the diluent.

2.4.2. PCR amplification

The 5-HTTLPR, *MAOA*, and the *DRD4* exon III VNTR loci have high GC content (Chang et al., 1996; Sabol et al., 1998) which can limit traditional PCR amplification. To improve amplification, a slow-down PCR procedure (Frey et al., 2008) was leveraged using Accuprime GC-rich polymerase (Invitrogen, USA). PCR reaction mixtures contained 1 unit of *Taq* polymerase, primers (forward and reverse) at 0.2 μM, 1X Accuprime GC-rich buffer A (containing thermostable AccuPrime proteins, dNTPs and $MgSO_4$), 100 μg DNA template and double distilled water up to a final reaction volume of 25 μL. Cycling conditions began with an initial denaturation at 95 °C for 5:00 min, followed by 48 cycles of denaturation (95 °C; 0:40), annealing (70 °C, decreasing by 0.4 °C per cycle; 0:30) and extension (72 °C; 0:40). A further 15 cycles followed at which the annealing temperature was kept stable at 53 °C, followed by a final extension at 72 °C for 5:00 min. The ramp speeds for heating and cooling were set at 2.5 °C/s and 1.5 °C/s respectively. All PCRs were conducted on BioRad T100 thermocycler instruments (BioRad, USA).

Primer sequences for 5-HTTLPR (Heils et al., 1996), *DRD4* (Lichter et al., 1993), and *MAOA* (Sabol et al., 1998) amplification were obtained from previously published sources. For 5-HTTLPR, the amplicon sizes were 469 bp (14-repeat S allele) and 512 bp (16-repeat L allele). For *DRD4*, amplicon sizes ranged from 379 bp (2-repeat allele) to 811 bp (11-repeat allele) in 48 bp increments. Lastly, for *MAOA*, amplicon sizes ranged from 290 bp (2-repeat allele) to 380 bp (5-repeat allele) in 30 bp increments.

2.4.3. Electrophoresis and genotype calling

All amplicons were resolved via gel electrophoresis on a 2% agarose gel run at 5 V/cm in 1X TBE. Amplicons were sized using a 50 bp ladder. Gels were viewed using the OmegaFluor gel documentation system (aplegen, USA) and software.

Genotypes were called independently by two researchers. In the case of discrepant or uncertain calls, amplicons were resolved again alongside samples of similar genotype to improve the accuracy of detecting small (one unit) repeat size differences. Where genotypes could still not be called with confidence, samples were reamplified until either a call could be made, or the sample was removed from the study due to repeated lack of amplification. Example gel images with genotype calls are displayed in Figs. S1 and S2.

2.5. Statistical analyses

To mitigate issues with longitudinal data incompleteness, we excluded individuals for whom no behavioural ratings were recorded (i.e. 100% missing item responses for the SACAS; $n = 318$) and/or no DNA sample was available for genotyping ($n = 226$). Of the 1402 HSPS respondents, 858 were retained after these exclusions. A small percentage of missing data were noted for some variables, including birthweight (0.35%), gestational age (1.40%), maternal education (6.29%), *DRD4* (0.82%), and 5-HTTLPR (0.12%). A smaller subset of these 858 individuals ($n = 357$) had available PMS data, with only one missing item at most (which was imputed using the median item value).

The remaining 501 participants had 80–100% missing item responses for the PMS instrument. For statistical analysis, we focused on the larger sample ($n = 858$; without PMS data) because it afforded better power. In an exploratory analysis, we then examined the smaller subsample (with PMS data). Although multiple imputation could have been used to restore item responses for the SACAS and PMS instruments (Eekhout et al., 2014; Madley-Dowd et al., 2019), imputing over 40% of missing data has been discouraged (Mirzaei et al., 2022), or otherwise suggested for only hypothesis-generating exercises (Jakobsen et al., 2017).

Instruments were scored and descriptive statistics prepared using the *psych* (version 2.1.6, Revelle, 2018) package. For each continuous variable, comparisons between male and female individuals were made using the Wilcoxon rank-sum test. For comparisons based on level of sensitivity, we categorised participants as either HSPs or low sensitive individuals using a high-low breakpoint on HSPS total scores of 117, as suggested by Aron and Aron (2013). Multiple linear regression modeling was used to investigate which early life predictor variables were associated with adult SPS levels (as a continuous variable). We examined genetic loci in a separate set of regression models, after filtering out non-Black African participants, in order to avoid potential issues with population stratification (Cardon & Palmer, 2003). A previous study using principal component analysis (based on over 460 000 single nucleotide polymorphisms) revealed that Black African Bt20+ members cluster together sufficiently to be regarded as a homogeneous ethnolinguistic group (May et al., 2013). Hence, we took no further actions to control for genetic ancestry. For each locus, participants were categorised as either carriers of an allele previously associated with high sensitivity (14-repeat allele for 5-HTTLPR; 7-repeat or longer allele for *DRD4*; 2- and 3-repeat alleles for *MAOA*) or non-carriers.

In any case where multiple tests were run, resulting p-values were adjusted using the Benjamini-Hochberg correction (Benjamini & Hochberg, 1995). All analyses were conducted using R (version 4.1.0) and RStudio (1.4.1103). Plots were prepared via the *ggplot2* (version 3.3.5, Wickham, 2016) and *sjPlot* (version 2.6.1; Lüdecke, 2018) packages. Other packages employed included *bestNormalize* (version 3.4.1, Peterson, 2017), *mice* (version 3.13.0, van Buuren & Groothuis-Oudshoorn, 2011), and *car* (version 3.0–2, Fox & Weisberg, 2011).

3. Results

3.1. Descriptive statistics

Descriptive statistics for the study are summarised in Table 1, except for age, given that all participants were 28–29 years old. The majority of participants were Black African (88.22%), followed by mixed ancestry (10.96%), Indian (0.7%) and White (0.12%). Females accounted for 53.03% of the sample. None of the continuous variables (birth weight, gestational age, SES, and scale total scores) were normally distributed ($p < .05$) and were not predicted to benefit substantially from common transformation procedures (using the *bestNormalize* package). Using Levene's test, all variables were found to have equal variance across

Table 1
Descriptive statistics.

Mean (standard deviation)					
	n	Total sample	Males	Females	*p* value [a]
Birth weight (kg)	855	3.03 (0.52)	3.10 (0.54)	2.98 (0.50)	**0.002**
Gestational Age (weeks)	846	37.98 (1.83)	38.07 (1.85)	37.90 (1.96)	0.228
SES (z-scored)	858	0.13 (0.96)	0.13 (0.92)	0.12 (0.99)	0.907
Maternal Education (average item)	804	4.28 (0.94)	4.31 (0.91)	4.26 (0.96)	0.773
SPS (average item)	858	3.91 (0.83)	3.69 (0.83)	4.10 (0.79)	**<0.001**
Internalising (average item)	858	0.47 (0.21)	0.46 (0.21)	0.48 (0.21)	0.238
Externalising (average item)	858	0.66 (0.29)	0.69 (0.30)	0.64 (0.27)	0.075

[a] Significant p-values are shown in bold. All p-values were adjusted using the Benjamini-Hochberg correction.

males and females. In line with known trends (Voldner et al., 2009), males were significantly heavier at birth than females (but with small effect size, $r = 0.11$), and females scored higher on the HSPS compared to males (Aron & Aron, 1997; Aron & Aron, 2013), with small effect size ($r = 0.24$). Compared to the rest of the original Bt20+ cohort, the study sample ($n = 858$) had lower average birth weight (3.03 vs 3.08 kg), lower average gestational age (37.98 vs 38.21 weeks), higher average SES score (0.13 vs −0.04), and higher average item scores for both internalising (1.25 vs 1.23) and externalising (1.25 vs 1.20) behaviours.

