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Co-Recovery of Physical Size and Cognitive Ability from Infancy to Adolescence: A Twin Study

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Abstract

This study tested phenotypic and biometric associations between physical and cognitive catch-up growth in a community sample of twins ($n = 1,285$, 51.8% female, 89.3% White). Height and weight were measured at up to 17 time points between birth and 15 years and cognitive ability was assessed at up to 16 time points between 3 months and 15 years. Weight and length at birth were positively associated with cognitive abilities in infancy and adolescence (r 's = .16-.51). More rapid weight catch-up growth was associated with slower, steadier cognitive catch-up growth. Shared and nonshared environmental factors accounted for positive associations between physical size at birth and cognitive outcomes. Findings highlight the role of prenatal environmental experiences in physical and cognitive co-development.

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Physical size in infancy, toddlerhood, and early childhood is generally considered to be an indicator of healthy development, and height and weight measurements are aspects of routine well-child check-ups over the first years of life. Children who fall below population norms for physical development are at risk for poor neurodevelopment including poor cognitive performance and gross and fine motor deficits (Cooke, 2006; Upadhyay et al., 2019). Among young children who are physically undersized at birth relative to population norms, catch-up growth in height and weight is associated with gains in cognitive ability (Ghods et al., 2011; Scharf et al., 2016). However, the majority of studies examining associations between physical and cognitive catch-up growth are limited to two time points; no research has leveraged latent growth curve models to explore co-catch-up growth in physical size and cognitive ability. Therefore, it is unclear how latent factors such as the rate and developmental timing of physical and cognitive catch-up growth relate to one another. Understanding the extent to which physical and cognitive growth patterns are related can inform our understanding of how interventions targeted at early physical growth (e.g., nutritional supplementation) may have additional benefits for cognitive development.

Low birth weight is associated with lower cognitive abilities in infancy (Kohlhauser et al., 2000) and into childhood (Antonioni et al., 2013; Edmonds et al., 2010). A recent meta-analysis found that infants born at low birth weight (less than 2,500 grams) scored approximately 5-6 points lower than typical birth weight infants on standardized cognitive assessments in childhood (Upadhyay et al., 2019). In a study of 71 pairs of identical twins, within-pair discrepancies in birth weight were associated with within-pair discrepancies in verbal IQ performance in childhood (Edmonds et al., 2010). Additionally, even among samples restricted to typical birthweight (> 2500 grams) and gestational age (37-40 weeks), there is a small, but positive association between birth weight and cognitive ability (Shenkin et al., 2004).

Less work has focused on associations between length in infancy and cognitive development, despite research suggesting a modest correlation between height and cognitive ability at later developmental stages (Silventoinen et al., 2012). However, in a sample of Singaporean children, Brokerman and colleagues (2009) observed a modest, but positive association between birth length and performance on the Raven's Standard Progressive Matrices test in late childhood, accounting for gestational age, birth weight, and head circumference. Likewise, among infants from rural Guatemala, length for age z-scores at birth were positively associated with Bayley mental development scores at 6, 24, and 36 months (r^2 's = .17-.25) (Kuklina et al., 2006).

Among samples of children born lighter or shorter than population norms, children displaying catch-up growth typically perform better on cognitive assessments relative to children who do not catch up (Fattal-Valevski et al., 2009; Scharf et al., 2016; Sudfeld et al., 2015). In a sample of American children born at very low birth weight, children who were stunted (height for age z-score < -2) at 9 months were more than twice as likely to have 9-month Bayley scores 2 SD below the population mean compared to children who were not stunted (Scharf et al., 2016). Moreover, children who were stunted at 24 months were nearly 3 times more likely to have 24-month Bayley scores \geq 2 SD below the population mean, suggesting that sustained height deficits elevate the risk for poor cognitive development

(Scharf et al., 2016). Likewise, in a sample of Israeli children born with intrauterine growth restriction, children who caught up in height and weight by age 2 displayed higher IQs at ages 9 and 10 years than children who did not catch up (Fattal-Valevski et al., 2009). Thus, among children born at high biological risk, failure to catch up in the first 24 months appears to elevate the risk for a poor cognitive trajectory.

In terms of developmental timing, earlier catch-up growth appears to be a strong predictor of later cognitive ability. Among small-for-gestational-age infants in the National Collaborative Perinatal Project, Varella and Moss (2015) observed a positive association between the rate of catch-up growth in the first 12 months and IQ scores at 4 years; children who displayed faster physical recovery performed better cognitively. Likewise, conditional growth in height in the first year, but not between 1 and 9 years, was associated with higher Full Scale, Verbal, and Performance IQ scores at 9 years among a sample of Thai singletons (Pongcharoen et al., 2012). Compelling evidence of the importance of early catch-up comes from a meta-analysis of 68 studies examining associations between linear growth and cognitive ability in childhood (Sudfeld et al., 2015). A one SD increase in height-for-age z-score before age 2 was associated with a 0.2 SD increase in cognitive ability whereas a one SD increase in height-for-age z-score after 2 years was associated with a 0.1 SD increase in cognitive ability (Sudfeld et al., 2015).

The extant body of literature demonstrating positive associations between early physical size and cognitive outcomes has primarily focused on between-family studies using samples of singletons (Fattal-Valevski et al., 2009; Varella & Moss, 2015). The between-family approach confounds any “true effect” of physical growth on cognitive development with genetic and environmental factors that also vary between families (e.g., socioeconomic status, length of gestation). Studies may attempt to control for important environmental factors, such as socioeconomic status, but controlling for all potentially confounding variables presents a significant challenge. The twin study design can be used to approximate an experimental relationship between physical and cognitive catch-up growth by focusing on within-pair associations, which controls for confounding genetic and shared environmental factors that vary between families (Turkheimer & Harden, 2014). For example, in a pair of identical twins raised together, the regression of within-pair differences in cognition on within-pair differences in physical size shows the extent to which the larger identical twin of the pair also has a higher cognitive score. This association cannot be caused by genetics, since they are identical, or by the family environment since they are raised together. The within-pair association leverages natural random variability that exists within a pair of twins and can be used to “experimentally” test associations in situations where true random assignment is neither ethically nor practically feasible (e.g., birth weight). Therefore, the within-pair relationship is sometimes referred to as a “quasi-experimental” relationship (Turkheimer & Harden, 2014).

Beyond the methodological strength of the twin study design to approximate quasi-experimental associations, twins are a naturally occurring population uniquely suited to study patterns of catch-up growth. Relative to singletons, twins are at elevated risk to experience a host of prenatal stressors, including competition for nutrients, uterine size constraints, and an increased rate of maternal health complications (Blickstein, 2004;

Goldenberg et al., 2008; Van Baal & Boomsma, 1998). Consequently, twins tend to be physically undersized relative to population norms as infants (Estourgie-van Burk et al., 2010; Wilson, 1979) and perform poorly on tests of early cognitive development (Datar & Jackowitz; 2009; Wilson, 1972). On average, twins demonstrate substantial catch-up growth in physical size and cognitive ability across infancy and toddlerhood and are average physically and cognitively by early childhood (Wilson, 1979; Womack et al., 2022, 2023). Understanding patterns of physical and cognitive co-development in twins can inform expectations for development among singletons exposed to early bio-environmental disadvantage (e.g., premature birth, preeclampsia, etc.). Using data from the Louisville Twin Study, we explore the quasi-experimental relationship between catch-up growth in height and weight and catch-up growth in cognitive ability from infancy to adolescence.

Leveraging twin studies, phenotypic associations can be decomposed into additive genetic, shared environmental, and nonshared environmental components. Genetically informed research designs have primarily focused on cross-sectional associations between height and cognitive ability in samples of adolescents and adults. Previous research has not come to a consensus on whether genetic or environmental factors account for the overlap between height and cognitive ability. Some research suggests additive genetic factors account for the majority of the association between physical size and cognitive ability (Silventoinen et al., 2006; Silventoinen et al., 2012). Alternatively, other researchers have noted the relative importance of the shared environment in the association between height and cognitive ability (Sundet, 2005).

Studies using samples recruited at earlier historical periods (e.g., Sundet, 2005) generally find a greater influence of the shared environment on the association between height and cognitive ability whereas samples recruited at later historical periods often find a greater genetic influence (Silventoinen et al., 2006; Silventoinen et al., 2012). This discrepancy may reflect differences in access to nutrition at different historical periods. The historical timeline of the Louisville Twin Study (1957-2000; Davis et al., 2019) generally overlaps with that of studies that have found a stronger additive genetic correlation between height and cognitive ability than a shared environmental correlation. However, without previous research examining associations between the rate of change in physical size and the rate of change in cognitive ability, it is unclear the extent to which physical and cognitive growth overlap. Additionally, it is unclear how much additive genetics or environmental experiences contributes to the overlap between the rate and shape of physical and cognitive catch-up growth trajectories.

Despite the large body of literature dedicated to examining associations between physical size and cognitive ability, major gaps exist. No studies have examined associations between the rate of growth in physical size and the rate of growth in cognitive ability. Pediatricians and other healthcare providers often use measurements of physical size as broad indicators of developmental health, and closely track physical growth patterns among children who are born physically undersized (e.g., low birth weight). Therefore, understanding the overlap in the developmental processes of physical and cognitive growth can be informative of the utility of physical measurements as an indicator of future wellbeing more globally among children exposed to early bio-environmental adversity. Additionally, using the twin study

design can inform the extent to which associations between physical and cognitive catch-up growth are quasi-experimental or mediated by genetic and familial environmental factors.

We fit a series of parallel-process growth models to age-standardized measurements of physical size (i.e., height and weight) and age-standardized cognitive measurements in a large community sample of twins followed from birth to 15 years. As no previous research that we are aware of has tested associations between physical and cognitive co-development in a prospective sample spanning infancy to adolescence, we did not have a-priori hypotheses about whether height or weight catch-up growth would be more strongly associated with cognitive development. Therefore, we tested associations between height and weight and cognitive ability in separate models.

