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**Regular Article**

# Developmental programming of shyness: A longitudinal, prospective study across four decades

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**Abstract**

Although shyness is a ubiquitous phenomenon with early developmental origins, little research has examined the influence of prenatal exposures on the developmental trajectory of shyness. Here, we examined trajectories of shyness from childhood to adulthood in three groups ($N = 254$), with varying degrees of prenatal adversity as indicated by the number of stressful exposures: extremely low birth weight (ELBW; <1000 g) survivors prenatally exposed to exogenous corticosteroids (ELBW+S, $n = 56$); ELBW survivors not prenatally exposed to exogenous corticosteroids (ELBW+NS, $n = 56$); and normal birth weight (NBW, $n = 142$) controls. Multilevel modeling revealed that the ELBW+S individuals exhibited the highest levels of childhood shyness, which remained stable into adulthood. The ELBW+NS and NBW controls had comparably low levels of childhood shyness; however, the ELBW+NS individuals experienced patterns of increasing shyness, while NBW controls displayed decreases in shyness into adulthood. We speculate that individuals exposed to multiple prenatal stressors (i.e., ELBW+S) may be developmentally programmed to be more sensitive to detecting social threat, with one manifestation being early developing, stable shyness, while increasing shyness among ELBW+NS individuals may reflect a later developing shyness influenced by postnatal context. We discuss the implications of these findings for understanding the developmental origins and developmental course of human shyness from childhood through adulthood.

**Keywords:** longitudinal studies, personality, prematurity, prenatal programming, shyness

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Development is a dynamic process in which very early perturbations can alter the psychobiological systems responsible for temperament and personality (Gartstein & Skinner, 2017; Krzeczkowski & Van Lieshout, 2018). Developmental programming hypotheses posit that individuals who are exposed to prenatal and early postnatal stressors may have altered maturation of central and peripheral physiological systems responsible for the regulation of stress, such as the prefrontal cortex, amygdala, and hypothalamic–pituitary–adrenal (HPA) axis (Gluckman, Hanson, & Buklijas, 2010; Harris & Seckl, 2011). These biological changes are thought to be adaptive in that they increase the fetus' *immediate* survival within a stressful prenatal environment (Bateson, Gluckman, & Hanson, 2014; Van Den Bergh, 2011). However, when the postnatal context is not comparably harsh and threatening as the prenatal environment, the individual manifests *long-term* programmed changes in the ability to handle stress and a predisposition to stress-reactivity and threat sensitivity (e.g., Bolten et al., 2013; Davis et al., 2007; DiPietro, Hodgson, Costigan, & Johnson, 1996; Pesonen et al., 2006; Werner et al., 2007; see also Gartstein & Skinner, 2017, for a review).

While emerging work has highlighted general behavioral domains impacted by early exposure to stressors, we know relatively little about *specific* temperaments or personalities that may be developmentally programmed. Some work has found that prenatal stress and administration of glucocorticoids in utero have resulted in fearful behaviors in response to novelty in rodent models (Dickerson, Lally, Gunnel, Birkle, & Salm, 2005, Van den Hove et al., 2005, Weinstock, 2005) and among nonhuman primates (Schneider, 1992; Schneider, Coe, & Lubach, 1992). These behavioral manifestations are comparable to behavioral inhibition in humans (Garcia-Coll, Kagan, & Reznick, 1984). Limited work in humans has likewise found that early exposure to glucocorticoids can predict cortisol dysregulation, behavioral inhibition, fearfulness, and anxiety in offspring (Davis et al., 2004; de Weerth, van Hees, & Buitelaay, 2003; Trautman, Meyer-Bahlburg, Postelnek, & New, 1995). Collectively, these data suggest that prenatal exposure to glucocorticoids may result in programmed changes in the infant with one of the main behavioral outcomes being inhibition and fear in response to novelty (early developmental precursors of shyness). Therefore, we hypothesized that one ubiquitous temperamental

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characteristic that may be particularly relevant to study in the context of developmental programming is shyness.

Shyness is a trait characterized by fear and inhibition in response to social novelty and/or situations of perceived social evaluation (Melchior & Cheek, 1990). Shyness is associated with physiological and behavioral stress reactivity (e.g., Kagan, Reznick, & Snidman 1987, 1988; Poole & Schmidt, 2018; Schmidt, 1999; Schmidt & Fox, 1994; Schmidt et al., 1997; Schmidt, Fox, Schulkin, & Gold, 1999) as well as increased detection of threat-related stimuli (LoBue & Pérez-Edgar, 2014; Pérez-Edgar et al., 2010, 2011). Likewise, exposure to prenatal adversity, including glucocorticoids, can result in altered development of neural systems, including limbic regions and prefrontal cortex, involved in fear regulation as well as increase the sensitivity of the HPA axis (see Bock, Rether, Gröger, Xie, & Braun, 2014; McEwen et al., 2015, for a review). Thus, it is possible that the experience of early adversity may result in alterations of stress response systems and the expression of negative affect, behavioral inhibition, and hypervigilance, which could lay the developmental foundation for prospective shyness (Gartstein & Skinner, 2017).