Several variables had significant, positive zero-order correlations (Table 2), including female sex and SPS, birth weight and gestational age, birth weight and maternal education, gestational age and maternal education, maternal education and SES, and internalising and externalising behaviour.

Significant negative correlations were observed for female sex and externalising behaviour, female sex and birth weight, birth weight and internalising behaviour, and SPS and gestational age.

3.2. The HSPS

The psychometric properties of the HSPS were previously explored in this sample (May et al., 2020) and found to be adequate and comparable to other samples worldwide. Internal consistency reliability in this study was sufficiently high (Cronbach's α = 0.85). Highly sensitive persons (n = 270) had significantly lower mean gestational age (37.68 vs 38.11 weeks; $W = 83802$, $p = .04$) and higher mean ratings of internalising behaviour (0.49 vs 0.46 average item score; $W = 71693$, $p = .03$) compared to non-highly sensitive persons ($n = 588$), but both comparisons fell short of significance after Benjamini-Hochberg correction ($p = .08$ for both comparisons). Mean birth weights were not significantly different between the two groups.

3.3. Behaviour ratings

Internal consistency reliability estimates for the two behaviour subscales were acceptable (internalising = 0.73, externalising = 0.82). Average item correlations were fairly low, in keeping with the broad nature of each scale (0.08 and 0.12 for internalising and externalising respectively).

3.4. Genotypes

Allele frequencies for the 5-HTTLPR, *DRD4*, and *MAOA* loci were similar to other published reports for continental Black African individuals (Table 3). Although 5-HTTLPR and *MAOA* genotypes were in Hardy-Weinberg equilibrium, the same was not true for *DRD4* genotypes, but this was not suspected to be a result of laboratory error given the similar allele frequencies in other studies (Table 3). Importantly, genotypes were not correlated to other study variables (i.e. no rGE).

Table 3
Black African Birth to Twenty Plus participant allele frequencies, compared to other Black South African samples.

Frequency (%)			
	Present study	Esau et al., 2008	Morgan et al., 2017
5-HTTLPR	*n* = 756	*n* = 68	*n* = 220
14	20.17	16.18	23.64
16	79.83	83.82	76.36
	Present study	Chang, Kidd, Livak, Pakstis, & Kidd, 1996	Malan, 2014
DRD4	*n* = 750	*n* = 80	*n* = 49
2	4.60	5.00	3.10
3	1.07	0.00	0.00
4	64.20	61.00	67.30
5	6.07	4.00	3.10
6	0.93	0.00	2.00
7	18.53	19.00	17.40
8	4.40	11.00	6.10
10	0.40	0.00	1.00
	Present study	Hemmings et al., 2018	
MAOA (males)	*n* = 350	*n* = 275	
2	7.43	6.90	
3	40.00	41.10	
4	49.71	48.70	
5	2.86	1.80	
	Present study		
MAOA (females)	*n* = 407		
2	6.39		
3	40.29		
4	52.21		
5	1.11		

Table 2
Zero-order correlations between study variables.

	1	2	3	4	5	6	7	8[a]
Sex (female)	–							
Birth weight	**−0.12*****	–						
Gestational age	−0.04	**0.46*****	–					
SES (z-scored)	0.00	0.02	−0.04	–				
Maternal education	−0.02	**0.15*****	**0.11*****	**0.25*****	–			
Internalising	0.05	**−0.08****	−0.03	−0.01	−0.05	–		
Externalising	**−0.09*****	−0.03	−0.03	0.02	−0.05	**0.45*****	–	
SPS	**0.25*****	−0.04	**−0.10*****	−0.02	0.03	0.02	−0.03	–

[a] Significant values are bolded. $^{*}p < .1$; $^{**}p < .05$; $^{***}p < .01$.

3.5. Regression modelling

3.5.1. Excluding prenatal maternal stress

Regression modelling results for the study sample (n = 858) are summarised in Table 4. A model incorporating all of the study predictors (except prenatal maternal stress) fit well, $F(782) = 9.80$, $p < .001$, suggesting a significant role for gestational age in predicting adult SPS levels, although of small effect (adjusted $R^2 = 0.07$). Specifically, lower gestational age predicted a higher average item score on the HSP scale (Fig. 1). Other non-significant predictors had coefficients in anticipated directions.

Given the suggested importance of gestational age in our regression results, we examined this variable in more detail. Approximately 13.95% ($n = 118$) of our participants were born pre-term in 1990 (< 37 weeks; slightly higher than the original Bt20+ cohort at 12.24%). Unfortunately, the prevalence of preterm birth in South Africa remains unknown (Ramokolo et al., 2019), but it is expected to be higher than the global average of approximately 10.6% (Chawanpaiboon et al., 2019). Of those born preterm, 61% ($n = 72$) were categorised as low sensitive individuals, but their mean gestational age (34.99 weeks) remained significantly higher than HSPs (33.93 weeks; $W = 83802$, $p = 0.03$). Chi-square testing suggested there were more HSPs born pre-term than expected, although this observation fell short of significance, $\chi^2(1) = 3.62$, $p = .06$.

3.5.2. Including genotypes

After filtering out non-Black African individuals (n = 101) and including three genetic loci as potential predictors, none of these loci significantly predicted adult SPS (Table 5). Notably, gestational age remained a significant predictor in the presence of these genetic variables.

3.5.3. Exploratory analysis: Including prenatal maternal stress

A smaller subset (n = 357) of study participants had comprehensive PMS data. In a separate set of analyses, we examined the potential influence of this additional variable, under the hypothesis that PMS is a more direct measure of early adversity and would thus have a more significant association with HSP scale scores. The smaller subset comprised of predominantly Black African individuals (91.6%), of which 51% were female. Compared to the larger study sample ($n = 858$), this smaller subset were on average heavier at birth (3.11 kg vs 3.03 kg), had a longer gestational age (38.10 vs 37.98 weeks), had better maternal education (4.42 vs 4.28), but poorer SES (0.00 vs 0.13). Scores for internalising and externalising behaviour, and the HSPS, were comparable between samples (Table S1).

Internal consistency reliability for the PMS instrument was adequate ($\alpha = 0.61$), and in line with other reports (Ramchandani et al., 2010). The average item score for PMS was 1.97, suggesting that most mothers had fairly benign levels of stress during pregnancy. A significant zero-order correlation emerged between low birth weight and higher PMS ($r = 0.12$, $p = .02$). There were no significant differences between male and female children, however, highly sensitive adults had mothers who were significantly more stressed during pregnancy (mean = 1.94) compared to mothers of low sensitive adults (mean = 1.98, $p = .03$).

Hierarchical regression modeling was used to explore the impact of including PMS as a predictor. In the first step (Table S2), a model was fitted with all predictors except PMS. Gestational age emerged as the strongest predictor of adult SPS levels, but unlike in the larger study sample, this relationship did not reach significance. In the second step (Tables 4 & S2), PMS was added as a predictor to the model. PMS had significant predictive power, suggesting that higher levels of stress (lower stress scores) resulted in higher levels of SPS. The model fit was significant, $F = 2.06$, $p = .04$, explaining a small proportion of variance (adjusted $R^2 = 0.02$).

3.6. Case-wise diagnostics

Case-wise diagnostics revealed no issues with the preceding regression models. No predictor, in any model, had a variance inflation factor (VIF) larger than 1 nor tolerance statistics (1/VIF) < 0.10 (Cohen et al., 2003). Use of the Durbin-Watson test revealed no issues with autocorrelation. Cook's distances and hat values were all within reason.