Methods

Participants

We used data from the Louisville Twin Study, a longitudinal study of temperament and cognitive development in twins (Beam et al., 2020). All twins were recruited from the Louisville, Kentucky metropolitan area. Participants were primarily White (89.3%) and were recruited to represent the socioeconomic composition of the Louisville metropolitan area. Twin zygosity was determined by blood serum analysis when the twins were 36 months or older as a part of the study protocol (Wilson, 1970). Before 36 months, zygosity was determined by examiner ratings of twins' physical features. Wilson and Matheny (1986) observed a 98% agreement between examiner ratings and blood typing results among twins in the Louisville Twin Study with repeated measurements between 6 and 36 months.

Over the 36-year course of the Louisville Twin Study, 1,770 individuals (885 pairs) participated at least once. Twins missing zygosity information were not included in analyses ($n = 120$). Of those remaining, 1,642 had at least one physical or cognitive measurement. As is typical in twin studies of physical or cognitive development, we restricted analyses to monozygotic and same-sex dizygotic twins ($n = 1,292$). Finally, given our interest in typical development, we removed individuals that had a physical measurement greater than four standard deviations above or below the population mean ($n = 7$). The final study sample was 1,285 participants (51.8% female).

Procedure

Data were collected between 1957 and 1993. Cognitive testing and physical measurements were completed by trained examiners during laboratory visits at the University of Louisville at 16 time points between 3 months and 15 years (0.25, 0.5, 0.75, 1, 1.5, 2, 2.5, 3, 4, 5, 6, 7, 8, 9, 12, and 15 years). Twins were typically assessed within one week of their birthdays and the testing schedule was arranged such that examiners did not administer cognitive assessments to the same individuals at consecutive assessments.

Measures

Physical Development.—Birth length and weight measurements were obtained from birth certificates. Infant weights between birth and 24 months were taken with the infant

lying undressed on a balance scale. After 24 months, infants were weighed wearing a light garment using a platform scale calibrated in four-ounce increments. All weights were recorded to the nearest ounce and were subsequently converted to kilograms. Height data, measured to the nearest millimeter, were collected during the same appointment as weight data. Recumbent length was used as a proxy for height between birth and 24 months. After 24 months, standing height was measured using a wall-mounted metric scale. Raw height and weight measurements were converted into age-standardized z-scores using CDC growth charts based on 2000 norms (Kuczmarski, 2000). The 2000 CDC growth charts were based on United States population surveys conducted between 1963 and 1994 (see Kuczmarski 2000 for further details). Thus, the 2000 CDC norms overlap considerably with the historical timeline of data collection in the Louisville Twin Study.

Cognitive Development.—Several age-standardized cognitive assessments were used over the course of the Louisville Twin Study as the twins aged and new test versions were published. Between 3 and 24 months, twins were administered the Bayley Scales of Mental Development, with most twins completing the first edition (Bayley, 1969) and some twins completing the second edition (Bayley, 1993). At 30 months, a minority of twins completed the Bayley. Most twins at 30 months and all twins at 36 months completed the Stanford-Binet Intelligence Scale – Third Edition with norms based on the 1972 re-standardization (Freides, 1972). At 4 years, twins completed either the Stanford-Binet – Third Edition, the McCarthy Scales of Children’s Abilities (McCarthy, 1972), or the WPPSI (Wechsler, 1967). Cognitive assessments administered at age 5 were either the McCarthy, the WPPSI, or a revised version of the WPPSI (WPPSI-R; Wechsler, 1989). At age 6, twins completed either the WPPSI or WPPSI-R. At ages 7, 8, and 9, twins completed either the WISC (Wechsler & Kodama, 1949), the WISC revised (WISC-R; Wechsler, 1974), or the WISC – Third Edition (WISC-III; Wechsler, 1991). At ages 12 and 15 years, twins either completed the WISC-R or WISC-III. A breakdown of each measure administered is presented in Supplementary Table 1.

All of the cognitive scales administered over the course of the Louisville Twin Study have an age-standardized mean of 100, which provides a common reference point for cognitive ability across measures and time. The Bayley and McCarthy Scales were standardized to have a standard deviation of 16; the Stanford-Binet and Wechsler scales were standardized to have a standard deviation of 15. Standardized cognitive scores were converted to z-scores so that they were on the same scale as the standardized physical growth measurements.

Data Analyses

Descriptive Statistics.—Data preparation, descriptive statistics, and the calculation of intercorrelations between study variables were conducted using the Base package in R version 4.2.2 (R Core Team, 2022).

Parallel Process Growth Models.—The parallel process growth curve models were guided by recent work using the Louisville Twin Study to model trajectories of catch-up growth in height, weight, and cognitive ability. Catch-up growth in height and weight was best described using an approximately exponential-shaped Weibull curve (Womack et al.,

2023). A sigmoid-shaped curve (Gompertz curve) best described cognitive catch-up growth in the Louisville Twins (Womack et al., 2022).

The Weibull Growth curve, which is derived from a Weibull distribution (Ratkowsky, 1983), can be expressed using the following formula.

$$Y_{it} = b_{1i} - (b_{1i} - b_{0i}) \cdot \exp(-b_{2i} \cdot t^{b_{3i}}) + e_{it}$$

In this model, the predicted outcome (e.g., height) for individual i and time t is a function of an intercept (b_0), an upper asymptote (b_1), the rate of growth (b_2), and an inflection point, or time at which growth is most rapid (b_3). In this model, the intercept (b_0) refers to the predicted value of Y when time equals 0, or birth in the present study.

The four-parameter Gompertz curve can be defined using the following equation (Tjørvæ & Tjørvæ, 2017).

$$Y_{it} = b_{0i} - (b_{0i} - b_{1i}) \cdot \exp(-\exp(-b_{2i} \cdot (t - b_{3i}))) + e_{it}$$

In the four-parameter Gompertz model, the estimated outcome (e.g., cognitive ability) for individual i and time t is a function of a lower asymptote (b_0), an upper asymptote (b_1), the rate of growth (b_2) and an inflection point, or time at which growth is most rapid (b_3). The lower asymptote (b_0) differs slightly from the intercept parameters in the Weibull growth models in that it reflects an individual's lowest cognitive ability score as opposed to their initial score. For readers unfamiliar with asymptotic growth models, see Appendix A in the supplement for an illustration of how different parameter estimates affect the shape of the curve.

All growth models were fit in a structural equation modeling framework using Mplus version 8.4 (Muthén & Muthén, 2017). Factor loadings for the latent growth variables were fixed to the partial derivative of each parameter in the target function ($b_0 - b_3$). We could then generate a linear combination of the latent growth variables and approximate each target growth function (i.e., Weibull curve for height and weight and Gompertz curve for cognitive ability; Grimm et al., 2013). We first tested phenotypic associations between physical and cognitive catch-up growth by specifying correlations between the growth parameters. We used the “cluster” function in Mplus to adjust standard errors as there were multiple twins within each family (Muthén & Muthén, 2017). Parallel process growth models were fit separately for height and weight.

We then fit a series of genetically-informed parallel process growth models to test biometric associations between physical and cognitive catch-up growth. The variance of each growth parameter was decomposed into three latent variables corresponding to the additive genetic (A), shared environmental (C), and nonshared environmental (E) variance components using a standard multilevel twin design with individual twins nested within families (McArdle & Prescott, 2005). Additive genetic factors refer to linear independent genetic effects that are

transmissible between generations. Shared environmental experiences reflect environmental experiences that make individuals within a family more similar (e.g., types of foods or books available in a home). Nonshared environmental experiences are aspects of the environment that make individuals within the same family different (e.g., one twin being prescribed a nutrient-enriched diet to promote weight gain). For monozygotic (MZ) twins, who share 100% of the same genes, the nonshared environmental factors account for all within-pair variance whereas a combination of additive genetic and shared environmental factors accounts for between-pair variance. For dizygotic (DZ) twins, who share on average 50% of the segregating genes, within-pair variance is due to a combination of the nonshared environmental factor and half of the additive genetic factor (i.e., the 50% of genes DZ twins do not share). A combination of the shared environmental factor and 50% of the additive genetic factor (i.e., the 50% of genes DZ twins share) accounts for between-pair variance. The variance decomposition can be represented using the following equations.

$$\begin{aligned} MZ_{\text{within}} &= E \\ MZ_{\text{between}} &= A + C \\ DZ_{\text{within}} &= 0.5^*A + E \\ DZ_{\text{between}} &= 0.5^*A + C \end{aligned}$$

We regressed the cognitive growth parameters onto the A, C, and E factors of the weight growth parameters (see Figure 1 for a simplified path diagram) following the “quasi-experimental” twin approach (Turkheimer and Harden, 2014). This approach can be conceptualized as dividing each physical growth parameter into three separate variables: an additive genetic variable, a shared environmental variable, and a nonshared environmental variable. The additive genetic and shared environmental factors can be thought of as covariates that account for genetic factors and environmental experiences shared by members of a family (paths labeled r_a and r_c in Figure 1, respectively). What is leftover is the quasi-experimental within-pair association between physical size and cognitive ability (labeled r_e in Figure 1). Following the example depicted in Figure 1, the additive genetic path (r_a) indicates the extent to which twins from families genetically more likely to have a higher weight intercept (i.e., heavier birth weight) also have a higher lower asymptote of cognitive ability (i.e., cognitive ability in infancy). The shared environmental path (r_c) reflects the extent to which twins from the “type” of family environment associated with higher birth weights (e.g., longer gestation, higher family SES) also have higher cognitive scores. In this illustration focused on birth weight, it is important to recognize that shared environmental components of birth weight reflect shared prenatal experiences (e.g., gestational age, maternal smoking).

