Genetic and Environmental Correlates of Physical and Cognitive Development in Twins: A Prospective Study of Recovery from Early Bio-Environmental Adversity

Sean Richard Womack
Lake Orion, Michigan

B.S., University of Pittsburgh, 2014
M.A., University of Virginia, 2018

A Dissertation Presented to the Graduate Faculty of the University of Virginia in Candidacy for the Degree of Doctor of Philosophy

Department of Psychology
August, 2023

Committee Members:
Eric Turkheimer Ph.D. (Chair)
Melvin N. Wilson Ph.D.
Xin (Cynthia) Tong Ph.D.
Rebecca Scharf M.D. (Department of Pediatrics)

Abstract

Relative to singletons, twins are at an elevated risk to be born prematurely and at low birth weight. Consequently, twins are small relative to population norms on anthropomorphic measurements in infancy and score about a standard deviation below the population mean on early cognitive assessments. However, by middle childhood twins are average physically and cognitively. Although twins make gains toward the population mean physically and cognitively over the first years of life, the recovery process is not well understood. The goal of this dissertation is to explore the process of physical and cognitive recovery using prospective data from a community sample of twins followed from infancy to adolescence. To that end, I fit a series of growth models to age-standardized measurements of height, weight, head circumference, and cognitive ability to identify typical trajectories of physical and cognitive recovery in twins. Leveraging the genetically informative portion of the data, I conducted biometric analyses to determine the proportion of the variance of recovery associated with genetic and environmental factors. I then tested associations between aspects of the environment (e.g., gestational age, family socioeconomic status) and patterns of recovery. Finally, I explored the co-development of physical size and cognitive ability using models of parallel development and dynamic development.

Recovery of height, weight, and head circumference was most rapid in the first year of life whereas cognitive recovery accelerated in toddlerhood. Shared environmental factors explained the majority of the variance in early physical size and cognitive ability. The rate, shape, and magnitude of physical and cognitive recovery was related to a combination of genetic and environmental factors. Associations between physical and cognitive development were moderate to strong in infancy, but nonsignificant by adolescence. There was some evidence of a dynamic relationship between physical recovery and cognitive development in infancy with larger height and head circumference measurements leading to increases in the rate of cognitive growth. Although the present study focuses on twins, twins can serve as a model for singleton development. Findings from this dissertation inform our understanding of typical patterns of physical and cognitive recovery following exposure to early bioenvironmental adversity, which can be used to identify children displaying atypical development.

Acknowledgments

There are so many people who have helped play a role in this project coming to fruition.

I have been incredibly fortunate to have excellent mentorship through my training. I want to thank Danny Shaw, who took me on as an undergraduate research assistant and fostered my early interest in developmental psychopathology. I am indebted to Melvin Wilson and Eric Turkheimer for their mentorship and support over my graduate career. Mel has always encouraged me to think about the real-world applications of my research and has helped me become a much stronger writer. Mel, you will be pleased with how few uses of "this" and "these" are in this document. Through Eric I have learned a great deal about the utility and limitations of genetically-informed methods and, more importantly, how to think critically about science. Cynthia Tong has taught me a considerable amount about longitudinal methods and their applications. I am grateful for Rebecca Scharf's perspective as a pediatrician and her input on this project.

I am most grateful for my family, who has been my biggest source of support and encouragement throughout my educational journey. To my parents, Rich and Cathy Womack, who instilled the values and work ethic necessary to make it to this point, I would not be here without you. Brice, Erin, Avery, and Gaven, I am incredibly lucky to have four younger siblings who I can say I look up to. I especially want to shout out my youngest brothers, Gaven and Avery, who will forever be a slide in my talks as the pinnacle model of fraternal twins. Finally, I want to thank my fiancée, Allie Silverman, who has provided endless love and support through the highs and lows of this process. I could not ask for a better life partner!

Table of Contents

Overview and Study Aims

Relative to singletons, twins are exposed to elevated rates of perinatal stressors including premature birth, low birth weight, and neonatal diseases (Giuffrè et al., 2012; Martin et al., 2021). According to the National Vital Statistics Report of 2019 births (Martin et al., 2021), 61% of twins were born prematurely (defined as less than 37 weeks gestation) and 56% were born at low birth weight (defined as less than 2,500 grams). Relative to singletons, rates of premature birth and low birth weight in twins were 7.2 and 8.3 times higher, respectively (Martin et al., 2021). Consequently, in infancy twins tend to fall about one standard deviation below the population mean in terms of height, weight, head circumference, and standardized cognitive ability (Estourgie-van Burk et al., 2010; Koeppen-Schomerus et al., 2000; Price et al., 2000; Wilson, 1979). Throughout childhood twins make rapid progress toward the population mean in physical size and cognitive ability and tend to be average physically and cognitively by middle childhood (Estourgie-van Burk et al., 2009; Estourgie-van Burk et al., 2010; Wilson, 1974; Wilson, 1979).

In a series of studies using prospective data from the Louisville Twin Study, Ronald Wilson documented early deficits in cognitive ability and physical size in twins as well as subsequent recovery to the population mean (Wilson, 1972; Wilson, 1974; Wilson, 1974b; Wilson, 1975; Wilson, 1979). Although it is well understood that twins fall below population norms physically and cognitively in infancy and are average physically and cognitively by middle childhood, little is known about the *process* by which twins recover. Specifically, the rate and shape of recovery of physical size and cognitive ability have not been documented in previous research. Thus, it is unclear how quickly twins recover and if there are developmental periods where the rate of recovery is more rapid. Additionally, the relative contribution of genetic and environmental factors to the recovery of physical size and cognitive ability are currently unknown. Finally, it is unclear the extent to which recovery in physical size is related to the recovery of cognitive ability. Although previous research generally suggests that physical and cognitive recovery occurs within the first five years of life (Estourgie-van Burk et al., 2009; Estourgie-van Burk et al., 2010; Wilson, 1974; Wilson, 1979), it is unclear if physical and cognitive recovery are strongly associated or largely independent processes.

Understanding the processes of physical and cognitive recovery in twins is important for several reasons. Both physical size and cognitive ability are strong predictors of numerous indicators of wellbeing (Calvin et al., 2007; Ceci & Williams, 1997; Roh et al., 2014). Physically undersized infants have an increased likelihood of premature mortality and are at risk to display deficits in early motor development and cognitive tasks (Belanger & Caron, 2018; Luke & Keith, 1992). Moreover, adults who are clinically underweight are at an increased risk for premature mortality and other health complications (Roh et al., 2014). Likewise, performance on intelligence tests in adolescence and adulthood is positively associated with educational attainment, employment, and physical health in adulthood and negatively associated with delinquency and risk for premature mortality (Batty et al., 2007; Calvin et al., 2007; Ceci & Williams, 1997; Farrington, 2005). From a developmental psychopathology perspective (Rutter & Sroufe, 2000), understanding typical patterns of recovery among children displaying early physical and cognitive deficits can inform our understanding of atypical development, or children not on track to catch up physically or cognitively. Knowing the typical magnitude of initial deficits as well as when and how quickly recovery occurs can be used to identify infants who are at a greater initial risk or who are not recovering as expected. This information can then be used to guide placement in intervention

services (e.g., nutrition supplementation to promote physical growth, Early Intervention to foster cognitive development).

Using prospective data from the Louisville Twin Study (Davis et al., 2019; Wilson, 1983) I aim to address gaps in the literature related to physical and cognitive recovery in twins across childhood. Since the publication of Wilson's seminal papers documenting physical and cognitive recovery in the Louisville Twins, the sample size of the Louisville Twin Study has nearly doubled and standardized cognitive data and physical growth data have been collected to age 15 years (Davis et al., 2019), and are currently being collected in midlife (Beam et al., 2020). My dissertation can be broken down into three aims, which are described in detail below. I aim to extend early Louisville Twin Study research and more broadly shed light on the *process* by which twins recover physically and cognitively across childhood. Moreover, as twins can serve as a model for singleton development, findings may provide insight into the process of recovery of cognitive ability and physical size for singletons born at bio-environmental risk (e.g., anoxic, prematurely, at low birth weight).

Aim 1: Model the Recovery of Height, Weight, and Head Circumference in Twins Across Childhood

Observations of average height and weight in twin samples at different developmental periods suggest that the mean height and weight of twins falls significantly below the population mean in infancy, but that twins generally catch up to the population mean by early childhood (Estourgie-van Burk, 2010; Wilson, 1979). However, that I am aware of, there has been no published work using growth models to describe the recovery of physical size in twins across childhood. Growth curve models can provide information on the average rate of growth over time as well as acceleration or deceleration in the average rate of growth over time, information that cannot be obtained from examining mean differences across study waves. Thus, the first aim of the

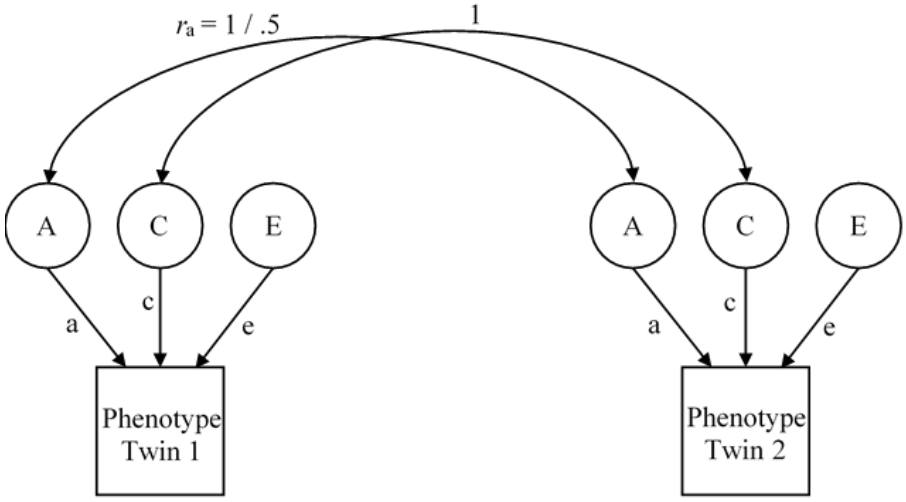


Figure 1. Path diagram for a classic ACE decomposition where a, c, and e represent factor loadings for the additive genetic, shared environmental, and nonshared environmental factors, respectively. Correlations between the additive genetic (A) factors are constrained to 1 for monozygotic twins and 0.5 for dizygotic twins. Correlations between the shared environmental (C) factors are constrained to 1 and correlations between the nonshared environmental factors (E) are not estimated.

dissertation seeks to describe the typical rate and shape of "recovery" of height-for-age and weight for-age z-scores in twins from birth to 15 years. In modeling the growth in height and weight, I considered nonlinear exponential and sigmoid-shaped (S-shaped) asymptotic growth models (Grimm & Ram, 2009; Grimm et al., 2013). I then leveraged the genetically informative portion of the study to decompose the variance of the growth parameters into additive genetic (A) variance, shared environmental (C) variance, and nonshared environmental (E) variance. Additive genetic variance refers to the sum of genetic contributions, the shared environment refers to features of the rearing environment that increase twin similarity (e.g., shared parenting experiences), and the nonshared environment refers to features of the rearing environment that reduce twin similarity including measurement error (e.g., having different teachers). As monozygotic (identical) twins share 100% of the same genes and dizygotic (fraternal) twins share 50% of the same genes, the variance in any phenotype can be decomposed into additive genetic, shared environmental, and nonshared environmental components through structural equation modeling (see Figure 1 for a generic ACE factor model). Finally, I examined measured shared and nonshared biological and environmental correlates of the recovery of height and weight, including socioeconomic status (SES), gestational age, and maternal age.

Aim 2: Model the Recovery in Cognitive Ability in Twins from Infancy to Adolescence

Similar to Aim 1, the second aim of the proposed dissertation focuses on the recovery of cognitive ability in twins across childhood. I focused on general cognitive ability as participants in the study sample completed a variety of age-appropriate standardized cognitive assessments over the course of the study (see Davis et al., 2019), all of which include a comparable general cognitive ability score. As with models of physical growth, I considered nonlinear exponential and sigmoid-shaped (S-shaped) asymptotic growth models. Once the best-fitting growth model for cognitive ability is established, I conducted biometric analyses to decompose the variance of the growth parameters into additive genetic, shared environmental, and nonshared environmental terms. Finally, I examined measured shared and nonshared environmental correlates to the recovery of cognitive ability across childhood including socioeconomic status (SES), gestational age, birth weight, and maternal age.

Aim 3: Examine the Co-Development of Physical Size and Cognitive Ability in Twins from Infancy to Adolescence

Previous research has established associations between physical size and cognitive ability across childhood. Children born at low birth weight or small stature tend to display poorer cognitive ability in infancy and childhood (Koeppen-Schomerus et al., 2000; Kohlhauser et al., 2000; Shenkin et al., 2004; Swamy et al., 2018). However, catch-up growth in height and weight across childhood has been associated with better performance on standardized cognitive assessments in adolescence and adulthood among samples born at low birth weight or small stature (Antoniou et al., 2013; Varella & Moss, 2015). Although associations between physical size and cognitive ability have been established in the literature, there has been no research that has examined the parallel growth in physical size and cognitive ability across multiple developmental stages in childhood.

The third aim of the proposed dissertation is to examine associations between the parallel recovery in physical size and recovery in cognitive ability across childhood by incorporating the best-fitting growth curve models observed in aims 1 and 2 into the same model and calculating phenotypic,

additive genetic, shared environmental, and nonshared environmental correlations between the growth parameters. To examine transactional associations between recovery in physical size and cognitive ability, I fit a bivariate dual change score model, which links changes in cognitive ability to previous values of physical size and vice versa. Such an approach elucidates the extent to which a child's physical size at time *t* is predictive of the amount of change in their rate of growth of cognitive ability between time *t* and time *t+1*.

Study 1:
Recovery of Physical Size in Twins Across Childhood: Genetic and Environmental Correlates

As early as 24 weeks gestation, twins display a dampened rate of prenatal physical growth relative to singletons (Hiersch et al., 2020). Researchers have hypothesized that discrepancies in prenatal growth rates are due to constraints of the uterine size (Blickstein, 2004) or increased nutrition requirements of two fetuses in the third trimester (Liao et al., 2012). Consequently, at birth twins have been observed to have marked deficits on measurements of physical growth including height, weight, and head circumference (Estourgie-van Burk et al., 2010; van Dommelen et al., 2008; Wilson, 1979; Wilson, 1974b). For example, in a large sample of Dutch infants, birth lengths in twins were 0.42 SD below the population mean and birth weights were 0.87 SD below the population mean whereas average birth lengths and weights for their singleton siblings were within 0.01 SD of the population mean (Estourgie-van Burk et al., 2010). Likewise, using data from the Louisville Twin Study, Wilson (1979) observed that twins were 17% shorter and 30% lighter at birth relative to singleton siblings. Deficits in birth length and weight in twins were observed across percentiles (i.e., twins in the 10^{th} percentile were substantially smaller than singletons in the 10^{th} percentile, and twins in the 90^{th} percentile were smaller than singletons in the 90^{th} percentile). However, deficits were more pronounced at lower height and weight percentiles (Wilson, 1979). Additionally, accounting for gestational age, Wilson (1974b) found that twins born at the 50^{th} percentile in terms of head circumference were comparable to singletons at the 10^{th} percentile.

Deficits in physical size at birth are linked to a myriad of negative developmental outcomes including delays in early cognitive and gross and fine motor development (Kuklina et al., 2006; Scharf et al., 2016; Scharf et al., 2018; Upadhyay et al., 2019). Sustained deficits in physical size increases the risk for poor neurodevelopment and other serious health concerns across the lifespan. For example, length, weight, and head circumference measurements greater than 2 SD below the population mean in infancy are risk factors for poor neurodevelopment including receiving a diagnosis of global developmental delay or intellectual disability (Bélanger & Carone, 2015). Likewise, being clinically underweight in adulthood (BMI < 18.5 kg/m^2) is a risk factor for all-cause mortality (Flegal et al., 2005; Roh et al., 2014). Therefore, identifying the typical rate and shape of catch-up growth in height, weight, and head circumference is important to identify children that are at risk for abnormal physical development and subsequent neurodevelopmental and health outcomes. Likewise, identifying environmental correlates of physical recovery in children exposed to early bio-environmental adversity is a useful first step in informing interventions to promote healthy physical development.

Catch-Up Growth in Height, Weight, and Head Circumference

In singletons born small for gestational age, most catch-up growth in height occurs in the first 12 months, suggesting that rapid recovery in physical size may begin in infancy (Brandt et al., 2003; Itabashi et al., 2007). Accordingly, twins make rapid gains toward singleton norms across infancy, but deficits in both height, weight, and head circumference remain apparent in toddlerhood (Buckler & Green, 2004; Schulte et al., 2016; van Dommelen et al., 2008; Wilson, 1979). By late childhood, most samples of twins suggest there are minimal to no discrepancies in physical size between twins and singletons (Estourgie-van Burk et al., 2010; Wilson, 1979). For example, despite early deficits in physical size, twins in the Louisville Twin Study were observed to be within 5% of singleton norms for height and weight by 5 years and exhibited no differences relative to singleton norms by 8 years (Wilson, 1979). As with length and weight at birth, recovery of height and weight was consistent across percentiles (i.e., twins in the 10^{th} percentile at birth were at the 10^{th} percentile singleton norms by age 8; Wilson, 1979). Although Wilson (1979) did not fit

growth models to the physical growth data, plots of sample means over time appear exponential with rapid recovery occurring in the first months and leveling off around the singleton means by early childhood, consistent with observations in small for gestational age singletons (Itabashi et al., 2007).

Studies examining associations between intra-pair discrepancies in birth weight and physical development outcomes can yield important information about physical growth outcomes of infants born at small weight and stature while holding constant shared prenatal and postnatal experiences. Across childhood, the smaller twin typically catches up to the larger twin in terms of height, weight, and head circumference (Buckler & Green, 2011, Morag et al., 2018; Ross et al., 2012; Schulte et al., 2016; Swamy et al., 2018), although some research suggests that slight discrepancies may persist into adulthood (Koziel, 1998). Using a small sample of monozygotic twins from the Louisville Twin Study born at discrepant birthweights (>= 750 grams), Wilson (1979) observed no differences in height in early childhood, but the smaller twin at birth was significantly lighter. In a longitudinal study of premature twins born at discordant birth weight (>15% discrepancy in birth weight), there were no intra-pair differences in height or weight at 3 years, indicating significant catch-up in the smaller twin within a discordant pair (Ross et al., 2012). Although all twins in the study were low birth weight, height and weight for age z-scores were within the normal range by age 3 years (Ross et al., 2012). In a study of birth weight discordant (>20% discrepancy in birth weight) monochorionic twins[1], the smaller twins were on average within 8% of the larger twin in terms of weight and within 2% in terms of height by early childhood (Swamy et al., 2018). Both the lighter and heavier twin were within the normal weight-for-age range in early childhood (Swamy et al., 2018). Thus, after accounting for shared prenatal and postnatal experiences as well as shared genetics, discrepancies in physical size tend to dissipate across the first few years of childhood. Moreover, results provide additional evidence that twins catch up to the population mean physically by early childhood (Ross et al., 2012; Swamy et al., 2018).

In later developmental stages, twins typically maintain gains in height, weight, and head circumference relative to the population mean (Estourgie-van Burk et al., 2010; Wilson, 1979). For example, Estourgie-van Burk and colleagues (2010) observed age-standardized heights and weights of Dutch twins to be at the population mean at age 18. Similarly, using data from Norwegian military conscripts, Sundet and colleagues (2005) found that twins were slightly shorter than nontwins in adulthood, although the effect size was extremely small (Cohen's d = .07). Thus, the typical recovery pattern of height and weight appears to be asymptotic with gains in height and weight stabilizing around the population mean in middle childhood and sustaining into adulthood.

Biometric Analyses of Height, Weight, and Head Circumference

Biometric twin studies that decompose the variance of a phenotype into genetic and environmental components generally suggests that additive genetic factors account for an increasing proportion of the variance in height, weight, and head circumference across the lifespan (Dommelen et al., 2004; Estourgie-van Burk et al., 2006; Mook-Kanamori et al., 2012; Silventoinen et al., 2003; Smit et al., 2010). The heritability of birth weight, length, and head circumference tends to be relatively low (Antoniou et al., 2013; Mook-Kanamori et al., 2012; Smit et al., 2010), which may be due to competition for nutrients between MZ twins (Van Baal & Boomsma, 1998). Using data from the Netherlands Twin Register, Mook-Kanamori and colleagues (2012) found additive genetics to

[1] Monochronic twins are twins that develop in a single fetus and are monozygotic by definition.

account for approximately 27% of the variance in birth length and 29% of the variance in birth weight whereas the shared environment accounted for 46% and 22% of the variance in birth length and weight, respectively. By 36 months, additive genetics accounted for 72% of the variance in height and 71% of the variance in weight and the shared environment accounted for 17% and 14% of the respective variance in height and weight at 36 months (Mook-Kanamori et al., 2012). Similarly, in a cohort study of Chinese children, genetic factors accounted for 12% of the variance in height for children 0-2 years old and 87% of the variance for children 15-17 years old (Liu et al., 2015). Alternatively, shared environmental factors accounted for 83% of the variance in height for children 0-2 years and 11% of the variance for children 15-17 years old (Liu et al., 2015). Analysis of four samples of twins from the Netherlands and Australia found the shared environment to contribute to between 71% and 82% of the variance in head circumference among infants younger than four months and additive genetics to contribute to between 84% and 92% of the variance in head circumference for infants between 5 and 13 months (Smit et al., 2010). Studies of adult samples have found height and weight to be highly heritable with heritability estimates for height, weight, and head circumference ranging from .68 to .90 (Silventoinen et al., 2000; Silventoinen et al., 2003; Smit et al., 2010; Yang et al., 2007).

Although numerous studies have conducted biometric analyses on height and weight in twins at specific developmental periods, biometric analyses of the *change in height or weight over time* are relatively uncommon and no studies have conducted biometric analyses on the latent growth of head circumference. In a prospective study of over 2,000 British twins, the total change in weight-for-age z-scores between birth and six months was primarily accounted for by additive genetic factors (57%) whereas shared and nonshared environmental contributions were marginal (22% and 21%, respectively) (Johnson et al., 2011). However, in a prospective study of physical growth in Israeli twins between birth and 12 months, shared environmental factors accounted for most of the variance in the velocity of growth in height and weight (56.4% and 49.5%, respectively) (Livshits et al., 2000). Additive genetics did not account for a significant proportion of the variance of velocity of height or weight growth (Livshits et al., 2000). Studies extending into later developmental stages consistently find additive genetics to explain most of the variance in the rate and shape of growth in height and weight over time (Demerath et al., 2007; Dommelen et al., 2004; Hauspie et al., 1994; Hjelmborg et al., 2008; Johnson et al., 2011).

Previous biometric analyses of physical development in twins have several limitations which are addressed in the present dissertation. First, as growth of head circumference is an important indicator of healthy child development, understanding the relative genetic and environmental contributions to the development of head circumference is important for our understanding of early human development. Understanding the recovery process of head circumference in high-risk samples (e.g., twins) is especially important provided the established association between poor head circumference growth and cognitive functioning and neurodevelopmental disorders (Bélanger & Carone, 2015; Scharf et al., 2018). A second limitation of previous research is that previous studies of physical development in twins have focused on a limited developmental period. That I am aware of, no studies have examined growth trends from infancy to adolescence, limiting our understanding of the role of genetics and environmental influences in long-term growth of height and weight in twins. Third, previous research has focused on change in raw measures of height or weight (Dommelen et al., 2004; Hauspie et al., 1994; Johnson et al., 2011). Such a focus does not provide insight into rates of growth relative to the population mean which is of interest

when considering typical and atypical patterns of physical development among children exposed to early bio-environmental adversity (e.g., premature birth, low birth weight, etc.). Thus, the relative genetic and environmental contributions to the *recovery* of physical size in twins are currently unknown.

Specific Environmental Contributions to Physical Recovery

Exposure to prenatal stressors may reflect one aspect of the shared environment that contributes to the extent to which twins catch up to the population mean in terms of physical size. Gestational age and birth weight represent crude but useful indicators of exposure to prenatal insults (Kline et al., 1989). In Israeli twins, gestational age accounted for approximately 40% of the total variance in birth weight and length as well as growth in height and weight over the first year, highlighting the importance of prenatal experiences in early physical development (Livshits et al., 2000). In a sample of singletons born small for gestational age, 90% of the full-term infants caught up to the population mean for height by 5 years, whereas 74% of the preterm infants caught up by 5 years (Itabashi et al., 2007), suggesting that premature birth may amplify the likelihood that small infants will fail to catch up to age-appropriate size.

Socioeconomic status, which may reflect access to higher-quality nutrition and medical care, is an aspect of the postnatal environment that has been associated with physical development (Ashworth et al., 1997; Batista et al., 2012; Jelenkovic et al., 2020). For example, among Brazilian singletons born prematurely, community-level SES, but not family-level SES positively predicted catch-up growth[2] in height between birth and school entry (Batista et al., 2012). However, among children living in poor communities, low family SES was associated with a greater likelihood of catch-down growth[3] (Batista et al., 2012). Results from a large aggregation of twin samples from 15 countries revealed a modest, positive correlation between height and parental education (Jelenkovic et al., 2020). Among Brazilian singletons born at low birthweight, family SES explained 21.4% of the variance in catch-up weight gain over the first year and 24.4% of the variance in catch-up height, accounting for maternal height, maternal smoking, and prenatal illness (Ashworth et al., 1997). In Western cultures, some research suggests an inverse relationship between SES and early weight gain such that infants in poorer households gain weight more rapidly than infants in wealthier families (Wijlaars et al., 2011). Thus, in the process of recovering to the population mean in terms of weight, twins in poorer homes may demonstrate a steeper recovery in weight and ultimately overshoot the population mean, ending up overweight in early childhood.

The Present Study

The present study seeks to describe the process by which twins recover to the population mean in terms of height, weight, and head circumference. Specifically, I fit nonlinear growth models (Grimm & Ram, 2009) to height for age, weight for age, and head circumference for age z-scores from birth to 15 years (birth to three years for head circumference) to model the magnitude, rate, and shape of the recovery of physical size in twins. I then conducted biometric analyses on the best-fitting growth models to determine relative additive genetic, shared environmental, and nonshared environmental contributions to physical recovery. Finally, I examined measured

[2] Catch-up growth refers to a change height-for-age z-score $\geq$ 0.67 standard deviations.
[3] Catch-down growth refers to a change height-for-age z-score $\geq$ -0.67 standard deviations.

prenatal and postnatal correlates to the recovery of physical size and cognitive ability across childhood. The study design and hypotheses were preregistered (https://osf.io/dsyac/).

Methods

Participants

Participants were 1,210 individual twins recruited as a part of the Louisville Twin Study, an ongoing longitudinal study of temperament and intellectual development in twins (Davis et al., 2019; Wilson, 1983). 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.

Over the 36-year course of the Louisville Twin Study, there were 1,770 individuals (885 pairs) listed as participating at least once. Twins missing zygosity information will not be included in analyses (n = 120). Of those remaining, 1,637 had at least one physical measurement. As is typical in twin studies, I restricted analyses to monozygotic and same-sex dizygotic twins (*n* = 1,287). Finally, provided my focus on typical physical development, I removed all individuals with a physical measurement greater than 4 SD above or below the population mean (*n* = 77). Thus, the final sample is 1,210 (52.3% 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. Most individuals in the study did not have data at all 16 points, but 89.0% had four or more physical assessments and 79.3% had four or more cognitive assessments (see Table 1).

Table 1
Number of Assessments Available

Number of Measurements	Weight	Height	Head Circumference
1	74	78	90
2	85	83	62
3	34	34	57
4	46	58	109
5	43	40	151
6	57	45	281
7	47	55	329
8	53	65	407
9	112	100	108
10	80	92	
11	85	103	
12	98	95	

13	132	132
14	189	204
15	291	245
16	130	121
17	80	69
Median	12	12

Note. Head circumference z-scores are only available until 36 months. Therefore, there were only 9 possible measurements for head circumference.

Measures

Physical development. Physical measurement procedures are described in detail elsewhere (Wilson, 1974b, Wilson, 1979). Birth length, weight, and head circumference measurements were obtained from birth certificates. All subsequent physical measurements were taken during assessments conducted at the University of Louisville. Infant weights between 3 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 was measured to the nearest millimeter. Recumbent length was used as a proxy for height between 3 and 24 months. After 24 months, standing height was measured using a wall-mounted metric scale. Head circumference was measured to the nearest eighth of an inch and converted to metric measurements for analyses (Wilson, 1974b). Raw height, weight, and head circumference measurements were converted into age-standardized z-scores using Center for Disease Control (CDC) growth charts based on 2000 norms (Kuczmarski, 2000). Z-scores are calculated separately for males and females and, therefore, are also standardized by sex. The 2000 CDC growth charts were based on United States population surveys conducted between 1963 and 1994 (see Kuczmarski 2000 for further details), which overlaps with the timeline of data collection in the Louisville Twin Study.