4. Discussion

Sensory processing sensitivity, and the broader Environmental Sensitivity metaframework, have potential ramifications for a range of disciplines including childhood development (Lionetti et al., 2018; Nocentini et al., 2018), psychosocial interventions (de Villiers et al., 2018; Morgan et al., 2017; Pluess & Boniwell, 2015), work-related stress (Evers et al., 2008), anthropology (Rappaport & Corbally, 2018), epidemiology (Goldberg et al., 2018), and gene × environment research (Belsky et al., 2013; Keers & Pluess, 2017). Consequently, there is substantial value in identifying early predictors of heightened sensitivity, which may help to identify individuals who would benefit from tailored and/or more intense intervention efforts. Unfortunately, few such predictors are currently known. We sought to investigate whether early life variables captured for the Birth to Twenty Plus cohort members were predictive of their sensory processing sensitivity (SPS) level at age 28–29.

To reflect the composite of factors influencing fetal/childhood growth (Bollen, Noble, & Adair, 2013), we tested a range of environmental and genetic variables. We found statistical support for gestational age as a predictor of adult SPS. While controlling for other study variables, shorter gestational age predicted a significantly greater chance of adult high sensitivity. On average, HS adults were born slightly earlier (37.68 weeks) than low sensitive adults (38.11 weeks), but no significant differences in birth weight were detected. In

Table 4
Multivariate regression models (excluding genotypes) examining potential predictors of average-item HSP scale scores (dependent variable) Excluding PMS ($n = 858$)[a] Including PMS ($n = 357$).

Predictors	B	Std Error	β	p	B	Std Error	β	p[b]
Intercept	5.52	0.59	–	**<0.001**	5.76	1.21	–	**<0.001**
Sex (female)	0.41	0.06	0.25	**<0.001**	0.24	0.08	0.16	**<0.01**
Birth weight	0.06	0.06	0.04	0.33	0.09	0.09	0.06	0.32
Gestational age	−0.06	0.02	−0.13	**<0.001**	−0.04	0.03	−0.07	0.21
Maternal Education	0.04	0.03	0.04	0.25	0.06	0.05	0.07	0.22
SES (z-scored)	−0.03	0.02	−0.03	0.36	−0.05	0.04	−0.06	0.27
Internalising behaviour	0.09	0.15	0.02	0.57	−0.22	0.21	−0.06	0.30
Externalising behaviour	−0.12	0.11	−0.04	0.29	0.00	0.16	0.00	0.97
PMS (average score)	–	–	–	–	−0.05	0.27	−0.11	**0.05**
Model		$F(782) = 9.80$, $p <$ **.001**				$F(341) = 2.06$, $p =$ **.04**		
Adjusted R^2		0.07				0.02		

[a] PMS: prenatal maternal stress.
[b] Significant p values are bolded.

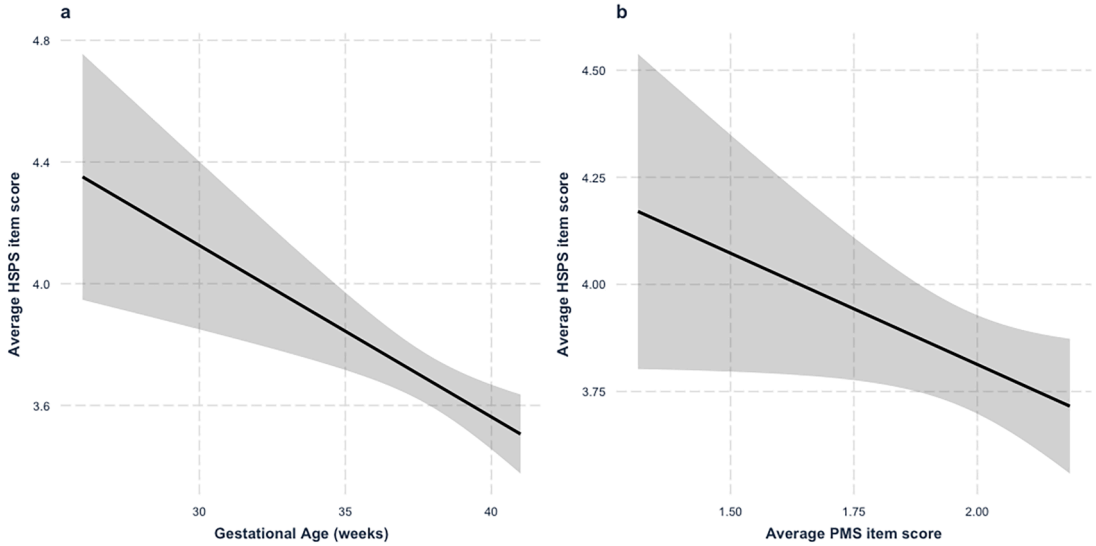


Fig. 1. Early predictors of adult SPS levels. While controlling for the effects of other variables, including sex, birthweight, maternal education, socioeconomic status, and internalising and external- ising behaviour (at age 7), (a) gestational age significantly ($p < .001$) predicted SPS levels assessed at ages 28–29 amongst members of the Birth to Twenty Plus cohort ($n = 858$). A longer gestational period correlated with a lower average item score on the Highly Sensitive Person scale. (b) In a smaller subset of participants with available data ($n = 357$), prenatal maternal stress (PMS) emerged as a marginal predictor of SPS ($p = .05$), suggesting that highly sensitive adults were more likely to be born to mothers experiencing stressful pregnancies. The grey shaded area represents the 95% confidence interval.

Table 5
Multivariate regression model (including genotypes) examining potential predictors of average-item HSP scale scores (dependent variable).

Predictors	B	Std Error	β	p[a]
Intercept	5.40	0.65	–	**<0.001**
Sex (female)	0.42	0.06	0.26	**<0.001**
Birth weight	0.09	0.06	0.06	0.16
Gestational age	−0.06	0.02	−0.13	**<0.01**
Maternal Education	0.04	0.03	0.04	0.26
SES (z-scored)	−0.03	0.03	−0.03	0.36
Internalising behaviour	0.20	0.16	0.05	0.20
Externalising behaviour	−0.17	0.12	−0.06	0.14
5-HTTLPR (short allele)	−0.02	0.06	−0.01	0.79
DRD4 (long allele)	0.06	0.06	0.03	0.46
MAOA (low activity)	0.03	0.07	0.02	0.61
Model		$F(684) = 6.36$, $p <$**.001**		
Adjusted R^2		0.07		

[a] Significant p values are bolded.

exploratory analyses using a smaller subset of participants with available prenatal maternal stress data, gestational age no longer emerged as significant, but prenatal maternal stress attained significance ($p = .05$). Notably, SES did not appear to predict levels of SPS. The Bt20+ cohort had uniformly poor SES in the early 1990s (Barbarin & Khomo, 1997), thus the lack of variance in this variable may have masked a stronger predictive role for SES. Low SES is likely to have been an appreciable stressor during pregnancy, the effects of which might be indirectly observed through earlier gestational age (Ruth et al., 2012).

Shorter gestational period and lower birth weight have been extensively used as proxy indicators of unfavourable fetal growth conditions (Bollen et al., 2013), often resulting from maternal prenatal stress (Bussières et al., 2015; Lima et al., 2018). Although both variables are related (and were once used interchangeably), they are treated independently given that low birth weight may be the result of interuterine growth retardation, whilst shorter gestational age babies are not always underweight (Loret De Mola et al., 2014; Wilcox, 2001). As such, gestational age and birth weight can be independently associated with phenotypic outcomes. For example, following a systematic review and meta-analysis, Loret De Mola et al., 2014 reported an association between low birth weight and adult depression, but no clear association between depression and gestational age. Conversely, Khashan et al. (2015) found an association between gestational age (between 33 and 36 weeks, and between 37 and 38 weeks) and type 1 diabetes, but no connection to birth weight. Our results appear to continue this trend, marking gestational age as an independent predictor of sensitivity, without involvement of birth weight. In contrast to our findings, Pluess and Belsky (2011) identified a possible role for low birth weight (but not gestational age) in promoting sensitivity, although the latter was measured via negative childhood temperament, an assumed proxy of high sensitivity. Additionally, their study involved children of European ancestry, whilst ours involved children of mostly African ancestry. Nevertheless, the inference remains that poorer prenatal quality appears to be related to heightened adult sensitivity. Our exploratory analysis on a smaller subset of the sample supported this notion, with a direct measure of prenatal maternal stress emerging as a better predictor of SPS than gestational age.