In all growth models, sex was included as a covariate as males and females were observed to demonstrate different trajectories of height and weight catch-up growth (Womack et al., 2023).

Missing Data

Rates of missing cognitive, weight, and height measures at each age are presented in Supplementary Tables 1-3, respectively. Additionally, longitudinal patterns of missingness for each measure are depicted in Supplementary Figures 1-3. We used full information maximum likelihood (FIML) estimation to model missing data. An assumption of FIML is that data are missing at random (MAR). Under MAR conditions, missingness may be related to other observed variables (e.g., family SES), but missingness is unrelated to the missing value itself (e.g., shorter children are not more likely to be missing height measurements) (Rubin, 1976). To explore patterns of missingness, we fit a series of logistic regression models predicting missingness from each variable (e.g., height, weight, and cognitive ability) at each age from the study covariates and the previous measure of the outcome variable (e.g., height at 3 months predicting missingness on height at 6 months). To the extent height, weight, and cognitive ability measurements are stable over time, including previous measurements in the logistic regression models allowed us to approximate if missingness was related to the missing value itself. Results from the missing data analyses provide support for our use of FIML to handle missingness under assumptions that data are MAR (Enders, 2013). See Supplementary Tables 4-6 for height, weight, and cognitive ability missingness analyses, respectively. Birth year emerged as a consistent predictor of missingness with children born in later years more likely to have missing data. This may reflect changes in study protocols over the course of the study or a loss of study funding in the 1990s (Beam et al., 2020). Measured height, weight, and cognitive ability scores were regressed onto birth year in all growth models to avoid generating biased parameter estimates (Enders, 2013). Accounting for birth year also controls for the secular rise in intelligence scores across birth cohorts (the Flynn Effect), which has been observed in the Louisville Twin Study (Giangrande et al., 2022).

Power Analyses

A Monte Carlo simulation was conducted in *Mplus* (Muthén & Muthén, 2017) to test the power of detecting the observed phenotypic associations between physical and cognitive catch-up growth. Power analyses were based on 1,000 simulated datasets of 1,285 individuals.

Results

Descriptive Statistics and Intercorrelations

Descriptive statistics for cognitive ability scores are presented in Supplementary Table 1 and descriptive statistics for height and weight are presented in Supplementary Tables 2-3, respectively. Concurrent correlations between physical measurements and cognitive ability scores are presented in Figure 2. There was a modest positive association between cognitive ability scores and measurements of height and weight across infancy and toddlerhood. After 36 months, concurrent correlations between cognitive ability and height and weight were weak and predominantly non-significant.

Phenotypic Correlations Between Physical and Cognitive Catch-Up Growth

Phenotypic associations between weight and cognitive catch-up growth are presented in Table 1. The intercept of weight (birth weight) was positively associated with the lower and upper asymptote of cognitive ability (r 's = .51 and .16, respectively, p 's < .001). Additionally, heavier children at birth had a faster rate of cognitive catch-up growth ($r = .18, p = .009$) and a later inflection point ($r = .14, p = .005$). The upper asymptote of weight was not significantly related to the upper asymptote of cognitive ability ($r = .01, p = .908$). Faster weight catch-up was associated with a slower rate of cognitive growth ($r = -.16, p = .018$) and a later inflection point ($r = -.13, p = .023$).

See Table 2 for phenotypic correlations between height and cognitive catch-up growth parameters. There was a modest, positive association between birth length and the lower asymptote of cognitive abilities ($r = .32, p < .001$) and a weak but statistically significant association between birth length and the upper asymptote of cognitive abilities ($r = .16, p = .001$). Individuals with a higher height upper asymptote had a later inflection point of cognitive catch-up growth ($r = -.12, p = .024$). The rate of height catch-up growth was not significantly associated with any of the cognitive catch-up growth parameters. A later inflection point of height growth was associated with a higher lower asymptote of cognitive ability ($r = .23, p = .003$) and a faster rate of cognitive growth ($r = .21, p = .026$). Phenotypic correlations between height and cognitive catch-up growth parameters are presented in Table 2.

Post-hoc power analyses revealed that there was sufficient power to detect the observed associations between height and weight at birth and the lower and asymptotes of cognitive ability (>94.1%). Power to detect the observed associations between the rate of weight catch-up growth and the rate and inflection point of cognitive catch-up growth were 47.2% and 44.1%, respectively. Power for all phenotypic associations tested are presented in Tables 1 and 2. Given the wide range of power available in the current study, some analyses (e.g., biometric analyses) are viewed as exploratory.

Biometric Components of Weight, Height, and Cognitive Growth

Exploring the variance decomposition of the height, weight, and cognitive ability growth parameters into A, C, and E components was not the goal of the present study as this information has been presented elsewhere (see Womack et al., 2022, 2023). Unstandardized and standardized A, C, and E variance components for the height, weight, and cognitive ability growth parameters estimated from univariate growth models are presented in Supplementary Table 7. This information is presented to provide context for the biometric regression analyses. Shared environmental factors were primarily associated with individual differences in initial height, weight, and cognitive ability measurements (59%-81% of the variance) whereas additive genetic factors accounted for the majority of the variance in the upper asymptotes of each variable (69%-79% of the variance). Individual differences in the rate of height, weight, and cognitive catch-up growth were primarily associated with a combination of additive genetic (57%-63% of the variance) and shared environmental factors (28%-43% of the variance). Likewise, a combination of additive genetic (17%-50%)

and shared environmental factors (42%-81%) were associated with individual differences in the inflection points of height, weight, and cognitive ability.

Biometric Associations: Weight and Cognitive Catch-Up Growth

The additive genetic variance component of the weight intercept was constrained to 0 as it was initially estimated to be negative and nonsignificant. Therefore, all regression paths from the additive genetic factor to cognitive growth were also constrained to 0. There were significant, positive associations between the shared environmental component of the intercept of weight (i.e., birth weight) and all growth parameters of cognitive ability. Importantly, all environmental experiences related to birth weight were experienced by the twins prenatally. Therefore, twins from “types” of prenatal environment associated with higher birth weights (e.g., longer gestation) also demonstrated higher cognitive scores in infancy, higher cognitive scores in adolescence, and had a faster and more prolonged rate of cognitive catch-up growth. Additionally, there was a significant positive association between the nonshared environmental component of birth weight and the lower and upper asymptotes of cognitive ability, indicating a quasi-experimental effect of birth weight on cognitive ability that persists into adolescence. Figure 3 depicts the quasi-experimental effect of birth weight on cognitive development trajectories into adolescence.

Additive genetic components of the rate of weight growth were negatively associated with the inflection point of cognitive growth. However, nonshared environmental components of weight growth were *positively* associated with the rate and inflection point of cognitive growth. Therefore, additive genetic factors shared by members of a family associated with faster weight catch-up growth were also associated with earlier cognitive growth. However, within a pair, the twin that gained weight the fastest made cognitive gains later. See Table 3 for all associations between the ACE components of weight recovery and cognitive recovery. Parameter estimates for the mean structure of the growth curve models are presented in Supplementary Table 8.

Biometric Associations: Height and Cognitive Catch-Up Growth

To address model convergence issues, several regression paths had to be constrained to 0 (see Table 4 for all regression coefficients between height and cognitive growth). This may be related to the complexity of the growth models and the relatively limited phenotypic overlap observed between the physical and cognitive growth parameters.

Length at birth was quasi-experimentally associated with a higher lower asymptote of cognitive ability and a faster rate of cognitive recovery. Within a pair, the longer twin at birth had higher cognitive scores in infancy and demonstrated faster catch-up growth toward the population mean. There was a significant negative association between the additive genetic factors related to the intercept of height and the lower asymptote of cognitive abilities, suggesting that genetic factors associated with a longer length at birth are associated with *lower* cognitive scores in infancy. Shared environmental factors associated with the intercept of height were associated with the lower and upper asymptote of height. As shared environmental factors associated with the intercept of height reflect prenatal experiences, this finding suggests that prenatal environmental exposures that are associated with greater

length at birth are also associated with higher cognitive abilities into adolescence. Figure 4 depicts the quasi-experimental effect of birth length on cognitive development.

Genetic factors associated with the upper asymptote of height were positively associated with the upper asymptote of cognitive ability and negatively associated with the rate and inflection point of cognitive growth. Shared environmental factors associated with the upper asymptote of height were negatively associated with the upper asymptote of cognitive ability and positively associated with the inflection point of cognitive growth. These shared environmental associations should be interpreted with caution given the wide confidence interval around the estimates and the relatively small proportion of the variance in the upper asymptote of height associated with shared environmental factors. Parameter estimates for the mean structure of the growth curve models are presented in Supplementary Table 9.

Discussion

Using prospective data from the Louisville Twin Study spanning birth to adolescence, we explored the longitudinal relationship between physical and cognitive catch-up growth in twins. Previous research has found that twins display substantial deficits in physical size and cognitive ability in infancy, but recover to population norms by middle childhood (Wilson, 1974, 1979; Womack et al., 2022, 2023). However, this study is the first to explore associations between patterns of catch-up growth in physical size and cognitive ability. Additionally, we leveraged an under-utilized strength of the twin study design: the ability to approximate quasi-experimental associations between patterns of physical growth (an exposure that is not practically or ethically feasible to manipulate) and cognitive development by controlling for potentially confounding genetic and environmental factors shared by members of a family.