Some cross-sectional work has examined the influence of prenatal stressors on the development of shyness or closely related constructs. These studies found evidence that lower birth weight (Pesonen et al., 2009) and prenatal corticosteroid exposure (dexamethasone; Trautman et al., 1995) were correlated with more shyness and avoidance at 2 years of age. Additional work has found that prematurity (Tessier, Nadeau, Boivin, & Tremblay, 1997) and prenatal corticosteroid exposure (betamethasone; Erni, Shaqiri, La Marca, Zimmermann, & Ehlert, 2012) are correlated with more social withdrawal and social-evaluative stress, respectively, in childhood (age 10–12 years). Still other studies have found that lower birth weight was associated with higher levels of introversion (Allin et al., 2006; Hertz, Mathiasen, Hansen, Mortensen, & Greisen, 2013; Pesonen et al., 2008), behavioral inhibition (Pyhälä et al., 2009), social withdrawal (Eryigit-Madzwamuse, Strauss, Baumann, Bartmannm, & Wolke, 2015; Hack et al., 2004) and shyness in young adulthood (early 20s; Schmidt, Miskovic, Boyle, & Saigal, 2008) and adulthood (early 30s; Waxman, Van Lieshout, Saigal, Boyle, & Schmidt, 2013; Xu Poole, Van Lieshout, Saigal, & Schmidt, 2018), as well as harm avoidance in later adulthood (age 60 years; Lahti et al., 2008).

Although individuals exposed to early stressors may display behavioral profiles characterized by shyness in cross-sectional studies, considerably less is known about prospective *longitudinal* development of shyness from childhood to adulthood. The reason for this is that existing studies generally measure or analyze shyness or related behavior during single developmental periods and employ cross-sectional analyses. While such approaches are important, they do not reveal whether these phenotypes exhibit *stability* or *change* across development. It is important to have information on development starting in childhood and measured across repeated assessments, as this allows a valuable opportunity to examine *trajectories* of personality and information on the emergence of specific traits and the generation of hypotheses around factors involved in altering these trajectories. This is particularly relevant in the study of shyness, as previous work has illustrated heterogeneity in the developmental onset of shyness, including both early and later developing subtypes (Booth-LaForce & Oxford, 2008; Oh et al., 2008; Tang et al., 2017), which may have different underlying biological origins, different contextual influences, and different developmental outcomes (see Poole, Tang, & Schmidt, 2018, for a review). Thus, studying early developmental influences on prospectively and longitudinally measured shyness has implications for understanding heterogeneity in its developmental onset and course.

An additional gap in the context of developmental programming is that it remains unclear if the relative number of prenatal exposures may influence personality development. There is some evidence of possible cumulative effects of early adversity on levels of social-evaluative stress in a cross-sectional study of children (Erni et al., 2012). However, to our knowledge, no work has tested this within a longitudinal framework. Doing so may reveal whether multiple prenatal exposures can result in cumulative effects of early adversity that contribute to differing long-term effects on the development, stability, or change in shyness across time (Gartstein & Skinner, 2017).

It appears that independent exposure to prematurity or prenatal corticosteroids may result in shyness, and thus it is plausible that individuals who experience *both* of these exposures may be particularly susceptible to experience developmental programming effects. Infants born at extremely low birth weight (ELBW; <1000 g) are the tiniest and most at-risk babies and are susceptible to a number of stress-related problems across development (see Mathewson et al., 2017, for a review), and also exhibit physiological correlates of stress-vulnerability (Krzeczkowski, Schmidt, Savoy, Saigal, & Van Lieshout, 2018; Schmidt, Miskovic, Boyle, & Saigal, 2010). In addition, some individuals born at ELBW are *also* exposed prenatally to exogenous corticosteroids, which are given to women at risk for preterm labor (Waffrin & Davis, 2012). Although these synthetic corticosteroids are an effective and important therapy for reducing infant mortality, exposure to these steroids prenatally can alter the developing brain and stress regulation systems (Davis, Waffran, & Sandman, 2011; Savoy, Ferro, Schmidt, Saigal, & Van Lieshout, 2016; see Welberg & Seckl, 2001, for an exhaustive review). For example, exposure to glucocorticoids can influence developing neural systems involved in fear regulation and detection (see Bock et al., 2014; McEwen et al., 2015, for a review). The reason is that while approximately 80% of maternal cortisol is metabolized prior to entering fetal circulation, synthetic corticosteroids such as those administered for preterm labor *are not* metabolized (Waffrin & Davis, 2012). Consequently, the developing fetus brain is exposed to abnormally high levels of synthetic cortisol, which can lead to HPA axis dysregulation (Moisiadis & Matthews, 2014). There exists some work suggesting that ELBW infants who are also exposed prenatally to corticosteroids may exhibit greater stress-vulnerability relative to those who were not exposed, as indicated by greater relative right frontal brain activity (Krzeczkowski et al., 2018) and increased risk for anxiety disorders (Savoy et al., 2016; Van Lieshout, Boyle, Saigal, Morrison, & Schmidt, 2015); these are also physiological and psychological correlates of shyness.