Importantly, sensitivity was associated with shorter gestational age, not necessarily preterm birth. Of our study participants born preterm, HSPs were somewhat overrepresented amongst babies classified as preterm, but not significantly so ($p = .06$). This observation would appear to align with sensitivity (and broader evolutionary) theorising, in that only *moderately* adverse pregnancies may encourage adaptive developmental responses in the fetus. The sorts of severe adversity that might lead to preterm birth and/or low birth weight (< 2.5 kg) may not be adaptively overcome, and thus might foster pathological sequelae without compensatory survival advantages (Bateson et al., 2004), such as cognitive impairment (Oudgenoeg-Paz et al., 2017), higher blood pressure (Huxley et al., 2000), asthma, and anxiety/depression (Raju et al., 2017). As a neutral, non-pathological personality trait, it seems reasonable that we find SPS associated with shorter gestational age, but not specifically preterm birth or low birth weight. Nevertheless, in studies that have focused on preterm birth, several have reported a consistent set of non-pathological personality features that tend to characterise individuals born preterm. These features included being more cautious, shy, dutiful, risk-averse, and ranking higher on neuroticism (Raju et al., 2017). Coincidentally, these too are well-described features of HSPs, and are behaviours specifically tapped by items on

the HSP scale (Aron & Aron, 1997). Considered together with our study, there would appear to be evidence that shorter gestational age, *including* preterm gestation to at least some extent, favours adaptive calibration towards heightened sensitivity.

Some caution should be applied to this reasoning however. Due to seemingly shared subsets of behavioural characteristics, sensory processing sensitivity is frequently confused with more pathological conditions such as sensory processing disorder, post-traumatic stress disorder (PTSD), autism spectrum disorders, and schizophrenia, despite being qualitatively different (Acevedo et al., 2018; Aron & Aron, 2013; Levit-Binnun et al., 2014). It is not known to what extent the HSP scale misclassifies individuals with these disorders, and we did not have any relevant data for our sample in order to investigate further. The Bt20+ cohort is not clinical in nature, with members meant to represent a typical cross-section of society (evidenced by the expected distribution of gestational ages, for example), suggesting a low overall likelihood for pathological disorder. Nevertheless, given their challenging rearing environment and the culture of violence associated with South Africa (Abrahams & Jewkes, 2005; Ward et al., 2012), only an estimated 1 % of the cohort was not exposed to some form of violence during childhood (Richter et al., 2018), and it is reasonable to assume that exposure to violence likely continued throughout the lives of a substantial proportion of cohort members. Such high levels of exposure may have significantly confounded our identification of HS persons, especially if sensitive behaviours were conditioned through PTSD. Whilst this does highlight a limitation of our study, it is also a strong reminder for why early, effective interventions are needed in South Africa (and other impoverished settings), and why knowledge of SPS might be particularly important.

A second caution is that our study design rests on the assumption that levels of sensitivity remained stable from birth to 28–29 years of age. However, the long-term stability of SPS is not currently known (Greven et al., 2019), and theory would suggest that early childhood affords ample opportunity for recalibration of sensitivity levels (Belsky & Pluess, 2009a; Pluess & Belsky, 2011). As discussed earlier, however, gestational age predicts numerous other adult phenotypes (including personality traits) that are similarly open to modification during the developmentally malleable period of childhood. This would suggest that while such modifications are possible, pre-natal forecasts completely mismatched to postnatal environments might be rare. The same reasoning may be applicable to SPS, but substantially more research is required to test this assumption.

We did not detect any association between genetic loci and adult sensitivity in our Black African participants. Alleles of all three of the included loci have been argued as potential markers of high sensitivity, but generally following certain environmental exposures (Belsky et al., 2009; Pluess & Belsky, 2011). Our lack of main effect findings is then anticipated, since main effect modelling fails to account for interactions with the environment. Moreover, sensitivity is likely a polygenic trait, determined by potentially hundreds of loci throughout the genome, which reduces the likelihood of single variants having significant predictive power (Keers et al., 2016; Pluess et al., 2022).

Unfortunately, we were not powered sufficiently to warrant serious investigation of possible G×E effects, but these should certainly be explored in larger cohorts especially of African-ancestry, given the unparalleled genetic diversity of African individuals (Fan et al., 2019). Arguably, our power to detect main genetic effects was also limited, but higher than similar, previous investigations (Chen et al., 2011; Licht et al., 2011; Licht et al., 2020).

Several limitations to this study warrant discussion. Firstly, ours was an observational study, meaning that no causality can be inferred. Secondly, our study sample differed appreciably from the original Bt20+ cohort, reflecting a possible ascertainment bias that might prevent our results from being generalisable. Our exploratory analysis with PMS involved a smaller subset of participants who further differed from the main cohort, suggesting added caution when interpreting the presented findings. Thirdly, the use of proxy variables, like birth weight and gestational age, to infer fetal conditions is not without issue. In practical terms, recording of these variables can be biased by inaccurate measurement scales and techniques, and differences in timing and delivery setting (Bollen et al., 2013). Even when measured with accuracy and precision, these variables do not perfectly correlate with the assumed fetal conditions that they proxy. Future studies should aim to include variables more closely reflecting maternal prenatal stress, rather than relying on indirect measures.

Similarly, our analysis might have been strengthened if we could control for other variables more proximal to the time point at which SPS was assessed (e.g. PTSD symptoms). Fourthly, although our findings were significant, they were of small effect size, suggesting that other unaccounted for mechanisms may contribute more substantially to the development of adult SPS. Finally, we resorted to strongly discouraged complete case analysis, rather than attempting multiple imputation (Mirzaei et al., 2022), given the large volume of missing instrument data (> 40%) for a substantial proportion of the Bt20+ cohort.

5. Conclusion

In conclusion, our study provides tentative support to the hypothesis that early, mild adversity (measured via shorter gestational age) is associated with higher sensitivity in individuals, possibly extending well into adulthood. If true, this would further encourage intervention efforts offered to individuals with shorter gestational age, not only to mitigate chances for known pathological complications, but also because these individuals may be more receptive to such intervention by virtue of a heightened sensitivity to environmental stimulation. To the best of our knowledge, this is the first study to investigate long-term predictors of SPS, and so our results await validation in other independent studies.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

To protect the privacy of Bt20+ cohort members, raw data and analysis code for this study will be made upon reasonable request to, and approval by, the study authors.

Acknowledgements

The authors would like to thank Feziwe Mpondo and her team of research assistants for administering the HSPS to Bt20+ cohort members, along with all cohort members who participated in this study. The DSI-NRF Center of Excellence in Human Development and the National Health Laboratory Service Research Trust are thanked for their funding contribution.

Funding

The support of the DSI-NRF Centre of Excellence in Human Development towards this research/activity is hereby acknowledged [Opportunity Grant OPP1640]. Opinions expressed and conclusions arrived at, are those of the authors and are not necessarily to be attributed to the CoE in Human Development. This work was also funded by the National Health Laboratory Service Research Trust Grant [grant number GRANT004 94582].

Author contributions

Andrew May, Zané Lombard, and Michael Pitman contributed to the

study conception and design. Data preparation and analysis was performed by Andrew May and Stephan Wessels. Shane Norris and Linda Richter provided access to the Birth to Twenty Plus cohort and prepared the cohort data. The first draft of the manuscript was written by Andrew May and reviewed by the other authors. All authors read and approved the final manuscript.

Appendix A. Supplementary material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jrp.2023.104370.