Consistent with study hypotheses and previous research (Kohlhauser et al., 2000; Kuklina et al., 2006), the intercepts of height and weight were positively associated with the lower asymptote of cognitive ability. Associations between weight and length at birth and early cognitive abilities were significant through shared environmental and quasi-experimental (within-pair) paths. The shared environmental association between length and weight at birth and early cognitive abilities indicate that shared prenatal experiences (e.g., gestational age, maternal BMI, maternal health complications, exposure to teratogens) that are related to higher (or lower) birth weights and lengths were also related to higher (or lower) cognitive scores in infancy. Additionally, accounting for shared environmental experiences (e.g., prenatal experiences) and shared genetic factors, the longer and heavier twin at birth had higher early cognitive scores compared to their co-twin. The quasi-experimental (within-pair) associations provide robust evidence that weight and length at birth are important indicators of early cognitive development. Contrary to expectations, additive genetic factors associated with greater length at birth were associated with lower early cognitive scores, which may suggest a genetic trade-off between prenatal linear growth and prenatal neurological development.

Phenotypically, weight and length at birth were positively associated with the upper asymptote of cognitive abilities, suggesting a small, but significant cognitive benefit of

being born larger is maintained into adolescence. Biometric analyses revealed significant positive shared environmental and quasi-experimental associations between birth weight and cognitive abilities. Additionally, length at birth was positively associated with the upper asymptote of cognitive ability through shared environmental pathways. Numerous extant studies have demonstrated positive associations between physical size at birth and cognitive abilities in childhood, adolescence, and adulthood (Antoniou et al., 2013; Flensburg-Madsen & Mortensen, 2017). However, many of these studies have utilized a between-family design (by following a single child per family) which confounds associations with genetic and environmental factors shared by a family. Our analytic approach of testing genetic, shared environmental, and quasi-experimental nonshared environmental associations simultaneously demonstrated that both shared and nonshared (prenatal) environmental experiences are important factors in understanding associations between birth weight and cognitive development.

Nonshared prenatal environmental experiences in a twin pregnancy may seem paradoxical, but differences in fetal placement may correspond with differences in access to nutrition or exposure to teratogens (Marceau et al., 2016). These findings have implications for pre- and postnatal care. Interventions to improve gestational health and length of gestation for high-risk pregnancies (e.g., twins) may have downstream effects on infant wellness including healthier physical size and early cognitive development. Additionally, the quasi-experimental association between birth weight and long-term cognitive development suggests that fetal growth restriction is a robust risk factor for poor cognitive development. Children falling behind fetal growth standards may benefit from postnatal interventions designed to promote early cognitive growth.

Shared (prenatal) environmental experiences associated with higher birth weight were associated with faster and more prolonged cognitive growth and ultimately higher cognitive scores. In the only other similar study, Cheadle and Goosby (2010) observed a positive association between birth weight and the rate of academic achievement growth from 5 to 14 years in a sample of singletons. As the majority of physical and cognitive recovery in the Louisville Twin sample occurs before age 5 (see Womack et al., 2022, 2023), the present study extends associations between birth weight and cognitive development to earlier developmental stages. Additionally, we demonstrate that prenatal environmental factors associated with higher birth weights are also associated with faster and greater cognitive catch-up growth.

Neither the rate of height nor weight growth were associated with the upper asymptote of cognitive ability. This finding stands in contrast to previous work which has found that faster catch-up growth in height and weight is associated with greater cognitive catch-up growth (Fattal-Valevski et al., 2009; Scharf et al., 2016). However, all of these previous studies have used the total amount of weight or height growth between two time points as a proxy for the rate of growth. This approach conflates the total amount of physical growth with the rate of growth. In using latent growth models, we were able to disentangle the rate of growth from the total amount of growth and observed no association between how quickly a child grew physically and their peak cognitive scores.

Phenotypically, a faster rate of weight catch-up growth was associated with a steadier, more prolonged trajectory of cognitive catch-up growth, and a faster rate of height growth was associated with a later cognitive inflection point. Genetic factors associated with faster height and weight growth were associated with earlier rapid cognitive catch-up growth. The pattern of findings differed somewhat within pairs; faster weight growth was associated with more rapid cognitive growth and a later inflection point (i.e., more concentrated, rapid growth) and faster height growth was associated with a later cognitive inflection point. This nuance in findings highlights the methodological strength of the twin study design to identify developmental differences within and between families. Compared to their co-twin, the faster physically growing twin may display a slight lag in their cognitive catch-up. However, compared to a pair of twins in another family, faster-growing twins may demonstrate earlier accelerated cognitive catch-up growth.

The upper asymptotes of height and weight were not significantly associated with the upper asymptote of cognitive ability, indicating that children who ended up larger did not necessarily demonstrate higher cognitive scores. The null association between the upper asymptotes of height and cognitive ability stands in contrast to previous research that has found a positive association between height and cognitive ability in adults (Silventoinen et al., 2012; Sundet et al., 2005). Measurements of height and weight in the Louisville Twin Study are currently only available to 15 years. There were low rates of extreme deficits in physical size after toddlerhood; after age 4, only 3.5% of the twins had a height measurement greater than 2 SD below the population mean and 5.8% had a weight measurement greater than 2 SD below the population mean. Therefore, it is possible that the significant cognitive deficits associated with deficits in physical size are only apparent in the minority of children who continue to be stunted or clinically underweight into childhood and adolescence. Typical variability in height or weight around the upper asymptote may not represent a significant enough deviation to correspond with deviations in cognitive scores.

Limitations and Future Directions

The Louisville Twin Study is a predominantly White (89.3%) sample of American children. Therefore, it is unclear the extent to which findings generalize to other racial and ethnic groups within the United States or to children developing in other states or countries. Rates of extreme deficits in height or weight were very low in the present sample; less than 6% of children in the Louisville Twin Study were more than two SD below the population mean in terms of height-for-age or weight-for-age z-scores after age 4 years. Children born in low-resource countries with higher rates of malnutrition and infectious diseases may experience higher rates of extreme growth deficits (e.g., stunting, wasting; Ssentongo et al., 2021). In such countries, there may be more variability around the upper asymptotes of physical size and a stronger relationship between physical size and cognitive ability at later developmental stages than was observed in this study. Moreover, in countries with greater food scarcity, shared environmental factors may account for a greater proportion of the association between physical growth and cognitive development.

In the United States, rates of premature birth, low birth weight, and adverse birth outcomes are substantially higher in Black, Indigenous, and Latinx populations relative to White and

Asian populations (Martin et al., 2021). Experiences of discrimination and discrimination-related stress partly account for the discrepancies in birth outcomes along racial lines. Additionally, owing to centuries of segregation and discrimination, Black, Latinx, and Indigenous populations may face structural barriers to accessing medical care, which may further contribute to racial discrepancies in birth outcomes and early postnatal development (Bailey, 2017). A critical direction for future human development research is to replicate the intensive prospective study designs in racially and ethnically diverse samples.

Over the duration of the Louisville Twin Study, a variety of cognitive assessments were used based on the age of the children and the release of new test versions. Therefore, different tests and test versions were given within wave and across waves. Early assessments of cognitive ability (e.g., the Bayley) are more reliant on motor skills than assessments conducted at later ages (e.g., the WISC). This may contribute to scores on the assessments in infancy being more sensitive to early deficits in physical size. By fitting growth models to different cognitive tests within wave and across waves, we relied on the assumption that the same underlying cognitive ability was being measured with each assessment. Different tests and different test versions often have very different items and tasks, which prohibited us from testing measurement invariance across assessments. However, wave-to-wave correlations between cognitive ability scores were moderate to high in the Louisville Twin Study (r 's = .49 to .91) and scores on the Bayley at 18 and 24 months correlated significantly with FSIQ scores at 15 years (r 's = .40-.41; Womack et al., 2022). Previous research has observed strong reliability between different versions of the Bayley (r = .76; Gagnon & Neal, 2000) and WISC (r = .84; Slate & Saarnio, 1995). Moreover, performance on the WPPSI-R has been found to correspond with performance on the Stanford Binet (McCrowell & Nagle, 1994), McCarthy (Karr et al., 1993), and WISC-III (Allen & Thorndike, 1995). Thus, there appears to be reasonable overlap in terms of the abilities assessed across test and test versions to permit exploration of longitudinal change in scores over time. Additionally, the use of standardized cognitive scores provided a common reference point to understand a child's abilities relative to a typically developing child their age.

Relatedly, as we used different cognitive assessments and different versions of each assessment, we were restricted to using the overall cognitive ability score. However, specific areas of cognitive development may be more closely related to physical growth. For example, in a sample of monozygotic twins, Edmonds and colleagues (2010) found that within-pair differences in birth weight were associated with verbal intelligence scores, but not performance intelligence scores. There is an ongoing effort to synchronize scores across test versions (e.g., across WISC versions; Beam et al., 2020), which will make it possible to explore associations between physical growth and specific cognitive abilities.

Although the twin study design allowed us to estimate common genetic and shared environmental factors associated with physical and cognitive catch-up growth and approximate quasi-experimental relationships between physical and cognitive development, twin studies have several limitations. First, the pathway from genotype to even "simple" phenotypes like height consists of an incredibly complicated series of interactions between numerous genes and environmental experiences (Turkheimer, 2000; Chabris et al., 2015).

That we observed significant associations between additive genetic portions of physical growth and cognitive growth does not mean a particular gene or constellation of genes is directly responsible for patterns of both physical and cognitive development. Second, the twin study design does not shed light on the specific shared environmental experiences that are related to individual differences in patterns of physical and cognitive development. Identifying specific environmental factors associated with physical and cognitive development is an important step for informing interventions. Although we were able to approximate quasi-experimental relationships by holding constant genetic and environmental factors shared among members of a family, twin studies cannot be used to prove causality and the within-pair (quasi-experimental) associations do not provide any information on potential causal mechanisms relating physical and cognitive development. Within-pair associations may also be confounded by measurement error. However, as it is not practically or ethically feasible to randomly assign children to different birth weights or patterns of physical growth, the twin-study design provides a powerful tool to approximate experimental relationships in human development. As the Louisville Twin Study is currently collecting cognitive data at midlife (Beam et al., 2020), an important future direction will be to test if quasi-experimental associations between birth weight and cognitive ability persist into adulthood.