## The Present Study

We conducted a longitudinal, prospective study spanning four decades to test whether increasing levels of perinatal adversity, specifically extreme prematurity and prenatal exposure to exogenous corticosteroids, were associated with the developmental onset and developmental course of shyness from childhood to adulthood. To this end, we used multilevel modeling to delineate the trajectory of shyness from age 8 to age 32 in three groups with varying degrees of exposure to prenatal adversity. From the highest to lowest levels of prenatal adversity, these groups included

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ELBW survivors who were also prenatally exposed to exogenous corticosteroids (ELBW+S); ELBW survivors who were not prenatally exposed to exogenous corticosteroids (ELBW+NS), and normal birth weight (NBW; >2500 g) controls.

We tested three hypotheses. First, because ELBW+S individuals had ostensibly more stressful early exposures relative to the other groups, we hypothesized they would manifest high levels of shyness in childhood that remain stable into adulthood, resulting from a predisposition to experience heightened postnatal stress-reactivity, threat sensitivity, and emotion dysregulation (Gartstein & Skinner, 2017). Second, we hypothesized that ELBW+NS individuals would display moderate, stable levels of shyness given their intermediate level of early stress exposure. Third, we hypothesized that NBW controls would display low, stable levels of shyness, or decreases in shyness across development in line with previous work in typically developing individuals (Dennissen, Asendorpf, & Van Aken, 2008).

## Method

### Sample overview

The ELBW sample was recruited at birth and comprised 397 predominantly Caucasian infants born at less than 1000 g between 1977 and 1982 to residents of central-west and has been prospectively followed since birth. Of these, 179/397 (45%) survived to hospital discharge. In the present study, all participants with neurosensory impairment (NSI; $n = 51$), defined as the presence of at least one of cerebral palsy, blindness, deafness, intellectual disability, or microcephaly diagnosed in childhood by a neonatologist or developmental pediatrician, were excluded because they have unique challenges that are not generalizable to the majority of those born preterm.

Follow-up assessments on this group have been conducted during childhood (8 years), adolescence (12 to 16 years), young adulthood (22 to 26 years), and adulthood (30 to 35 years). We will use the developmental period to describe each assessment for the remainder of the paper. At the childhood assessment, 108 (84.4%) ELBW survivors had complete shyness data; at the adolescent assessment, 102 (80.0%) ELBW survivors had complete shyness data; at the young adulthood assessment, 109 (85.2%) ELBW survivors had complete shyness data; and at the adulthood assessment, 71 (55.4%) ELBW survivors had complete shyness data. Participants who had shyness data for at least one visit ($n = 112$) were included in the analyses. Of these ELBW participants, a total of 56 (50%) mothers received prenatal administration of betamethasone (ELBW+S), whereas 56 (50%) mothers did not receive prenatal administration of betamethasone (ELBW+NS).

Participants in the NBW control group were recruited when they and the ELBW survivors were 8 years old. These 145 children were selected from a random sample of students in the Hamilton Public School System (Ontario) who were born at full term and matched with the ELBW participants on age, sex, and family socioeconomic status (SES) at the childhood assessment. Subsequent assessments have occurred at the same ages as the ELBW cohort. At the childhood assessment, 139 (95.9%) NBW children had complete shyness data; at the adolescent assessment, 120 (82.8%) NBW adolescents had complete shyness data; at the young adulthood assessment, 129 (90.0%) NBW participants had complete shyness data; and at the adulthood assessment, 85 (58.6%) NBW controls had complete shyness data. Participants who had shyness data for at least one visit ($n = 142$) were included in the analyses.

### Procedures

Study assessments were conducted at McMaster Children's Hospital for the childhood and adolescent assessment, and at the Child Emotion Laboratory at McMaster University for both adulthood assessments. After a complete description of the study was provided, written informed consent was obtained from the parents of all participants during the childhood and adolescent assessments, and by the participants themselves during the adult assessments. The Hamilton Health Sciences Research Ethics Board approved all study procedures.