References

Abrahams, N., & Jewkes, R. (2005). Effects of South African men's having witnessed abuse of their mothers during childhood on their levels of violence in adulthood. *American Journal of Public Health, 95*(10), 1811–1816. https://doi.org/10.2105/AJPH.2003.035006

Acevedo, B. P., Aron, E. N., Pospos, S., & Jessen, D. (2018). The functional highly sensitive brain: A review of the brain circuits underlying sensory processing sensitivity and seemingly related disorders. *Philosophical Transactions of the Royal Society B: Biological Sciences, 373*, Article 20170161. https://doi.org/10.1098/rstb.2017.0161

Achenbach, T. M., Howell, C. T., Quay, H. C., & Conners, C. K. (1991). National survey of problems and competencies among four- to sixteen-year-olds: Parents' reports for normative and clinical samples. *Monographs of the Society for Research in Child Development, 56*(3), 1–131. https://doi.org/10.1111/j.1540-5834.1991.tb01174.x

Achenbach, T. M., & Ruffle, T. M. (2000). The child behavior checklist and related forms for assessing behavioral/emotional problems and competencies. *Pediatrics in Review, 21*(1), 265–271. https://doi.org/10.1542/pir.21-8-265

Aron, E. N., & Aron, A. (2013). Tips for SPS research (Revised November 21, 2013). Retrieved March 22, 2018, from http://hsperson.com/pdf/Tips %7B %5C %7Dfor %7B %5C %7DSPS %7B %5C %7DResearchers %7B %5C %7DNov21 %7B %5C %7D2013.pdf.

Aron, E. N., & Aron, A. (1997). Sensory-processing sensitivity and its relation to introversion and emotionality. *Journal of Personality and Social Psychology, 73*(2), 345–368. https://doi.org/10.1037/0022-3514.73.2.345

Aron, E. N., Aron, A., & Jagiellowicz, J. (2012). Sensory processing sensitivity: A review in the light of the evolution of biological responsivity. *Personality and Social Psychology Review, 16*(3), 262–282. https://doi.org/10.1177/1088868311434213

Aron, A., Ketay, S., Hedden, T., Aron, E. N., Rose Markus, H., & Gabrieli, J. D. E. (2010). Temperament trait of sensory processing sensitivity moderates cultural differences in neural response. *Social Cognitive and Affective Neuroscience, 5*(2–3), 219–226. https://doi.org/10.1093/scan/nsq028

Aron, E. N. (2019). The highly sensitive person. Retrieved September 25, 2018, from http://hsperson.com/faq/evidence-for-does/.

Barbarin, O. A., & Khomo, N. (1997). Indicators of economic status and social capital in South African townships: What do they reveal about the material and social conditions in families of poor children? *Childhood, 4*(2), 193–222. https://doi.org/10.1177/0907568297004002001

Barbarin, O. A., & Richter, L. M. (1999). Adversity and psychosocial competence of South African children. *American Journal of Orthopsychiatry, 69*(3), 319–327. https://doi.org/10.1037/h0080406

Barbarin, O. A., & Richter, L. (2001a). Economic status, community danger and psychological problems among South African children. *Childhood, 8*(1), 115–133. https://doi.org/10.1177/0907568201008001007

Barbarin, O. A., & Richter, L. M. (2001b). *Mandela's children: Growing up in post-apartheid South Africa*. Abingdon, UK: Routledge.

Barker, D. J. P. (1998). In utero programming of chronic disease. *Clinical Science, 95*, 115–128. https://doi.org/10.1042/cs0950115

Bateson, P., Barker, D. J. P., Clutton-Brock, T., Deb, D., D'Udine, B., Foley, R., Gluckman, P., Godfrey, K., Kirkwood, T., Lahr, M. M., McNamara, J., Metcalfe, N. B., Monaghan, P., Spencer, H. G., & Sultan, S. E. (2004). Developmental plasticity and human health. *Nature, 430*(6998), 419–421. https://doi.org/10.1038/nature02725

Belsky, J., Jonassaint, C., Pluess, M., Stanton, M., Brummett, B., & Williams, R. (2009). Vulnerability genes or plasticity genes? *Molecular Psychiatry, 14*(8), 746–754. https://doi.org/10.1038/mp.2009.44

Belsky, J., & Pluess, M. (2009a). The nature (and Nurture?) of plasticity in early human development. *Perspectives on Psychological Science, 4*(4), 345–351.

Belsky, J., & Pluess, M. (2009b). Beyond diathesis stress: Differential susceptibility to environmental influences. *Psychological Bulletin, 135*(6), 885–908. https://doi.org/10.1037/a0017376

Belsky, J., Pluess, M., & Widaman, K. F. (2013). Confirmatory and competitive evaluation of alternative gene-environment interaction hypotheses. *Journal of Child Psychology and Psychiatry, 54*(10), 1135–1143. https://doi.org/10.1111/jcpp.12075

Belsky, J., Steinberg, L., & Draper, P. (1991). Childhood experience, interpersonal development, and reproductive strategy: An evolutionary theory of socialization. *Child Development, 62*, 647–670. https://doi.org/10.1111/j.1467-8624.1991.tb01558.x

Benjamini, Y., & Hochberg, Y. (1995). Controlling the false discovery rate: A practical and powerful approach to multiple testing. *Journal of the Royal Statistical Society, 57*(1), 289–300. https://doi.org/10.2307/2346101

Bollen, K. A., Noble, M. D., & Adair, L. S. (2013). Are gestational age, birth weight, and birth length indicators of favorable fetal growth conditions? A structural equation analysis of Filipino Infants. *Statistics in Medicine, 32*(17), 2950–2961. https://doi.org/10.1002/sim.5771

Booth, C., Standage, H., & Fox, E. (2015). Sensory-processing sensitivity moderates the association between childhood experiences and adult life satisfaction. *Personality and Individual Differences, 87*, 24–29. https://doi.org/10.1016/j.paid.2015.07.020

Boyce, W. T., Chesney, M., Alkon, A., Tschann, J. M., Adams, S., Chesterman, B., … Wara, D. (1995). Psychobiologic reactivity to stress and childhood respiratory illnesses: Results of two prospective studies. *Psychosomatic Medicine, 57*(5), 411–422. TNK0062.

Boyce, W. T., & Ellis, B. J. (2005). Biological sensitivity to context: I. An evolutionary–developmental theory of the origins and functions of stress reactivity. *Development and Psychopathology, 17*, 271–301. https://doi.org/10.1017/S0954579405050145

Bussières, E. L., Tarabulsy, G. M., Pearson, J., Tessier, R., Forest, J. C., & Gigue're, Y. (2015). Maternal prenatal stress and infant birth weight and gestational age: A meta-analysis of prospective studies. *Developmental Review, 36*, 179–199. https://doi.org/10.1016/j.dr.2015.04.001

Cardon, L. R., & Palmer, L. J. (2003). Population stratification and spurious allelic association. *Lancet, 361*(9357), 598–604. https://doi.org/10.1016/S0140-6736(03)12520-2

Chawanpaiboon, S., Vogel, J. P., Moller, A. B., Lumbiganon, P., Petzold, M., Hogan, D., … Gülmezoglu, A. M. (2019). Global, regional, and national estimates of levels of preterm birth in 2014: A systematic review and modelling analysis. *The Lancet Global Health, 7*(1), e37–e46. https://doi.org/10.1016/S2214-109X(18)30451-0

Carver, C. S., & White, T. L. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS scales. *Journal of Personality and Social Psychology, 67*(2), 319–333. https://doi.org/10.1037/0022-3514.67.2.319

Chang, F. M., Kidd, J. R., Livak, K. J., Pakstis, A. J., & Kidd, K. K. (1996). The world-wide distribution of allele frequencies at the human dopamine D4 receptor locus. *Human Genetics, 98*(1), 91–101. https://doi.org/10.1007/s004390050166

Chen, C., Chen, C., Moyzis, R., Stern, H., He, Q., Li, H., … Dong, Q. (2011). Contributions of dopamine-related genes and environmental factors to highly sensitive personality: A multi-step neuronal system-level approach. *PLoS ONE, 6*(7), Article e21636. https://doi.org/10.1371/journal.pone.0021636

Cohen, J., Cohen, P., West, S. G., & Aiken, L. S. (2003). *Applied linear regression/correlation analysis for the behavioural sciences (3rd edition)*. Mahwah, NJ: Lawrence Erlbaum Associates.