Finally, although power was sufficient to test associations between physical size and birth and the lower and upper asymptotes of cognitive development, power was lower than desired to test associations between physical development and the rate and shape of cognitive development (i.e., the rate and inflection point of cognitive growth). Replication of findings that birth weight and the rate of weight catch-up growth is associated with the rate and shape of cognitive catch-up growth is warranted in larger samples.

Conclusion

Findings suggest that there is a small to medium association between physical size at birth and early cognitive abilities. Within-pair, the twin that was longer and heavier than their co-twin at birth had higher cognitive scores in infancy. Additionally, within pair, the heavier twin at birth had faster and more prolonged cognitive growth and ultimately had higher cognitive scores in adolescence. Between-pair associations between weight and length at birth and cognitive growth trajectories were mediated by shared environmental factors, highlighting the role of early prenatal experiences (e.g., premature birth) in early physical development and long-term cognitive outcomes.

Prematurity is normative in twin pregnancies (Martin et al., 2021), and it may not be possible to ensure full gestation in all twin pregnancies. In cases where twins are born prematurely or physically undersized, interventions designed to promote height and weight catch-up growth postnatally may encourage early cognitive development. Within pair, a faster rate of weight growth was associated with a faster rate of cognitive growth. Protein- and calcium-enriched nutritional interventions for premature, undersized infants have been found to promote lean body mass gain and healthy bone development (Marini et al., 2003). Such postnatal interventions may have downstream benefits for emerging cognitive development. However, as rapid postnatal weight gain among low birth weight infants has

been linked to cardiovascular disease in adulthood (Kelishadi et al., 2015), it is important to monitor postnatal weight gain trajectories and encourage preventative behaviors, such as physical activity, which may mitigate the health risks associated with catch-up growth (Cesa et al., 2014).

Although this study focused on physical and cognitive development in a sample of twins, it is important to recognize that twins can serve as a developmental model for singletons exposed to early bioenvironmental adversity (e.g., premature birth, low birth weight, etc.). Indeed, singletons born at low birth weight demonstrate a similar pattern of catch-up growth in weight and height between birth and school age (Belfort et al., 2011). It is our hope that findings from this study can inform our understanding of physical and cognitive development in both singletons and multiples.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Data Availability Statement:

The data necessary to reproduce the analyses presented here are not publicly accessible. Analytic scripts are available on the Open Science Framework (<https://osf.io/2xwdc/>). The materials necessary to attempt to replicate the findings presented here are not publicly accessible. The analyses presented here were not preregistered.

References

- Allen SR, & Thorndike RM (1995). Stability of the WPPSI-R and WISC-III factor structure using cross-validation of covariance structure models. *Journal of Psychoeducational Assessment*, 13(1), 3–20. 10.1177/073428299501300101
- Antoniou EE, Fowler T, Thiery E, Southwood TR, Van Gestel S, Jacobs N, Vlietinck R, van Os J, Rijdsdijk FV, Derom C, & Zeegers MP (2013). Intrauterine environment and cognitive development in young twins. *Journal of Developmental Origins of Health and Disease*, 4(6), 513–521. 10.1017/s2040174413000287 [PubMed: 24924230]
- Bailey ZD, Krieger N, Agénor M, Graves J, Linos N, & Bassett MT (2017). Structural racism and health inequities in the USA: evidence and interventions. *The Lancet*, 389(10077), 1453–1463. 10.1016/s0140-6736(17)30569-x
- Bayley N. (1969). Manual for the Bayley scales of infant development. Psychological Corporation.
- Bayley N. (1993). Manual for the Bayley scales of infant development – Second edition. Psychological Corporation.
- Beam CR, Turkheimer E, Finkel D, Levine M, Zandi E, Guterbock T, Giangrande EJ, Ryan L, Pasquenza N, & Davis DW (2020). Midlife study of the Louisville twins: Connecting cognitive development to biological and cognitive aging. *Behavior Genetics*, 50(2), 73–83. 10.1007/s10519-019-09983-6 [PubMed: 31820295]

- Belfort MB, Gillman MW, & McCormick MC (2012). Prenatal and perinatal predictors of blood pressure at school age in former preterm, low birth weight infants. *Journal of Perinatology*, 32(4), 265–269. 10.1038/jp.2011.88 [PubMed: 21738122]
- Blickstein I. (2004). Is it normal for multiples to be smaller than singletons?. *Best practice & research Clinical Obstetrics & Gynaecology*, 18(4), 613–623. 10.1016/j.bpobgyn.2004.04.008 [PubMed: 15279820]
- Cesa CC, Sbruzzi G, Ribeiro RA, Barbiero SM, de Oliveira Petkowicz R, Eibel B, Machado NB, Marques RV, Tortato G, dos Santos TJ, Leiria C, Schaan BD& Pellanda LC (2014). Physical activity and cardiovascular risk factors in children: meta-analysis of randomized clinical trials. *Preventive Medicine*, 69, 54–62. 10.1016/j.ypmed.2014.08.014 [PubMed: 25175591]
- Chabris CF, Lee JJ, Cesarini D, Benjamin DJ, & Laibson DI (2015). The fourth law of behavior genetics. *Current Directions in Psychological Science*, 24(4), 304–312. 10.1177/0963721415580430 [PubMed: 26556960]
- Cheadle JE, & Goosby BJ (2010). Birth weight, cognitive development, and life chances: A comparison of siblings from childhood into early adulthood. *Social Science Research*, 39(4), 570–584. 10.1016/j.ssresearch.2010.03.003
- Cooke RW (2006). Are there critical periods for brain growth in children born preterm?. *Archives of Disease in Childhood-Fetal and Neonatal Edition*, 91(1), F17–F20. 10.1136/adc.2005.077438 [PubMed: 16223756]
- Datar A, & Jackowitz A (2009). Birth weight effects on children’s mental, motor, and physical development: evidence from twins data. *Maternal and Child Health Journal*, 13(6), 780. 10.1007/s10995-009-0461-6 [PubMed: 19308711]
- Davis DW, Turkheimer E, Finkel D, Beam C, & Ryan L (2019). The Louisville twin study: Past, present and future. *Twin Research and Human Genetics*, 22(6), 735–740. 10.1017/thg.2019.37 [PubMed: 31362801]
- Edmonds CJ, Isaacs EB, Cole TJ, Rogers MH, Lanigan J, Singhal A, Birbara T, Gringas P, Denton J, & Lucas A (2010). The effect of intrauterine growth on verbal IQ scores in childhood: a study of monozygotic twins. *Pediatrics*, 126(5), e1095–e1101. 10.1542/peds.2008-3684 [PubMed: 20937654]
- Enders CK (2013). Dealing with missing data in developmental research. *Child Development Perspectives*, 7(1), 27–31. 10.1111/cdep.12008
- Estourgie-van Burk GF, Bartels M, Boomsma DI, & Delemarre-van de Waal HA (2010). Body size of twins compared with siblings and the general population: From birth to late adolescence. *The Journal of Pediatrics*, 156(4), 586–591. 10.1016/j.jpeds.2009.10.045 [PubMed: 20036377]
- Fattal-Valevski A, Toledano-Alhadeef H, Leitner Y, Geva R, Eshel R, & Harel S (2009). Growth patterns in children with intrauterine growth retardation and their correlation to neurocognitive development. *Journal of Child Neurology*, 24(7), 846–851. 10.1177/0883073808331082 [PubMed: 19617460]
- Flensburg-Madsen T, & Mortensen EL (2017). Birth weight and intelligence in young adulthood and midlife. *Pediatrics*, 139(6), e20163161. 10.1542/peds.2016-3161 [PubMed: 28562263]
- Freides D. (1972). Review of the Stanford-Binet intelligence scale, third revision. *The seventh mental measurements yearbook*, 1, 772–773.
- Gagnon SG, & Nagle RJ (2000). Comparison of the revised and original versions of the Bayley Scales of Infant Development. *School Psychology International*, 21(3), 293–305. 10.1177/0143034300213006
- Ghods E, Kreissl A, Brandstetter S, Fuiko R, & Widhalm K (2011). Head circumference catch-up growth among preterm very low birth weight infants: effect on neurodevelopmental outcome. *Journal of Prenatal Medicine* 39(5), 579–86. 10.1515/jpm.2011.049
- Giangrande EJ, Beam CR, Finkel D, Davis DW, & Turkheimer E (2022). Genetically informed, multilevel analysis of the Flynn Effect across four decades and three WISC versions. *Child Development*, 93(1), e47–e58. 10.1111/cdev.13675 [PubMed: 34762291]
- Goldenberg RL, Culhane JF, Iams JD, & Romero R (2008). Epidemiology and causes of preterm birth. *The Lancet*, 371(9606), 75–84. 10.1016/s0140-6736(08)60074-4