### Shyness measure

#### Childhood assessment

Shyness was measured in childhood using seven items from the parent-report Child Behavior Checklist (Achenbach & Edelbrock, 1983). Sample items included "Child is shy or timid" and "Child is self-conscious or easily embarrassed," and parents rate how characteristic each item is on a 3-point Likert scale (0 = *not true*, 1 = *sometimes or somewhat true*, 2 = *very true or often true*). This scale demonstrated acceptable internal reliability in our sample ($\alpha = 0.63$).

#### Adolescent assessment

Shyness was measured in adolescence using five items from the parent-reported Ontario Child Health Study—Revised (Boyle et al., 1987) Questionnaire. Sample items from this included "Adolescent is self-conscious/easily embarrassed" and "Adolescent is withdrawn," and parents rate how characteristic each item is on a 3-point Likert scale (0 = *not true*, 1 = *sometimes or somewhat true*, 2 = *very true or often true*). This scale demonstrated acceptable internal reliability in our sample ($\alpha = 0.66$).

#### Young adulthood and adulthood assessments

At both adult visits, participants completed the Young Adult Self-Report (YASR; Achenbach, 1997), and the seven items that comprised the shyness scale. Sample items included "I am too shy or timid" and "I am self-conscious or easily embarrassed," and participants rate how characteristic each item is on a 3-point Likert scale (0 = *not true*, 1 = *sometimes or somewhat true*, 2 = *very true or often true*). The scale demonstrated good internal reliability at both visits (age 22–26: $\alpha = 0.77$; age 30–35: $\alpha = 0.78$). This shyness scale demonstrated concurrent validity with the Cheek and Buss Shyness Scale (CBSS; Cheek, 1983; Cheek & Buss, 1981) that was administered at each adult visit (age 22–26: $r = .60$, $p < .001$; age 30–35: $r = .68$, $p < .001$).[1]

### Prenatal corticosteroid exposure

ELBW survivors were identified as being exposed to steroids prenatally if they were born to mothers who received one or two doses

1. Given that the CBSS was only administered at the adult visits, it was not included in our composite shyness score, and was reported to provide convergent validity with our shyness measure using items from the YASR. However, when computing a shyness composite including both the CBSS and YASR shyness items for the adult visits and using this as the dependent measure, the statistical significance and direction of the reported results remain unchanged.

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(12 mg/dose) of betamethasone administered intramuscularly in a single 24-hr period. Mothers were administered betamethasone at the discretion of their attending physician if they were at risk for preterm delivery. Information regarding the dose and exposure was obtained from medical records.

### Covariates

We considered a number of covariates in our analyses that may alter socioemotional development, including participant sex, intellectual quotient (IQ), and SES.

#### Sex

Data on the biological sex of the participants were drawn from the ELBW participants' medical charts and were reported by parents of NBW participants at age 8.

#### IQ

IQ was assessed by using the Wechsler Intelligence Scale for Children—Revised, which was administered at age 8. The Wechsler Intelligence Scale for Children—Revised consists of 10 subtests and combining these subtests creates a performance IQ scale (i.e., assessment of fluid intelligence) and a verbal IQ scale (i.e., assessment of reading, verbal, and language abilities). Together, these two subscales are combined to reflect an overall IQ, estimating overall intelligence, which was used in the present study (Wechsler, 1974).

#### SES

SES was modeled as a time-varying covariate, meaning that we accounted for changes in SES across each of the four visits. At the childhood and adolescent assessments, parental SES was assessed via parental reports using the Hollingshead two-factor index that uses educational attainment and occupational prestige as indicators of social position (Hollingshead, 1969). This index has five levels, and for comparison with our adult measures of SES, we recoded SES such that 1 indicated the lowest SES level and 5 indicated the highest SES level. In order to attempt to remain as consistent as possible, for both adulthood assessments we used educational attainment and household income as our primary indicators of SES. Educational attainment was self-reported and calculated by summing the years of education completed at the time of testing, and mean household income was self-reported. These two indices of SES were averaged and combined into a composite measure at each adult visit. To ensure consistency across measures, indicators of SES at each assessment were *z*-scored and included as a time-varying covariate.

### Statistical analyses

Sample characteristics between groups (i.e., ELBW+S, ELBW+NS, and NBW) were examined using analyses of variance for continuous measures, and chi-squared tests for categorical variables. Pearson's correlations assessed the relation of shyness scores across visits.

Shyness trajectories from childhood to adulthood were delineated using multilevel modeling in which repeated measures (i.e., shyness) are regressed on the timing of these assessments (i.e., participant age) to estimate rates of change at an individual level. Growth curve analysis provides estimates pertaining to variability in baseline shyness (i.e., intercept variance) as well as the possibility that individuals' shyness changes at different rates (i.e., slope variance; DeLucia & Pitts, 2005). Because the items comprising the measures of shyness varied slightly across some visits, *z* scores were computed and used as the dependent measure.