Conley, D., Rauscher, E., & Siegal, M. L. (2013). Beyond orchids and dandelions: Testing the 5HTT "risky" allele for evidence of phenotypic capacitance and frequency dependent selection. *Biodemography and Social Biology, 144*(5), 724–732. https://doi.org/10.1038/jid.2014.371

Craik, F. I. M., & Lockhart, R. S. (1972). Levels of processing: A framework for memory research. *Journal of Verbal Learning and Verbal Behavior, 11*, 671–684.

de Villiers, B., Lionetti, F., & Pluess, M. (2018). Vantage sensitivity: A framework for individual differences in response to psychological intervention. *Social Psychiatry and Psychiatric Epidemiology, 53*(6), 545–554. https://doi.org/10.1007/s00127-017-1471-0

Eekhout, I., De Vet, Twisk, J. W., Brand, J. P., De Boer, & Heymans, M. W. (2014). Missing data in a multi-item instrument were best handled by multiple imputation at the item score level. *Journal of Clinical Epidemiology, 67*(3), 335–342. https://doi.org/10.1016/j.jclinepi.2013.09.009

Ellis, B. J., Boyce, W. T., Belsky, J., & Bakermans-Kranenburg, M. J. (2011). Differential susceptibility to the environment: An evolutionary–neurodevelopmental theory. *Development and Psychopathology, 23*, 7–28. https://doi.org/10.1017/S0954579410000611

Fan, S., Kelly, D. E., Beltrame, M. H., Hansen, M. E., Mallick, S., Ranciaro, A., … Tishkoff, S. A. (2019). African evolutionary history inferred from whole genome sequence data of 44 indigenous African populations. *Genome Biology, 20*(1), 1–14. https://doi.org/10.1186/s13059-019-1679-2

Esau, L., Kaur, M., Adonis, L., & Arieff, Z. (2008). The 5-HTTLPR polymorphism in South African healthy populations: a global comparison. *Journal of Neural Transmission, 115*(5), 755–760. https://doi.org/10.1007/s00702-007-0012-5

Evers, A., Rasche, J., & Schabracq, M. J. (2008). High sensory-processing sensitivity at work. *International Journal of Stress Management, 15*(2), 189–198. https://doi.org/10.1037/1072-5245.15.2.189

Feeley, A. B., Musenge, E., Pettifor, J. M., & Norris, S. A. (2013). Investigation into longitudinal dietary behaviours and household socio-economic indicators and their association with BMI Z-score and fat mass in South African adolescents: The Birth to Twenty (Bt20) cohort. *Public Health Nutrition, 16*(4), 693–703. https://doi.org/10.1017/S1368980012003308

Filmer, D., & Pritchett, L. (1999). The effect of household wealth on educational attainment: Evidence from 35 countries. *Population and Development Review, 25*(1), 85–120. https://doi.org/10.1111/j.1728-4457.1999.00085.x

Fox, J., & Weisberg, S. (2011). *An R companion to applied regression (2nd edition)*. Thousand Oaks, CA: Sage.

Frey, U. H., Bachmann, H. S., Peters, J., & Siffert, W. (2008). PCR-amplification of GC-rich regions: 'slowdown PCR'. *Nature Protocols, 3*(8), 1312–1317. https://doi.org/10.1038/nprot.2008.112

Gerstenberg, F. X. (2012). Sensory-processing sensitivity predicts performance on a visual search task followed by an increase in perceived stress. *Personality and Individual Differences, 53*(4), 496–500. https://doi.org/10.1016/j.paid.2012.04.019