- Grimm K, Zhang Z, Hamagami F, & Mazzocco M (2013). Modeling nonlinear change via latent change and latent acceleration frameworks: Examining velocity and acceleration of growth trajectories. *Multivariate Behavioral Research*, 48(1), 117–143. 10.1080/00273171.2012.755111 [PubMed: 26789211]
- Karr SK, Carvajal H, Elser D, Bays K, Logan RA, & Page GL (1993). Concurrent validity of the WPPSI—R and the McCarthy Scales of Children's Abilities. *Psychological Reports*, 72(3), 940–942. 10.2466/pr0.1993.72.3.940
- Kuczumski RJ (2002). 2000 CDC Growth Charts for the United States: methods and development (No. 246). Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics.
- Kohlhauser C, Fuiko R, Panagl A, Zadra C, Haschke N, Brandstetter S, Weninger M, & Pollak A (2000). Outcome of very-low-birth-weight infants at 1 and 2 years of age: the importance of early identification of neurodevelopmental deficits. *Clinical Pediatrics*, 39(8), 441–449. 10.1177/00092280003900801 [PubMed: 10961816]
- Kuklina EV, Ramakrishnan U, Stein AD, Barnhart HH, & Martorell R (2006). Early childhood growth and development in rural Guatemala. *Early Human Development*, 82(7), 425–433. 10.1016/j.earlhumdev.2005.10.018 [PubMed: 16431042]
- Marceau K, McMaster MT, Smith TF, Daams JG, van Beijsterveldt CE, Boomsma DI, & Knopik VS (2016). The prenatal environment in twin studies: a review on chorionicity. *Behavior Genetics*, 46(3), 286–303. 10.1007/s10519-016-9782-6 [PubMed: 26944881]
- Marini A, Vegni C, Gangi S, Benedetti V, & Agosti M (2003). Influence of different types of post-discharge feeding on somatic growth, cognitive development and their correlation in very low birthweight preterm infants. *Acta Pædiatrica*, 92, 18–33. 10.1111/j.1651-2227.2003.tb00642.x
- Martin JA, Hamilton BE, Osterman MJ, Driscoll AK (2021) Births: Final data for 2019. *National Vital Statistics Report*, 70(2), 1–51. 10.15620/cdc:100472
- McArdle JJ, & Prescott CA (2005). Mixed-effects variance components models for biometric family analyses. *Behavior Genetics*, 35(5), 631–652. 10.1007/s10519-005-2868-1 [PubMed: 16184490]
- McCarthy D. (1972). *Manual for the McCarthy Scales of Children's Abilities*. New York: Psychological Corporation.
- McCrowell KL, & Nagle RJ (1994). Comparability of the WPPSI-R and the SB: IV among preschool children. *Journal of Psychoeducational Assessment*, 12(2), 126–134. 10.1177/073428299401200202
- Muthén LK, & Muthén BO (2017). *Mplus. Statistical analysis with latent variables. User's guide*, 8.
- Pongcharoen T, Ramakrishnan U, DiGirolamo AM, Winichagoon P, Flores R, Singkhornard J, & Martorell R (2012). Influence of prenatal and postnatal growth on intellectual functioning in school-aged children. *Archives of Pediatrics & Adolescent Medicine*, 166(5), 411–416. 10.1001/archpediatrics.2011.1413 [PubMed: 22566539]
- R Core Team (2022). *R: A language and environment for statistical computing*. R Foundation for Statistical Computing, Vienna, Austria. URL <https://www.R-project.org/>.
- Ratkowsky D. (1983). *Nonlinear regression modelling*. Marcel Dekker, New York.
- Rubin DB (1976). Inference and missing data. *Biometrika*, 63(3), 581–592. 10.2307/2335739
- Scharf RJ, Stroustrup A, Conaway MR, & DeBoer MD (2016). Growth and development in children born very low birthweight. *Archives of Disease in Childhood-Fetal and Neonatal Edition*, 101(5), F433–F438. 10.1136/archdischild-2015-309427 [PubMed: 26627552]
- Shenkin SD, Starr JM, & Deary IJ (2004). Birth weight and cognitive ability in childhood: a systematic review. *Psychological Bulletin*, 130(6), 989–1013. 10.1037/0033-2909.130.6.989 [PubMed: 15535745]
- Silventoinen K, Iacono WG, Krueger R, & McGue M (2012). Genetic and environmental contributions to the association between anthropometric measures and IQ: a study of Minnesota twins at age 11 and 17. *Behavior Genetics*, 42(3), 393–401. 10.1007/s10519-011-9521-y [PubMed: 22139438]
- Silventoinen K, Posthuma D, Van Beijsterveldt T, Bartels M, & Boomsma DI (2006). Genetic contributions to the association between height and intelligence: evidence from Dutch twin data from childhood to middle age. *Genes, Brain and Behavior*, 5(8), 585–595. 10.1111/j.1601-183x.2006.00208.x [PubMed: 17081263]

- Slate JR, & Saarnio DA (1995). Differences between WISC-III and WISC-R IQs: a preliminary investigation. *Journal of Psychoeducational Assessment*, 13(4), 340–346. 10.1177/073428299501300401
- Ssentongo P, Ssentongo AE, Ba DM, Ericson JE, Na M, Gao X, Fronterre C, Chinchilli VM, & Schiff SJ (2021). Global, regional and national epidemiology and prevalence of child stunting, wasting and underweight in low-and middle-income countries, 2006–2018. *Scientific Reports*, 11(1), 1–12. 10.1038/s41598-021-84302-w [PubMed: 33414495]
- Sudfeld CR, McCoy DC, Danaei G, Fink G, Ezzati M, Andrews KG, & Fawzi WW (2015). Linear growth and child development in low-and middle-income countries: a meta-analysis. *Pediatrics*, 135(5), e1266–e1275. 10.1542/peds.2014-3111 [PubMed: 25847806]
- Sundet JM, Tambs K, Harris JR, Magnus P, & Torjussen TM (2005). Resolving the genetic and environmental sources of the correlation between height and intelligence: A study of nearly 2600 Norwegian male twin pairs. *Twin Research and Human Genetics*, 8(4), 307–311. 10.1375/1832427054936745 [PubMed: 16176713]
- Tjørve KM, & Tjørve E (2017). The use of Gompertz models in growth analyses, and new Gompertz-model approach: An addition to the Unified-Richards family. *PloS one*, 12(6), e0178691. 10.1371/journal.pone.0178691 [PubMed: 28582419]
- Turkheimer E. (2000). Three laws of behavior genetics and what they mean. *Current Directions in Psychological Science*, 9(5), 160–164. 10.1111/1467-8721.00084
- Turkheimer E, & Harden KP (2014). Behavior genetic research methods: Testing quasi-causal hypotheses using multivariate twin data. In Reis HT & Judd CM (Eds.), *Handbook of research methods in social and personality psychology* (pp. 159–187). Cambridge University Press.
- Upadhyay RP, Naik G, Choudhary TS, Chowdhury R, Taneja S, Bhandari N, Martines JC, Bahl R, & Bhan MK (2019). Cognitive and motor outcomes in children born low birth weight: a systematic review and meta-analysis of studies from South Asia. *BMC pediatrics*, 19(1), 1–15. 10.1186/s12887-019-1408-8 [PubMed: 30606158]
- Varella MH, & Moss WJ (2015). Early growth patterns are associated with intelligence quotient scores in children born small-for-gestational age. *Early Human Development*, 91(8), 491–497. 10.1016/j.earlhumdev.2015.06.002 [PubMed: 26100090]
- van Baal CG, & Boomsma DI (1998). Etiology of individual differences in birth weight of twins as a function of maternal smoking during pregnancy. *Twin Research and Human Genetics*, 1(3), 123–130. 10.1375/136905298320566258
- Wechsler D. (1967). *Manual for the Wechsler preschool and primary scale of intelligence*. Psychological Corporation.
- Wechsler D. (1974). *Wechsler intelligence scale for children—revised*. New York: Psychological Corporation.
- Wechsler D (1989). *WPPSI-R: Wechsler Preschool and Primary Scale of Intelligence-Revised*. San Antonio: Psychological Corporation.
- Wechsler D. (1991). *The Wechsler intelligence scale for children—third edition*. San Antonio, TX: The Psychological Corporation.
- Wechsler D, & Kodama H (1949). *Wechsler intelligence scale for children (Vol. 1)*. New York: Psychological corporation.
- Wilson RS (1970). Bloodtyping and twin zygosity. *Human Heridity*, 20, 30–56. 10.1159/000152292
- Wilson RS (1972). Twins: Early mental development. *Science*, 175(4024), 914–917. 10.1126/science.175.4024.914 [PubMed: 5061798]
- Wilson RS (1974). Twins: Mental development in the preschool years. *Developmental Psychology*, 10(4), 580–588. 10.1037/h0036596
- Wilson RS (1979). Twin growth: initial deficit, recovery, and trends in concordance from birth to nine years. *Annals of Human Biology*, 6(3), 205–220. 10.1080/03014467900007212 [PubMed: 573983]
- Wilson RS & Matheny AP (1986). Behavior-genetics research in infant temperament: The Louisville Twin Study. In: Plomin R & Dunn J (Eds.) *The study of temperament: changes, continuities and challenges* (pp. 81-97). Erlbaum, Hillsdale, pp 81–97. Lawrence Erlbaum Associates.

- Womack SR, Beam CR, Davis DW, Finkel D, Turkheimer E (2021). Genetic and Environmental Correlates of the Nonlinear Recovery of Cognitive Ability in Twins. *Developmental Psychology*, 58(3), 535–550. 10.1037/dev0001305 [PubMed: 34881967]
- Womack SR, Beam CR, Giangrande EJ, Scharf RJ, Tong X, Ponnappalli M, Davis DW, Turkheimer E (2023). Nonlinear catch-up growth in height, weight, and head circumference from birth to adolescence: A longitudinal twin study. *Behavior Genetics* 10.1007/s10519-023-10151-0