We examined whether prenatal exposure status (i.e., ELBW+S, ELBW+NS, and NBW) affected the initial status (i.e., shyness at childhood) and the rate of change of shyness across assessments (i.e., trajectory of shyness from childhood to adulthood). The reference category for this analysis was the ELBW+NS group. Maximum likelihood was used to account for missing data in the growth models to present unbiased estimates. We examined two models including an unadjusted model, and a fully adjusted model controlling for covariates (i.e., sex, IQ, and SES).

## Results

### Descriptive statistics

Sample characteristics are presented in Table 1. Pearson's correlations and mean levels of shyness scores across each of the four visits are presented in Table 2. Shyness scores at each visit were normally distributed.

### Trajectories of shyness

The parameter estimates for the unadjusted and adjusted growth curve models are shown in Table 3. Results indicated that ELBW+S participants had significantly higher levels of shyness in childhood relative to the ELBW+NS participants ($\beta = 0.42$; $p < .05$), with a stable trajectory into adulthood (as indicated by a nonsignificant slope: $\beta = -0.01$; $p = ns$). The NBW and ELBW+NS participants did not significantly differ on childhood shyness ($\beta = 0.34$; $p = ns$), but there was divergence in shyness levels from childhood to adulthood in the ELBW+NS and NBW participants. As illustrated in Figure 1, the NBW participants demonstrated decreases in shyness into adulthood relative to the ELBW+NS participants ($\beta = -0.02$; $p < .05$), who displayed relative increases, with levels comparable to the ELBW+S participants by adulthood.[2]

## Discussion

Previous theoretical frameworks have posited that in utero exposure to adversity and stressful environments can exert long-term influences on behavior and socioemotional development (Gartstein & Skinner, 2017; Gluckman et al., 2010). Our findings converge with this notion and suggest that the relative severity of early prenatal stressors may influence the emergence and trajectory of shyness across different developmental periods. Using a prospective, longitudinal study, we found that individuals who were born at ELBW *and* exposed prenatally to corticosteroids exhibited the highest levels of shyness in childhood that remained stable into their early 30s. We further report that ELBW survivors who *were not* exposed prenatally to corticosteroids had comparably low levels of childhood shyness relative to the NBW controls; however, whereas the NBW controls displayed decreases in shyness from childhood to adulthood, the ELBW+NS individuals experienced patterns of increasing shyness into their 30s.

2. Because previous research has suggested that males and females may be differentially vulnerable to the physiological and behavioral effects of prenatal environmental exposures (Gartstein & Skinner, 2017), we also considered sex as a *moderator* on the influence of prenatal stress and trajectories of shyness, and no significant effects were observed.

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**Table 1.** Sample characteristics

| | ELBW+S ($n = 56$) | ELBW+NS ($n = 56$) | NBW ($n = 142$) | |
|---|---|---|---|---|
| Birth weight in grams, mean (*SD*) | 836.33 (112.26)[a] | 836.50 (128.73)[b] | 3376.18 (492.11)[a,b] | $p < .001$ |
| Gestational age in weeks, mean (*SD*) | 26.98 (1.64)[a] | 27.60 (2.89)[b] | 40[a,b] | $p < .001$ |
| Small for gestational age, *n* (%) | 18 (32.14%) | 15 (26.78%) | N/A | $p > .05$ |
| Males, *n* (%) | 25 (41.66%) | 29 (42.65%) | 66 (45.52%) | $p > .05$ |
| Childhood IQ, mean (*SD*) | 94.00 (11.81)[a] | 94.88 (13.38)[b] | 104.16 (12.01)[a,b] | $p < .001$ |
| Age, mean (*SD*) | | | | |
| Childhood assessment | 7.64 (0.34)[a] | 7.74 (0.39)[b] | 8.10 (0.50) [a,b] | $p < .001$ |
| Adolescent assessment | 13.52 (1.51)[a] | 14.00 (1.62)[b] | 14.50 (1.30) [a,b] | $p < .001$ |
| Young adulthood assessment | 23.05 (1.09)[a] | 23.25 (1.22)[b] | 23.63 (1.02) [a,b] | $p < .01$ |
| Adulthood assessment | 31.80 (1.63) | 32.11 (1.69) | 32.47 (1.36) | $p > .05$ |
| Household SES (*z*-scored), mean (*SD*) | | | | |
| Childhood assessment | −0.05 (0.90) | −0.11 (1.03) | 0.09 (1.02) | $p > .05$ |
| Adolescent assessment | −0.07 (0.88) | −0.12 (1.01) | 0.07 (1.00) | $p > .05$ |
| Young adulthood assessment | −0.04 (0.63)[a] | −0.13 (0.66)[b] | 0.19 (0.62)[a,b] | $p < .01$ |
| Adulthood assessment | −0.09 (0.63)[a] | −0.31 (0.68)[b] | 0.21 (0.83)[a,b] | $p < .01$ |

*Note*: Identical superscripts denote significant group differences. ELBW+S, extremely low birth weight and prenatal steroid exposure. ELBW+NS, extremely low birth weight and no prenatal steroid exposure. IQ, intellectual quotient. NBW, normal birth weight. SES, socioeconomic status.