Covariates. Baseline household SES was measured based on the Hollingshead Four Factor Index of Socioeconomic Status, which is based on parental occupation, education, sex, and income (Hollingshead, 1975). Hollingshead scores are based on a continuous zero to 100-point scale. Gestational age (in weeks) was calculated based on maternal report of last menses. Maternal age (in years) at birth was also included as a study covariate. Child sex was also included as a covariate in growth models. Continuous covariates were standardized to have a mean of 0 and SD of 1, and sex was centered. A quadratic SES term was created by squaring the standardized SES term. I then centered the quadratic SES term to ensure it had a mean of 0, which is a requirement to ensure that the predicted means of the latent growth terms are 0 (Grimm, personal communication, February 18, 2021).

Proposed Analyses

Descriptive Statistics. Data preparation, descriptive statistics, and the calculation of intercorrelations between study variables will be conducted using the Base package in R (R Core Team, 2020).

Nonlinear growth modeling. In modeling the recovery of height, weight, and head circumference in twins, I considered polynomial (linear, quadratic, and cubic), exponential, sigmoid (Logistic, Gompertz, Richards), Weibull, and Morgan-Mercer-Flodin growth models. I discuss the Weibull and Morgan-Mercer-Flodin models in more detail below as these models fit the physical growth data best.

Weibull Growth Model. The Weibull growth model is commonly used in the biological sciences and has been found to describe the vertical growth of trees (Mahanta & Borah, 2014), the weight of birds (Wen et al., 2019), and the growth of brain volume in children (Peterson et al., 2018). The Weibull growth model is an extension of the Weibull distribution and can be defined by the following equation (Ratkowsky, 1983).

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

In the Weibull model, b_0 is often described as the "biological constant" and is analogous to the intercept (measurement when time equals zero). b_1 represents the upper asymptote, and b_2 represents the average rate of change. b_3 is a shaping parameter that controls the overall shape of the curve. Values of b_3 less than 1 describe a model that follows an exponential shape whereas values greater than 1 describe a sigmoid shape. In the case that b_3 equals 1, the Weibull curve simplifies to a traditional exponential curve.

Morgan-Mercer-Flodin Model. The Morgan-Mercer-Flodin growth model is also frequently used in the biological sciences to describe the growth of organisms (Tariq et al., 2013). The Morgan-Mercer-Flodin model can be expressed using the following equation (Morgan et al., 1975).

$$Y_{ij} = b_{1i} - \frac{b_{1i} - b_{0i}}{(1 + (b_2 \cdot \mathrm{t})^{b_{3i}})} + \mathrm{e}_{it}$$

In the Morgan-Mercer-Flodin model, b_0 corresponds to the intercept, b_1 is the upper asymptote, b_2 is the average rate of growth. The inflection point scaling parameter ($b3$) can be used to calculate the age at which growth is the fastest using the following equation:

$$\text{Age of Inflection} = \left[\frac{b_3 - 1}{b_3 + 1}\right]^{\frac{1}{b_3}}$$

All growth models were fit within a structural equation modeling framework using Mplus version 8.4 (Muthén & Muthén, 2017). As described by Grimm and colleagues (2013) nonlinear growth functions can be approximated in a structural equation modeling by using a Taylor Series expansion to generate a linear function of the target function (e.g., Weibull curve). This can be done by fixing the loadings for each growth factor to the partial derivative of each term in the target function.

As models comparing different growth functions were not nested, the best-fitting model was determined using the Root Mean Square Error of Approximation (RMSEA), Tucker-Lewis Index (TLI), Akaike Information Criterion (AIC), and Bayesian Information Criterion (BIC). Because individual twins are nested within families, the genetically-informative portion of the analysis was conducted using a two-level model, which requires the estimation of growth models at the child

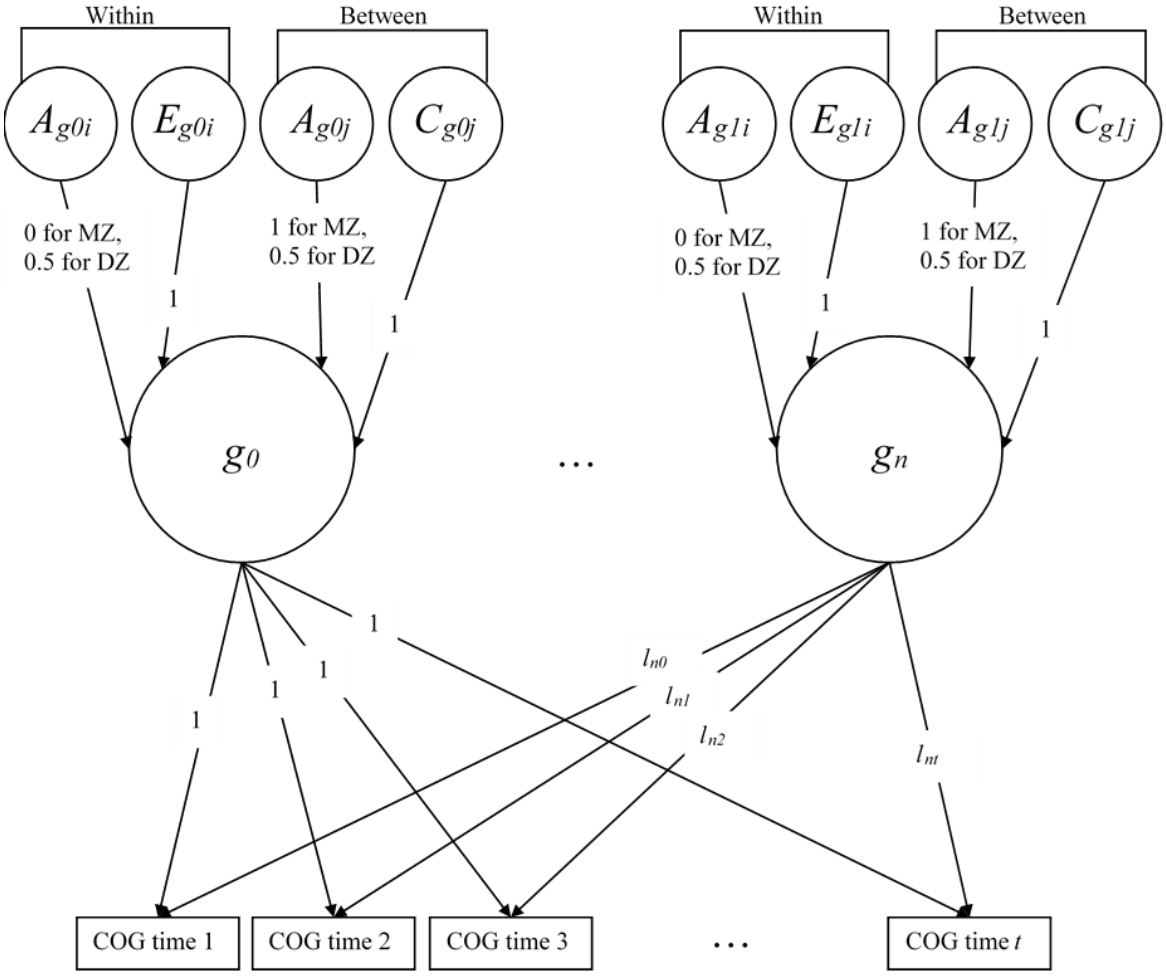
level (within-pair) and family level (between-pair). Variances within and between MZ and DZ twins were then transformed into the genetic and environmental variances of the classical twin model. All models were fit using maximum likelihood estimation with robust standard errors.

Genetic analyses. Once the best fitting growth model was identified, the growth parameters were decomposed into additive genetic (A), shared environmental (C), and nonshared environmental effects plus measurement error (E) following a standard multilevel approach (McArdle & Prescott, 2005). The ACE decomposition takes advantage of the fact that MZ twins share 100% of their DNA sequence, whereas DZ twins share, on average, 50% of their segregating genes. The between- and within-pair variances can be decomposed into environmental and genetic components following.

$$MZ_{within} = E$$
$$MZ_{between} = A + C$$
$$DZ_{within} = 0.5*A + E$$
$$DZ_{between} = 0.5*A + C$$

See Figure 2 for a sample path diagram of the multilevel ACE decomposition.

Figure 2
Generic ACE Decomposition of a Multilevel Growth Model



Note. Biometric components of growth parameters g_0 to g_n for twin i in family j are estimated for within- and between-pair. Factor loadings l represent the appropriate factor loadings for growth parameter g_n at time t. COG stands for cognitive ability.

Environmental correlates to physical and cognitive recovery. I then regressed the growth parameters onto covariates to identify environmental correlates to the recovery of height, weight, and head circumference in twins. All study covariates were constant across twins within a family (i.e., SES, gestational age, and maternal age) and were included at the between-pair level of the model. To aid model convergence, I standardized all covariates prior to inclusion.

Missing Data

Due to the longitudinal nature of the Louisville Twin Study, there were missing data on all measures. Rates of missingness for each study variable are presented in Table 2.

Table 2

Proportion of Missingness for Each Study Variable

Age	% Missing Height	% Missing Weight	% Missing Head Circumference
Birth	20.8%	0.3%	56.5%
3 months	46.7%	46.4%	44.7%
6 months	31.2%	31.3%	31.4%
9 months	31.9%	32.0%	32.1%
12 months	17.4%	17.4%	17.9%
18 months	20.2%	20.4%	21.3%
24 months	20.5%	20.5%	21.8%
30 months	46.9%	46.7%	47.1%
36 months	21.8%	21.6%	22.3%
4 years	26.6%	26.4%	
5 years	28.0%	28.0%	
6 years	29.9%	29.9%	
7 years	41.2%	41.2%	
8 years	42.2%	42.2%	
9 years	46.2%	46.2%	
12 years	83.1%	83.1%	
15 years	57.1%	57.1%	
Average	36.0%	34.7%	33.0%

Note. CDC charts to calculate Z-scores for head circumference after 36 months were not available. Therefore, head circumference is only measured to 36 months.

Rubin (1976) describes three patterns that may describe missing data: missing completely at random (MCAR), missing at random (MAR), and missing not at random (MNAR). Data are MCAR when missingness is unrelated to any observed variables (e.g., other variables in the study) or unobserved variables (e.g., the missing value). In the present study, data would be MCAR if missingness on weight measurements was unrelated to study covariates (e.g., SES), previous weights, and the would-be weight from the missing timepoint. MAR occurs when missingness is related to other observed variables, but not the missing value itself. For example, if children from lower SES households are more likely to be missing weight measurements and heavier children are not more likely to be missing, data are considered MAR. When missingness is related to the value of the missing variable (e.g., if extremely light children did not participate at a particular

wave due to health concerns), data would be considered MNAR. There are a variety of strategies to handle missing data when data are MCAR or MAR, however, parameter estimates are likely to be biased in cases where data are MNAR (Enders, 2011a).

There is no statistical test to definitively determine if data are MCAR or MAR as both of these data patterns require knowledge of an unknown variable (i.e., the value of the missing data point). Several strategies have been derived to approximate the missing data pattern. Little's MCAR test tests the null hypothesis that missingness is unrelated to observed values on all other variables in a dataset (Little, 1988). Little's MCAR test was significant for height (X^2 = 12,429 $_{df = 6,894}$, p < .001), weight (X^2 = 11,563 $_{df = 6,402}$, p < .001), and head circumference (X^2 = 5,411 $_{df = 2,368}$, p < .001). Therefore, none of the physical growth parameters were MCAR as missingness was predicted by at least one variable in the dataset.

To further explore missing data patterns, I fit a series of logistic regression models with missingness of each physical growth indicator (height, weight, and head circumference) at each age predicted by the study covariates as well as zygosity. Additionally, as study procedures changed throughout the course of the Louisville Twin Study, birth year was included as a predictor of missingness to determine if there were systematic differences in missingness by cohort (e.g., were later-born cohorts less likely to be assessed at certain ages). Finally, the observed measure at the previous assessment was included in the logistic regression models (i.e., weight at 3 months predicting missing weight at 6 months). To the extent that concurrent physical measurements across childhood are stable, I was able to approximate whether or not missingness is related to the missing value (i.e., are heavier children more likely to be missing at 6 months).

In general birth year was positively associated with missingness on the physical growth measures, indicating that children born later (and enrolled in the study later) were less likely to have height, weight, and head circumference measurements. Socioeconomic status was positively related to missing head circumference at birth, but negatively related to missingness at 12 months. Children with longer gestation were less likely to be missing weight measurements at 8 years. In four instances, a physical measurement was significantly predicted by the previous measurement. A larger head circumference at 24 months predicted a greater likelihood of missingness at 30 months (B = 0.05, SE = 0.02, p = .004). Children who were taller at 6 years were more likely to be missing height measurements at 7 years (B = 0.05, SE = 0.02, p < .001). Finally, shorter children at 12 years were more likely to be missing height measurements at 15 years (B = -0.10, SE = 0.03, p = .002), and lighter children at 12 years were more likely to be missing weight measurements at 15 years (B = 0.11, SE = 0.03, p < .001).

Although there are a few occasions where physical size at time t predicts missingness at time $t + 1$, these associations do not appear to be systematic. That is, observed measurements are not biased by consistently larger or smaller children. Birth year does appear to be an important indicator of missingness, and likely reflects changes in study protocols over the years. This suggests that physical growth data in the LTS are generally MAR. I used full information maximum likelihood estimation (FIML) in Mplus to handle missing data. FIML is appropriate for handling data under MAR conditions and performs comparably to other strategies of handling missingness (e.g., multiple imputation) and is computationally simpler (Enders, 2013). When using FIML to handle missingness, it is important to include predictors of missingness in the model to avoid generating

biased parameter estimates (Graham, 2001; Enders, 2013). As birth year was the only consistent predictor of missingness, I include birth year as a predictor of measured height, weight, and head circumference at each age in all growth curve models.

Results

Descriptive Statistics and Intercorrelations

Hollingshead SES scores indicated that, overall, the sample was of average SES with families approximately evenly distributed across SES quintiles (22.2%, 20.1%, 18.8%, 25.7%, and 13.1% in the first through fifth quintiles, respectively). The mean gestational age (37.4 weeks SD 2.3) suggests that the average twin pair was born on the cusp of prematurity. The majority of twins (69.3%) were born at typical gestation (greater than 37 weeks), but 28.4% of the twins were born prematurely (32-37 weeks gestation) and 2.3% were born very prematurely (less than 32 weeks gestation). Correlations among study covariates were generally weak suggesting that they represent independent aspects of the shared environment. Family SES was moderately correlated with maternal age, and birth weight was moderately correlated with gestational age. See Table 3 for descriptive information and intercorrelations between study covariates.

Descriptive Statistics Physical Size

The average birth weight for the study sample was 5.6 pounds (5.5 pounds is the cutoff for low birth weight). Most twins were born at typical birth weight (55.9%), but 41.4% of twins were born at low birth weight (3.3 to 5.5 pounds) and 2.7% were born at very low birth weight (less than 3.3 pounds). Although premature birth and low birth weight were common in the study sample, relatively few twins were born very prematurely or at very low birth weight. Therefore, the study sample largely reflects children born at relatively moderate risk in terms of birth weight and gestational age.

Average weight, height, and head circumference z-scores at each age are presented in Tables 4, 5, and 6, respectively. Mean birth weights, lengths, and head circumferences were between 1.52 standard deviations below the mean (for weight) to 0.77 standard deviations below the mean (for length). Between birth and 15 years (3 years for head circumference), mean scores reflected a pattern of early deficits and recovery to the mean (see Figures 3, 4, and 5 for spaghetti plots of trajectories of change in weight, height, and head circumference across time).

Underweight is a medical term used to describe children more than 2 standard deviations below the population mean in terms of weight, and stunting refers to children more than two standard deviations below the population mean in terms of height. At birth, 27.0% of the twins were underweight and 11.7% were stunted. However, the proportion of twins that were underweight or stunted decreased dramatically over time, and by 15 years only 0.4% were underweight and 0.7% were stunted. See Figure 6 for a plot of the proportion of children underweight or stunted at each age.

Twin correlations and rough estimates of the heritability of weight, height, and head circumference at each age are presented in Tables 4-6, respectively. Heritability estimates of weight, height, and head circumference at birth were remarkably low (0.02 – 0.10), which likely reflects the strong effects of the early prenatal environment on early physical size. However, heritability estimates of all of the physical growth parameters rapidly increased over time. Thus, more genetically similar

individuals (i.e., identical twins) became more similar physically than less genetically related individuals (i.e., fraternal twins) at later developmental stages.

Table 3
Descriptive Statistics for Study Covariates

						Correlations	
Variable	*n*	Mean (*SD*)	Range	Skew	Kurtosis	SES	Chaos	Gestational Age
Family SES	1,388	47.83 (*26.60*)	1-96	0.00	-1.18			
Household Chaos	438	5.28 (*2.75*)	0.39-13.00	0.45	-0.32	-.09		
Gestational Age (weeks)	1,109	37.39 (*2.35*)	29-45	-0.75	0.90	.00	-.05	
Mom Age (years)	1,375	27.59 (*5.48*)	14-42	0.23	-0.43	.35*	.01	-.05

Note. The *n* reflects the number of individual twins each covariate is available for. * denotes $p < .05$.

Table 4
Descriptive Statistics of Weight

Age	*n* (MZ)	*n* (DZ)	Mean (*SD*)	Range	*r* MZ	*r* DZ	h^2
Birth	729	477	-1.53 (0.78)	-3.97 – 1.29	.63	.60	0.06
3 months	379	269	-1.03 (0.95)	-3.95 – 2.03	.79	.49	0.60
6 months	505	326	-0.75 (0.98)	-3.64 – 2.40	.81	.54	0.54
9 months	511	312	-0.73 (1.00)	-3.77 – 2.08	.83	.50	0.66
12 months	619	381	-0.69 (1.02)	-3.59 – 2.42	.87	.56	0.62
18 months	601	362	-0.69 (1.03)	-3.97 – 2.39	.86	.53	0.66
24 months	605	357	-0.57 (1.03)	-3.95 – 2.28	.88	.53	0.66
30 months	392	253	-0.31 (1.00)	-3.51 – 2.14	.86	.54	0.64
36 months	586	363	-0.24 (0.97)	-3.31 – 3.11	.88	.54	0.64
4 years	555	336	-0.23 (0.93)	-3.05 – 3.15	.87	.54	0.64
5 years	534	323	-0.26 (0.94)	-3.13 – 3.54	.88	.54	0.64
6 years	534	314	-0.24 (0.97)	-3.46 – 2.93	.88	.59	0.58
7 years	450	262	-0.20 (0.99)	-3.05 – 2.97	.88	.56	0.64
8 years	440	259	-0.09 (1.00)	-2.89 – 2.91	.90	.57	0.66
9 years	415	236	-0.05 (1.03)	-2.65 – 2.86	.90	.62	0.56
12 years	127	77	0.20 (1.18)	-2.49 – 2.68	.96	.58	0.76
15 years	328	191	0.20 (0.95)	-3.48 – 3.45	.88	.64	0.48

Note. MZ indicated monozygotic and DZ indicates dizygotic. Heritability (h^2) was estimated by multiplying the difference between the MZ and DZ correlations by 2.

Figure 3
Individual trajectories of weight z-scores from birth to 180 months. The sample mean is represented by the solid black line. The dashed black line represents the population mean of 0.

Table 5
Descriptive Statistics of Height

Age	*n* (MZ)	*n* (DZ)	Mean (*SD*)	Range	*r* MZ	*r* DZ	h^2
Birth	598	360	-0.77 (1.17)	-3.97 – 2.70	.67	.65	0.04
3 months	377	268	-1.32 (1.03)	-3.98 – 1.19	.77	.63	0.28
6 months	505	327	-0.74 (0.93)	-3.32 – 1.78	.78	.62	0.32
9 months	5011	313	-0.63 (0.90)	-3.47 – 1.99	.82	.58	0.48
12 months	619	380	-0.45 (0.90)	-3.59 – 2.48	.84	.65	0.38
18 months	602	363	-0.34 (0.89)	-3.94 – 2.13	.88	.66	0.44
24 months	605	357	-0.33 (0.88)	-3.00 – 2.24	.89	.57	0.64
30 months	389	254	-0.33 (0.87)	-3.38 – 2.20	.92	.57	0.70
36 months	585	361	-0.32 (0.92)	-3.46 – 2.22	.93	.57	0.72
4 years	553	335	-0.20 (0.94)	-3.04 – 2.38	.94	.60	0.68
5 years	585	323	-0.10 (0.92)	-2.85 – 2.49	.94	.56	0.76
6 years	534	314	-0.11 (0.93)	-2.68 – 2.46	.94	.55	0.78
7 years	450	262	-0.21 (0.93)	-2.97 – 2.36	.94	.52	0.84
8 years	440	259	-0.19 (0.95)	-2.93 – 2.50	.95	.51	0.88
9 years	415	236	-0.11 (0.97)	-2.80 – 3.57	.94	.54	0.80
12 years	127	77	0.01 (1.04)	-2.21 – 3.74	.96	.50	0.92
15 years	328	191	0.03 (0.97)	-2.67 – 2.98	.94	.39	1.10*

Note. MZ indicated monozygotic and DZ indicates dizygotic. Heritability (h^2) was calculated by multiplying the difference between the MZ and DZ correlations by 2. *At 15 years, the MZ twin correlation was more than twice the DZ correlation, resulting in a heritability estimate greater than one.

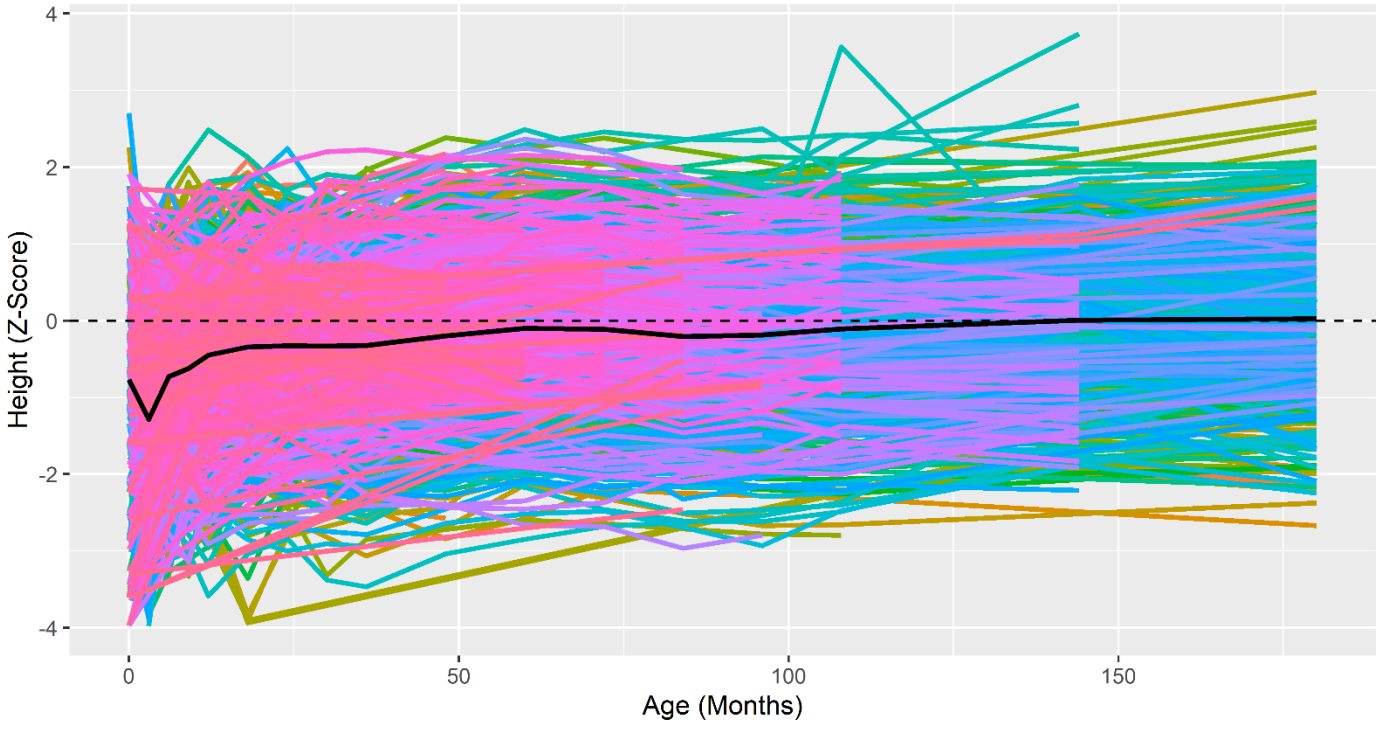


Figure 4
Individual trajectories of height z-scores from birth to 180 months. The sample mean is represented by the solid black line. The dashed black line represents the population mean of 0.

Table 6
Descriptive Statistics of Head Circumference

Age	*n* (MZ)	*n* (DZ)	Mean (*SD*)	Range	*r* MZ	*r* DZ	h^2
Birth	332	194	-1.29 (0.86)	-3.86 – 1.20	.63	.57	0.12
3 months	378	267	-1.13 (0.90)	-3.87 – 3.30	.83	.44	0.78
6 months	504	326	-0.44 (0.91)	-3.28 – 2.04	.84	.40	0.88
9 months	509	311	-0.06 (0.96)	-3.79 – 3.21	.87	.43	0.88
12 months	616	377	0.25 (0.97)	-2.82 – 3.59	.83	.41	0.84
18 months	595	357	0.31 (1.01)	-2.48 – 3.40	.90	.45	0.90
24 months	592	354	0.35 (0.94)	-2.25 – 3.37	.89	.44	0.90
30 months	389	251	0.41 (0.89)	-2.08 – 3.49	.90	.49	0.82
36 months	579	361	0.44 (0.88)	-1.93 – 3.55	.89	.47	0.84

Note. MZ indicated monozygotic and DZ indicates dizygotic. Heritability (h^2) was calculated by multiplying the difference between the MZ and DZ correlations by 2.