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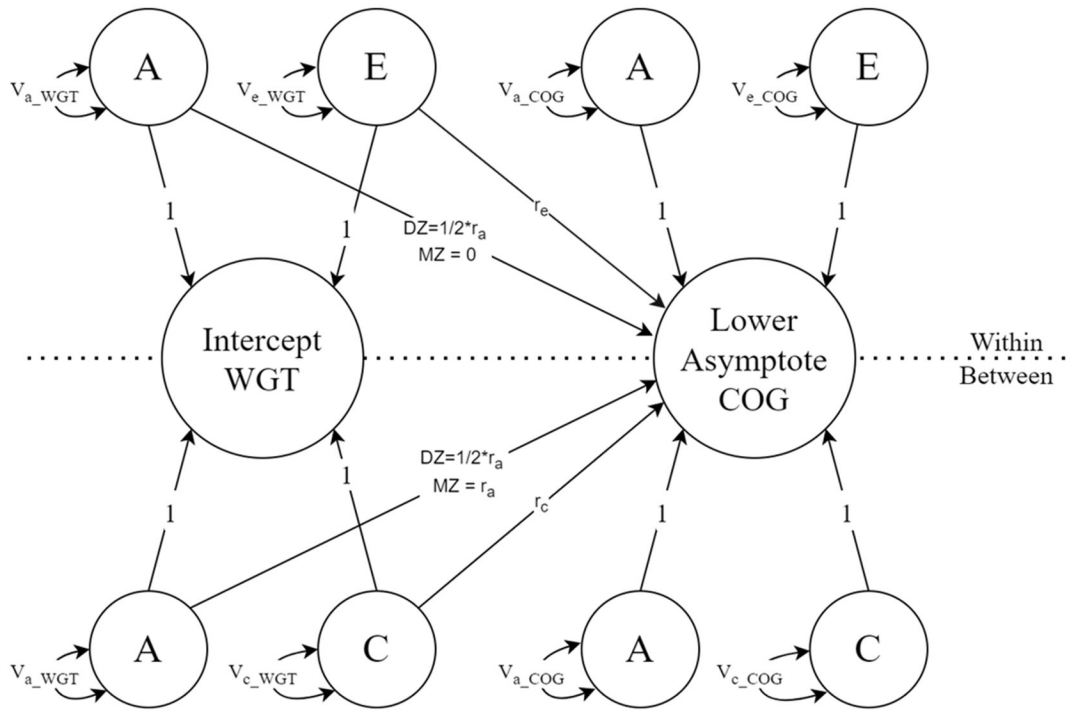


Figure 1. Simplified Path Diagram of the Quasi-Experimental Multilevel Twin Model
Note. For clarity, only the association between the intercept of weight and the lower asymptote of cognitive ability is shown. The study models included regression paths from the ACE parameters for all physical growth parameters to all cognitive growth parameters. WGT = weight, COG = cognitive, A = additive genetic, C = shared environment, E = nonshared environment.

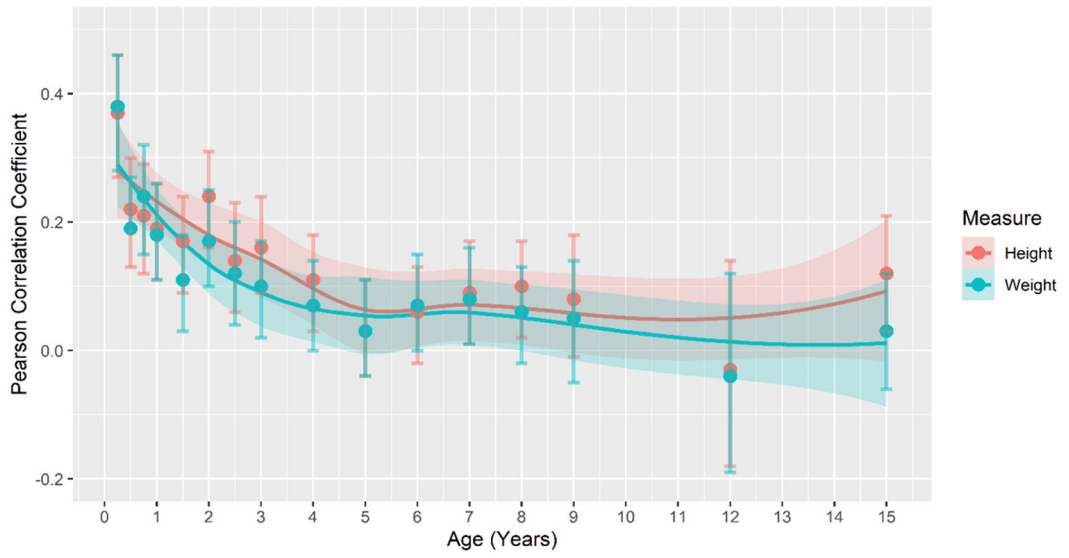


Figure 2. Concurrent Correlations between Physical Size and Cognitive Ability
Note. Each point reflects the Pearson correlation coefficient (r) between the physical size measurement and cognitive ability score at that age. The error bars reflect the 95% confidence interval around the correlation coefficient estimate.

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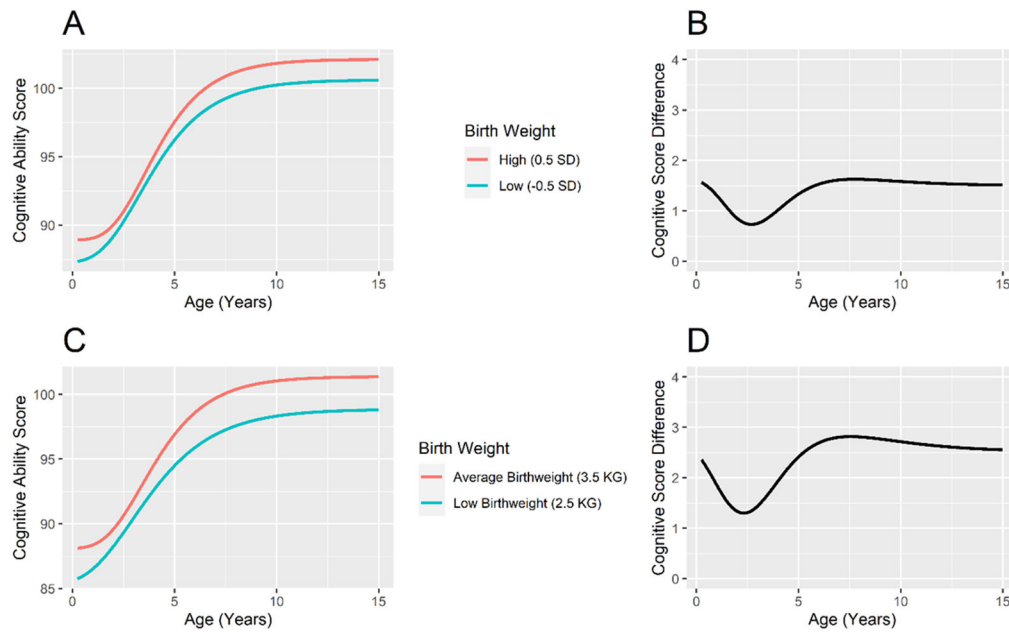


Figure 3. Illustration of the Quasi-Experimental Effect of Birth Weight on Cognitive Development

Note. The top left plot (A) depicts expected cognitive catch-up growth trajectories for a twin with a birth weight 0.5 SD above the population mean and a twin with a birth weight 0.5 SD below the population mean. This 1-unit within-pair difference in standardized birth weight measurements corresponds to a within-pair difference in birth weight of 1.19 kg for male twins and a 1.07 kg within-pair difference for female twins. The within-pair difference in cognitive scores from infancy to adolescence for a 1-unit within-pair difference in standardized birth weight is depicted in the upper right corner (B). For every 1-unit within-pair difference in birth weight Z-scores, there is a corresponding 1.5-point within-pair difference in adolescent intelligence quotient scores. The bottom left plot (C) depicts trajectories of cognitive catch-up growth for a twin born at average birth weight (3.5 kg) and a twin born at low birth weight (2.5kg). The bottom right plot (D) depicts the within-pair difference in cognitive ability scores between a twin born at average birth weight and their co-twin born at low birth weight. Relative to a child born at average birth weight, the quasi-casual effect of being born at low birth weight corresponds to about a 2.5-point deficit in intelligence quotient scores in adolescence.

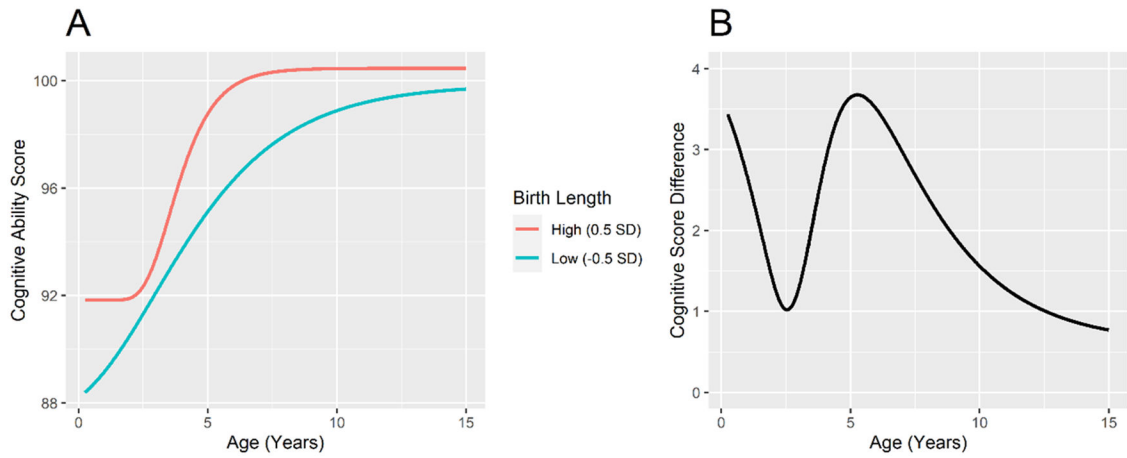


Figure 4. Illustration of the Quasi-Experimental Effect of Birth Length on Cognitive Development

Note. The diagram on the left (A) shows expected cognitive catch-up growth trajectories for a twin with a birth length 0.5 SD above the population mean and a twin with a birth length 0.5 SD below the population mean. This 1-unit within-pair difference in standardized birth length measurements corresponds to a within-pair difference in birth weight of 2.65 cm for male twins and a 2.47 cm within-pair difference for female twins. The within-pair difference in cognitive scores from infancy to adolescence for a 1-unit within-pair difference in standardized birth weight is depicted in the plot on the right (B).