Gesten, E. L. (1976). A health resources inventory: The development of a measure of the personal and social competence of primary-grade children. *Journal of Consulting and Clinical Psychology, 44*(5), 775–786. https://doi.org/10.1037/0022-006X.44.5.775
Goldberg, A., Ebraheem, Z., Freiberg, C., Ferarro, R., Chai, S., & Gottfried, O. D. (2018). Sweet and sensitive: Sensory processing sensitivity and type 1 diabetes. *Journal of Pediatric Nursing, 38*, e35–e38. https://doi.org/10.1016/j.pedn.2017.10.015
Greven, C. U., Lionetti, F., Booth, C., Aron, E. N., Fox, E., Schendan, H. E., … Homberg, J. (2019). Sensory processing sensitivity in the context of environmental sensitivity: A critical review and development of research agenda. *Neuroscience and Biobehavioral Reviews, 98*(January), 287–305. https://doi.org/10.1016/j.neubiorev.2019.01.009
Gutteling, B. M., De Weerth, C., & Buitelaar, J. K. (2005). Prenatal stress and children's cortisol reaction to the first day of school. *Psychoneuroendocrinology, 30*(6), 541–549. https://doi.org/10.1016/j.psyneuen.2005.01.002
Heils, A., Teufel, A., Petri, S., Stöber, G., Riederer, P., Bengel, D., & Lesch, K. P. (1996). Allelic variation of human serotonin transporter gene expression. *Journal of Neurochemistry, 66*(6), 2621–2624. https://doi.org/10.1046/j.1471-4159.1996.66062621.x
Heindel, J. J., & Vandenberg, L. N. (2015). Developmental origins of health and disease: A paradigm for understanding disease cause and prevention. *Current Opinion in Pediatrics, 27*(2), 248–253. https://doi.org/10.1097/MOP.0000000000000191
Hemmings, S. M. J., Xulu, K., Sommer, J., Hinsberger, M., Malan-Muller, S., Tromp, G., Elbert, T., Weierstall, R., & Seedat, S. (2018). Appetitive and reactive aggression are differentially associated with the STin2 genetic variant in the serotonin transporter gene. *Scientific Reports, 8*(1), 1–9. https://doi.org/10.1038/s41598-018-25066-8
Homberg, J. R., Schubert, D., Asan, E., & Aron, E. N. (2016). Sensory processing sensitivity and serotonin gene variance: Insights into mechanisms shaping environmental sensitivity. *Neuroscience and Biobehavioral Reviews, 71*, 472–483. https://doi.org/10.1016/j.neubiorev.2016.09.029
Huizink, A. C., de Medina, Mulder, E. J. H., Visser, G. H. A., & Buitelaar, J. K. (2002). Psychological measures of prenatal stress as predictors of infant temperament. *Journal of the American Academy of Child & Adolescent Psychiatry, 41*(9), 1078–1085. https://doi.org/10.1097/01.CHI.0000020276.43550.4B
Huizink, A. C., Bartels, M., Rose, R. J., Pulkkinen, L., Eriksson, C. J., & Kaprio, J. (2008). Chernobyl exposure as stressor during pregnancy and hormone levels in adolescent offspring. *Journal of Epidemiology and Community Health, 62*(4), 2–7. https://doi.org/10.1136/jech.2007.060350
Huxley, R. R., Shiell, A. W., & Law, C. M. (2000). The role of size at birth and postnatal catch-up growth in determining systolic blood pressure: A systematic review of the literature. *Journal of Hypertension, 18*(7), 815–831. https://doi.org/10.1097/00004872-200018070-00002
Jagiellowicz, J., Aron, A., & Aron, E. N. (2016). Relationship between the temperament trait of sensory processing sensitivity and emotional reactivity. *Social Behavior and Personality, 44*(2), 185–200. https://doi.org/10.2224/sbp.2016.44.2.185
Jakobsen, J. C., Gluud, C., Wetterslev, J., & Winkel, P. (2017). When and how should multiple imputation be used for handling missing data in randomised clinical trials - A practical guide with flowcharts. *BMC Medical Research Methodology, 17*(1), 1–10. https://doi.org/10.1186/s12874-017-0442-1
Keers, R., Coleman, J. R., Lester, K. J., Roberts, S., Breen, G., Thastum, M., … Eley, T. C. (2016). A genome-wide test of the differential susceptibility hypothesis reveals a genetic predictor of differential response to psychological treatments for child anxiety disorders. *Psychotherapy and Psychosomatics, 85*(3), 146–158. https://doi.org/10.1159/000444023
Keers, R., & Pluess, M. (2017). Childhood quality influences genetic sensitivity to environmental influences across adulthood: A life-course Gene x Environment interaction study. *Development and Psychopathology, 29*(5), 1921–1933. https://doi.org/10.1017/S0954579417001493
Khashan, A. S., Kenny, L. C., Lundholm, C., Kearney, P. M., Gong, T., McNamee, R., & Almqvist, C. (2015). Gestational age and birth weight and the risk of childhood type 1 diabetes: A population-based cohort and sibling design study. *Diabetes Care, 38*(12), 2308–2315. https://doi.org/10.2337/dc15-0897
Levit-Binnun, N., Szepsenwol, O., Stern-Ellran, K., & Engel-Yeger, B. (2014). The relationship between sensory responsiveness profiles, attachment orientations, and anxiety symptoms attachment orientations, and anxiety symptoms. *Australian Journal of Psychology, 66*(4), 233–240. https://doi.org/10.1111/ajpy.12064
Licht, C. L., Mortensen, E. L., & Knudsen, G. M. (2011). Association between Sensory Processing Sensitivity and the 5-HTTLPR Short / Short Genotype. Biological Psychiatry, 69, 152S–153S, Abstract 510.
Licht, C. L., Mortensen, E. L., Hjordt, L. V., Stenbæk, D. S., Arentzen, T. E., Nørremølle, A., & Knudsen, G. M. (2020). Serotonin transporter gene (SLC6A4) variation and sensory processing sensitivity—Comparison with other anxiety-related temperamental dimensions. *Molecular Genetics and Genomic Medicine, 8*(8), 1–10. https://doi.org/10.1002/mgg3.1352
Lichter, J. B., Barr, C. L., Kenney, J. L., Van Tol, H. H. M., Kidd, K. K., & Livak, K. J. (1993). A hypervariable segment in the human dopamine D4 (DRD4) gene. *Human Molecular Genetics, 2*(6), 767–773. https://doi.org/10.1093/hmg/2.6.767
Lima, S. A. M., El Dib, R. P., Rodrigues, M. R. K., Ferraz, G. A. R., Molina, A. C., Neto, C. A. P., De Lima, M. A. F., & Rudge, M. V. C. (2018). Is the risk of low birth weight or preterm labor greater when maternal stress is experienced during pregnancy? A systematic review and meta-analysis of cohort studies. *PLoS ONE, 13*(7), 1–15. https://doi.org/10.1371/journal.pone.0200594
Lionetti, F., Aron, A., Aron, E. N., Burns, G. L., Jagiellowicz, J., & Pluess, M. (2018). Dandelions, tulips and orchids: Evidence for the existence of low-sensitive, medium-sensitive and high-sensitive individuals. *Translational Psychiatry, 8*(24). https://doi.org/10.1038/s41398-017-0090-6
Lionetti, F., Aron, E. N., Aron, A., Klein, D. N., & Pluess, M. (2019). Observer-rated environmental sensitivity moderates children's response to parenting quality in early childhood. *Developmental Psychology, 55*(11), 2389–2402. https://doi.org/10.1037/dev0000795.supp
Loret De Mola, C., De Franca, De Avila Quevedo, L., & Horta, B. L. (2014). Low birth weight, preterm birth and small for gestational age association with adult depression: Systematic review and meta-analysis. *British Journal of Psychiatry, 205*(5), 340–347. https://doi.org/10.1192/bjp.bp.113.139014
Lüdecke, D. (2018). sjPlot: Data visualization for statistics in social science. https://cran.r-project.org/package=sjPlot.
Madley-Dowd, P., Hughes, R., Tilling, K., & Heron, J. (2019). The proportion of missing data should not be used to guide decisions on multiple imputation. *Journal of Clinical Epidemiology, 110*, 63–73. https://doi.org/10.1016/j.jclinepi.2019.02.016
Malan, C. (2014). *Allelic diversity of selected human neurotransmitter genes in South African ethnic groups*. University of the Free State. Doctoral dissertation.
May, A., Hazelhurst, S., Li, Y., Norris, S., Govind, N., Tikly, M., … Ramsay, M. (2013). Genetic diversity in black South Africans from Soweto. *BMC Genomics, 14*(1). https://doi.org/10.1186/1471-2164-14-644
May, A., Norris, S. A., Richter, L. M., & Pitman, M. (2020). A psychometric evaluation of the Highly Sensitive Person Scale in ethnically and culturally heterogeneous South African samples. *Current Psychology*. https://doi.org/10.1007/s12144-020-00988-7
Mehrabian, A. (1977). Individual differences in stimulus screening and arousability. *Journal of Personality, 45*(2), 237–250. https://doi.org/10.1111/j.1467-6494.1977.tb00149.x
Miller, S. A., Dykes, D., & Polesky, H. (1988). A simple salting out procedure for extracting DNA from human nucleated cells. *Nucleic Acids Research, 16*(3), 1215. https://doi.org/10.1016/B978-0-7506-7152-1.50011-4
Mirzaei, A., Carter, S. R., Patanwala, A. E., & Schneider, C. R. (2022). Missing data in surveys: Key concepts, approaches, and applications. *Research in Social and Administrative Pharmacy, 18*(2), 2308–2316. https://doi.org/10.1016/j.sapharm.2021.03.009
Morgan, B., Kumsta, R., Fearon, P., Moser, D., Skeen, S., Cooper, P., … Tomlinson, M. (2017). Serotonin transporter gene (SLC6A4) polymorphism and susceptibility to a home-visiting maternal-infant attachment intervention delivered by community health workers in South Africa: Reanalysis of a randomized controlled trial. *PLoS Medicine, 14*(2), Article e1002237. https://doi.org/10.1371/journal.pmed.1002237
National Center for Education Statistics. (1993). *Public school kindergarten teachers' views on children's readiness for school* (tech. rep.). Washington, DC: U.S. Department of Education, Office of Educational Research and Improvement.
Nocentini, A., Menesini, E., & Pluess, M. (2018). The personality trait of environmental sensitivity predicts children's positive response to school-based antibullying intervention. *Clinical Psychological Science, 6*(6), 848–859. https://doi.org/10.1177/2167702618782194
O'Connor, T. G., Ben-Shlomo, Y., Heron, J., Golding, J., Adams, D., & Glover, V. (2005). Prenatal anxiety predicts individual differences in cortisol in pre-adolescent children. *Biological Psychiatry, 58*(3), 211–217. https://doi.org/10.1016/j.biopsych.2005.03.032
O'Donnell, K. J., & Meaney, M. J. (2017). Fetal origins of mental health: The developmental origins of health and disease hypothesis. *American Journal of Psychiatry, 174*(4), 319–328. https://doi.org/10.1176/appi.ajp.2016.16020138
Oudgenoeg-Paz, O., Mulder, H., Jongmans, M. J., Ham, I. J. M. V. D., & Stigchel, S. V. D. (2017). The link between motor and cognitive development in children born preterm and / or with low birth weight : A review of current evidence. *Neuroscience and Biobehavioral Reviews, 80*(May), 382–393. https://doi.org/10.1016/j.neubiorev.2017.06.009
Pesonen, A. K., Räikkönen, K., Strandberg, T. E., & Järvenpää, A. L. (2005). Continuity of maternal stress from the pre- to the postnatal period: Associations with infant's positive, negative and overall temperamental reactivity. *Infant Behavior and Development, 28*(1), 36–47. https://doi.org/10.1016/j.infbeh.2004.09.001
Peterson, J. L., & Zill, N. (1986). Marital disruption, parent-child relationships, and behavior problems in children. *Journal of Marriage and Family, 48*(2), 295–307. https://doi.org/10.2307/352397
Peterson, R. A. (2017). bestNormalize: A suite of normalizing transformations. https://cran.r-project.org/web/packages/bestNormalize/bestNormalize.pdf.
Pluess, M. (2015). Individual differences in environmental sensitivity. *Child Development Perspectives, 9*(3), 138–143. https://doi.org/10.1111/cdep.12120
Pluess, M., & Belsky, J. (2011). Prenatal programming of postnatal plasticity? *Development and Psychopathology, 23*, 29–38. https://doi.org/10.1017/S0954579410000623
Pluess, M., & Boniwell, I. (2015). Sensory-processing sensitivity predicts treatment response to a school-based depression prevention program: Evidence of vantage sensitivity. *Personality and Individual Differences, 82*, 40–45. https://doi.org/10.1016/j.paid.2015.03.011
Pluess, M., Assary, E., Lionetti, F., Lester, K. J., Krapohl, E., Aron, E. N., & Aron, A. (2018). Environmental sensitivity in children: Development of the highly sensitive child scale and identification of sensitivity groups. *Developmental Psychology, 54*(1), 51–70. https://doi.org/10.1037/dev0000406
Pluess, M., Rhoades, G., Keers, R., Knopp, K., Belsky, J., Markman, H., & Stanley, S. (2022). Genetic Sensitivity Predicts Long-Term Psychological Benefits of a Relationship Education Program for Married Couples. *Journal of Consulting and Clinical Psychology, 90*(2), 195–207. https://doi.org/10.1037/ccp0000715
Raju, T. N., Buist, A. S., Blaisdell, C. J., Moxey-Mims, M., & Saigal, S. (2017). Adults born preterm: A review of general health and system-specific outcomes. *Acta Paediatrica, 106*(9), 1409–1437. https://doi.org/10.1111/apa.13880
Ramchandani, P. G., Richter, L. M., Norris, S. A., & Stein, A. (2010). Maternal prenatal stress and later child behavioral problems in an urban South African setting. *Journal of the American Academy of Child and Adolescent Psychiatry, 49*(3), 239–247. https://doi.org/10.1016/j.jaac.2009.11.013