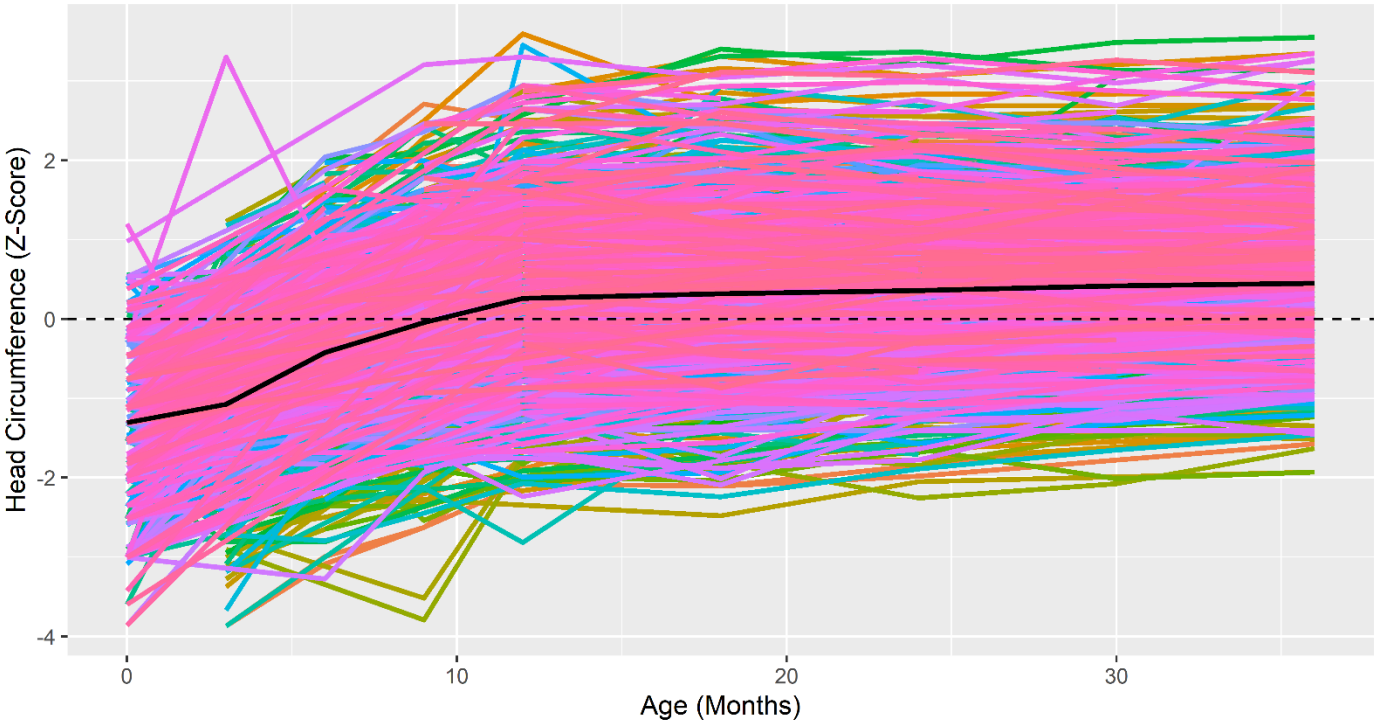
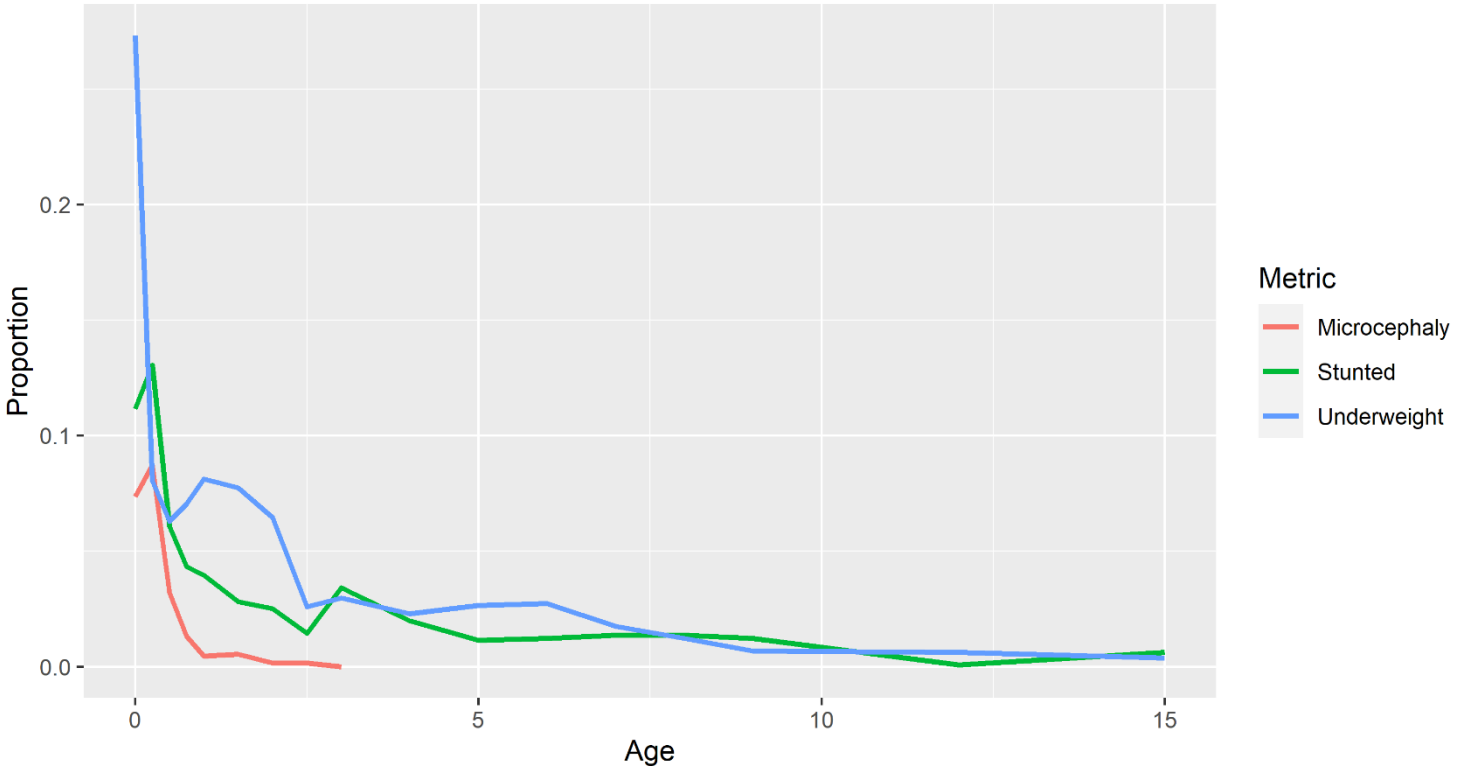


Figure 5
Individual trajectories of head circumference z-scores from birth to 36 months. The sample mean is represented by the solid black line. The dashed black line represents the population mean of 0.

Figure 6
Proportion of the study sample that are underweight, stunted, or microcephalic at each age.



Intercorrelations

Longitudinal intercorrelations between weights, heights, head circumferences, and cognitive scores are presented in Figures 7-9, respectively. Intercorrelations between consecutive weights, heights, and head circumferences were strong, indicating stability in physical size across relatively brief periods from infancy to adolescence. Birth weight was weakly to moderately correlated with weight after the first year (*r*'s = .15-.35). Likewise, length at birth was weakly to moderately correlated with length (height) after the first year (*r*'s = .17-.35). Head circumference at birth was moderately correlated with head circumference measurements within the first three years (*r*'s = .35-.47). Measurements of head circumference after birth were highly intercorrelated (*r*'s = .69-.96).

Figure 7
Longitudinal Intercorrelations between Weight Z-Scores

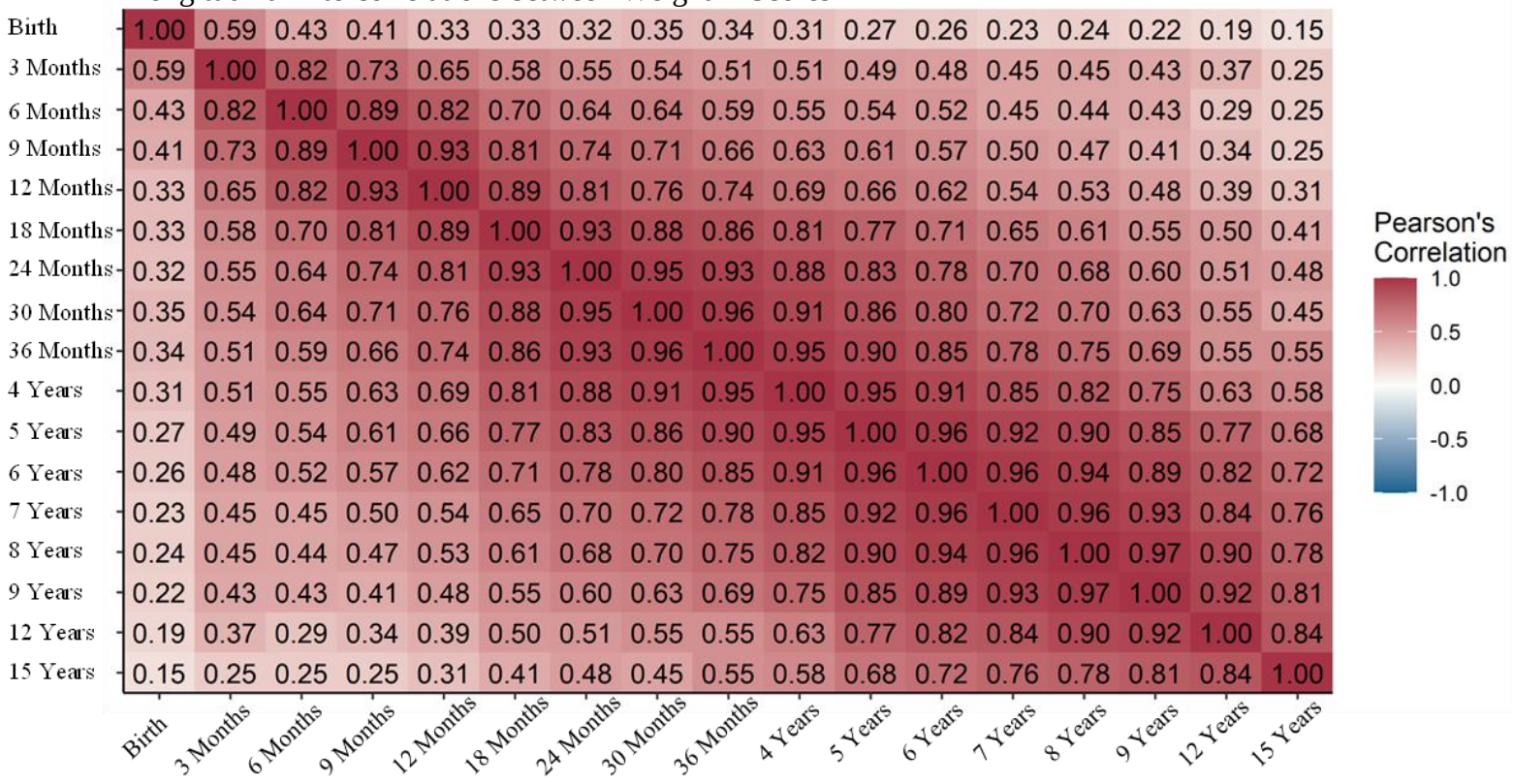


Figure 8
Longitudinal Intercorrelations between Height Z-Scores

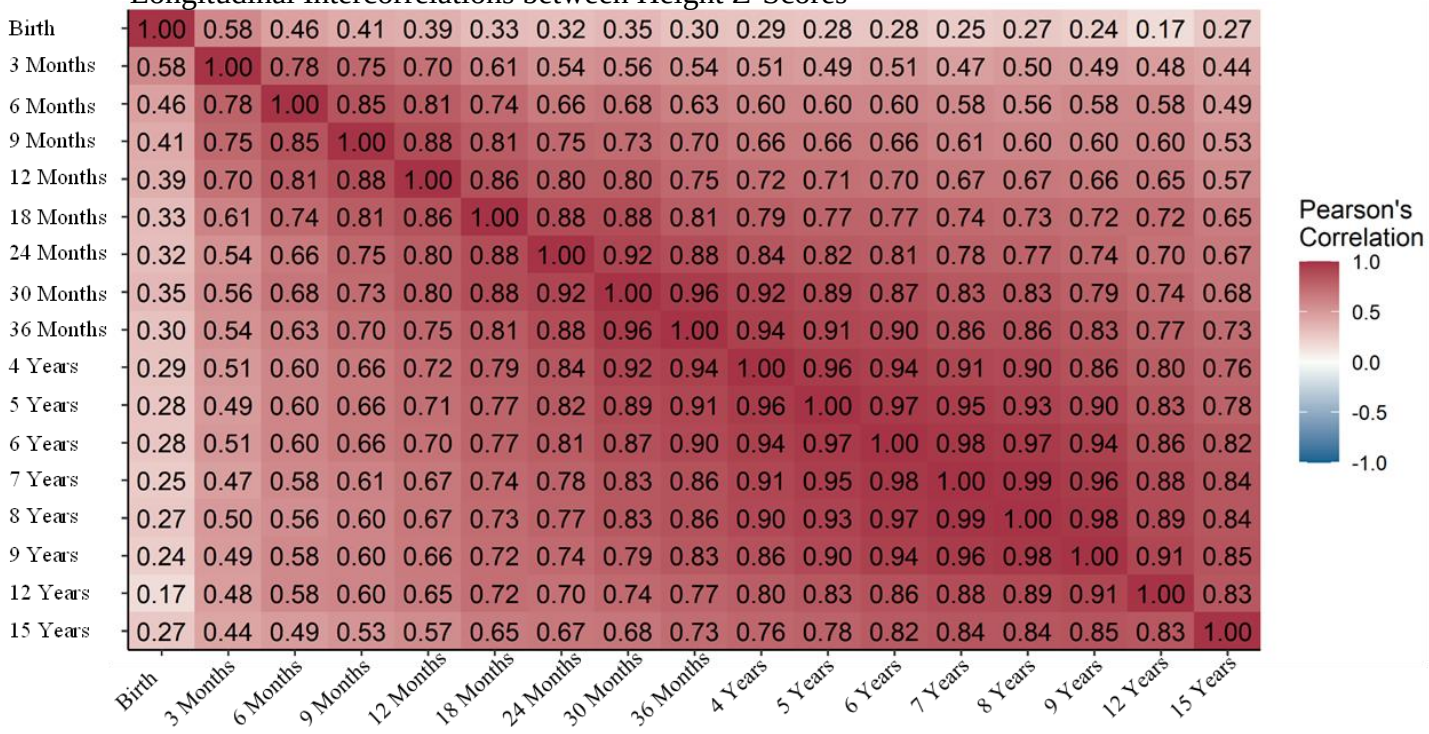
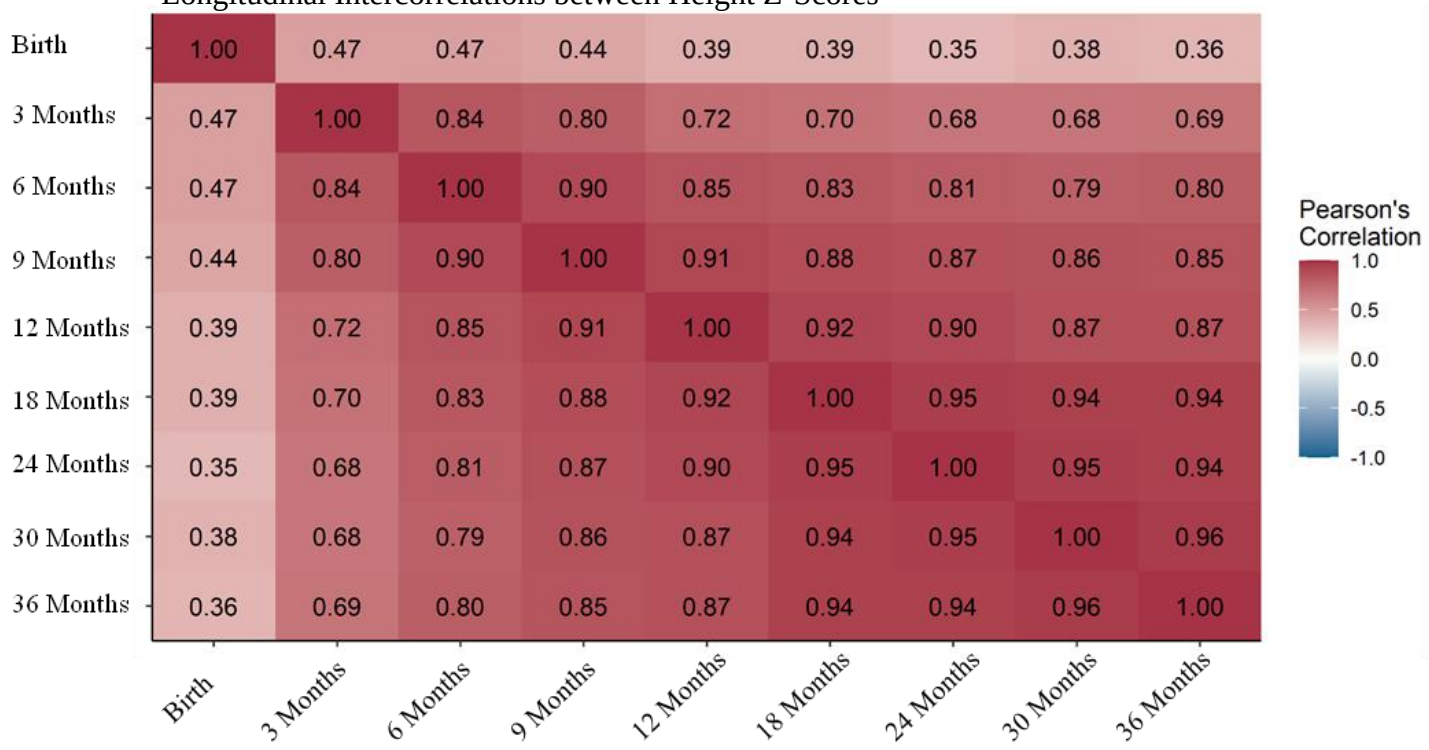


Figure 9
Longitudinal Intercorrelations between Height Z-Scores



Study Aim 1a: Model the Recovery of Weight

Model Identification: Recovery of Weight. Model fit information for the growth curve models fit to weight can be found in Table 7. The cubic, logistic, and Richard's growth curve models did not converge, and fit statistics were not estimated. Based on the BIC values, the Weibull growth model fit the data best. Although the Weibull model fit the data best relative to the other growth models, the RMSEA value (.10) and TLI value (.88) indicate that the Weibull model did not fit the data well.

Table 7
Model Fit Statistics - Weight

Fit Statistic	Linear	Quadratic	Cubic	Exponential	Weibull	Morgan-Mercer-Flodin	Logistic	Gompertz	Richards
X^2/df	4,739/149	3,849/145		2,540 /145	1,800/141	1,836/140		2,206/140	
TLI	.66	.72		.82	.87	.87		.84	
RMSEA	.16	.15		.12	.10	.10		.11	
AIC	24,795	23,361		21,245	20,075	20,129		20,633	
BIC	24,968	23,586		21,469	20,319	20,379		20,882	

Growth models were initially fit using a traditional approach of omitting correlations between the residuals. Although omitted residual correlations is the most common approach used in empirical research (Marcoulides, 2019), assuming that residuals are uncorrelated is often an unrealistic

assumption, and miss-specifying the residual structure in a growth model can lead to bias in the standard error estimates (Kwok et al., 2007). Adding autocorrelations between residuals in a growth curve model can also lead to an improvement in model fit (Grimm & Widaman, 2010).

I used a banded structure (Wolfinger, 1993) to model the residual structure of the Weibull growth model. This approach strikes a balance between parsimony and maximizing model fit. A simplified matrix of the banded structure model used is depicted below. Specifically, residual variances for each measured variable are freely estimated and covariances between sequential residuals (e.g., between 3 and 6 months) are freely estimated. However, all other residuals remain uncorrelated (e.g., 3 months and 15 years).

$$\begin{bmatrix} \sigma_1^2 & & & \\ \sigma_5 & \sigma_2^2 & & \\ & \sigma_6 & \sigma_3^2 & \\ & & \sigma_7 & \sigma_4^2 \end{bmatrix}$$

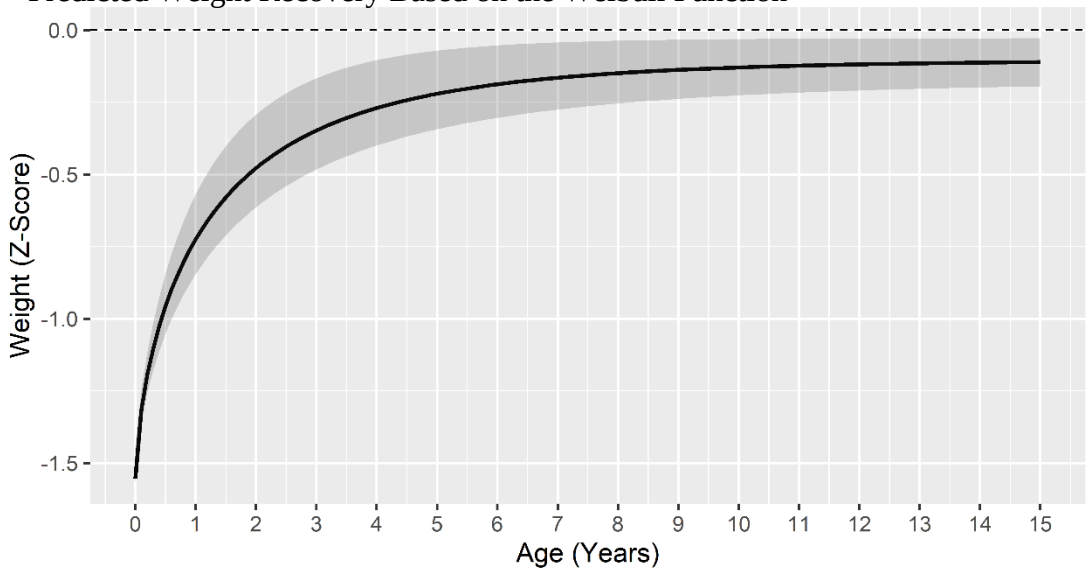
The model including structured autocorrelations fit significantly better than the model without (X^2 = 829.8, df = 17, p < .001). The final Weibull growth model fit the data acceptably (X^2 = 970.4, df = 124, p < .001; RMSEA = .08; TLI = .92). Parameter estimates for the final Weibull model are presented in Table 8. The average twin had an intercept of -1.50, indicating that at birth the average twin was able 1.5 standard deviations lighter than the population mean. The upper asymptote was -0.09, indicating that the average twin recovered to within a tenth of a standard deviation of the population mean (see Figure 10).

Table 8
Parameter Estimates of the Recovery of Weight

			Correlation Coefficient [95% C.I.]		
Parameter	Mean [95% C.I.]	Variance [95% C.I.]	Intercept (b_0)	Upper Asymptote (b_1)	Rate of Growth (b_2)
Intercept (b_0)	**-1.50 [-1.56, -1.44]**	**0.56 [0.25, 0.88]**			
Upper Asymptote (b_1)	**-0.09 [-0.17, -0.02]**	**1.11 [0.95, 1.28]**	**.18 [.07, .29]**		
Rate of Growth (b_2)	**0.85 [0.72, 0.97]**	**3.38 [1.42, 5.35]**	-.01 [-.21, .19]	**-.33 [-.45, -.21]**	
Shaping Parameter (b_3)	**0.73 [0.66, 0.81]**	**1.06 [0.81, 1.31]**	.15 [-.03, .33]	**-.36 [-.45, -.26]**	**.27 [.12, .41]**

Note. Estimates that are significantly different from 0 at p < .05 are presented in bold.

Figure 10
Predicted Weight Recovery Based on the Weibull Function



Note. The dashed line represents the population mean of 0. The shaded area represents the 95% confidence interval of the growth curve.

Biometric analysis of the recovery of weight. Unstandardized and standardized biometric components are presented in Table 9. Shared environmental influences (C) contributed to the majority of the variance in the lower asymptote (68%) and did not contribute significantly to the upper asymptote. On the other hand, additive genetics did not contribute significantly to the lower asymptote of weight but accounted for the majority of the variance in the upper asymptote (76%). Additive genetics and shared environmental factors accounted for a significant portion of the variance in the rate of growth and inflection point. A Wald test revealed that the proportion of the variance in the rate of growth and inflection point attributed to additive genetics is not significantly different from the proportion of variance due to shared environmental factors (X^2 = 3.76, *df* = 2, *p* = .153). Finally, nonshared environmental factors made relatively small but significant contributions to all of the growth parameters.

Table 9
Variance in Weight Growth Parameters Due to Genetic and Environmental Components

Parameter	Additive Genetic (A) [95% C.I.]	Shared Environment (C) [95% C.I.]	Nonshared Environment (E) [95% C.I.]	Proportion A	Proportion C	Proportion E
Intercept (b_0)	0.03 [-0.26, 0.32]	**0.35 [0.06, 0.64]**	**0.13 [0.07, 0.20]**	.06	.68	.26
Upper Asymptote (b_1)	**0.84 [0.58, 1.09]**	0.17 [-0.07, 0.42]	**0.09 [0.07, 0.11]**	.76	.16	.08
Rate of Change (b_2)	**1.67 [0.76, 2.59]**	**0.70 [0.04, 1.35]**	**0.11 [0.04 – 0.18]**	.67	.28	.05
Inflection Point (b_3)	**0.66 [0.31, 0.95]**	**0.39 [0.04 – 0.73]**	**0.07 [0.03 – 0.11]**	.58	.36	.06

Note. Proportions that are significant at the $p < .05$ level are highlighted in bold.

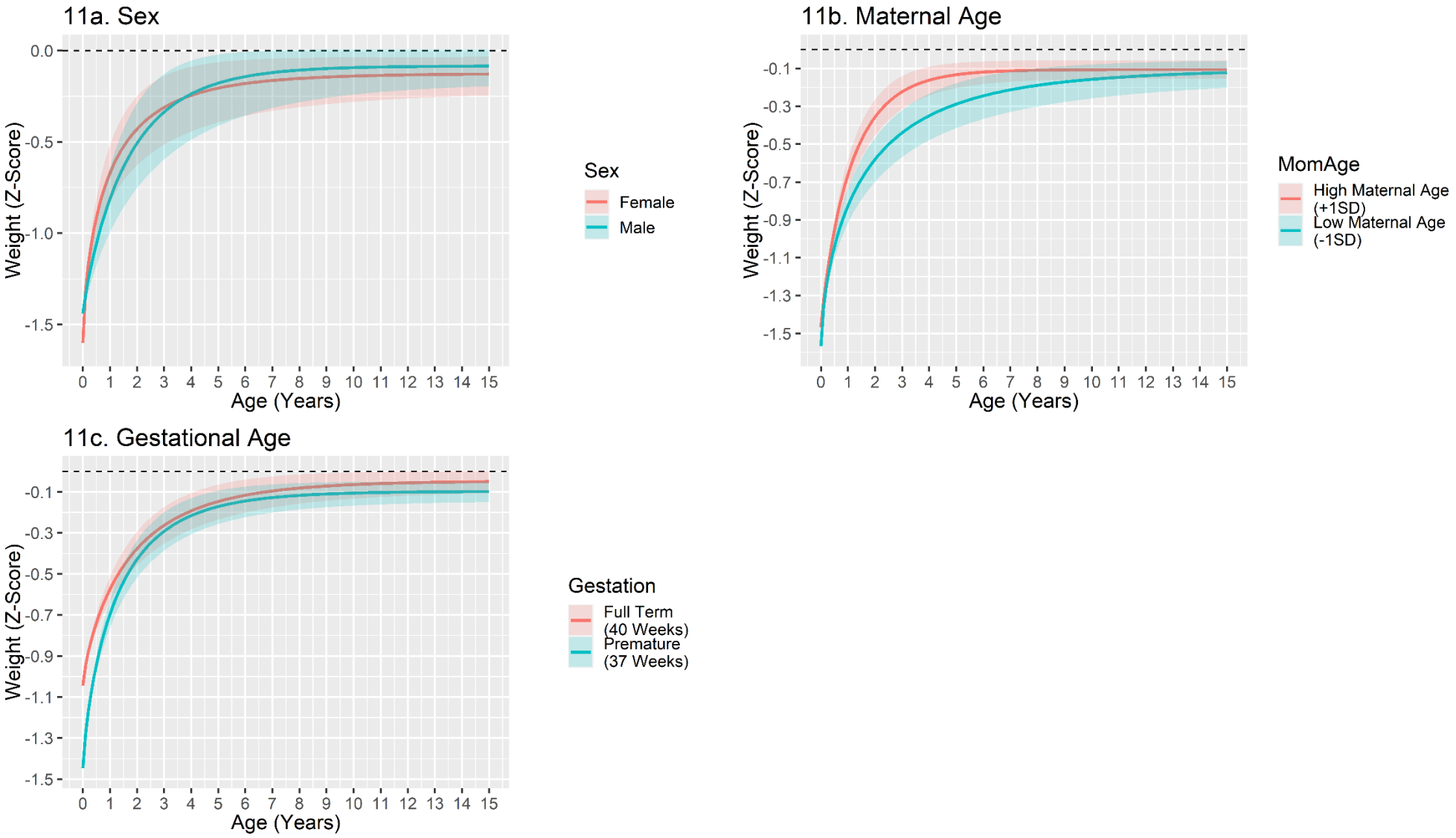
Environmental correlates of weight recovery. The growth parameters for weight recovery were regressed on gestational age, SES, the residual of SES squared, sex, and maternal age at birth. All parameter estimates are resented in Table 10. Males had a significantly higher intercept than females, suggesting that male twins are born closer to the population mean. However, males and females were statistically indistinguishable at the upper asymptote, indicating that they recovered consistently relative to the population mean. Females had a faster rate of recovery and an earlier inflection point than males. Figure 17a depicts the predicted trajectory of weight recovery by sex. Maternal age was positively associated with the inflection point of weight recovery; children born to older mothers had a later inflection point (see Figure 17b). Gestational age was significantly associated with the intercept of weight; children born later were closer to the population mean in terms of weight. However, twins born full term still had lower birth weights compared to the population mean, suggesting that prematurity does not entirely account for early deficits in weight among twins (see Figure 17c). Twins born at a lower gestational age had a quicker rate of growth, and there was no association between gestational age and the upper asymptote of weight. Thus, any effects of gestational age on weight appear to dissipate across infancy and are negligible by toddlerhood.

Table 10
Correlates of Weight Recovery

	Between-Pair Effects			
Predictor Variable	Intercept (b_0) *B* [95% C.I.]	Upper Asymptote (b_1) *B* [95% C.I.]	Rate of Change (b_2) *B* [95% C.I.]	Inflection Point (b_3) *B* [95% C.I.]
Sex	**0.16 [0.07, 0.25]**	0.04 [-0.14, 0.22]	**-0.37 [-0.67, -0.08]**	**0.24 [0.04, 0.45]**
Family SES	0.04 [-0.02, 0.09]	-0.08 [-0.18, 0.02]	0.04 [-0.12, 0.19]	0.05 [-0.06, 0.15]
Family SES^{2*}	**0.08 [0.02, 0.13]**	**0.11 [0.01, 0.22]**	-0.07 [-0.23, 0.10]	0.03 [-0.15, 0.09]
Gestational Age	**0.43 [0.38, 0.47]**	0.05 [-0.04, 0.15]	**-0.21 [-0.36, -0.06]**	0.01 [-0.11, 0.13]
Maternal Age	0.05 [-0.00, 0.10]	-0.00 [-0.09, 0.09]	0.10 [-0.05, 0.24]	**0.12 [0.02, 0.23]**

Note. Coefficients that are significant at the $p < .05$ level are highlighted in bold. *Family SES^2 is the residual of family SES squared regressed onto family SES.