Table 1

Phenotypic Correlations between Catch-Up Growth of Weight and Cognitive Ability

| | Intercept WGT | Upper Asymptote WGT | Rate WGT | Inflection Point WGT | Lower Asymptote COG | Upper Asymptote COG | Rate COG | Inflection Point COG |
|----------------------|--------------------------|------------------------------------|---------------------|-------------------------------------|------------------------------------|------------------------------------|---------------------|-------------------------------------|
| Intercept WGT | - | 99.7% | 11.8% | 94.1% | 100% | 94.1% | 43.4% | 43.4% |
| Upper Asymptote WGT | .19* | - | 100% | 100% | 93.8% | 5.2% | 24.7% | 3.5% |
| Rate WGT | -.02 | -.28* | - | 99.7% | 86.1% | 4.9% | 47.2% | 44.1% |
| Inflection Point WGT | .14* | -.36* | .17* | - | 79.9% | 39.9% | 41.3% | 22.9% |
| Lower Asymptote COG | .51* | .19* | -.17* | -.16* | - | 19.4% | 77.8% | 47.9% |
| Upper Asymptote COG | .16* | .01 | .01 | .06 | .07 | - | 96.5% | 99.3% |
| Rate COG | .18* | .10* | -.17* | -.15* | .44* | -.40* | - | 56.2% |
| Inflection Point COG | .14* | .00 | -.13* | -.08 | .51* | -.35* | .59* | - |

Note. Pearson correlation coefficients are presented below the diagonal. 95% confidence intervals are presented below the correlation coefficients. Significant correlations are bolded for clarity. Results from the Monte Carlo power analysis are presented above the diagonal. The power analyses refer to the percent of the 1,000 simulated datasets detected a statistically significant correlation at $p < .05$.

Table 2

Phenotypic Correlations between Catch-Up Growth of Height and Cognitive Ability

| | Intercept HGT | Upper Asymptote HGT | Rate HGT | Inflection Point HGT | Lower Asymptote COG | Upper Asymptote COG | Rate COG | Inflection Point COG |
|----------------------|--------------------------|------------------------------------|---------------------|-------------------------------------|------------------------------------|------------------------------------|---------------------|-------------------------------------|
| Intercept HGT | - | 100% | 100% | 100% | 99.5% | 100% | 3.9% | 3.4% |
| Upper Asymptote HGT | .53* | - | 21.7% | 100% | 29.6% | 81.3% | 17.2% | 43.3% |
| Rate HGT | -.28* | -.04 | - | 100% | 63.5% | 3.4% | 24.6% | 5.4% |
| Inflection Point HGT | .37* | -.18* | -.30* | - | 93.6% | 9.4% | 61.1% | 40.4% |
| Lower Asymptote COG | .32* | .08 | -.13 | .23* | - | 55.2% | 38.9% | 27.6% |
| Upper Asymptote COG | .16* | .09 | .00 | -.01 | .15 | - | 99.0% | 100% |
| Rate COG | -.01 | -.09 | -.12 | .21* | .32* | -.42* | - | 41.4% |
| Inflection Point COG | .01 | -.12* | -.02 | .13 | .38* | -.35* | .53* | - |

Note. Pearson correlation coefficients are presented below the diagonal. 95% confidence intervals are presented below the correlation coefficients. Significant correlations are bolded for clarity. Results from the Monte Carlo power analysis are presented above the diagonal. The power analyses refer to the percent of the 1,000 simulated datasets detected a statistically significant correlation at $p < .05$.

Table 3**Biometric Associations between Weight and Cognitive Catch-Up Growth**

| | Additive Genetic | Shared Environmental | Nonshared Environmental |
|---|--------------------------------|--------------------------------|--------------------------------|
| Intercept WGT → Lower Asymptote COG Ability | 0 | 0.79 [0.59, 1.00] | 0.11 [0.03, 0.20] |
| Intercept WGT → Upper Asymptote COG Ability | 0 | 0.24 [0.09, 0.40] | 0.10 [0.01, 0.19] |
| Intercept WGT → Rate COG Ability | 0 | 0.28 [0.04, 0.52] | 0.06 [-0.10, 0.21] |
| Intercept WGT → Inflection Point COG Ability | 0 | 1.38 [0.29, 2.48] | 0.28 [-0.24, 0.80] |
| Upper Asymptote WGT → Lower Asymptote COG Ability | -0.02 [-0.18, 0.22] | -0.12 [-1.24, 1.00] | 0.11 [-0.05, 0.27] |
| Upper Asymptote WGT → Upper Asymptote COG Ability | 0.06 [-0.07, 0.19] | -0.28 [-0.83, 0.27] | 0.00 [-0.14, 0.14] |
| Upper Asymptote WGT → Rate COG Ability | 0.06 [-0.17, 0.29] | -0.45 [-1.47, -0.56] | 0.16 [-0.12, 0.44] |
| Upper Asymptote WGT → Inflection Point COG Ability | -0.55 [-1.10, 0.07] | 0 | 0.29 [-0.69, 1.28] |
| Rate WGT → Lower Asymptote COG Ability | -0.15 [-0.27, -0.03] | 1.30 [-2.57, 5.17] | 0.19 [0.07, 0.32] |
| Rate WGT → Upper Asymptote COG Ability | 0.07 [-0.08, 0.19] | -1.81 [-6.14, 2.53] | -0.03 [-0.12, 0.06] |
| Rate WGT → Rate COG Ability | -0.19 [-0.38, 0.00] | 2.22 [-3.48, 7.92] | 0.25 [0.04, 0.45] |
| Rate WGT → Inflection Point COG Ability | -0.95 [-1.85, -0.04] | 12.67 [-22.48, 47.82] | 0.86 [0.21, 1.52] |
| Inflection Point WGT → Lower Asymptote COG Ability | 0.18 [0.00, 0.36] | -0.55 [-0.90, -0.19] | -0.17 [-0.32, -0.02] |
| Inflection Point WGT → Upper Asymptote COG Ability | 0.32 [0.06, 0.59] | -0.27 [-0.57, 0.02] | -0.21 [-0.41, 0.00] |
| Inflection Point WGT → Rate COG Ability | -0.23 [-0.53, 0.07] | 0.02 [-0.45, 0.48] | -0.10 [-0.38, 0.18] |
| Inflection Point WGT → Inflection Point COG Ability | 0.04 [-1.05, 1.13] | -0.96 [-2.84, 0.92] | -0.75 [-1.63, 0.12] |

Note. Results are unstandardized regression coefficients. 95% confidence intervals are presented in brackets below the parameter estimates. Significant associations are bolded for clarity. Estimates that are 0 without a confidence interval were initially estimated to be negative and were constrained to equal 0.

Table 4

Biometric Association between Height and Cognitive Catch-Up Growth

| | Additive Genetic | Shared Environmental | Nonshared Environmental |
|---|--------------------------------|--------------------------------|--------------------------------|
| Intercept HGT → Lower Asymptote COG Ability | -0.83 [-1.61, -0.05] | 0.64 [0.27, 1.00] | 0.29 [0.15, 0.43] |
| Intercept HGT → Upper Asymptote COG Ability | -0.86 [-2.48, 0.76] | 0.42 [0.21, 0.63] | 0.04 [-0.13, 0.20] |
| Intercept HGT → Rate COG Ability | 0.11 [-1.46, 1.66] | -0.06 [-0.62, 0.50] | 0.67 [0.18, 1.15] |
| Intercept HGT → Inflection Point COG Ability | 0 | 0.48 [-0.92, 1.88] | 0.57 [-0.37, 1.52] |
| Upper Asymptote HGT → Lower Asymptote COG Ability | -0.08 [-0.25, 0.09] | 0.32 [-0.40, 1.04] | -0.05 [-0.24, 0.15] |
| Upper Asymptote HGT → Upper Asymptote COG Ability | 0.23 [0.09, 0.37] | -0.90 [-1.79, -0.02] | 0.06 [-0.12, 0.25] |
| Upper Asymptote HGT → Rate COG Ability | -0.50 [-0.80, -0.21] | 1.89 [0.01, 3.78] | 0.11 [-0.55, 0.78] |
| Upper Asymptote HGT → Inflection Point COG Ability | -1.04 [-1.67, -0.41] | 0 | 0.32 [-0.97, 1.57] |
| Rate HGT → Lower Asymptote COG Ability | -0.14 [-0.27, -0.02] | 0.06 [-0.12, 0.26] | 0.13 [0.03, 0.22] |
| Rate HGT → Upper Asymptote COG Ability | 0.15 [-0.00, 0.30] | -0.15 [-0.30, 0.01] | -0.11 [-0.01, 0.22] |
| Rate HGT → Rate COG Ability | 0 | -0.16 [-0.44, 0.13] | -0.25 [-0.60, 0.10] |
| Rate HGT → Inflection Point COG Ability | -1.10 [-1.99, -0.20] | 0 | 0.97 [0.32, 1.62] |
| Inflection Point HGT → Lower Asymptote COG Ability | -0.01 [-0.12, 0.10] | -0.03 [-0.20, 0.14] | -0.25 [-0.49, -0.01] |
| Inflection Point HGT → Upper Asymptote COG Ability | 0 | 0.03 [-0.09, 0.15] | -0.48 [-0.90, -0.07] |
| Inflection Point HGT → Rate COG Ability | -0.10 [-0.33, 0.12] | 0 | 0.41 [-0.46, 1.29] |
| Inflection Point HGT → Inflection Point COG Ability | 0 | 0 | -0.62 [-2.46, 1.22] |

Note. Results are unstandardized regression coefficients. 95% confidence intervals are presented in brackets below the parameter estimates. Significant associations are bolded for clarity. Estimates that are 0 without a confidence interval were initially estimated to be negative and were constrained to equal 0.