Ramokolo, V., Malaba, T., Rhoda, N., Kauchali, S., & Goga, A. (2019). A landscape analysis of preterm birth in South Africa: Systemic gaps and solutions. In T. Moeti, & A. Padarath (Eds.), *South African Health Review 2019* (pp. 133–144).

Rappaport, M. B., & Corbally, C. (2018). Evolution of religious capacity in the genus homo: Cognitive time sequence. *Zygon, 53*(1), 159–197. https://doi.org/10.1111/zygo.12387

Revelle, W. (2018). psych: Procedures for personality and psychological research. https://cran.r-project.org/package=psych.

Richter, L. M., Mathews, S., Kagura, J., & Nonterah, E. (2018). A longitudinal perspective on violence in the lives of South African children from the Birth to Twenty Plus cohort study in Johannesburg-Soweto. *South African Medical Journal, 108*(3), 181–186. https://doi.org/10.7196/SAMJ.2018.v108i3.12661

Richter, L. M., Norris, S. A., & De Wet, T. (2004). Transition from birth to ten to birth to twenty: The South African cohort reaches 13 years of age. *Paediatric and Perinatal Epidemiology, 18*(4), 290–301. https://doi.org/10.1111/j.1365-3016.2004.00572.x

Richter, L., Norris, S., Pettifor, J., Yach, D., & Cameron, N. (2007). Cohort profile: Mandela's children: The 1990 birth to twenty study in South Africa. *International Journal of Epidemiology*, 1–8. https://doi.org/10.1093/ije/dym016

Ruth, C. A., Roos, N., Hildes-Ripstein, E., & Brownell, M. (2012). The influence of gestational age and socioeconomic status on neonatal outcomes in late preterm and early term gestation: A population based study. *BMC Pregnancy and Childbirth, 12*, 9–11. https://doi.org/10.1186/1471-2393-12-62

Sabol, S. Z., Hu, S., & Hamer, D. (1998). A functional polymorphism in the monoamine oxidase A gene promoter. *Human Genetics, 103*(3), 273–279. https://doi.org/10.1007/s004390050816

Sengül-Inal, G., & Sümer, N. (2017). Exploring the multidimensional structure of sensory processing sensitivity in Turkish samples. *Current Psychology, 39*, 194–206. https://doi.org/10.1007/s12144-017-9751-0

Smith, H. L., Sriken, J., & Erford, B. T. (2019). Clinical and research utility of the highly sensitive person scale. *Journal of Mental Health Counseling, 41*(3), 221–241. https://doi.org/10.17744/mehc.41.3.03

Smolewska, K. A., McCabe, S. B., & Woody, E. Z. (2006). A psychometric evaluation of the Highly Sensitive Person Scale: The components of sensory-processing sensitivity and their relation to the BIS/BAS and "Big Five". *Personality and Individual Differences, 40*(6), 1269–1279. https://doi.org/10.1016/j.paid.2005.09.022

van Buuren, S., & Groothuis-Oudshoorn, K. (2011). mice: Multivariate imputation by chained equations in R. *Journal of Statistical Software, 45*(3), 1–67. 10.18637/jss.v045.i03.

Verkuijl, N. E., Richter, L., Norris, S. A., Stein, A., Avan, B., & Ramchandani, P. G. (2014). Postnatal depressive symptoms and child psychological development at 10 years: A prospective study of longitudinal data from the South African Birth to Twenty cohort. *The Lancet Psychiatry, 1*(6), 454–460. https://doi.org/10.1016/S2215-0366(14)70361-X

Voldner, N., Frey Frøslie, K., Godang, K., Bollerslev, J., & Henriksen, T. (2009). Determinants of birth weight in boys and girls. *Human Ontogenetics, 3*(1), 7–12. https://doi.org/10.1002/huon.200900001

Ward, C. L., Artz, L., Berg, J., Boonzaier, F., Crawford-Browne, S., Dawes, A., Foster, D., Matzopoulos, R., Nicol, A., Seekings, J., van As, A. B., & Van Der Spuy, E. (2012). Violence, violence prevention, and safety: A research agenda for South Africa. *South African Medical Journal, 102*(4), 215–218. https://doi.org/10.7196/SAMJ.5544

Wickham, H. (2016). *ggplot2: Elegant graphics for data analysis*. New York, NY: Springer-Verlag.

Wilcox, A. J. (2001). On the importance-and the unimportance-of birthweight. *International Journal of Epidemiology, 30*(6), 1233–1241. https://doi.org/10.1093/ije/30.6.1233

World Health Organisation (2016). ICD-10: *International statistical classification of diseases and related health problems: Tenth revision, fifth edition* (tech. rep.). https://apps.who.int/iris/handle/10665/246208.

Wu, X., Zhang, R., Li, X., Feng, T., & Yan, N. (2021). The moderating role of sensory processing sensitivity in the link between stress and depression: A VBM study. *Neuropsychologia, 150*(September 2020), Article 107704. https://doi.org/10.1016/j.neuropsychologia.2020.107704

Yano, K., Kase, T., & Oishi, K. (2020). Sensory processing sensitivity moderates the relationships between life skills and depressive tendencies in university students. *Japanese Psychological Research, 63*(3), 152–163. https://doi.org/10.1111/jpr.12289