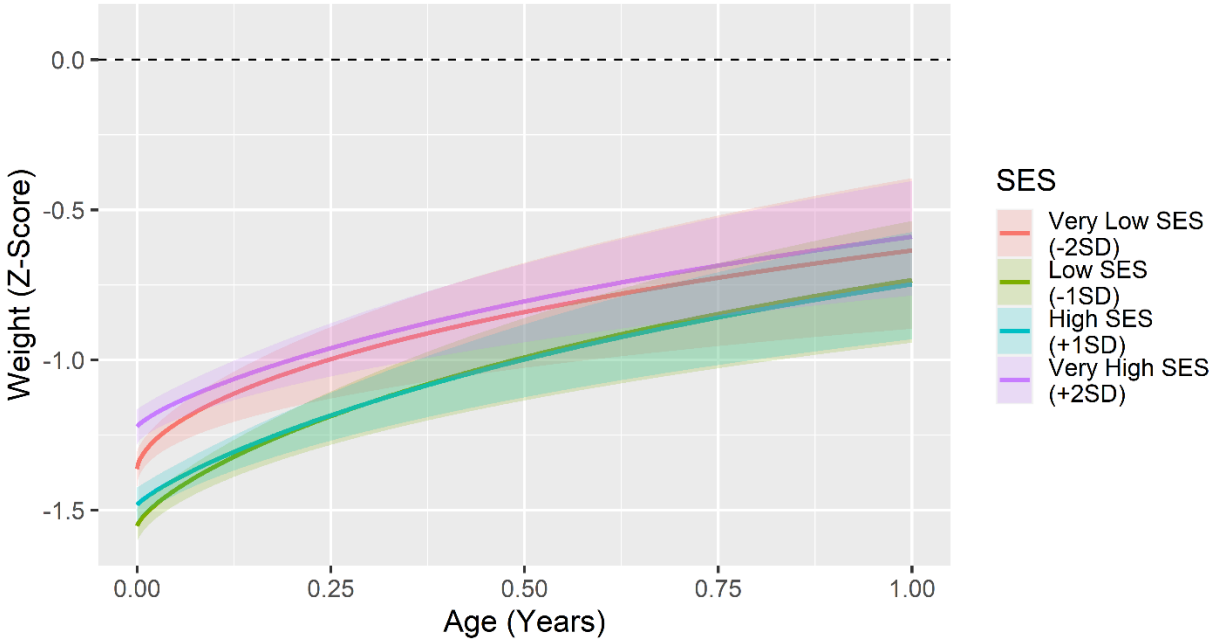
Figure 11
Correlates of Weight Recovery



Note. The dashed line represents the population mean of 0. The shaded area around each trajectory represents the standard error.

The quadratic, but not the linear SES term was associated with the intercept and upper asymptote of weight recovery. This suggests that the associated between SES and weight recovery becomes greatest at the extremes (high and low) of SES. Figure 12 depicts the predicted trajectories of weight recovery by SES across the first year, which highlights differences in the intercept by SES. At birth, children in very low (-2 SDs) or very high (+2 SDs) SES are born the heaviest.

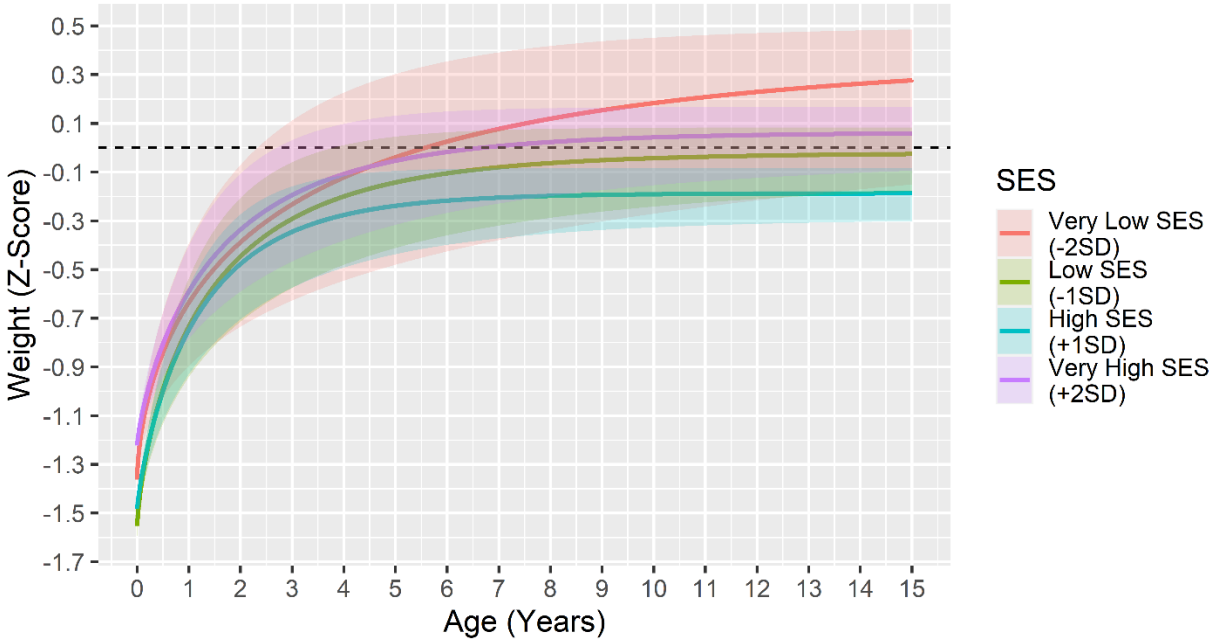
Figure 12
Predicted Weight Recovery by SES across the First Year



Note. The dashed line represents the population mean of 0.

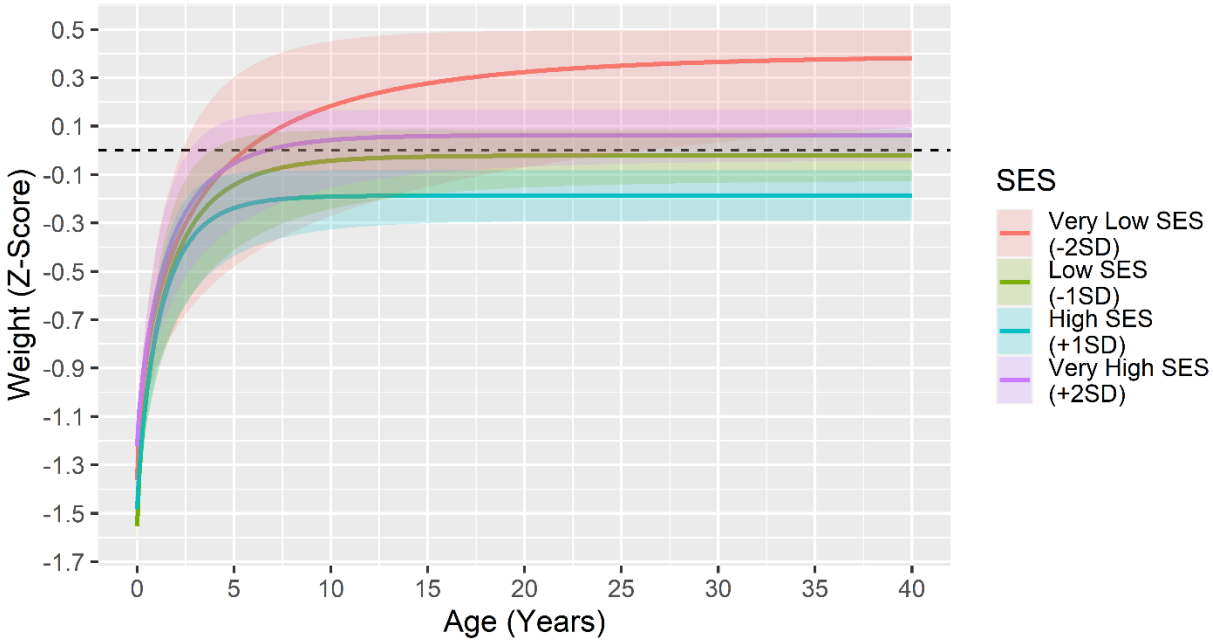
By adolescence, children at very low and very high SES have the highest upper asymptotes (see Figure 13). Notably, children at very low SES have the highest weight Z-scores and are still approaching their upper asymptote of weight relative to the population mean in late adolescence. The discrepancy between children born at very low SES and other children become more extreme if the predicted trajectories are extended to middle adulthood (see Figure 14) and suggest a relationship between poverty and unhealthy weight gain across the lifespan.

Figure 13
Predicted Weight Recovery by SES from Birth to Adolescence



Note. The dashed line represents the population mean of 0.

Figure 14
Predicted Weight Recovery by SES from Birth to Middle Adulthood



Note. The dashed line represents the population mean of 0. Estimates beyond 15 years are predicted based on the parameter estimates of the model fit to 15 years and do not reflect actual measured weight.

As an approximation of effect size, I calculated the proportion of the shared environmental (C) variance explained by measured shared environmental constructs (i.e., family SES, gestational age, etc.). This was calculated by subtracting the unstandardized shared environment (C) variance in the constrained model from the C variance in the unconstrained model and dividing the difference by the C variance in the unconstrained model (Singer et al., 2003). The study covariates accounted for 48% of the variance in C for the intercept, 20% of the variance in the upper asymptote, 17% of the variance in the rate of growth, and 8% of the variance in the inflection point.

Study Aim 1b: Model the Recovery of Height

Model Identification: Recovery of Height. The growth curve models for height yielded a similar story to the growth curve models for weight. The Weibull growth model fit the data better than the other growth models according to the BIC, but the TLI and RMSEA values indicate that the fit to the data was poor (see Table 11). As with the model of weight recovery, adding the banded structure autocorrelations to the residuals of the Weibull growth model improved the model fit to an acceptable level (X^2 = 1,095.1, df = 123, $p < .001$; RMSEA = .08; TLI = .91). The average twin was 0.92 SD below the population mean in terms of height at birth and recovered to within 0.13 SD of the population mean. Growth parameter estimates are presented in Table 12. The mean trajectory of height recovery is depicted in Figure 15.

Table 11
Model Fit Statistics - Height

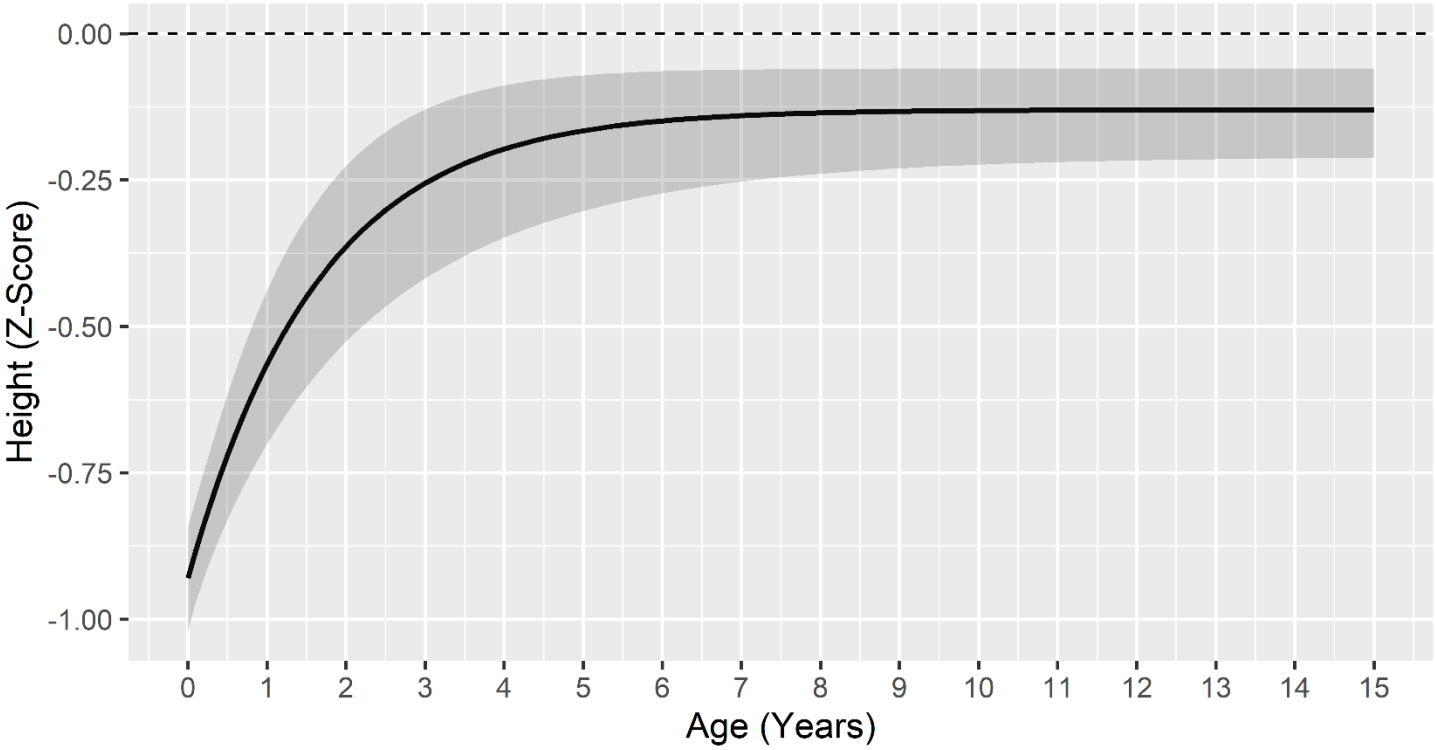
Fit Statistic	Linear	Quadratic	Cubic	Exponential	Weibull	Morgan-Mercer-Flodin	Logistic	Gompertz	Richards
X^2/df	3,599/148	2,699/144		1,841/144	1,565/139	1,773/139	1,571/139	1,666/139	
TLI	.74	.80		.87	.88	.87	.88	.88	
RMSEA	.13	.12		.10	.09	.10	.09	.10	
AIC	18,689	17,283		15,838	15,357	15,530	15,363	15,432	
BIC	18,803	17,418		15,973	15,517	15,690	15,523	15,592	

Table 12
Parameter Estimates from the Recovery of Height

			Correlation Coefficient [95% C.I.]		
Parameter	Mean [95% C.I.]	Variance [95% C.I.]	Intercept (b_0)	Upper Asymptote (b_1)	Rate of Growth (b_2)
Intercept (b_0)	**-0.93 [-1.02, -0.84]**	**0.75 [0.55, 0.95]**			
Upper Asymptote (b_1)	**-0.13 [-0.21, -0.06]**	**0.92 [0.83, 1.01]**	**.41 [.30, .53]**		
Rate of Growth (b_2)	**0.61 [0.50, 0.72]**	**1.01 [0.49, 1.53]**	**-.20 [-.36, -.03]**	**-.17 [-.28, -.06]**	
Inflection Point Scaling Parameter (b_3)	**1.01 [0.91, 1.10]**	**2.45 [1.43, 3.47]**	**.24 [.04, .43]**	**-.19 [-.31, -.06]**	-.13 [-.33, .07]

Note. Estimates that are significantly different from 0 at $p < .05$ are presented in bold.

Figure 15
Predicted Height Recovery Based on the Weibull Function



Note. The dashed line represents the population mean of 0. The shaded area represents the 95% confidence interval of the growth curve.

ACE decomposition for height. Biometric analyses of the growth parameters for the recovery of height indicate that the shared environment accounts for the majority of the variance in the intercept whereas additive genetics account for the majority of the variance in the upper asymptote. Additive genetics and the shared environment account for relatively comparable proportions of the rate of recovery and inflection point. A Wald test revealed that the proportion of the variance in the rate of growth and inflection point attributed to additive genetics is not significantly different from the proportion of variance due to shared environmental factors (X^2 = 0.86, df = 2, p = .650). See Table 13 for the unstandardized and standardized ACE estimates.

Table 13
Variance in Height Growth Parameters Due to Genetic and Environmental Components

Parameter	Additive Genetic (A) [95% C.I.]	Shared Environment (C) [95% C.I.]	Nonshared Environment (E) [95% C.I.]	Proportion A	Proportion C	Proportion E
Intercept (b_0)	0.19 [-0.20, 0.57]	**0.72 [0.31, 1.13]**	**0.13 [0.05, 0.21]**	.18	.70	.13
Upper Asymptote (b_1)	**0.82 [0.63, 1.01]**	0.08 [-0.12, 0.27]	**0.05 [0.04, 0.06]**	.87	.08	.05
Average Rate of Change (b_2)	**0.80 [0.25, 1.34]**	**0.70 [0.11, 1.29]**	0.06 [-0.01, 0.14]	.51	.44	04
Inflection Point (b_3)	**0.90 [-0.09, 1.88]**	**1.68 [0.58 – 2.78]**	0.11 [-0.04 – 0.25]	.33	.63	.04

Note. Proportions that are significant at the p < .05 level are highlighted in bold. Spaces shaded black indicate ACE parameters that had to be constrained to 0. Proportions that do not add up to 1 are due to rounding.

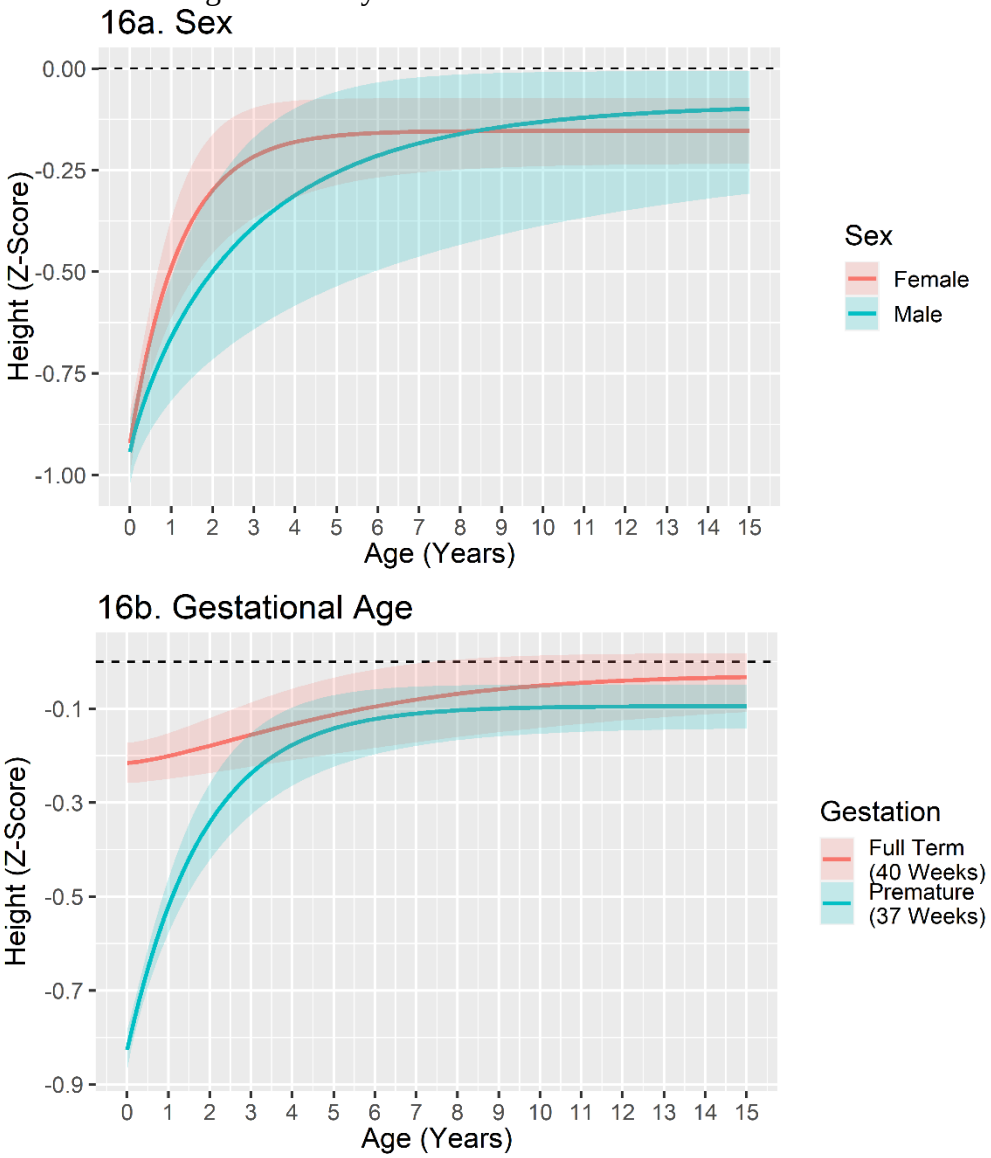
Environmental correlates of height recovery. Maternal age was not significantly related to any of the height recovery parameters. Females had a faster rate of recovery than males, but there were no differences in the intercept or upper asymptote by sex (see Figure 16a). Gestational age was a strong correlate of height recovery. Children born at full term were within a quarter standard deviation of the population mean at birth and gradually grew toward the population mean. On the other hand, premature children were substantially below the population mean at birth and recovered rapidly toward the population mean. By early childhood, premature and full-term children were statistically indistinguishable in terms of height (see Figure 16b). As with weight, linear SES was not significantly associated with recovery of height. However, there was a significant quadratic relationship between SES and the upper asymptote of height, indicating that children in both very wealthy and very poor homes were taller relative to the population mean in adolescence (see Figure 16c).

Table 14
Correlates of Height Recovery

	Between-Pair Effects			
Predictor Variable	Intercept (b_0) *B* [95% C.I.]	Upper Asymptote (b_1) *B* [95% C.I.]	Rate of Change (b_2) *B* [95% C.I.]	Inflection Point (b_3) *B* [95% C.I.]
Sex	-0.02 [-0.18, 0.14]	0.07 [-0.09, 0.22]	**-0.43 [-0.67, -0.18]**	-0.14 [-0.49, 0.21]
Family SES	0.07 [-0.02, 0.16]	-0.02 [-0.11, 0.07]	-0.05 [-0.17, 0.08]	0.15 [-0.05, 0.34]
Family SES^{2*}	0.06 [-0.04, 0.14]	**0.12 [0.03, 0.20]**	-0.10 [-0.23, 0.03]	0.01 [-0.19, 0.22]
Gestational Age	**0.65 [0.56, 0.73]**	0.07 [-0.02, 0.16]	**-0.39 [-0.53, -0.26]**	**0.43 [0.22, 0.64]**
Maternal Age	0.08 [-0.00, 0.17]	-0.04 [-0.04, 0.12]	0.08 [-0.04, 0.20]	0.11 [-0.08, 0.30]

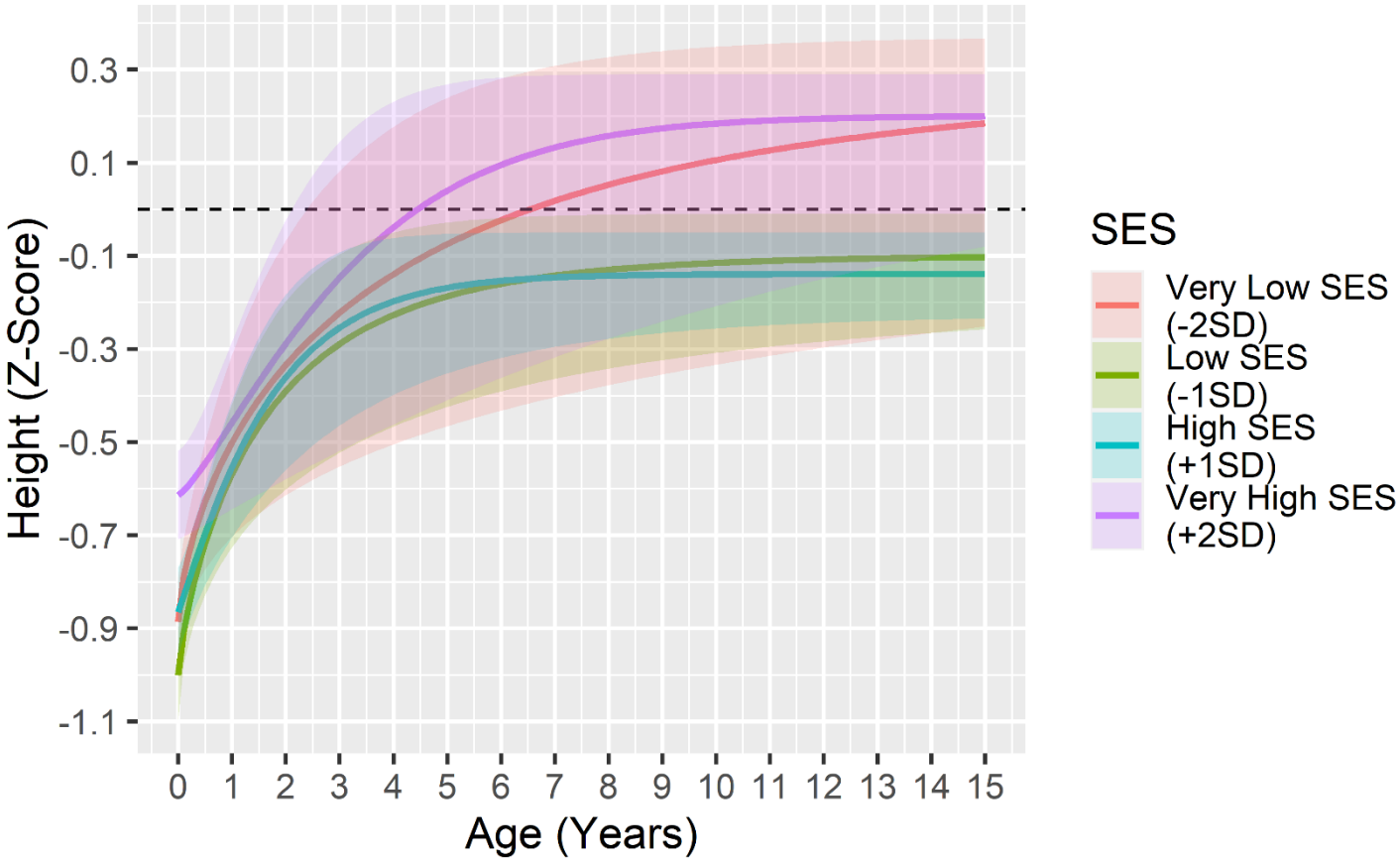
Note. Coefficients that are significant at the $p < .05$ level are highlighted in bold. *Family SES^2 is the residual of family SES squared regressed onto family SES.

Figure 16
Correlates of Height Recovery



Note. The dashed line represents the population mean of 0. The shaded area around each trajectory represents the standard error.

Figure 17
Height Recovery by SES



Note. The dashed line represents the population mean of 0. The shaded area around each trajectory represents the standard error.

Study Aim 1c: Model the Recovery of Head Circumference

Model Identification: Recovery of Head Circumference. Fit indices for the growth curve models fit to head circumference are presented in Table 15. The Morgan-Mercer-Flodin growth curve provided the best fit to the data. Moreover, the Morgan-Mercer-Flodin model provided an acceptable fit to the data based on the RMSEA value (.07) and TLI value (.96). I compared the Morgan-Mercer-Flodin model without residual autocorrelations to a model with residual autocorrelations. Correlations between birth and 3 months and between 3 and 6 months were initially estimated to be greater than 1 and had to be constrained to 0. Even with these constraints, the model with residual autocorrelations fit significantly better than the model without the constraints (X^2 = 54.5, df = 6, p < .001). Therefore, residuals were allowed to correlate with a banded structure. Parameter estimates from the Morgan-Mercer-Flodin model are presented in Table 16.

The average twin had a lower asymptote of -1.44 and grew to an upper asymptote of 0.48. Thus, the average twin's head circumference grew approximately 1.92 standard deviations relative to the population mean. The age of inflection was calculated to be 0.63. Therefore, the average twin is

growing most rapidly at approximately 7.5 months of age. The average twin reached the population mean (i.e., had an estimated z-score of 0) by 6 months (95% C.I. 5.5, 7.0 months). Finally, the average twin reached their upper asymptote by approximately 20 months (95% C.I. 17, 24 months). Therefore, most twins can be expected to recover their head circumference by 7 months, but catch-up growth may continue as late as 24 months albeit extremely gradually after 12 months (see Figure 18).