**Table 2.** Descriptive statistics and correlations for shyness across assessments from childhood to adulthood

| | 1 | 2 | 3 | Mean (*SD*) |
|---|---|---|---|---|
| 1. Childhood | — | | | 0.03 (0.99) |
| 2. Adolescence | .27** | — | | −0.04 (0.97) |
| 3. Young adulthood | .23** | .22** | — | −0.01 (0.98) |
| 4. Adulthood | .17* | .34** | .69** | −0.06 (0.94) |

Shyness scores are *z*-scored. *$p < .05$. **$p < .001$.

**Table 3.** Growth curve models of shyness predicted by birth weight status and prenatal corticosteroid exposure

| | Unadjusted model β (*SE*) | Adjusted model β (*SE*) |
|---|---|---|
| *Initial status* | | |
| Intercept | −0.20 (0.14) | 0.14 (0.61) |
| ELBW+S | 0.44 (0.22)* | 0.42 (0.22)* |
| NBW | 0.30 (0.19) | 0.34 (0.20) |
| Sex | — | −0.05 (0.15) |
| IQ | — | 0.00 (0.01) |
| *Slope* | | |
| Age | 0.01 (.01) | −0.03 (0.03) |
| ELBW+S × Age | −0.01 (.01) | −0.01 (0.01) |
| NBW × Age | −0.02 (.01)* | −0.02 (0.01)* |
| Sex × Age | — | 0.00 (0.01) |
| IQ × Age | — | 0.00 (0.00) |
| SES | — | −0.15 (0.04)* |

*Note*: ELBW+S, extremely low birth weight and prenatal steroid exposure. IQ, intellectual quotient. NBW, normal birth weight. SES, socioeconomic status. ELBW+NS, extremely low birth weight and no prenatal steroid exposure, is the reference category. $N = 254$. *$p < .05$.

The origins of shyness are multifaceted, with both biological and contextual influences affecting its developmental course (e.g., Poole, Tang, et al., 2018; Schmidt, Polak, & Spooner, 2005; Stevenson-Hinde, 2002). One line of evidence argues that some typically developing infants enter the world with a biological predisposition to become physiologically and behaviorally aroused in response to novelty (i.e., behaviorally inhibited; Garcia-Coll et al., 1984). As these individuals undergo further sociocognitive development across early childhood, this inhibition can become particularly salient in novel *social* contexts for some children (i.e., shyness), resulting in the perception of social situations as threatening in nature. The typical biological modifications (e.g., increased HPA axis and amygdala activity) accompanying stressful prenatal environments are similar to those biological systems implicated in shyness and social threat processing (Beaton et al., 2008; Kagan et al., 1987, 1988; Schmidt et al., 1997; Tang et al., 2016), and we speculate that these developmental antecedents of shyness may be programmed in utero in response to prenatal stressors.

The pattern of shyness among the ELBW+S individuals likely reflects an early emerging shyness that is stable across development. It has been proposed that prenatal alterations that occur in order to increase postnatal survival can occur at the expense of physiological and behavioral flexibility (i.e., ability to change across development), and consequently may lead to behavioral phenotypes that are less likely to change across time (Duckworth, 2015). The selection of a stable system during a stressful prenatal period is viewed as adaptive as this allows the individual to optimize survival in an equally threatening postnatal environment through the programming of traits such as hypervigilance, threat-sensitivity, and stress-reactivity—key features of

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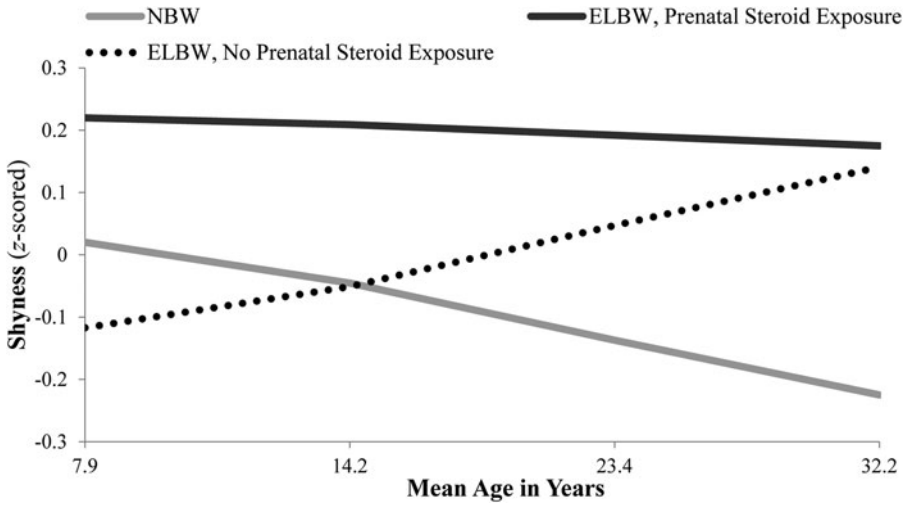