Table 15
Model Fit Statistics – Head Circumference

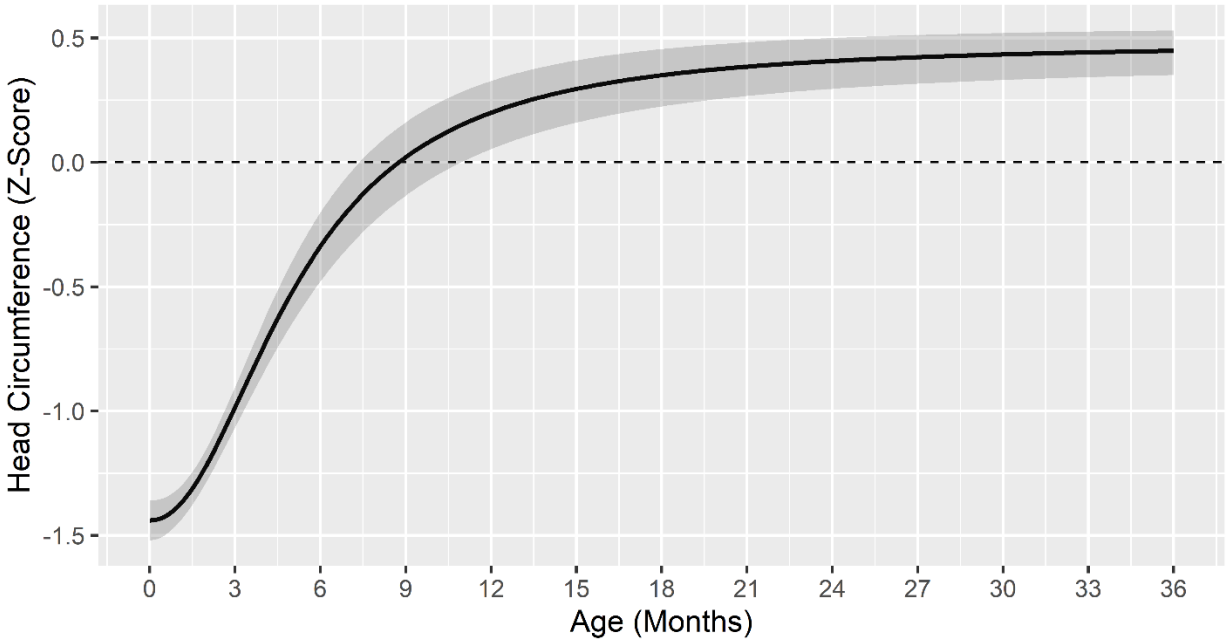
Fit Statistic	Linear	Quadratic	Cubic	Exponential	Weibull	Morgan-Mercer-Flodin	Logistic	Gompertz	Richards
X^2/df	2,041/40	1,141/36	313/31	410/36	279/33	230/32	343/39	337/31	
TLI	.69	.81	.94	.94	.95	.96	.95	.95	
RMSEA	.20	.15	.08	.09	.08	.07	.08	.08	
AIC	12,310	10,774	9,380	9,600	9,301	9,250	9,521	9,510	
BIC	12,382	10,867	9,498	9,542	9,414	9,364	9,598	9,587	

Table 16
Parameter Estimates from the Recovery of Head Circumference

			Correlation Coefficient [95% C.I.]		
Parameter	Mean [95% C.I.]	Variance [95% C.I.]	Lower Asymptote (b_0)	Upper Asymptote (b_1)	Rate of Growth (b_2)
Lower Asymptote (b_0)	**-1.45 [-1.52, -1.37]**	**0.82 [0.71, 0.93]**			
Upper Asymptote (b_1)	**0.44 [0.38, 0.51]**	**0.81 [.72, 0.90]**	**.38 [.29, .47]**		
Rate of Growth (b_2)	**2.27 [2.15, 2.37]**	**1.49 [1.02, 1.96]**	**-0.25 [-.36, -.14]**	**.23 [.11, .34]**	
Inflection Point Scaling Parameter (b_3)	**2.19 [2.01, 2.36]**	**1.21 [0.58, 1.84]**	**.63 [.45, .82]**	-.14 [-.29, .02]	**-.45 [-.65, -.25]**

Note. Estimates that are significantly different from 0 at $p < .05$ are presented in bold.

Figure 18
Predicted Head Circumference Recovery Based on the Weibull Function



Note. The dashed line represents the population mean of 0. The shaded area represents the 95% confidence interval of the growth curve.

Biometric analyses. To address convergence issues, a banded Topelitz structure had to be applied to autocorrelations between residuals (Wolfinger, 1993). A banded Topelitz structure involves constraining the residual variances to be equal over time and constraining the autocorrelations to be equal. This structure, which is presented below, is more parsimonious that the banded structure used in previous models.

$$\begin{bmatrix} \sigma_1^2 & & & \\ \sigma_2 & \sigma_1^2 & & \\ & \sigma_2 & \sigma_1^2 & \\ & & \sigma_2 & \sigma_1^2 \end{bmatrix}$$

The C component of the upper asymptote and the A component of the inflection point scaler were initially estimated to be negative, which violates the traditional ACE model. These components were constrained to be 0. Therefore, additive genetic factors are unrelated to variance in the inflection point of head circumference recovery, and shared environmental factors are unrelated to the upper asymptote of head circumference.

Unstandardized and standardized biometric estimates are presented in Table 17. Variance in the lower asymptote was predominantly due to shared environmental factors, although nonshared environmental factors contributed significantly. Additive genetics accounted for almost all of the variance in the upper asymptote and rate of growth. Shared environmental factors accounted for the majority of the variance in the inflection point scaler.

Table 17
Variance in Head Circumference Growth Parameters Due to Genetic and Environmental Components

Parameter	Additive Genetic (A) [95% C.I.]	Shared Environment (C) [95% C.I.]	Nonshared Environment (E) [95% C.I.]	Proportion A	Proportion C	Proportion E
Lower Asymptote (b_0)	0.16 [-0.04, 0.35]	**0.48 [0.27, 0.69]**	**0.23 [0.16, 0.29]**	.18	.56	.26
Upper Asymptote (b_1)	**0.77 [0.68, 0.87]**	0	**0.05 [0.03, 0.07]**	.94	0	.06
Average Rate of Change (b_2)	**1.06 [0.43, 1.39]**	0.13 [-0.49, 0.75]	**0.29 [0.11, 0.48]**	.71	.09	.20
Inflection Point Scaler (b_3)	0	**1.08 [0.66 – 1.51]**	**0.67 [0.40 – 0.94]**	0	.62	.38

Note. Proportions that are significant at the $p < .05$ level are highlighted in bold. Spaces shaded black indicate ACE parameters that had to be constrained to 0. Proportions that do not add up to 1 are due to rounding.

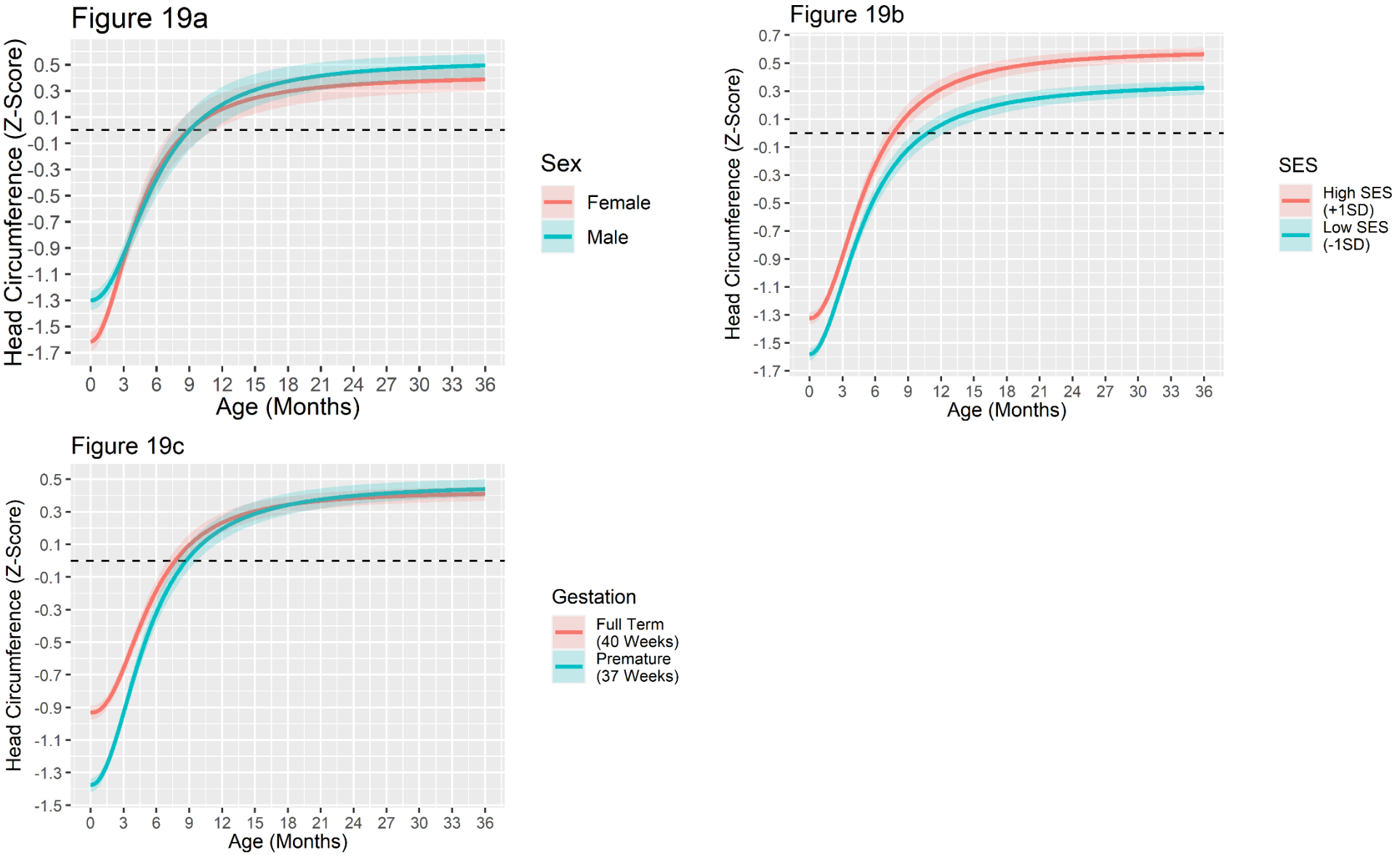
Environmental correlates of the recovery of head circumference. The growth parameters for head circumference recovery were regressed on gestational age, SES, SES squared, sex, and maternal age at birth (see Table 18). Males had a higher lower asymptote than females, but females recovered at a faster rate. SES was linearly related to the lower and upper asymptotes. Specifically, individuals a standard deviation above the mean in SES had a 0.13 standard deviation advantage in their head circumference in infancy and a 0.11 standard deviation advantage in the upper asymptote. Thus, the early advantages of SES associated with head circumference appear to be retained into early childhood. Gestational age was strongly associated with the lower asymptote of head circumference; a standard deviation increase in gestational age is associated with about a half standard deviation increase in head circumference in infancy. However, gestational age is not associated with the upper asymptote. Children born earlier have a slower rate of recovery, but an earlier inflection point, suggestive of a longer period of recovery. Maternal age was not significantly associated with any of the cognitive recovery parameters. See Figure 24 for plots of head circumference recovery trajectories by sex, SES, and gestational age.

Table 18
Correlates of Recovery of Head Circumference

	Between-Pair Effects			
Predictor Variable	Lower Asymptote (b_0) *B* [95% C.I.]	Upper Asymptote (b_1) *B* [95% C.I.]	Rate of Change (b_2) *B* [95% C.I.]	Inflection Point Scaler (b_3) *B* [95% C.I.]
Sex	**0.32 [0.17, 0.46]**	0.11 [-0.03, 0.25]	**-0.62 [-0.87, -0.36]**	0.14 [-0.16, 0.43]
Family SES	**0.13 [0.04, 0.21]**	**0.12 [0.04, 0.20]**	-0.04 [-0.17, 0.10]	0.09 [0.08, 0.26]
Family SES^2	0.02 [-0.07, 0.11]	0.06 [-0.02, 0.14]	0.03 [-0.10, 0.17]	-0.10 [-0.28, 0.07]
Gestational Age	**0.50 [0.41, 0.58]**	-0.06 [-0.13, 0.02]	**-0.15 [-0.29, -0.01]**	**0.20 [0.03, 0.36]**
Maternal Age	0.04 [-0.05, 0.13]	0.03 [-0.04, 0.11]	-0.02 [-0.11, 0.16]	0.06 [0.11, 0.23]

Note. Coefficients that are significant at the $p < .05$ level are highlighted in bold

Figure 19
Correlates of Head Circumference Recovery



Note. The dashed line represents the population mean of 0. The shaded area around each trajectory represents the standard error.

As shared environmental factors (C) accounted for a significant proportion of the variance in the lower asymptote (b_0) and inflection point scaling parameter (b_3), I was able to calculate the proportion of the C variance explained by measured shared environmental constructs (i.e., family SES, gestational age, and shared environmental factors associated with gender). This was calculated by subtracting the unstandardized shared environment (C) variance in the constrained model from the C variance in the unconstrained model and dividing the difference by the C variance in the unconstrained model (Singer et al., 2003). The study covariates accounted for 62% of the variance in C for the lower asymptote and about 1% of the variance in the inflection point scaling parameter.

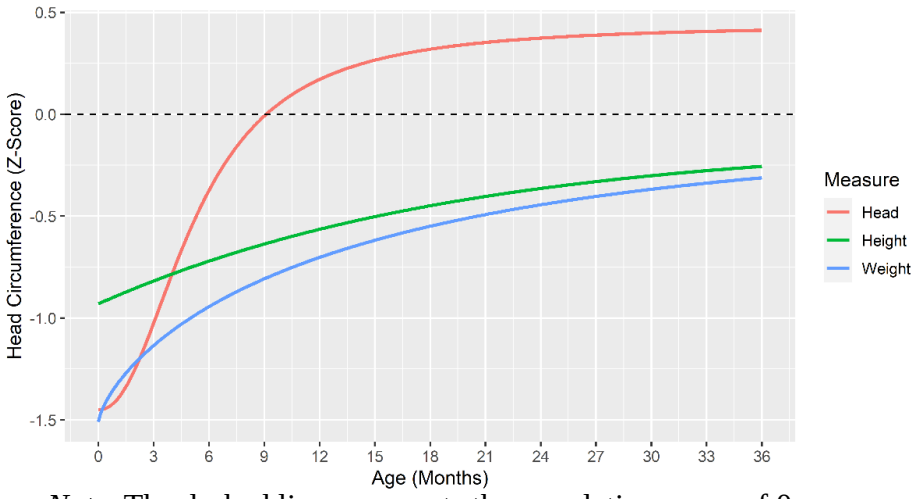
Discussion

Nonlinear growth models fit to age-standardized measurements of height, weight, and head circumference from birth to adolescence revealed a pattern of development characterized by large deficits in physical size at birth and rapid physical recovery across infancy. At birth, deficits in physical size ranged from 1.5 SD below the population mean for weight to 0.9 SD below the population mean for height. These findings suggest slightly larger physical deficits at birth than have been reported elsewhere (Estourgie-van Burk et al., 2010; Johnson et al., 2011), but are comparable to observations in the Netherland Twin Register (van Dommelen et al., 2008). Although the upper asymptotes for height and weight were statistically different from 0, the average twin was within 0.1 SD of the mean at their upper asymptote, suggesting that the difference is not clinically meaningful. Additionally, the upper asymptote for head circumference was slightly above the population mean. Thus, on average twins fully recovered to age-typical heights, weights, and head circumference, which is consistent with findings from previous research (Estourgie-van Burk et al., 2010; Wilson, 1974b, Wilson, 1979).

The rate of recovery for each growth metric was most rapid in infancy, highlighting the importance of recovery over the first few months of life. For example, the rate of head circumference recovery was most rapid at approximately 7.5 months, and on average twins had recovered half of their initial weight deficits by 12 months. Therefore, children born with significant deficits in their physical size would be expected to make rapid progress toward the population mean across the first year of life. Children who do not make progress toward the population mean over the first year of life may be at an increased risk for severe health problems related to physical size including stunting, wasting, microcephalia, or failure to thrive. Temporally, twins reached their upper asymptote of head circumference first (see Figure 20), suggesting that neurological recovery begins rapidly following birth and precedes recovery of other structures (i.e., bone structure, adipose) in infancy.

Figure 20

Early Trajectories of Height, Weight, and Head Circumference Recovery



Note. The dashed line represents the population mean of 0.

Biometric Contributions to Recovery

Biometric analyses of the recovery of height, weight, and head circumference revealed a consistent pattern across measurements. Aspects of the shared environment accounted for the majority of the variance in the intercept of physical size, additive genetics accounted for the majority of the variance in the upper asymptote, and the rate and shape of recovery were associated with a combination of additive genetic and shared environmental factors. The large association of shared environmental factors and nonsignificant association of genetic factors with variance in physical size at birth suggests that exposure to early bioenvironmental stress (e.g., premature birth) may override genetic contributions to early physical size. Interestingly, nonshared environmental factors explained a small but statistically significant portion of the variance in the intercepts of height, weight, and head circumference. Significant nonshared environmental contributions to early physical size may reflect differences in placental placement which lead to better nutritional access for one twin (Marceau et al., 2016) or in more extreme cases, twin-twin transfusion syndrome (Simpson et al., 2013).

On the other hand, additive genetics accounted for the majority of the variance in the upper asymptotes of height, weight, and head circumference whereas shared environmental factors did not account for a significant portion of the variance. As children age and early environmental stressors (e.g., perinatal stressors) become more distal experiences, genetic influences on physical size may have more room to operate. Findings that additive genetics contribute to the majority of the variance in the upper asymptote of physical size are consistent with previous research that has found physical size to be highly heritable in samples above the age of 3 years (Liu et al., 2015; Silventoinen et al., 2000; Silventoinen et al., 2003; Smit et al., 2010; Yang et al., 2007). The nonsignificant contribution of shared environmental factors suggests that environmental differences between families (e.g., between-family differences in the types of food available at home) do not significantly contribute to differences in the upper asymptote of height, weight, or head circumference. However, the significant nonshared environmental contributions to the variance of the upper asymptote of physical size suggest that within-family differences (e.g., within-pair differences in diet) contribute to the variance in physical size.

The present study made unique contributions to the literature by demonstrating that the variance in the latent rate and shape of physical recovery from early bioenvironmental adversity is attributable to a combination of genetic, shared environmental, and nonshared environmental factors. Additive genetics and shared environmental factors accounted for approximately equal proportions of the rate of recovery and inflection point of recovery. This pattern of findings is consistent with previous research which has found that the latent rate of growth of *raw* height and weight are due to both additive genetic and shared environmental factors (Demerath et al., 2007; Johnson et al., 2011; Livshits et al., 2000). Therefore, a combination of genetic factors (e.g., genes influencing the accumulation of body mass and bone growth) and shared environmental factors (e.g., feeding schedule, postnatal diet) are related to how quickly infant twins recover physically.

Patterns of Recovery by Sex

The intercepts of weight and head circumference were higher for males than females, suggesting that males may be more resilient to the perinatal stressors associated with twinning. However, females demonstrated a faster rate of growth in height, weight, and head circumference than males. The upper asymptotes for height, weight, and head circumference were statistically

indistinguishable between males and females. Therefore, early discrepancies in relative physical size between males and females do not persist into childhood, and early discrepancies are overcome by a faster rate of recovery among females. A similar pattern of height and weight recovery was observed among a sample of male and female singletons born at very low birth weight (< 1,500 grams) followed from birth to 20 years (Hack et al., 2003). At birth, males had higher weight-for-age and height-for-age Z-scores relative to females, but by young adulthood, females were indistinguishable from the population mean whereas males remained small (Hack et al., 2003). Previous research has found that females are less susceptible to neonatal complications compared to males, which may explain the faster rates of recovery in females (Stevenson et al., 2000).

Length of Gestation and Physical Recovery

Consistent with hypotheses, gestational age emerged as an important environmental correlate of early height, weight, and head circumference. Previous research has concluded that much of the discrepancy in size between twins and singletons is due to shorter gestational periods for twins (Buckler & Green, 2004). However, gestational age did not explain all of the variance in early physical size. Children born at full gestation (40 weeks) were predicted to have initial weights and head circumferences nearly 1 SD below the population mean. Regarding birth length, full term infants were only slightly below the population mean (0.2 SD) whereas premature twins were nearly three-quarters of a SD below the population mean. Factors associated with birth length (e.g., skeletal growth) may benefit more from full gestation. However, measurements of length at birth are known to be unreliable (Laar et al., 2018) and may not have been performed on the unhealthiest neonates due to the medical risks associated with stretching a medically-fragile newborn on a table to measure length (Hark et al., 2003). Indeed, rates of missing birth length were relatively high (20%) and there was a decline in length relative to the population mean between birth and 3 months when smaller infants could be safely measured (see Figure 4).

Across height, weight, and head circumference, gestational age was not associated with the upper asymptote, indicating that premature twins recovered early deficits in physical size both relative to their full-term twin peers and relative to the population mean. Gestational age does not appear to be a long-term predictor of physical size. Infants born earlier had a faster rate of height, weight, and head circumference growth, providing some insight into the process by which children recover physically following premature birth. That is, premature infants recover physically by growing more rapidly than full-term infants (as opposed to more slowly but for a longer period). A variety of prenatal stressors associated with premature birth are also associated with restricted prenatal growth (e.g., preeclampsia, gestational diabetes, uterine size restrictions) (Goldenberg et al., 2008). Once prenatal environmental stressors are removed following birth, biological mechanisms may take over and stimulate physical growth.

Nonlinear Associations between SES and Physical Recovery

In general, being from a higher-SES household is associated with better physical development from birth to adolescence. Accounting for gestational age, sex, and maternal age, children in wealthier families are born heavier, longer, and with larger heads, indicating more complete prenatal development. However, even children born in the highest-SES households displayed deficits in their early height, weight, and head circumference. Interestingly, SES was quadratically associated with the intercept of height and weight such that children born in very wealthy and very

poor homes were closest to the population mean at birth. Families at the highest end of the SES spectrum are likely able to afford high-quality prenatal care and may have access to better nutrition to support healthy prenatal development. On the other hand, the implementation of social programs in the 1960s including Medicaid and the Women, Infant, and Children Nutritional Supplement Centers may provide the poorest families access to regular prenatal medical care and quality nutrition. Within families, participation in WIC during pregnancy has been associated with increased birth weight, suggesting that programs targeted at improving prenatal nutrition among low-income families are effective in improving birth outcomes (Kowaleski-Jones & Duncan, 2002). Unfortunately, data on participation in WIC or other programs is unavailable in the Louisville Twin Study, prohibiting my ability to test associations between participation in WIC and birth outcomes.

Family SES was also quadratically related to the upper asymptote of height and weight. Specifically, children in the poorest homes were about 0.4 SD above the population mean in terms of weight, and children in the wealthiest and poorest homes were about 0.2 SD above the population mean in terms of height. Family SES was unrelated to the rate of height or weight recovery, indicating that children in the poorest and wealthiest homes grew relative to the population mean at the same rate as their average SES peers, but grew for a longer period. In the case of weight, the extended weight growth may highlight the developmental course of child- or adolescent-onset obesity among low birthweight children reared in poverty (Lee et al., 2014; Klebanov et al., 2014). In the context of poverty, parents trying to encourage catch-up growth in their young children may rely on high-sugar, calorie-dense foods which tend to be cheaper and more widely available in poor neighborhoods (Drewnowski & Specter, 2004; Hails & Shaw, 2019).

Limitations and Future Directions

Although there is socioeconomic variability in the Louisville Twin Study, it is an almost entirely White sample. Therefore, findings reflect the process of physical recovery in White twins born in the United States and do not necessarily generalize to populations outside of the United States or nonwhite populations within the United States. For example, rates of malnutrition are extremely low in the United States (United Nations Children's Fund, 2021), and social programs such as WIC centers and the Supplemental Nutrition Assistance Program allow even extremely poor families to meet the basic nutritional needs of their children (Bitler et al., 2015; Kowaleski-Jones & Duncan, 2002). However, in developing countries where rates of malnutrition are higher and supplementary nutritional programs do not exist, shared environmental factors may explain a greater proportion of the variance in the upper asymptote of physical recovery. Moreover, as structural barriers have contributed to inequities in access to socioeconomic resources and medical care in the United States along racial lines for centuries (Bailey et al., 2017; Corcoran et al., 2008), the process of physical recovery in Black, Latinx, and Indigenous twins may be different than in White twins. Future research extending findings from the present study to racially diverse samples within the United States as well as to samples from developing countries is necessary to further our understanding of how children recover physically from early bio-environmental adversity.

Findings also reflect typical patterns of physical recovery in a sample with relatively low rates of very premature birth and very low birth weight. In the study sample, only 2.3% of the twins were born very prematurely and 2.4% were born at very low birth weight. Very premature birth

reflects a more extreme degree of early biological disadvantage from which children have to recover. Although children born very prematurely or at very low birth weight demonstrate catch-up growth relative to the population mean, differences in physical size may persist into adolescence (Ford et al., 2000). Moreover, very premature or very low birth weight infants may demonstrate a different pattern of recovery. For example, Niklasson and colleagues (2003) observed that very premature infants initially fell further behind the population mean in terms of weight and length over the first month of life before making slow, steady progress toward the population mean over the next seven years. More work is needed to understand the process of physical development in very premature and extremely premature infants.

I was limited to broad measures of environmental factors associated with physical recovery, which prevented me from exploring more specific environmental correlates of physical recovery in twins. Gestational age was included in models as a crude indicator of prenatal health, and family SES at birth was included as a general indicator of the postnatal environment. Having data on more specific prenatal experiences such as exposure to substances or heavy metals, maternal health concerns (e.g., preeclampsia, gestational diabetes), or mental health concerns (e.g., maternal depression) would likely explain additional variance in physical size and birth and the subsequent pattern of recovery. Moreover, unmeasured postnatal experiences (e.g., diet, use of stimulant medications, neighborhood characteristics) are likely associated with patterns of physical recovery above and beyond family SES. As individual, family, and community (e.g., neighborhood) experiences all contribute to child development (Bronfenbrenner, 1989), having more detailed information on the child's environment would be helpful for future research to more accurately explain how children recover from early bioenvironmental adversity and inform interventions.

Relatedly, I was unable to test mechanisms by which exposure to broad environmental factors (e.g., SES) was associated with physical development. A potentially concerning finding is the quadratic association between family SES and weight where children in the poorest homes had the highest upper asymptote of weight. One of the hypothesized mechanisms by which family SES is associated with an elevated weight is through the consumption of more processed foods higher in fat and sugar content (Drewnowski & Specter, 2004). Community-level factors such as neighborhood dangerousness, available green space, and access to grocery stores that sell a variety of healthy foods may also mediate the association between poverty and increased weight gain in children (Lovasi et al., 2013). Unfortunately, data on the diets or neighborhood characteristics of children in the Louisville Twin Study are unavailable. Future research is warranted to test the specific mechanisms by which exposure to poverty in infancy is related to elevated weight gain across childhood among children born at low birth weight.

A primary assumption of the present study is that physical recovery to the population mean is a positive indicator of physical health. Indeed, among children born at low birth weight, small birth length, or small head circumference, catch-up growth is associated with healthier physical and cognitive development (Jensen et al., 2015; Scharf et al., 2016). However, research suggests that rapid catch-up growth in weight is associated with a greater risk for obesity and cardiovascular problems in adulthood (Kelishadi et al., 2015; Martin et al., 2017). Future research should consider the process by which children recover physically (i.e., the rate of recovery and inflection point) as a predictor of physical health in adulthood. Such questions will be able to be answered following

the recent extension of the Louisville Twin Study into adulthood and a renewed focus on biological aging (Beam et al., 2020).

Finally, I am unable to make any causal or quasi-causal claims regarding the "effects" of genetics or environmental factors on physical recovery in twins. Although the upper asymptotes of height, weight, and head circumference were highly heritable, it is not necessarily the case that one's upper asymptote of physical size is caused by their genetics. Active or evocative gene-environment or phenotype-environment correlations may inflate the proportion of variance attributed to additive genetics (Beam & Turkheimer, 2013). For example, infant appetite, a highly heritable characteristic (Llewellyn et al., 2010), may contribute to one twin consuming more or requesting more frequent feeding. Genetic differences related to early appetite could contribute to within-pair differences in the rate of growth or upper asymptote of physical size for dizygotic twins.

Conclusions

Recovery of physical size in twins begins immediately after birth as evidenced by the preferred fit of exponential-shaped functions (e.g., Weibull, Morgan-Mercer-Flodin) over S-shaped functions (e.g., Gompertz). Temporally, recovery of head circumference occurred before the recovery of height and weight. However, on average, substantial recovery of all physical measures occurred within the first year of life. Consistent with previous research, gestational age was an important early environmental correlate of height, weight, and physical size. However, with the exception of birth length, twins born at full term continued to display substantial deficits in their physical size at birth. Therefore, additional prenatal factors associated with twinning (e.g., competition for nutrients) likely also contribute to the relatively small physical size of twins at birth. Importantly, there were no differences in childhood physical size between premature and full-term twins. Family SES also emerged as an important environmental correlate to physical recovery in twins. Children in the wealthiest and poorest homes were closer to the population mean at birth in terms of weight and length. However, children in the poorest homes grew to be both heavier and taller than the population mean by adolescence. The relatively larger estimated weight among very poor children in adolescence (z-score = 0.4) compared to the estimated height (z-score = 0.2) suggests a possible like between poverty and later obesity among children born at low birth weight through extended weight gain across childhood.