**Figure 1.** The mean developmental trajectory of shyness from childhood to adulthood based on birth weight status and prenatal corticosteroid exposure. ELBW, extremely low birth weight. NBW, normal birth weight. Estimates from the growth curve analysis are plotted. $N = 254$.

shyness. Being born at ELBW is a significant early stressor resulting in programmed changes in the central and physiological systems responsible for the stress response (Gluckman et al., 2010; Harris & Seckl, 2011). Likewise, administration of prenatal synthetic steroids stimulates the fetal HPA axis and mimics the effects of a natural occurring stressor (Benediktsson, Lindsay, Noble, Seckl, & Edwards, 1993). Thus, exposure to synthetic glucocorticoids *in addition* to the stresses of being born at ELBW may result in a cumulative effect by which set points in the brain and body are programmed during sensitive periods of early development, and lead to stability in shyness across development. Given that these hypothesized mechanisms were not directly measured in the present study, it will be informative to systematically test these speculated mechanisms in future longitudinal work.

Our findings also illustrate the concept of equifinality, which is the notion that individuals may have different initial starting points but have similar later outcomes (Cicchetti & Rogosch, 1996). The ELBW+S and ELBW+NS groups had different initial starting points of steroid exposure versus no exposure and levels of childhood shyness, but the ELBW+NS group had patterns of increasing shyness with levels similar to the ELBW+S group by adulthood. We further find that the ELBW+NS and NBW groups had similarly low levels of childhood shyness, but there was divergence in shyness between the ELBW+NS and NBW groups around adolescence, such that the ELBW+NS individuals demonstrated patterns of increasing shyness into adulthood, while the NBW controls showed decreases in shyness. It is possible that this divergence in shyness occurs due to differences in developmental circumstances and processes between the groups (Cicchetti & Rogosch, 1996).

Beyond biological influences on the development of shyness, additional developmental models of shyness highlight the important role of social influences and context (Coplan, Arbeau, & Armer, 2008; Gazelle & Ladd, 2003; Hastings, Nuselovici, Rubin, & Cheah, 2010; Rubin, Bowker, & Gazelle, 2010; Schmidt et al., 2005; Stevenson-Hinde, 2002). Previous work has shown that those born prematurely are more prone to be the recipient of overprotective parenting (e.g., Indredavik, Vik, Heyerdahl, Romundstad, & Brubakk, 2005; Jaekel, Wolke, & Chernova, 2012; Wightman et al. 2007), victims of bullying, peer victimization, and social exclusion (see Day, Van Lieshout, Vaillancourt, & Schmidt, 2015, for a recent review), and have lower social competence and social skills (Dahl et al., 2006; Hoy et al., 1992; Ross, Lipper, & Auld, 1990) relative to their typically developing peers. These are key social influences that play a role in later developing or increasing patterns of shyness (Booth-LaForce & Oxford, 2008; Hastings et al., 2010; Karevold, Ystrom, Coplan, Sanson, & Mathiesen, 2012; Oh et al., 2008; Poole, Tang, et al., 2018; Rubin et al., 2010, Tang et al., 2017). These social factors may be particularly influential during adolescence as this coincides with the onset of puberty, increases in sociocognitive development, and an increased reliance on peers and need for social acceptance (Cheek, Carpentieri, Smith, Rierdran, & Koff, 1986). These hormonal, neural, and social changes can affect the development, expression, and regulation of emotions (Del Piero, Saxbe, & Margolin, 2016), and some work has found that shyness and social fears may increase during adolescence due to these factors (Cheek et al., 1986; Cheek & Krasnoperova, 1999; Tang et al., 2017; Westenberg, Drewes, Goedhart, Siebelink, & Treffers, 2004). Taken together, we predict that social influences may place ELBW+NS individuals on a path toward increasing shyness beginning in adolescence that reflects a later developing shyness that is influenced by postnatal context. Although we suspect that these social experiences are also present for ELBW+S individuals, these factors may play a role in the maintenance of shyness as opposed to increasing patterns given the relatively high initial childhood shyness of the ELBW+S group, likely resulting in ceiling effects.