Although the present study focused on a sample of twins, findings likely generalize to singletons who experience early bioenvironmental risk. As with twins, most singletons who are born at low birth weight, short birth length, or with a small head circumference at birth catch up to population norms (Lundgren et al., 2001), and substantial catch-up growth appears to occur across the first few months of life (Albertson-Wikland & Karlberg, 1997). Pediatricians and primary care physicians working with children who are born prematurely or physically small should expect to see rapid recovery in physical size across the first year. Children who are not making progress toward the population mean on age-standardized measurements over the first year may be at risk to remain physically small throughout their lives and may benefit from more rigorous intervention to encourage catch-up growth (e.g., nutritional supplementation).

Study 2:
Genetic and Environmental Correlates of the Nonlinear Recovery of Cognitive Ability in Twins

Twins display a well-documented delay in cognitive ability during infancy and the toddler years, scoring nearly one standard deviation below the population mean on the Mental Development Index (MDI) of Bayley Scales of Infant Development (Datar & Jacknowitz, 2009; Koeppen-Schomerus et al., 2000; Price et al., 2000; Wilson 1974). By early childhood, however, twins who previously exhibited deficiencies score at the population mean (Wilson, 1974). Although cognitive ability measured in infancy is generally a poor predictor of cognitive functioning in adolescence or adulthood (Bishop et al., 2003; Honzik et al., 1948; Schneider et al., 2014), infant cognitive ability is thought to set the stage for later cognitive development and correlates at least moderately with early language development (Siegel, 1981) and cognitive ability in early childhood (Bishop et al., 2003; Yu et al., 2018). Moreover, depressed cognitive functioning in infancy and toddler years is an early indicator of neurodevelopmental disorders (Johnson & Marlow, 2006). Thus, understanding typical and atypical patterns of early cognitive development in twins has implications for clinicians and educators seeking to differentiate children who are delayed at a level requiring intervention from those who have developmentally typical delays from which they are likely to recover.

In a series of studies using prospective data from the Louisville Twin Study (LTS), Ronald Wilson documented the delay in early cognitive ability in twins (Wilson, 1972; Wilson, 1974; Wilson, 1975) as well as the subsequent recovery to performance at the population mean, which was observed to occur by age 6 (Wilson, 1974; Wilson, 1975). Since the publication of Wilson's seminal papers, the sample size of the LTS has nearly doubled and standardized cognitive data have been collected to age 15 years (Davis et al., 2019), and are currently being collected in midlife (Beam et al., 2020). The present study builds on early LTS research and applies contemporary nonlinear growth methods to quantify the magnitude and shape of recovery of twins' standardized cognitive ability from 3 months to 15 years. I then leveraged the genetically informative portion of the study to decompose the variance in growth parameters into additive genetic (A) variance, shared environmental (C) variance, and nonshared environmental (E) variance. Additive genetic variance refers to the sum of genetic contributions, the shared environment refers to features of the rearing environment that increase twin similarity, and the nonshared environment refers to features of the rearing environment that reduce twin similarity including measurement error. Finally, I examine measured shared and nonshared biological and environmental correlates of the growth in standardized cognitive ability, including socioeconomic status (SES), household chaos, gestational age, birth weight, and maternal age.

Infant Cognitive Ability in Twins: The Role of the Shared Environment

The established delay in early cognitive ability among twin samples is frequently attributed to the fact that, relative to their singleton peers, twins are six times more likely to be born prematurely (defined as less than 37 weeks gestation) and, consequently, are frequently born at lower birth weights (Giuffrè et al., 2012). Adding credence to the hypothesis that prematurity and low birth weight contribute to lower cognitive scores in infancy, twin samples oversampled to be of typical gestational age and birth weight do not display the same deficits in Bayley MDI scores (Bishop et al., 2003; Cherny et al., 1994). However, as gestational age, prenatal stressors, and low birth weight are highly correlated (Wadhwa et al., 1993), it can be difficult to tease apart the effects of a single factor on early cognitive development. Using a within-pair study design, Datar and Jacknowitz (2009) observed a small effect of birth weight on Bayley MDI scores at 9 and 24 months for dizygotic (DZ) twins, and a nonsignificant effect of birthweight on Bayley scores for monozygotic

(MZ) twins. Thus, when shared prenatal environment (including length of gestation) and genetic effects are accounted for, birth weight appears to have a minimal impact on early cognitive ability, while gestational age and other shared environmental experiences appear to drive mean differences in infant cognitive scores for twins.

Biometric studies that decompose the variance of early cognitive ability into genetic and environmental components also highlight the importance of the early shared environment in infant cognitive ability (Koeppen-Schomeerus et al., 2000; Price et al., 2000). For example, in a sample of nearly 2,000 24-month-old twins, Price and colleagues (2000) found shared environmental factors to contribute to 58% and 72% of the variance in nonverbal and verbal abilities, respectively. Similarly, using data from twins born prematurely, Koeppen-Schomeerus and colleagues (2000) found that the shared environment explained a larger proportion of variance in 24-month verbal abilities for twins born at less than 32 weeks gestation (84%) than for twins born at greater than 33 weeks gestation (73%), indicating that prenatal complications may increase the proportion of early cognitive ability attributable to the shared environment.

Aspects of the shared environment that contribute to cognitive ability in infancy are most likely transmitted through the prenatal environment (Devlin et al., 1997). Specifically, factors including gestational diabetes (He et al., 2021), exposure to heavy metals (Shah-Kulkarni et al., 2020), maternal drug and alcohol consumption (Messinger et al., 2004; Testa et al., 2003), or length of gestation (Giuffrè et al., 2012) reflect specific aspects of the shared prenatal environment that are known to contribute to early cognitive ability. Additionally, maternal age represents a shared environmental feature that may confer additional risk for prenatal complications as the risk for gestational diabetes, preeclampsia, and preterm birth increase substantially in mothers older than 40 years (Luke & Brown, 2007). Although aspects of the postnatal environment become increasingly associated with cognitive abilities as children age, researchers have observed weak associations between the home environment and cognitive abilities within the first year of life (Bradley et al., 1989; Klebanov et al., 1998; Tucker-Drob et al., 2011). Thus, initial cognitive delays from which twins begin recovering to the population mean are most likely to be associated with features of the prenatal environment.

Patterns of Cognitive Development: Early Childhood through Adolescence

Although twins display significant deficits in early cognitive ability, it is also established that they catch up to the population mean by early childhood (Antoniou et al., 2013; Ross et al., 2012), making rapid gains in standardized cognitive scores across the toddler years (Wilson, 1974; Wilson, 1975). Wilson (1974) concluded that the recovery to the population mean was most pronounced between ages 4 and 6 years, observing mean Wechsler Preschool & Primary Scale of Intelligence (WPPSI) Full Scale Intelligence Quotient (FSIQ) scores of 92.6, 95.9, and 100.9 at ages 4, 5, and 6 years, respectively. From early childhood through adulthood, twins score commensurate with the population mean on standardized cognitive assessments (Giangrande et al., 2019; Webbink et al., 2008). For example, in a prospective population sample of Dutch twins, Webbink and colleagues (2008) observed that twins' intelligence scores at ages 8, 10, 12, and 38 years were within 3.5 points (0.23 SD) of the population mean. Likewise, in a recent publication using newly recovered data from the LTS, Giangrande and colleagues (2019) observed that mean FSIQ scores were within 2.82 points (0.19 SD) of the population mean for twins from ages 7 to 15.

As twins are more likely to be born smaller than their singleton peers, a critical part of early development may involve catch-up growth in physical size (Estourgie-van Burk et al., 2010; Wilson, 1979). Early catch-up growth in height and weight among small-for-gestational-age singletons has been positively associated with higher cognitive ability scores in early childhood (Varella et al., 2015). Rapid gains toward singleton norms in terms of length and weight over the first few years of life (Wilson, 1979) may afford twins increased motor abilities to explore their environments (Jeng et al., 2000), which have been associated with subsequent cognitive development (Ghassabian et al., 2016). Therefore, cognitive recovery in twins may parallel or succeed recovery in physical size.

Biometric Contributions to Cognitive Development

As children age, genetic factors account for an increasing proportion of variance in standardized cognitive scores, while shared environmental factors become less influential (Davis et al., 2009; Tucker-Drob & Briley, 2014). For example, in a prospective study of nearly 9,000 twin pairs, additive genetic variance accounted for 23% of the variance in early childhood (ages 2-4 years) cognitive ability, while shared environmental variance accounted for 74% of the variance (Davis et al., 2009). However, by middle childhood (ages 7-10), shared environmental variance accounted for 33% of the variance in cognitive ability, while additive genetic variance accounted for 62% of the variance (Davis et al., 2009). Likewise, in a meta-analysis of 21 studies and over 12,000 twin pairs, Tucker-Drob and Briley (2014) observed that the proportion of variance in the stability of cognitive scores attributable to additive genetic variance increased exponentially across childhood, reaching an asymptote around age 10. Correspondingly, the proportion of variance associated with shared environmental factors decreased exponentially across early childhood, reflecting the increasing role of genetics at later developmental stages. Finally, using LTS data, Finkel and colleagues (2015) observed that the contribution of additive genetics to cognitive abilities at the subtest level generally increases across early childhood and then stabilizes from middle childhood to adolescence.

Despite the declining influence of the shared environment on cognitive ability throughout childhood, specific features of the shared environment may be associated with the development of cognitive ability across childhood. For example, a meta-analysis of 27 studies covering over 7,000 children ranging in age from 3 to 16 years observed an 11.94-point difference in standardized intelligence scores for children born preterm compared to children born full term (Kerr-Wilson et al., 2012). Mangin and colleagues (2017) fit linear growth models to standardized cognitive data for singleton children born very prematurely (32 weeks or less gestation) and children born at term. Very premature children had a significantly lower intercept than full-term children, but prematurity status was not associated with interindividual rates of change (Mangin et al., 2017). Thus, associations between gestational age and cognitive ability in childhood appear to reflect stable effects on initial abilities rather than the rate of growth over time.

Family SES is a robust predictor of cognitive ability in childhood (Kainz et al., 2012; Tucker-Drob et al., 2011; von Stumm & Plomin, 2015). In a prospective study of over 1,200 children, family income below 200% of the poverty line was associated with an 11-point deficit in WPPSI scores at 3 years relative to children at or above 200% of the poverty line (Kainz et al., 2012). Similarly, Tucker-Drob and colleagues (2011) observed that a standard deviation increase in family SES corresponded with a 0.33 standard deviation increase in the change in Bayley scores between 10

and 24 months. Finally, in a sample of 14,853 individual twins, children in high-SES homes at age 2 (1 SD above the mean) exhibited an increase in standardized cognitive scores from age 2 to 16, while children in low-SES homes exhibited declines in standardized cognitive scores (von Stumm & Plomin, 2015).

Household chaos, or noise, disorganization, and confusion in the home represents another aspect of the home environment (Matheny et al., 1995). High levels of household chaos may promote a strategy of filtering out unwanted environmental stimulation, which may contribute to a child inadvertently filtering out information pertinent to cognitive development (Dumas et al., 2005; Gärling & Evans, 1991). Household chaos has been negatively associated with cognitive ability in childhood, even after accounting for SES (Deater-Deckard et al., 2009; Martin et al., 2012). For example, in a sample of 302 twin pairs, higher levels of household chaos were associated with lower Stanford Binet scores in kindergarten, accounting for parent IQ, parenting warmth, and poor housing conditions (Deater-Deckard et al., 2009). Finally, a genetically informed study found chaos to account for 5% of the shared environmental variance in verbal and nonverbal abilities at ages 3 and 4 independent of the effects of SES (Petrill et al., 2004).

Gaps in the Literature

Few studies focus on understanding twins' nonlinear recovery of cognitive ability from infancy through early childhood. Only one study has applied growth curve models to standardized cognitive ability across childhood in twins, in which fit quadratic growth models were fit to standardized cognitive scores from age 2 to 16 (von Stumm & Plomin, 2015). von Stumm and Plomin (2015) observed a positive slope and negative quadratic coefficient for boys and a negative slope and positive quadratic coefficient for girls, which yielded trajectories of cognitive ability that began to drift exponentially negatively for boys and exponentially positively for girls. Polynomial curves are well-known to have considerable difficulties in modeling the actual course of growth over age, especially at the extremes, because they, by definition, extend to positive or negative infinity in both directions (Grimm et al., 2011). Examining mean cognitive scores in twins from infancy to adulthood reveals a pattern of growth characterized by deficits in infancy (Datar & Jacknowitz, 2009; Koeppen-Schomerus et al., 2000), rapid recovery toward the population mean across the toddler and early childhood years (Wilson, 1974), and stabilization around the population mean from early childhood to adulthood (Webbink et al., 2008), suggesting that the growth in cognitive ability across childhood may be best captured by a nonlinear asymptotic model.

Previous research suggests that additive genetic factors account for an increasing proportion of the variability in both general (Davis et al., 2009) and specific cognitive abilities (Finkel et al., 2015) as children age. However, it is unclear the extent to which genetic and environmental factors contribute to the *rate and shape* of growth in cognitive ability across childhood.

No twin study has published findings on the genetic and environmental factors that contribute to twins' recovery of cognitive ability. von Stumm and Plomin (2015) did not conduct biometric analyses on the intercept growth terms to determine additive genetic, shared environmental, and nonshared environmental contributions to the variance in the growth of cognitive ability over time. Finkel and colleagues (2015) conducted biometric analyses on the growth of heritability of specific cognitive abilities across childhood but did not examine the growth of general cognitive ability.

Thus, the heritability of growth of general cognitive ability, and in particular the developmentally typical recovery of cognitive ability in twins is currently unknown. Understanding biometric contributions to the rate and shape of cognitive recovery will yield insight into the relative contributions of genes and the environment and, therefore, the amenability of recovery to interventions in the environment.

The Present Study

The present study had three primary aims. First, I fit nonlinear growth models (Grimm & Ram, 2009) to standardized cognitive ability scores for twins in the LTS from 3 months to 15 years to model the magnitude, rate, and shape of the recovery of cognitive ability in twins. By considering exponential and sigmoid (s-shaped) functions, I reveal the developmental timing of cognitive recovery in twins. Specifically, exponential growth would indicate immediate, rapid recovery of cognitive ability across infancy, whereas sigmoid growth would correspond to stable low scores in infancy and rapid recovery in early to middle childhood. Nonlinear growth models also provide estimates of parameters of interest to developmental scientists, including a lower asymptote (i.e., lowest cognitive score from which a twin begins recovering), the total amount of growth, and the average rate of growth (Grimm & Ram, 2009; Grimm et al., 2013). Second, I decomposed the variance in *growth* in cognitive ability across childhood into genetic and environmental components. Finally, I examined measured prenatal and postnatal correlates to cognitive deficits in infancy, and the rate and shape of recovery to the population mean.

Methods

Participants

Participants were a subset of 1,153 individual twins (47.9% male) from 578 families recruited as a part of the Louisville Twin Study, a longitudinal study of temperament and intellectual development in twins (Davis et al., 2019; Wilson, 1983). Of the 1,153 twins included in the present study, 710 were MZ (43.8% male) and 443 were same-sex DZ (53.7% male). Zygosity was determined by blood serum analysis.

All infants were from the Louisville, Kentucky, metropolitan area. Participants were primarily White (91.5%) and were recruited to represent the socioeconomic composition of the Louisville metropolitan area. Data on child cognitive ability were collected between 1957 and 1993. Twins were removed from the initial sample of 1,267 if they were missing information on zygosity (n = 114), yielding the final sample size of 1,153.

Procedure

Cognitive testing was 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). Most individuals in the study did not have data at all 16 points, but 75% had 4 or more assessments (see Table 1). Twins were typically tested within one week of their birthdays and the testing schedule was arranged such that examiners did not assess the same individuals at consecutive assessments. Institutional review board (IRB) approval was attained from the University of Louisville (protocol number 19.0989) as a part of the Contribution of Child Development, Biological Aging, and Beta-Amyloid to Cognitive Function of the Louisville Twins at Midlife study.

Measures

Cognitive Development. Over the course of the LTS, twins were administered a variety of age-appropriate cognitive batteries. A breakdown of each measure including sample size and means is presented in Table 2. Twins were administered the Bayley Scales of Mental Development between ages 3 and 24 months, with the majority of twins completing the first edition (Bayley, 1969) and a handful of infants completing the second edition (Bayley, 1993). At 30 months, a minority of twins completed the Bayley. However, 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 restandardization (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). At age 5, twins completed 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.

All of the cognitive scales administered over the course of the LTS have a 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. To allow for a more direct comparison of scores across time, scores on the Bayley and McCarthy were rescaled to a mean of 100 and a standard deviation of 15.

Covariates. Baseline household SES was measured based on the Hollingshead Four Factor Index of Socioeconomic Status, which is based on parental occupation, education, sex, and income (Hollingshead, 1975). Hollingshead scores are based on a continuous zero to 100-point scale.

Mothers and, when available, fathers completed the Confusion, Hubbub, and Order Scale (CHAOS), a 15-item measure of commotion and disorganization within the home (Matheny et al., 1995). Participants responded True or False to each item (e.g., "it's a real zoo in our home"), and the 15 items were summed to create a total chaos score with higher scores representing higher levels of chaos. The CHAOS scale has been found to have good psychometric properties (Matheny et al., 1995), with acceptable internal reliability among the items in the present study ($\alpha = .80$ for mothers, .79 for fathers). Due to high rates of missing CHAOS scores over time, CHAOS scores were averaged within family from 3 months to 15 years to provide a broad indicator of family chaos across childhood. CHAOS scores were relatively stable over time; the median inter-wave correlation for CHAOS scores was .66 with a range of -.57 to .95 (it should be noted that the negative correlation was between 18 and 96 months and included data from only 3 families). Limiting inter-wave correlations to observations with at least 25 families yielded a median inter-wave correlation of .72 with a range of .48 to .84.

Information on child birth weight was gathered from birth certificates. Gestational age (in weeks) was calculated based on maternal-report of last menses. Maternal age (in years) at birth was also included as a study covariate. As is common practice in twin research, sex effects were regressed out of all cognitive measures before genetic analyses.

Data Analysis

Descriptive Statistics. Descriptive statistics and intercorrelations between study variables were calculated using the Base package in R version 4.0.3 (R Core Team, 2020).

Nonlinear Growth Modeling. To model the growth in cognitive scores of twins, I considered polynomial (linear, quadratic, and cubic), exponential, and sigmoid (logistic, Gompertz, and Richards) growth models. (For a comprehensive discussion of nonlinear growth models including empirical examples, see Cameron et al., 2015; Grimm & Ram, 2009; Grimm et al., 2013). All models were fit within a structural equation modeling framework using Mplus version 8.4 (Muthén & Muthén, 2017), which entailed specifying constraints on the factor loadings of the growth parameters to specify nonlinear growth for the exponential and sigmoid models.

The Gompertz model was ultimately retained as the best-fitting growth model of cognitive ability across childhood, so the specific parameters of the Gompertz model are presented. The Gompertz function is defined by:

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

In the Gompertz function, shaped as a sigmoid, b_0 represents the lower asymptote, b_1 represents the difference between the lower and upper asymptote, b_2 represents the average rate of change in cognitive scores, and b_3 represents the "inflection point" or age at which the rate of cognitive growth is the fastest. The asymptotes in a sigmoid function refer to an individual's lowest and highest cognitive scores. Thus, b_0 differs from an intercept parameter in a traditional growth model in that it is not necessarily the score when time is 0 (i.e., 3 months in the present study). Gompertz functions define asymmetrical growth such that approximately 37% of the growth in cognitive ability occurs before the inflection point (Grimm & Ram, 2009). Thus, the inflection point in the present study represents the age at which a child has completed 37% of the growth between their lower and upper asymptotes of cognitive ability.

As models comparing different growth functions were not nested, the best-fitting model was identified using the Root Mean Square Error of Approximation (RMSEA), Tucker-Lewis Index (TLI), Akaike Information Criterion (AIC), and Bayesian Information Criterion (BIC). Because twins are nested within families, the genetically-informative portion of the data were modeled as a two-level model, which required the estimation of growth models at the child level (within-pair) and family level (between-pair). Variances within and between MZ and DZ twins were then transformed into the genetic and environmental variances of the classical twin model. All models were fit using maximum likelihood estimation with robust standard errors and missing data were handled using full information maximum likelihood estimation.

Genetic Analyses. Once the best-fitting growth model was identified, the growth parameters were decomposed into additive genetic (A), shared environmental (C), and nonshared environmental effects plus measurement error (E) following a standard multilevel approach (McArdle & Prescott, 2005). The ACE decomposition takes advantage of the fact that MZ twins share 100% of their DNA sequence, whereas DZ twins share, on average, 50% of their segregating genes. The between- and within-pair variances can be decomposed into environmental and genetic components following (2-5).

$$\mathrm{MZ}_{\mathrm{within}} = \mathrm{E} \quad (2)$$

$$MZ_{between} = A + C \quad (3)$$
$$DZ_{within} = 0.5*A + E \quad (4)$$
$$DZ_{between} = 0.5*A + C \quad (5)$$

Environmental Predictors of Cognitive Growth. Growth parameters were regressed on covariates to identify environmental correlates to the growth in cognitive ability in twins. Covariates that were constant across twins within a family (i.e., SES, household chaos, gestational age, and maternal age) were included at the between-pair level of the model. Birth weight, which varied within twin pairs and between families, was included at both the within-pair and between-pair levels of the model. To aid model convergence, all covariates were standardized before inclusion.

Missing Data

As denoted in Table 19, most children in the study did not have cognitive assessments at every wave. The number of assessments was not significantly correlated with SES or birth weight. The number of assessments was significantly but weakly correlated with gestational age (r = .06, p = .048), maternal age (r = .10, p < .001), and household chaos (r = -.14, p = .002). There were no significant differences in the number of assessments by child gender or by zygosity.

Gestational age was generally negatively associated with missingness of early cognitive scores, suggesting that twins born earlier were more likely to be missing. Before 5 years, birth year was negatively associated with missing cognitive scores whereas after 5 years birth year was generally positively associated with missingness. That suggests that twins enrolled in the study later were more likely to complete the early cognitive testing, but less likely to complete testing in childhood and adolescence. At three study waves, cognitive scores at the previous wave were significantly associated with missingness at the next wave after adjusting for multiple tests. Specifically, higher scores at 6 months were less likely to be missing at 9 months (B = -.04, SE = .01, p < .001), higher scores at 12 months were less likely to be missing at 18 months (B = -.03, SE = .01, p < .001), and higher scores at 30 months were less likely to be missing at 36 months (B = -.04, SE = .01, p < .001). Therefore, there may be some bias in the observed infant and toddler cognitive scores toward higher cognitive performance. However, as cognitive scores are only modestly stable wave to wave in infancy (see Figure 14), a child's score at wave t is not necessarily indicative of their score at time t + 1. Moreover, all observed cognitive scores would be incorporated in models assuming that data are MAR (e.g., full information maximum likelihood estimation, multiple imputation). Therefore, I proceeded to analyze the cognitive data under the assumption that data are MAR using full information maximum likelihood (FIML) estimation in Mplus (Muthén & Muthén, 2017).

Table 19
Number of Cognitive Assessments Available

Number of Assessments	Number of (Individual) Twins
1	87
2	47
3	104
4	102
5	66
6	98
7	77
8	50
9	43
10	50
11	61
12	37
13	74
14	126
15	68
16	63

Results

Descriptive Statistics and Intercorrelations

Descriptive statistics for the cognitive data are presented in Table 20 and descriptive statistics for study covariates are presented in Table 3 (Study 1). Consistent with previous observations of preliminary LTS data (Wilson, 1974), scores on the cognitive assessments in infancy and the toddler years fell significantly below the standardization mean of 100, but by age 5 years mean scores were within 3 points of the standardization mean.

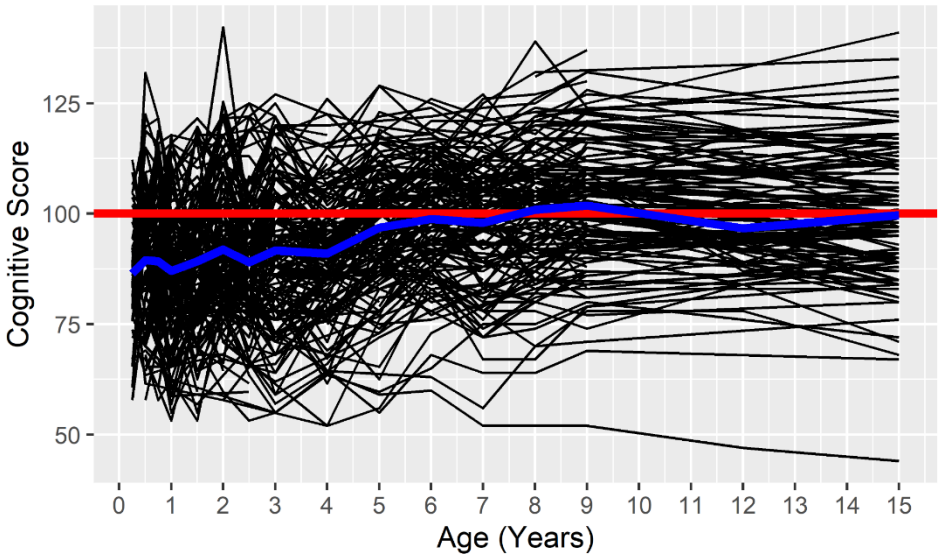
Table 20
Descriptive Statistics of Cognitive Measures

Age	Measure	*n* (MZ)	*n* (DZ)	Unscaled Mean (*SD*)	Range	Rescaled Mean (*SD*)*	*n* Missing	*r* MZ	*r* DZ	h^2
3 months	Bayley 1st Ed.	202	144	87.67 (*15.87*)	50-142	88.44 (*14.88*)	813	.687	.700	-0.03*
6 months	Bayley 1st Ed.	354	226	89.03 (*14.96*)	50-144	89.53 (*13.95*)	566	.774	.734	0.08
	Bayley 2nd Ed.	6	7	79.85 (*5.44*)	70-87					
9 months	Bayley 1st Ed.	335	175	88.81 (*15.78*)	50-136	89.47 (*14.75*)	647	.623	.661	-0.08*
	Bayley 2nd Ed.	4	0	84.00 (*6.27*)	76-89					
12 months	Bayley 1st Ed.	400	216	87.42 (*15.87*)	50-128	87.98 (*14.66*)	519	.696	.511	0.37
	Bayley 2nd Ed.	18	10	81.89 (*7.43*)	60-93					
18 months	Bayley 1st Ed.	396	234	90.82 (*15.88*)	50-136	91.11 (*14.78*)	487	.698	.580	0.24
	Bayley 2nd Ed.	33	12	86.33 (*13475*)	57-125					
24 months	Bayley 1st Ed.	388	231	93.27 (*16.41*)	50-145	93.18 (*15.22*)	486	.774	.737	0.07
	Bayley 2nd Ed.	39	14	86.49 (*12.46*)	56-122					
30 months	Bayley 1st Ed.	72	34	76.07 (*13.34*)	50-116	89.71 (*15.46*)	546	.860	.685	0.35
	Bayley 2nd Ed.	2	4	64.00 (*8.46*)	53-75					
	Stanford Binet	347	205	91.42 (*14.73*)	55-139					
36 months	Stanford Binet	465	270	91.87 (*16.52*)	45-139	91.87 (*16.52*)	422	.872	.767	0.21
4 years	Stanford Binet	35	25	77.43 (*11.45*)	52-106	91.67 (*14.05*)	448	.803	.691	0.22
	McCarthy	308	169	92.74 (*14.31*)	54-126					
	WPPSI	106	66	92.43 (*13.90*)	63-135					
5 years	McCarthy	44	14	93.00 (*17.54*)	52-121	97.21 (*13.64*)	468	.815	.596	0.44
	WPPSI	337	208	97.58 (*13.42*)	56-134					
	WPPSI-R	52	34	97.41 (*12.71*)	73-131					
6 years	WPPSI	391	232	100.86 (*12.92*)	59-144	100.50 (*12.96*)	436	.841	.595	0.49
	WPPSI-R	58	40	98.21 (*13.00*)	74-134					
7 years	WISC	112	100	101.36 (*11.06*)	71-126	98.76 (*13.32*)	496	.842	.586	0.51
	WISC-R	269	138	97.08 (*14.07*)	52-133					
	WISC-III	26	18	102.05 (*13.61*)	71-127					
8 years	WISC	174	116	104.80 (*11.25*)	79-134	102.30 (*13.17*)	418	.819	.608	0.42
	WISC-R	269	150	100.56 (*14.28*)	56-141					
	WISC-III	24	16	102.00 (*11.49*)	84-123					
9 years	WISC	48	28	109.96 (*11.04*)	82-137	103.35 (*13.73*)	639	.844	.650	0.39
	WISC-R	256	146	101.97 (*14.02*)	52-144					
	WISC-III	38	12	104.58 (*11.63*)	84-130					
12 years	WISC-R	92	54	100.23 (*14.38*)	47-139	100.47 (*13.56*)	985	.832	.688	0.29
	WISC-III	14	12	101.81 (*7.52*)	86-117					
15 years	WISC-R	309	178	100.04 (*13.79*)	63-141	100.18 (*13.82*)	622	.880	.563	0.63
	WISC-III	32	16	101.62 (*14.24*)	44-125					

Note. Unscaled scores represent the mean and standard deviation of the standardized score for a particular cognitive test at a particular age. All measures were standardized to have a mean of 100. *Because the Bayley and McCarthy Scales were standardized to have a standard deviation of 16, whereas the Stanford Binet and Wechsler scales were standardized to have a standard deviation of 15, Bayley and McCarthy scores were rescaled by subtracting 100 from individual scores, multiplying the residual by 15/16, and adding 100. Rescaled scores are an aggregation of scores across measures at a given age. Heritability (h^2) was calculated by multiplying the difference between the MZ and DZ correlations by 2. *At 3 and 9 months, the DZ twin correlation was slightly larger than the MZ correlation, resulting in a heritability estimate less than zero.

Figure 21 depicts individual trajectories of cognitive scores for a randomly selected subset of 250 participants. Although there is interindividual variability, individual trajectories appear to follow the same general pattern as trends in mean scores: deficits in infancy, improvement in scores across early childhood, and plateau around the population mean by late childhood.

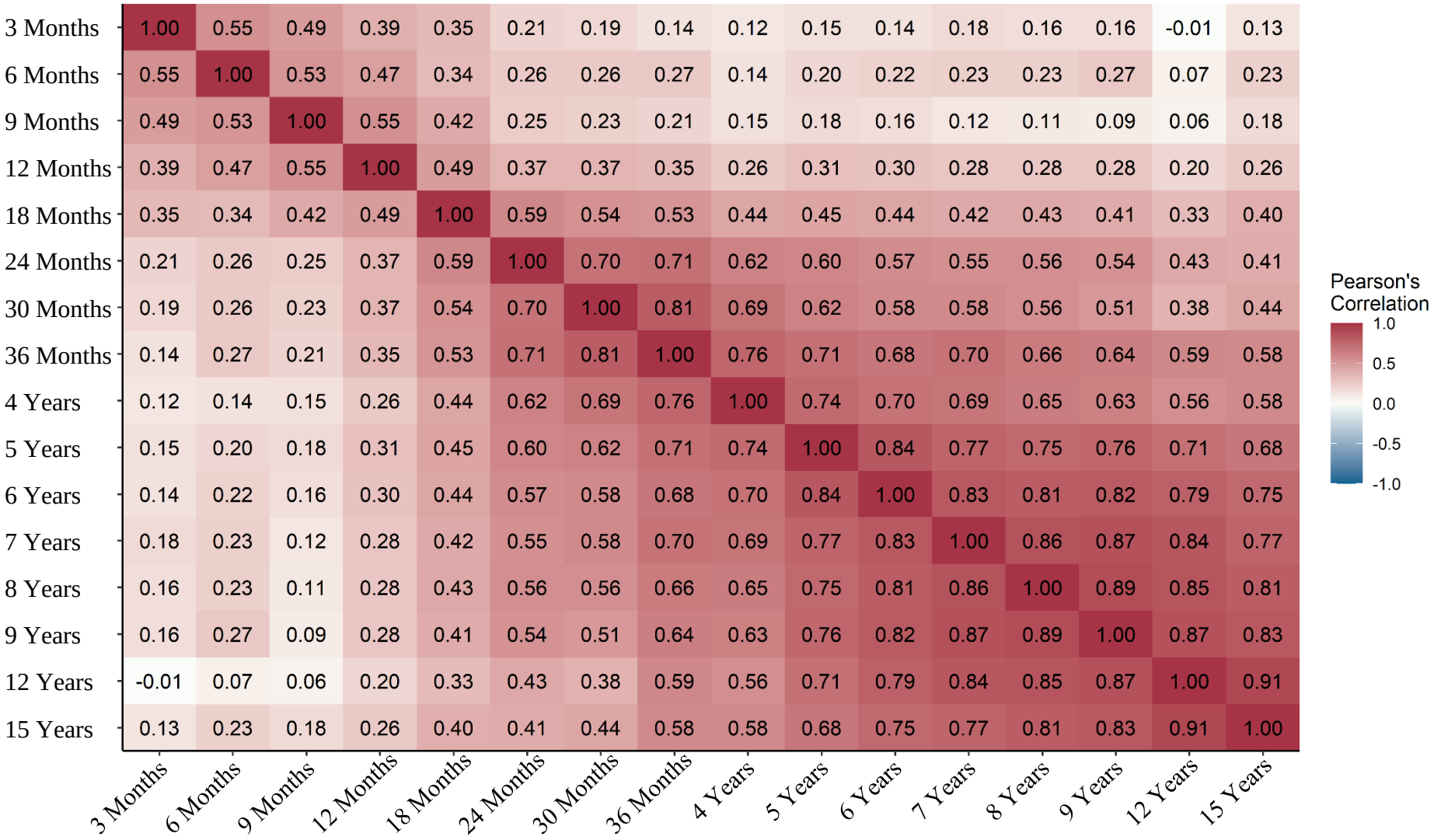
Figure 21
Individual Trajectories of Cognitive Abilities



Note. Trajectories are for a randomly selected subset of 250 study participants. The solid red line represents the population mean of 100. The solid blue line is the mean score of the randomly selected subset of participants.

Longitudinal intercorrelations between cognitive scores are presented in Figure 22. Correlations between consecutive cognitive assessments were modest in the first 24 months (*r*s = .49-.59) and were strong after 24 months (*r*s = .70-.91), indicating a general trend toward stabilizing cognitive ability across childhood. Cognitive ability in the first year was positively, but weakly correlated with cognitive ability in middle-childhood and adolescence.

Figure 22
Longitudinal Intercorrelations between Cognitive Measures



Model Identification: Growth of Cognitive Ability

Model fit information can be found in Table 21. The polynomial and exponential models had TLI values below 0.9, high RMSEA values, and higher AIC and BIC statistics than the S-shaped functions, indicating that growth in cognitive ability in twins was best characterized by relatively slow early growth, a steep incline, and a rapid stabilization. Due to convergence issues with the Richards model, the variances of the lower asymptote, rate of growth, and inflection point scaling parameter had to be set to 0. Consequently, the Richards model demonstrated higher AIC and BIC scores than the logistic and Gompertz models, indicating worse fit. The Gompertz model had lower AIC and BIC values than the logistic model, and was ultimately retained as the best fitting model.

Table 21
Model Fit Statistics

Fit Statistic	Linear	Quadratic	Cubic	Exponential	Weibull	Morgan-Mercer-Flodin	Logistic	Gompertz	Richards
X^2/df	2,247/131	1,241/127	907/122	1,137/140	884/122	897/122	857/122	818/122	1103/128
TLI	.72	.85	.89	.88	.89	.89	.893	.899	.865
RMSEA	.12	.09	.08	.08	.07	.07	.072	.070	.081
AIC	72,303	71,134	70,740	70,999	70,696	70,722	70,675	70,634	70,930
BIC	77,408	71,260	70,891	71,059	70,847	70,873	70,827	70,786	71,051

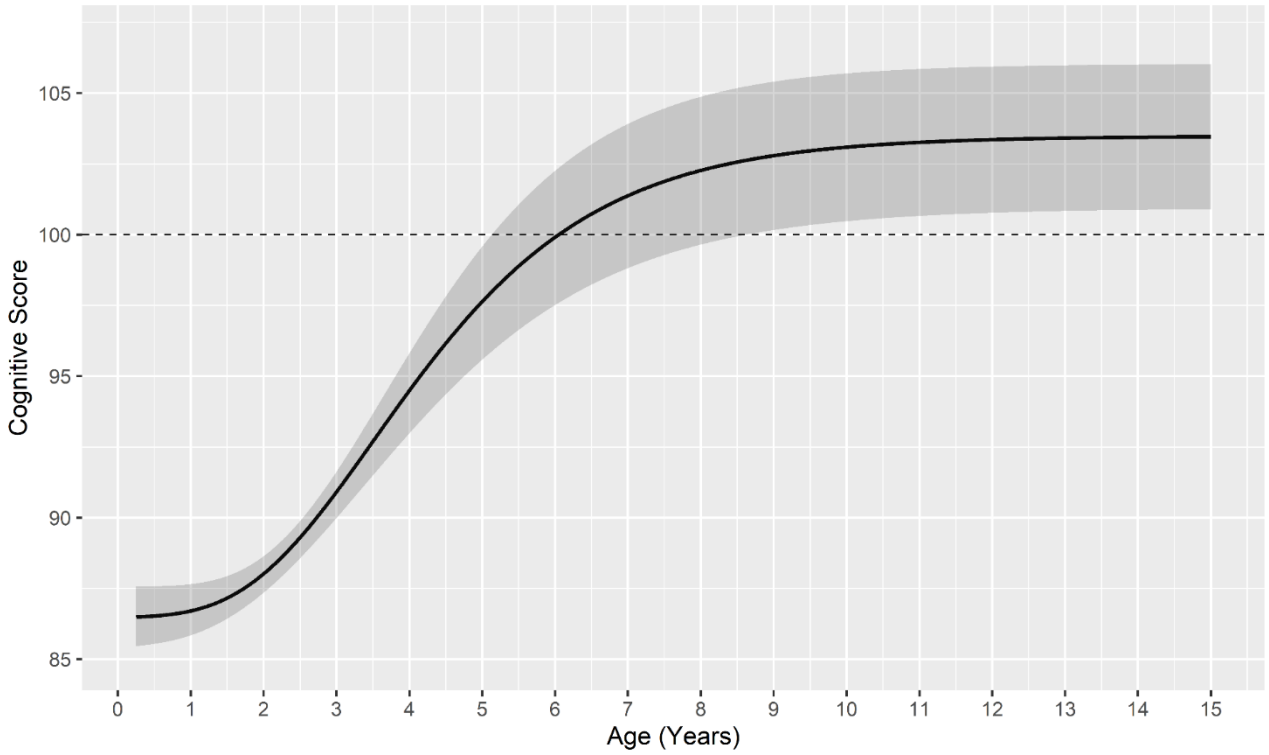
Parameter estimates for the final Gompertz model are presented in Table 22. The average twin had a lower asymptote equal to 86.5 points and grew an average of 17.0 points to an average upper asymptote of 103.5 points. The average rate of approach was 0.6 points per year, and the inflection point was at 3.3 years. There was significant between-pair variability on all of the growth parameters. Within pairs, MZ twins did not vary significantly on any of the growth parameters, and DZ twins varied only on their total change. The average growth in cognitive ability is depicted in Figure 23.

Table 22
Parameter Estimates from the Gompertz Model

Parameter	Mean [95% C.I.]	MZ Variance (Within) [95% C.I.]	MZ Variance (Between) [95% C.I.]	DZ Variance (Within) [95% C.I.]	DZ Variance (Between) [95% C.I.]
Lower Asymptote (b_0)	**86.47** **[85.38, 87.56]**	1.85 [-2.15, 5.86]	**119.01** **[93.44, 140.48]**	8.45 [-0.67, 17.58]	**112.41** **[85.96, 138.87]**
Growth from Lower Asymptote (b_1)	**17.01** **[15.55, 18.47]**	5.35 [-1.66, 12.36]	**245.95** **[191.17, 300.73]**	**48.97** **[24.51, 73.43]**	**202.33** **[145.65, 259.01]**
Average Rate of Change (b_2)	**0.58** **[0.53, 0.63]**	0.04 [-0.05, 0.09]	**0.52** **[0.35, 0.70]**	0.08 [-0.02, 0.17]	**0.49** **[0.31, 0.68]**
Age at Steepest Growth (b_3)	**3.26** **[3.12, 3.41]**	0.16 [-0.23, 0.54]	**10.95** **[7.48, 14.42]**	0.83 [-0.03, 1.68]	**10.28** **[6.84, 13.72]**

Note. MZ refers to MZ twins and DZ refers to DZ twins. Parameter means were constrained to be equal for MZ and DZ twins. Variances that are significantly different from 0 at $p < .05$ are presented in bold.

Figure 23
Predicted Cognitive Scores based on the Gompertz Function



Note. The dashed line at 100 corresponds to the standardized mean of the cognitive assessments included in the present study. The shaded area represents the 95% confidence interval.

Biometric Analyses

Unstandardized and standardized biometric components are presented in Table 23. Shared environmental influences (C) contributed to the majority of the variance in the lower asymptote (87.5%), total growth from the lower asymptote (63.2%), average rate of change (81.4%), and inflection point (86.5%). Additive genetic influences (A) significantly only contributed to the variance of the total growth from the lower asymptote (34.7%). The nonshared environment (E) did not contribute significantly to the variance of any growth parameters.

Table 23
Variance in Growth Parameters Due to Genetic and Environmental Components

Parameter	Additive Genetic (A) [95% C.I.]	Shared Environment (C) [95% C.I.]	Nonshared Environment (E) [95% C.I.]	Proportion A	Proportion C	Proportion E
Lower Asymptote (b_0)	13.20 [-6.72, 33.11]	**105.48 [75.10, 136.54]**	1.86 [-2.15, 5.86]	0.109	0.875	0.015
Growth from Lower Asymptote (b_1)	**87.24 [31.41, 138.08]**	**158.71 [90.03, 227.39]**	5.35 [-1.66, 12.36]	0.347	0.632	0.021
Average Rate of Change (b_2)	0.06 [-0.15, 0.27]	**0.46 [0.22, 0.71]**	0.04 [-0.01 – 0.09]	0.108	0.814	0.078
Age at Steepest Growth (b_3)	1.34 [-0.52, 3.20]	**9.61 [5.96 – 13.27]**	0.16 [-0.23 – 0.54]	0.121	0.865	0.014

Note. Proportions that are significant at the $p < .05$ level are highlighted in bold.

Environmental Correlates of Cognitive Growth

Parameter estimates for the environmental correlates of cognitive growth are presented in Table 24. There were no between-pair associations between birth weight and growth parameters, but within-pair, the heavier twin exhibited a slightly higher lower asymptote.

Table 24

Environmental Correlates of Cognitive Growth

Between-Pair Effects				
Predictor Variable	Lower Asymptote (b_0) *B* [95% C.I.]	Growth from Lower Asymptote (b_1) *B* [95% C.I.]	Average Rate of Change (b_2) *B* [95% C.I.]	Age at Steepest Growth (b_3) *B* [95% C.I.]
Birth Weight	1.77 [-0.90, 4.43]	2.37 [-1.39, 6.13]	-0.16 [-0.37, 0.05]	-0.71 [-1.58, 0.16]
Gestational Age	**5.26 [3.20, 7.32]**	**-7.38 [-10.28, -4.48]**	**0.32 [0.16, 0.48]**	**1.17 [0.50, 1.84]**
Mom Age	-0.51 [-1.75, 0.73]	1.20 [-0.54, 2.94]	-0.06 [-0.16, 0.04]	**-0.49 [-0.89, -0.09]**
Family SES	**-1.31 [-2.58, -0.05]**	**5.57 [3.78, 7.35]**	**-0.17 [-0.27, -0.07]**	**-1.00 [-1.43, -0.58]**
Household Chaos	0.67 [-1.18, 2.53]	**-3.07 [-5.42, -0.73]**	0.12 [-0.01, 0.26]	**0.82 [0.10, 1.53]**
Within-Pair Effects				
Predictor Variable	Lower Asymptote (b_0) *B* [95% C.I.]	Growth from Lower Asymptote (b_1) *B* [95% C.I.]	Average Rate of Change (b_2) *B* [95% C.I.]	Age at Steepest Growth (b_3) *B* [95% C.I.]
Birth Weight	**1.25 [0.39, 2.06]**	0.08 [-1.15, 1.31]	0.05 [-0.05, 0.14]	0.11 [-0.16, 0.38]

Note. Coefficients that are significant at the $p < .05$ level are highlighted in bold.

Children who had a gestational age 1 SD below the mean displayed a lower asymptote 5.26 points below the mean (95% C.I. 3.20, 7.32), but displayed a difference between the lower and upper asymptote of 7.38 points above the mean (95% C.I. 4.48, 10.28). Children with an earlier gestational age also displayed a slower average rate of growth and an earlier inflection point, indicating that, although initial cognitive ability fell substantially below the mean, cognitive recovery began earlier and was more consistent over time. See Figure 24a for a plot of predicted cognitive growth for a child born at normal gestational age (40 weeks) and premature (37 weeks). To further aid interpretation, Figure 25a depicts the difference in cognitive ability between a child born at 40 weeks and a child born at 37 weeks. As depicted, a child born at 37 weeks would be predicted to perform as well as a child born at 40 weeks by approximately 3.5 years of age. Maternal age was unrelated to the lower asymptote, upper asymptote, or average rate of change, but children with older mothers had a slightly earlier inflection point, which led to a slight advantage in cognitive ability in the toddler years (see Figures 24b and 25b).

Children from homes at 1 SD above the SES mean had a lower asymptote, which was 1.31 points below the mean (95% C.I. -2.58, -0.05), but gained an additional 5.57 points (95% C.I. 3.78, 7.35) at their upper asymptote than children at mean SES. Thus, by the upper asymptote, children at 1 SD above the SES mean displayed an 8.52-point advantage over children at 1 SD below the SES mean (see Figure 24c). SES was related to a lower average rate of growth, but an earlier inflection point. As depicted in Figure 24c, the cognitive ability of twins in high SES homes begin rising almost immediately with an average inflection point of 2.26 years, while abilities for twins in low

SES homes remained stable through about 2.5 years before beginning to rise with an average inflection point at 4.26 years.

Household chaos was associated with the total growth from the lower asymptote and inflection point above and beyond the effects of SES. Specifically, greater household chaos was significantly associated with a lower total growth from the lower asymptote and an older inflection point (see Figures 24d and 25d).

Figure 24
Environmental Correlates of Cognitive Development

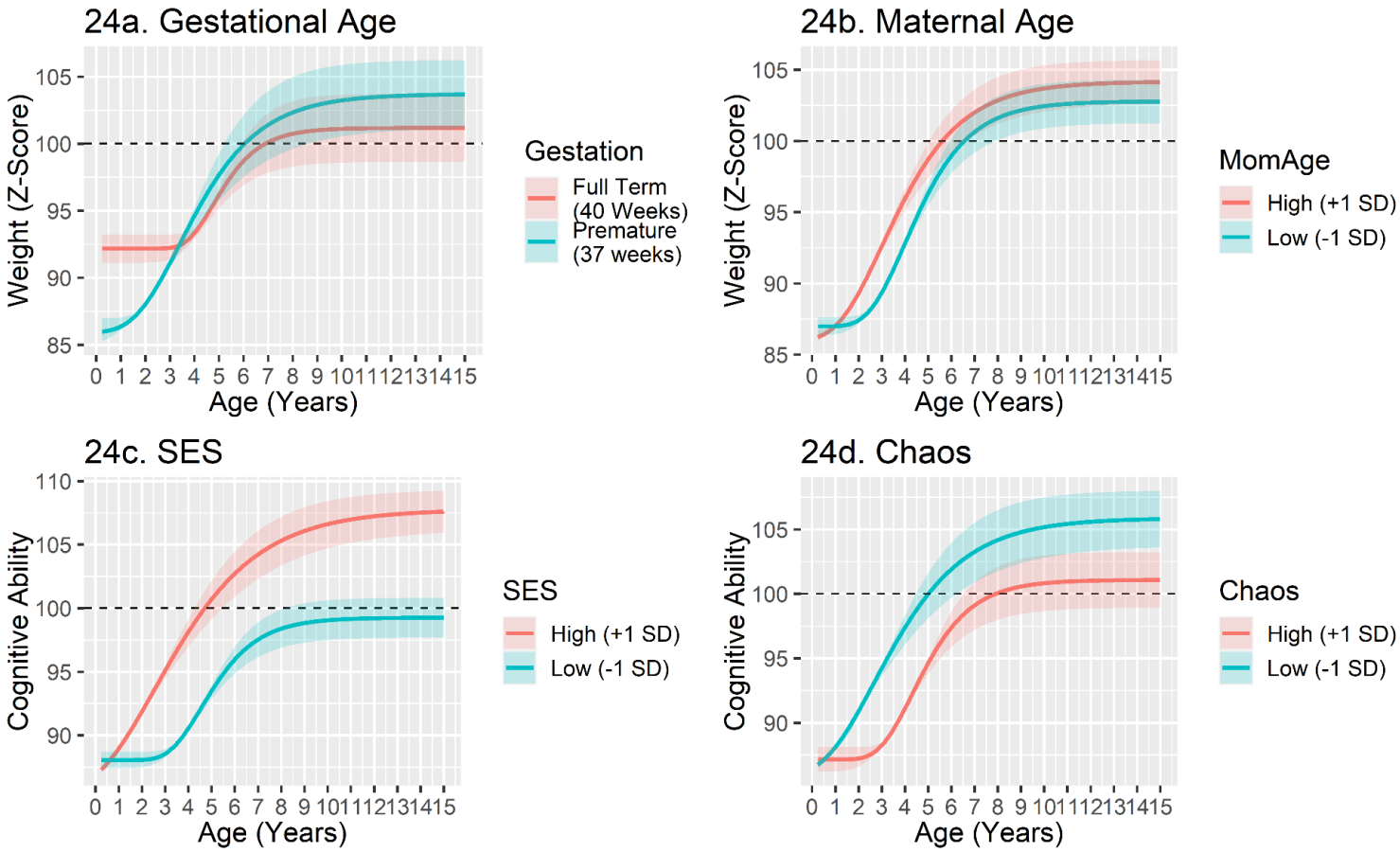
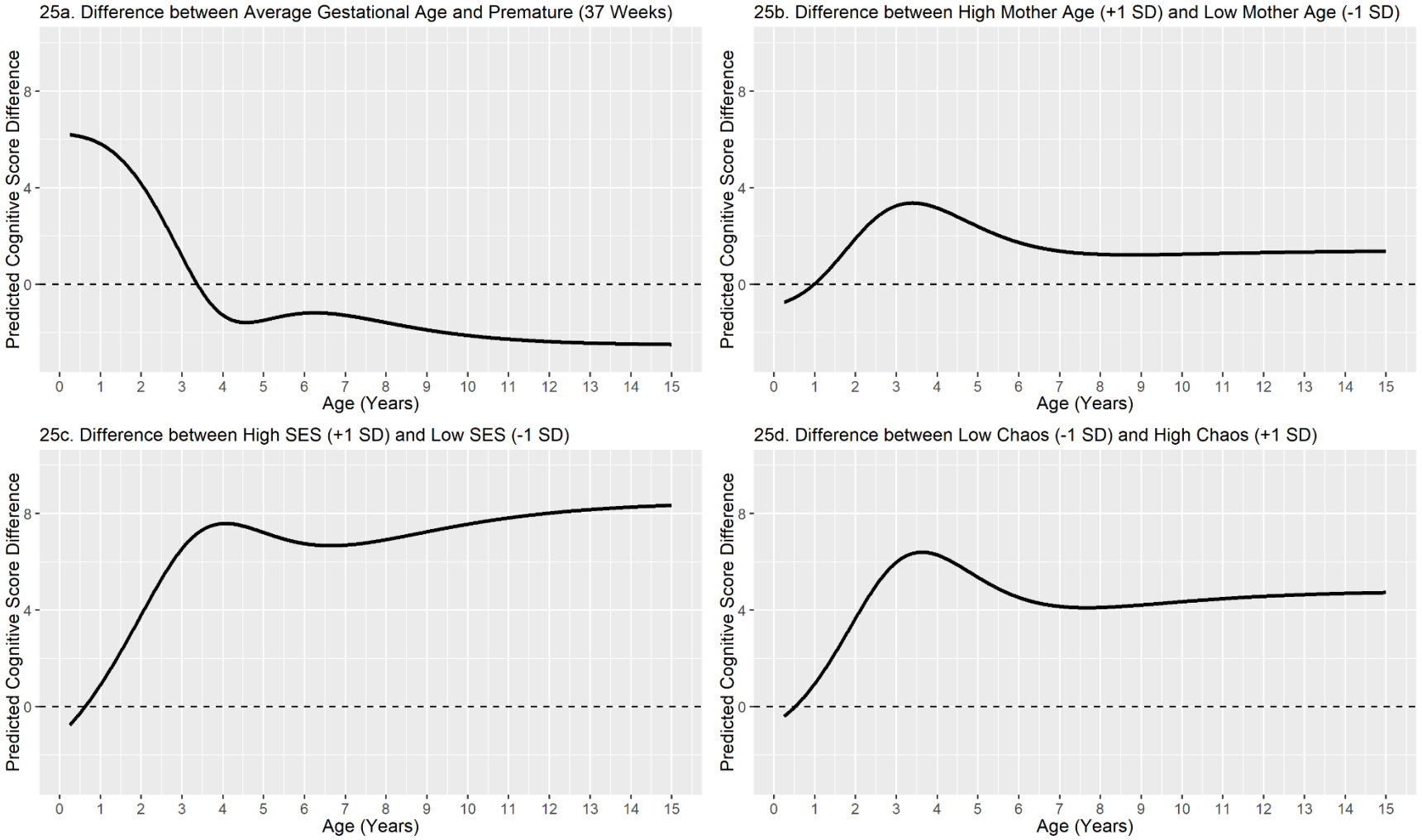


Figure 25
Stabilization of Mean Differences in Cognitive Ability by Environmental Correlate



Of particular interest was the proportion of shared environmental variance in the growth parameters accounted for by the study covariates. The change in the proportion of shared environmental variance was calculated by subtracting the unstandardized shared environment (C) variance in the constrained model from the C variance in the unconstrained model and dividing the difference by the C variance in the unconstrained model (Singer et al., 2003). The proportion of change between the unstandardized shared environment variances for the unconstrained and constrained models are presented in Table 25. The study covariates accounted for 41% of the variance in C for the lower asymptote, 54% for the total change between the lower and upper asymptotes, 22% for the average rate of growth, and 22% for the inflection point.

Table 25
Proportion of Change in Shared Environmental Variance Due to Study Covariates

Growth Parameter	Unstandardized Shared Environment Variance Unconstrained Model	Unstandardized Shared Environment Variance Constrained Model	Proportion of Change
Lower Asymptote	105.48	62.64	0.41
Change from Lower to Upper Asymptote	158.71	73.47	0.54
Average Rate of Change	0.46	0.36	0.22
Inflection Point	9.61	7.51	0.22

Discussion

Nonlinear growth curve models fit to standardized cognitive test scores from infancy to adolescence in a community sample of twins identified a pattern of growth characterized by deficits in infancy, rapid recovery through early childhood, and stabilization around the population mean from middle childhood through adolescence. Findings extend early research that identified mean differences between early cognitive scores in twins and the population mean (Wilson, 1972, Wilson, 1974) and provide information on the typical shape and rate of recovery of cognitive ability in twins.

The shape of cognitive recovery in twins was best captured by an S-shaped curve, which suggests that cognitive recovery to the population mean is minimal through infancy and increases steadily across the toddler years. The rate of growth was steepest at 3.26 years, highlighting the importance of toddlerhood in the development of cognitive ability in twins. Because cognitive tests are age-standardized, scores at a given age reflect an individual's performance relative to the population mean of individuals at that age, but do not necessarily capture the enormous gains in verbal and reasoning abilities that occur year to year across toddlerhood (O'Muircheartaigh et al., 2014). The rapid increase of the twins' standardized cognitive test scores relative to the population mean across toddlerhood suggest that the typical cognitive developmental process in twins entails the acquisition of both developmentally typical cognitive ability as well as prerequisite abilities that were not observed at previous ages. The delay in the inflection point of cognitive recovery to toddlerhood may reflect the fact that twins are frequently born prematurely and at low birth weight (Giuffrè et al., 2012), and spend the first few years of life catching up physically to their singleton peers (Estourgie-van Burk et al., 2010; Wilson, 1979; and see Study 1). Recovery in physical size may set the stage for cognitive recovery through advances in motor abilities, which allow toddler twins to explore their environment more completely. Future research is necessary to further

understand the temporal relationship between physical development, motor development, and mental development in twins.

Once reaching the population mean of 100 by approximately age 6 years, the predicted growth in cognitive ability, based on the Gompertz model, leveled off approaching an upper asymptote of 103.5. In the only other study to model the growth of standardized cognitive scores in twins across childhood, von Stumm and Plomin (2015) observed patterns of cognitive development that approached positive and negative infinity over time based on quadratic growth models. Ongoing LTS work has observed moderate correlations between cognitive ability in childhood and cognitive functioning in midlife in pilot data (Beam et al., 2020), and a central goal of ongoing work is to extend childhood trends in the growth of cognitive ability across childhood to cognitive functioning in midlife. However, polynomial models have extremely limited utility in predicting later-life outcomes from childhood data (Grimm et al., 2011). Indeed, based on the parameter estimates of the best-fitting polynomial in the present study (the cubic model), the predicted standardized cognitive score for twins in the LTS at age 40 is -608.6, whereas the asymptotic Gompertz model predicts an average standardized cognitive score of 103.5 at age 40.