Our findings also shed light on the relative trajectory of shyness from childhood to adulthood in typically developing samples (i.e., those born at NBW). Although the findings should be interpreted with appropriate caution as the trajectories are *relative* to the ELBW survivors, they nonetheless provide information on how shyness develops in typically developing individuals. This is important given that there exists relatively little work examining the mean-level developmental course of shyness from childhood to adulthood. One longitudinal study found decreasing patterns of shyness from age 4 to 23 years among "resilient" children (Dennissen et al., 2008), which is similar to what we found for the NBW group from age 8 to 32 years. As individuals enter developmental periods of stability and achieve important milestones (e.g., establish romantic relationships, obtain a career, and start a family), they may become more socially dominant and emotionally stable (Neyer & Asendorpf, 2001; Roberts, Walton, & Viechtbauer, 2006; Robins, Fraley, Roberts, & Trzesniewski, 2001). Relative to individuals who were born at

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ELBW, NBW individuals are more likely to obtain these milestones (Saigal et al., 2016), and this may be one mechanism underlying their relative decreases in shyness into adulthood.

Although it appears that survivors of ELBW exposed to synthetic glucocorticoids may be on a path toward the development of shyness and some work has shown that shyness is predictive of psychopathology and maladaptive outcomes (e.g., Clauss & Blackford, 2012), it is important to point out that shyness is not always inherently maladaptive. Other research has reported adaptive aspects of some shyness subtypes (e.g., Colonnesi, Napoleone, & Bögels, 2014; Poole & Schmidt, 2019, Schmidt & Poole, 2018). Likewise, although we hypothesize that the development of shyness may result from programmed changes in the physiological systems underlying hypervigilant and fearful reactions due to early stressors, we further reiterate that these changes are not necessarily pathological, but are thought to be functional adaptations that may promote resilience in the faces of future environmental challenges and stressors (Del Giudice, Ellis, & Shirtcliff, 2010; Gluckman, Hanson, & Pinal, 2005).

### Limitations

There were several limitations of the present study that should be acknowledged. First, shyness was assessed using different informants during the different developmental periods (i.e., parent vs. self-report). Although this is an inherent methodological challenge to long-term developmental research (Biesanz, West, & Kwok, 2003; Caspi, Roberts, & Shiner, 2005), and while we standardized measures for consistency, it nonetheless is prone to possible informant discrepancies across assessments and issues of measurement variance. Second, although this work provides valuable information on the development of shyness from childhood to adulthood, we do not have data pertaining to earlier developmental periods such as toddlerhood and so we cannot directly assess the early developmental precursors of shyness (e.g., behavioral inhibition) that may have influenced the trajectories. Third, we relied on questionnaire-based indices of shyness, and thus future work could aim to integrate observational measures of shyness, notwithstanding the challenges associated with accurately assessing the same phenotype across development given differences in the expression, experience, and overt behaviors of shyness depending on age. Fourth, although Cronbach's alphas were acceptable, they were somewhat low during the childhood and adolescent assessments; however, this is not uncommon for scales with fewer than 10 items, as internal consistency tends to be an underestimate in scales with fewer items (Taber, 2018). Fifth, we acknowledge that there were likely additional unmeasured influences on the development of shyness beyond those investigated in the present study including parental traits such as social anxiety, which likely exerts both biological (i.e., genetic) and environmental (i.e., social modeling) influences on the expression of shyness in offspring (Bögels, Stevens, & Majdandžić, 2011; Lieb et al., 2000; Poole, Van Lieshout, McHolm, Cunningham, & Schmidt, 2018; Smith et al., 2012), as well as additional early stressors aside from prematurity and prenatal steroid exposure. Sixth, given the cohort study design, women were not randomized to receive corticosteroids (i.e., NBW babies were not prenatally exposed to corticosteroids), and thus it is possible that there were unmeasured confounding factors influencing our findings. Finally, it is important to replicate our findings in contemporary samples to ensure generalizability to more recent birth cohorts given advancements in neonatal care and increased survival rates among individuals who are born extremely prematurely.

### Implications and conclusions

The present study highlights the importance of employing a longitudinal framework when testing developmental programming hypotheses as this allows for identification of how prenatal stressors may result in the prospective emergence of personality traits during different developmental periods. Our findings inform developmental models of shyness, and illustrate that while biological susceptibility may influence the development of shyness, postnatal contextual influences may also impact the development of shyness for some individuals. This study provides evidence for heterogeneity in the developmental origins, onset, and course of shyness. We recommend that future studies continue to investigate how early life experiences may influence the manifestation of shyness in particular, and personality styles in general across development, and how this may be related to the development of psychopathology.

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