Biometric analyses revealed that the majority of the variance of growth in cognitive ability across childhood was attributable to shared environmental experiences. Consistent with estimates from previous biometric analyses of cognitive scores in infants (Koeppen-Schomeerus et al., 2000; Price et al., 2000), I found that the shared environment accounted for 87.6% of the variance in the lower asymptote of cognitive ability. Shared environmental experiences also accounted for the majority of the variance in the total amount of growth, the average rate of growth, and the age of steepest growth. Of the four growth factors, additive genetics only accounted for a significant amount of the variance in the total growth from the lower asymptote, adding nuance to extant research that has found that the shared environment explains a decreasing proportion of the variance in cognitive ability at later developmental stages (Davis et al., 2009; Finkel et al., 2015). Specifically, additive genetics appear to become increasingly important in explaining the variance in cognitive ability at specific time points in late childhood and adolescence but account for a marginal portion of the variance in the developmental trajectory of cognitive ability across childhood.

Nonshared environmental factors accounted for a nonsignificant portion of the variance in all of the growth parameters. Another interpretation of the nonshared environmental factor is that it represents the extent to which MZ twins are different from each other within pairs. Nonsignificant nonshared environment coefficients indicate that, statistically, individuals in an MZ twin pair would be expected to exhibit the same pattern of growth in cognitive ability from infancy to adolescence. Previous research has found nonshared environmental factors to contribute a marginal, but statistically significant, portion of the variance in cognitive ability at different developmental stages across childhood (Davis et al., 2009; Price et al., 2000). Nonshared environmental variance tends to reflect age-specific variance, which is more strongly captured in the residual variances in a growth model. Therefore, factors that contribute to within-pair differences in cognitive ability for MZ twins (including measurement error) may be more apparent in age-specific measurements of cognitive ability rather than trends in growth. As the present study is the first to apply biometric analyses to the growth in standardized cognitive scores across childhood, replication of the present findings in other samples is warranted before concluding that MZ twins do not differ within-pair in their trajectories of cognitive development across childhood.

Examining associations between specific features of the environment and cognitive development yielded distinct patterns for prenatal and postnatal environmental experiences. Specifically, aspects of the prenatal environment were most strongly associated with early cognitive ability, whereas associations between postnatal features of the environment became increasingly apparent at later ages. With regards to gestational age, children born at full term (40 weeks) exhibited approximately a 5-point advantage in their lower asymptote relative to children born at 37 weeks (the cutoff for prematurity). However, children born more prematurely exhibited greater total growth, a slower rate of approach to the asymptote, and an earlier inflection point. As depicted in Figures 5a and 5a, the predicted trajectories of cognitive ability for twins born at 37 and 40 weeks converged at approximately 3.5 years of age.

Birth weight was not associated with any growth parameters at the between-pair level. Within-pair, the heavier twin displayed a slight advantage in their lower asymptote, but there were no significant within-pair birth weight associations with cognitive growth. Thus, results are consistent with observations by Datar and Jacknowitz (2009), as they indicate that associations between birth weight and cognitive development may be confounded by other nonshared environmental factors, in particular, prenatal environmental factors (e.g., uterine position). In the present study, sex was regressed out of all cognitive scores before models were fit and, therefore, associations with birth weight and cognitive development are independent of sex.

In the LTS, very few children were born very prematurely (defined as less than 32 weeks gestation; 2.4% of the study sample) and a minority were born at very low birth weight (defined as less than 3.31 pounds; 2.3% of the sample). Therefore, findings reflect developmental patterns for children born at typical gestation and/or birth weight or the lowest risk category of prematurity and/or low birth weight. Children born at very low birthweight or very prematurely demonstrate substantial deficits in cognitive ability which tend to persist across childhood and adolescence (Mangin et al., 2017). Therefore, the observed patterns of premature infants catching up to their full-term peers by toddlerhood and nonsignificant associations between birth weight and cognitive development may not apply to populations at greater risk based on gestational age, birth weight, or complications associated with those conditions.

Maternal age was only associated with the inflection point of cognitive ability. Children of older mothers hit their period of steepest growth earlier than children of younger mothers, contributing to a slight advantage in cognitive ability across toddlerhood for children born to older mothers. In the LTS, only a handful of mothers fell above age 40 at twin birth. Therefore, the majority of mothers in the present study fell within an age range in which I would not expect elevated rates of prenatal complications due to age (Luke & Brown, 2007). That greater maternal age was associated with an earlier inflection point may suggest that maternal age reflects aspects of the rearing environment not entirely captured by SES. For example, older maternal age has been associated with more proactive parenting practices, lower levels of household instability, and a more supportive learning environment (Fergusson & Woodward, 1999; Rodriguez & Tamis-LeMonda, 2011), which may contribute to cognitive development above and beyond a family's socioeconomic resources.

Consistent with previous research (Kainz et al., 2012; Tucker-Drob et al., 2011), higher family SES was associated with more positive cognitive developmental outcomes. Specifically, twins reared in higher-SES families exhibited an earlier inflection point and a slower average rate of growth, indicating that growth in cognitive ability relative to the population mean began at an earlier age and continued for a longer period of time. Children in families one SD above the sample SES mean were growing most rapidly at 2.26 years of age, whereas children in families one SD below the sample SES mean were growing most rapidly at 4.26 years of age. In other words, the period of most rapid cognitive growth for children in poorer homes appears to correspond with the beginning of public schooling, whereas children in more affluent homes develop most rapidly *before* entry into preschool. The SES-based divergence observed in early cognitive development trajectories appears to stabilize in early childhood at approximately an eight-point difference between children at one SD above and below the SES mean.

Household chaos was also associated with the growth in cognitive ability across childhood such that children reared in more chaotic households displayed less total growth in their cognitive ability and exhibited a later inflection point. In the present study, all covariates were entered into the growth model simultaneously indicating that household chaos was associated with cognitive development after accounting for SES. Building on extant research (Deater-Deckard et al., 2009; Martin et al., 2012), findings from the present study suggest that household organization, structure, and routine represent important aspects of a child's learning environment distinct from SES in the growth of cognitive ability.

Limitations

The findings from the present study must be considered in light of several limitations. First, a variety of instruments were used to assess cognitive ability across the developmental span from infancy to adolescence. Additionally, within each age, different cognitive assessment versions were used throughout the duration of the LTS as new test versions became available (e.g., cognitive ability across childhood were assessed using the WISC, WISC-R, or WISC-III depending on the most recently published test). My approach to aggregate cognitive assessments across tests and versions within age and to measure the change in the aggregated scores across time relied on the assumption that the same "cognitive ability" construct was captured with each assessment. Different cognitive assessments are comprised of very different subtests prohibiting us from testing measurement invariance across test versions or test types. Different test versions are generally highly correlated (Bayley, 1993; Wechsler, 1989; Wechsler, 1991), and correlations between different measures over time are moderate to high (Bode et al., 2014; Ramey et al., 1973; Yule et al., 1982), supporting my decision to aggregate across test version and different measures. Additionally, I re-ran analyses restricting the assessments to the primary assessment used at each age (i.e., the Bayley 1st edition for 3-24 months, the Stanford Binet at 30 and 36 months, the WPPSI at 4-6 years, and the WISC-R at 7-15 years) to reduce assumptions made by aggregating across different tests or test versions at each age. Parameter estimates for the restricted model as well as associations between covariates and growth parameters were essentially unchanged, increasing confidence in my approach of aggregating across versions and measuring within age to maximize the sample size.

Intercorrelations between the aggregated cognitive scores in the present study were moderate to strong with the exception of scores within the first year, which were modestly but positively

associated with cognitive ability in childhood and adolescence. Moreover, correlations between cognitive scores over time in the present study were stronger than those observed in previous longitudinal studies of cognitive development (Honzik et al., 1948; von Stumm & Plomin, 2015). Thus, there appears to be some meaningful overlap in the abilities assessed at each age.

A second limitation of the present study was the limited racial diversity in the study sample. The LTS sample is predominantly White, limiting my ability to generalize findings to Black, Latinx, or Asian populations. Therefore, the extent to which similar patterns of cognitive recovery observed in White twins play out in other racial and ethnic groups is unclear. As the results from the present study highlight the importance of the shared environment in cognitive development in twins, racial inequities in access to healthcare, socioeconomic resources, and community resources may exacerbate initial delays in cognitive ability or slow the recovery process. Additionally, there has been little research to date on patterns of cognitive development in more affluent Black and Latinx families, highlighting a critical avenue for future research needed to understand the role of racism and poverty in cognitive development.

Finally, due to the constructs available in the LTS, I had limited access to measures of specific features of the environment and, consequently, the environmental correlates in the present study represent broad generalizations about the child's environment. For example, birth weight and gestational age were used as a proxy for prenatal environmental experiences. However, more specific measures, such as prenatal exposure to heavy metals, substances, or maternal medical complications may provide a finer-grained picture as to specific prenatal stressors associated with cognitive development. Relatedly, family SES and household chaos were included as time-invariant covariates due to sporadic measurement of these constructs over time. Specifically, family SES was measured at birth and does not capture potential upward or downward socioeconomic mobility over time. Likewise, household chaos was measured by aggregating scores across all study waves as most families had at least one chaos score, but few families had multiple scores. Additionally, some twins may have been exposed to interventions designed to bolster neurodevelopment (e.g., Early Intervention), which could contribute to the recovery of cognitive ability. As the mean cognitive ability in the community-based Louisville Twin sample reached the average range by early childhood, the observed trend of deficits in infant cognitive ability and recovery to the population mean by early childhood likely reflects typical cognitive development in twins. However, without access to data indicating whether twins in the LTS received any cognitive intervention, it is impossible to definitively disentangle typical developmental processes from environmental experiences. As higher SES was associated with an earlier inflection point and greater overall cognitive recovery across childhood, it is likely that more targeted interventions designed to bolster cognitive development would be beneficial for cognitive recovery.

The broad environmental constructs used in the present study likely represent an underestimation of the strength of the association between prenatal and postnatal environmental experiences. Replication of the study findings using more precise environmental measures is warranted. Specifically, environmental covariates may be best represented as time-varying to capture the dynamic nature of early family environments. Additionally, future research may consider other aspects of the environment, such as organization and learning stimulation in the home

environment, exposure to early neurodevelopmental interventions, and the quality of early childcare and educational placements.

Conclusions

Typical cognitive development in twins relative to the population mean followed an S-shaped curve characterized by substantial deficits in infancy, rapid recovery in abilities across toddlerhood, and stabilization of scores around the population mean in early childhood. Twins can serve as a model for singleton development and, therefore, the results from the present study may provide some insight into the nonlinear recovery of cognitive ability for singletons born at bio-environmental risk (e.g., anoxic, prematurely, at low birth weight). The pattern of cognitive recovery observed in the present study has implications for clinicians conducting assessments of neurodevelopmental disorders in infant twins. Specifically, deficits that would typically be suggestive of a developmental delay in singletons (i.e., scores >2 SD below the population mean) may be more normative in infant twins relative to singletons. As twins were found to exhibit rapid recovery of abilities across the toddler years, a pattern of continued deficits below the population mean persisting into early childhood may be indicative of a neurodevelopmental disorder. Biometric analyses indicate that the majority of the variance in the growth of standardized cognitive scores is attributable to shared environmental factors. Specifically, length of gestation appears to be strongly associated with patterns of cognitive development across infancy, but associations were less apparent by early childhood. Alternatively, SES and household chaos were most strongly associated with cognitive development across early childhood.

Study 3:
Co-Recovery of Physical Size and Cognitive Ability across Childhood: A Study of Twins

Relative to singletons, twins are at an elevated risk to be exposed to a host of stressors prenatally including competition for nutrients, uterine size constraints, and an increased risk for maternal health complications (Blickstein, 2004; Goldenberg et al., 2008; Van Baal & Boomsma, 1998). Twins are about six times more likely than singletons to be born prematurely (Giuffrè et al., 2012) and, consequently, are substantially undersized in infancy (Estourgie-van Burk et al., 2010; Wilson, 1974b) and perform poorly on tests of early cognitive development (Datar & Jacknowitz; 2009; Wilson, 1972). Throughout infancy and toddlerhood, twins demonstrate substantial recovery in physical size (Wilson, 1979) and cognitive ability (Datar and Jacknowitz; 2009; Wilson, 1972). By early childhood, twins are average both physically (Wilson, 1979) and cognitively (Wilson, 1974). Previous research has found physical recovery to follow a roughly exponential trajectory with twins making rapid gains in their physical size toward the population mean across the first year (see Study 1; Wilson, 1979). Cognitive recovery in twins follows an s-shaped trajectory characterized by limited gains in infancy, rapid development across toddlerhood, and an asymptote at the population mean in early childhood (Womack et al., 2022).

Physical size in infancy, toddlerhood, and early childhood is generally considered to be an indicator of healthy development, and measurements of height, weight, and head circumference are aspects of routine well-child check-ups over the first years of life. Children who fall far behind population norms for height, weight, and head circumference are at risk for poor neurodevelopment including poor cognitive performance and gross and fine motor deficits (Cooke, 2006). Among young children who are substantially below population norms for physical size, catch-up growth in height, weight, and head circumference is associated with gains in cognitive ability (Ghods et al., 2011; Scharf et al., 2016). However, no research has examined associations between the latent rate and shape of physical recovery and the latent rate and shape of cognitive recovery. Therefore, it is unclear the extent to which the processes of early physical and cognitive development are related to one another.

Study three explored the relationship between physical and cognitive recovery. I considered two models which may describe the pattern of growth between physical and cognitive recovery: parallel process growth curve models and dual change score models. Parallel process growth curve models explore the extent to which aspects of physical recovery (e.g., rate of height recovery) are associated with aspects of cognitive recovery (e.g., rate of cognitive recovery). Dual change score models incorporate parallel growth as well as dynamic associations between physical and cognitive recovery. The dynamic associations in a dual change score framework allows for feedback between physical and cognitive recovery and can inform our understanding of how deviations from the mean trajectory of physical growth (e.g., growth spurts) relate to changes in the rate of cognitive recovery.

Understanding the strength of the relationship between physical and cognitive recovery is relevant for clinical practice and may be informative to clinicians prescribing interventions for children exposed to early bioenvironmental adversity. For example, a strong relationship between early physical recovery and cognitive recovery may indicate that interventions designed to promote physical growth may also promote early cognitive development. On the other hand, findings that physical and cognitive recovery are unrelated would suggest that these processes are unrelated and separate interventions may be necessary to promote physical and cognitive recovery.

Associations between Birth Size and Cognitive Ability. Extant research on associations between birth weight and cognitive ability has yielded discrepant findings. Children born small relative to population norms generally display lower cognitive abilities in infancy (Kohlhauser et al., 2000; Scharf et al., 2016) and childhood (Antoniou et al., 2013; Edmonds et al., 2010; Ross et al., 2012). Additionally, among children born at 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). However, some research on discordant birth weight monozygotic twins has found limited evidence of marked cognitive impairment in the smaller twin relative to their larger co-twin (Datar & Jacknowitz, 2009; Fujikura & Froehlich, 1974; Swamy et al., 2018; Wilson, 1979). Null findings from discordant birth weight monozygotic twins may reflect the importance of examining physical size relative to the population mean; intra-pair differences in physical size may not necessarily correspond with differences in cognitive performance if both twins are significantly above or below the population mean for birth weight. Accordingly, results from two meta-analyses revealed that infants categorized as low birth weight scored approximately 5-6 points lower than typical birth weight infants on standardized cognitive assessments in childhood (Aylward et al., 1989; Upadhyay et al., 2019). Thus, low birth weight appears to elevate the risk for slight reductions in cognitive performance in childhood, on average.

Less work has focused on birth *length* and cognitive development despite research suggesting a modest correlation between height and cognitive ability at later developmental stages (Silventoinen et al., 2012; Sundet et al., 2005). Gale and colleagues (2004) found birth weight, but not birth length to significantly predict higher WISC scores at 9 years. 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 performance on the Bayley MDI at 6, 24, and 36 months (*r*'s = .17-.25) (Kuklina et al., 2006).

Catch-Up Growth and Cognitive Development. As deficits in physical size at birth relative to population norms appear to be at least modestly associated with future cognitive outcomes, an important question concerns the association between catch-up growth (i.e., growth in physical size toward the population mean) and recovery of cognitive abilities. That is, among children who are born significantly below population norms (i.e., low birth weight), does recovery in physical size correspond with a commensurate increase in performance on cognitive assessments? Previous research has used various definitions for small physical size at birth (e.g., low birth weight, small for gestational age, stunting)[4] and various definitions for catch-up growth. Findings generally suggest that children who demonstrate growth in physical size toward population norms perform better on cognitive assessments relative to children who do not catch up (Li et al., 2019; Sudfeld et al., 2015; Scharf et al., 2016). However, many questions remain unanswered. Notably, I have not been able to find a study examining associations between catch-up growth and cognitive ability in *twins*; all of the research reviewed in this section is based on samples of singletons.

[4] Low birth weight refers to a birth weight less than 2,500 grams and greater than 1,500 grams (less than 1,500 grams is characterized as very low birth weight). Small for gestational age refers to infants born below the 10th percentile for their gestational age. Children are categorized as stunted if height-for-age z-scores 2 or more standard deviations below the World Health Organization Child Growth standards median.

A large body of research on the association between catch-up growth and cognitive development has focused on children reared in low- to middle-income countries where rates of low birth weight and stunting are higher than in high-income countries (Dicker et al., 2018). Findings from low- and middle-income countries provide evidence of a positive association between catch-up of physical size and improvement in standardized cognitive ability scores (Adair et al., 2013; Fink & Rockers, 2014; Horta et al., 2017; Pongcharoen et al., 2012; Poveda et al., 2021; Scharf, 2018). As change in height and weight are highly correlated, many studies use measures of *conditional growth*[5] to disentangle the relative contribution of growth in height from growth in weight. Studies of conditional growth provide robust evidence that growth in height, particularly in the first four years of life, is positively associated with cognitive recovery (Adair et al., 2013; Horta et al., 2017; Pongcharoen et al., 2012; Poveda et al., 2021). For example, in a large prospective study of children from six low- and middle-income countries, conditional growth in height between birth and 2 years and between 2 and 4 years predicted completion from secondary school (Adair et al., 2013) and IQ in adulthood (Poveda et al., 2021). Conditional growth in weight was not associated with years of schooling or cognitive ability in adulthood (Adair et al., 2013; Poveda et al., 2021). Likewise, in a prospective study of Thai singletons, conditional growth in height (but not weight) between 4 and 12 months was associated with higher Full Scale, Verbal, and Performance IQ scores on the WISC at age 9 years (Pongcharoen et al., 2012).

Studies of associations between catch-up growth and cognitive ability in samples from high-income countries reveal a similar pattern of findings (Fattal-Valevsk et al, 2009; Heinonen et al., 2008; Scharf et al, 2016; Silventoinen et al., 2014). Among American children born at very low birth weight, children who were stunted (height for age z-score < -2) or waisted (weight for age z-score < -2) at 9 months demonstrated lower MDI scores on the Bayley at 24 months, controlling for birth length and weight as well as 9-month Bayley MDI scores (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 (defined as height-for-age or weight-for-age z-score > -2) displayed higher IQs at ages 9 and 10 years than children who did not catch up (Fattal-Valevsk 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 addition to height and weight, other anthropomorphic measurements including head circumference are important physical indicators of early neurocognitive development (Cooke, 2006; Ghods et al., 2011; Scharf et al., 2016). Head circumference is highly correlated with brain volume in young children and moderately correlated with brain volume beyond early childhood (Bartholomeusz et al., 2002). In a sample of infants born very prematurely, infants who demonstrated head circumference catch-up growth in the first year were more likely to have Bayley Mental Development Index scores in the normal range at 24 months compared to children who did not catch up (Ghods et al., 2002). Similarly, in a sample of nearly 1,000 infants born very low birth weight, Scharf and colleagues (2016) observed concurrent associations between head

[5] Conditional growth refers to the change in physical size across two or more developmental periods and is calculated by regressing out previous physical measurements. For example, conditional growth in height between birth and 2 years would be calculated by storing the residuals from a model where height at age 2 is regressed on height at birth. Thus, any associations between conditional height at age 2 and cognitive ability are independent of height at birth.

circumference and Bayley mental and motor scores at ages 9 and 24 months. Additionally, head circumference z-scores at 9 months positively predicted mental development scores at 24 months.

In terms of developmental timing, earlier catch-up growth appears to be the strongest 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 a steeper recovery performed better. 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). The most 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 0.24 SD increase in cognitive ability whereas a one SD increase in height-for-age z-score after 2 years was associated with a 0.09 SD increase in cognitive ability (Sudfeld et al., 2015).

Dampened physical recovery during the first 24 months may correspond with poor neurological development during a developmental period of expansive brain growth (Johnson, 2001). It is possible that early catch-up growth in physical size coincides with catch-up in neurological development (e.g., cortical thickening, increasing gray matter). However, the neurological processes underlying physical and cognitive development in children exposed to early bio-environmental adversity remain unclear (Khan et al., 2017).

Motor development represents another potential mechanism by which early recovery in physical size may relate to cognitive development (Jeng et al., 2000; Sudfeld et al., 2015). Results from a large meta-analysis indicate a moderate association between height-for-age z-score and performance on motor assessments (r = .24) and a greater likelihood of walking before age 2. In addition to having poorer motor abilities, undersized children have been observed to be less active than typically sized children (Gardner et al., 1990) suggesting that undersized children may be less able to use the motor abilities they have developed to explore their environments. Rapid gains toward population norms in terms of length and weight over the first two years of life may afford children increased opportunities to interact with others and explore their environments, increasing early cognitive stimulation.

Biometric Contributions to the Association between Physical Size and Cognitive Ability. Phenotypically, height and IQ have been found to correlate modestly (r's = .1 to .2; Keller et al., 2013; Silventoinen et al., 2012; Sundet et al., 2005). Leveraging twin studies, phenotypic correlations can be decomposed into additive genetic, shared environmental, and nonshared components in a similar fashion as variances for a single variable (see Figure 26 for a sample path diagram). The handful of twin studies that have tested genetic and environmental correlations between height and cognitive ability have not come to a consensus on whether genetic or environmental factors account for the overlap between height and cognitive ability. Antoniou and colleagues (2013) found that *non-shared* environmental factors accounted for most of the covariance between birth weight and verbal and performance IQ (but not FSIQ) in late childhood. Alternatively, studies of adolescents have found additive genetics to account for the majority of the correlation between height and cognitive ability (Silventoinen et al., 2006; Silventoinen et al.,

2012) and head circumference and cognitive ability (Silventoinen et al., 2012). In the only study to examine associations between *change* in height and cognitive ability, Silventoinen and colleagues (2006) found additive genetics to account for the correlation between change in height between 16 and 18 years and cognitive ability at 18 years among Dutch males.

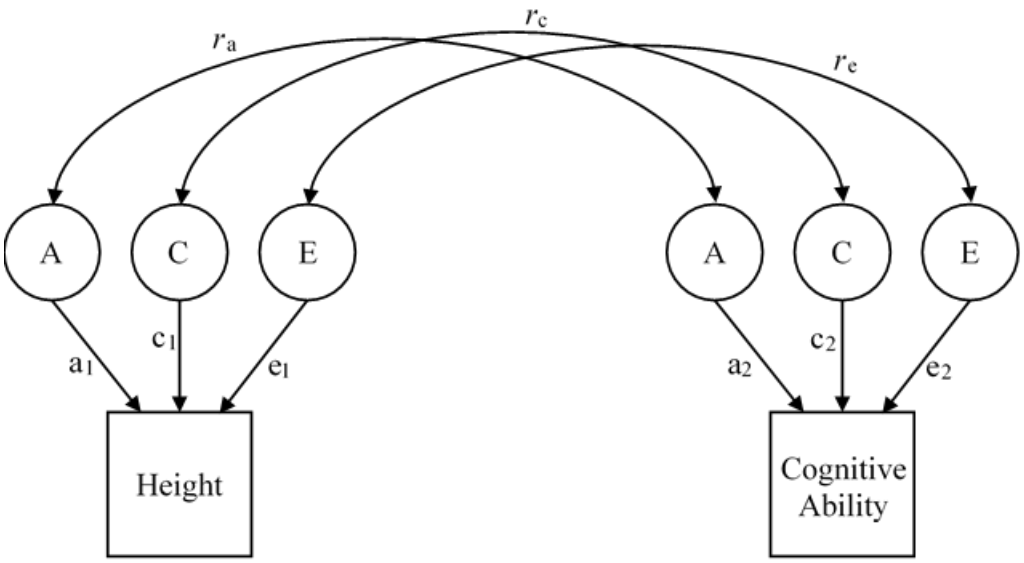


Figure 26. Path diagram for a bivariate correlated factors model where a, c, and e represent the loadings for the additive genetic, shared environmental, and nonshared environmental correlations, respectively. For simplicity, the diagram is shown for only one twin.

Other researchers have noted the relative importance of the *shared environment* in the association between height and cognitive ability. Shared environmental factors have been found to account for between 59% and 100% of the correlation between height and educational attainment whereas shared genetic factors did not account for a significant proportion of the association (Silventoinen et al., 2000a; Silventoinen et al., 2000b). Likewise, among adult Norwegian military conscripts, the shared environment accounted for most of the phenotypic correlation between height and cognitive ability (59%; Sundet et al., 2005). Genetic factors accounted for 35% of the correlation and nonshared environmental factors accounted for 6% of the correlation. Similarly, in a large sample of adult Swedish twins, shared environmental factors accounted for the majority of the phenotypic correlation between height and intelligence (59%) whereas additive genetics accounted for a modest proportion of the correlation (31%) (Beauchamp et al., 2011).

Some researchers have raised concerns that the failure to account for assortative mating[6] in classic twin models inflates shared environmental correlations between height and cognitive ability (Beauchamp et al., 2011; Keller et al., 2013). Once accounting for assortative mating in the association between height and cognitive ability, *additive genetics* explained the majority of the

[6] Assortative mating, which is assumed to be zero in the classic twin model, occurs when phenotypically similar individuals mate at a rate greater than chance (e.g., taller males mate with taller females or, by extension, taller males mate with more intelligent females).

correlation whereas the shared and nonshared environmental correlations between height and cognitive ability were nonsignificant (Beauchamp et al., 2011; Keller et al., 2013).

In sum, there is little consensus on the relative role of additive genetic, shared environmental, or nonshared environmental factors in the correlation between height and cognitive ability. Samples recruited before 1915 (e.g., Beauchamp et al., 2011; Sundet et al., 2005) generally suggest 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 (1957-1993) generally overlaps with that of studies that have found a stronger additive genetic correlation between height and weight 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 recovery.

Gaps in the Literature on Associations between Physical Size and Cognitive Ability. Despite the large body of literature dedicated to examining associations between physical size and cognitive ability, major gaps exist. That I am aware of, no studies have examined associations between the *rate of growth* in physical size and the *rate of growth* in cognitive ability. Considering that twins typically develop from initial deficits in physical size (Wilson, 1979) and cognitive ability (Wilson, 1974a), it is important to understand the relationship between recovery of physical size and recovery of cognitive ability as physical size is often used by healthcare providers as a broad indicator of developmental health. The extent to which recovery of physical size and cognitive ability are associated across childhood 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.

A related limitation of previous research on the association between physical size and cognitive ability has to do with temporal associations between growth in physical size and growth in cognitive ability. Studies of associations between catch-up growth and cognitive ability generally include measures of cognitive ability *after* measures of catch-up growth (e.g., Poveda et al., 2021; Sudfeld et al., 2015; Varella & Moss, 2015), suggesting a general hypothesis in the literature that physical catch-up precedes cognitive catch-up. However, it is possible that catch-up growth in physical size and cognitive ability occur in parallel or that cognitive recovery precedes physical recovery. Understanding the *temporal* relationship between the recovery of physical size and recovery of cognitive ability is the first step in better understanding the mechanisms that underly physical and cognitive recovery from perinatal stressors.

To date, there has been no research dedicated to understanding the biometric associations between *weight* and *cognitive ability*. As recovery of weight from early deficits has been positively associated with cognitive outcomes (e.g., Scharf et al., 2016), understanding the relative genetic and environmental correlations between catch-up weight gain and cognitive development is a first step in understanding the etiology of the association between weight recovery gain and cognitive

ability. Likewise, there has been little research on biometric associations between head circumference in infancy and early cognitive development. In the only study approximating a biometric analysis of the association between infant head circumference and cognitive development, Fujisawa and colleagues (2012) observed a negative additive genetic correlation and a positive shared environmental correlation between the rate of head circumference growth in the first year and sociocognitive development at age 2, as measured by an autism screener.

Additionally, no previous research has examined relative genetic and environmental contributions to the association between the *growth in cognitive ability* and *growth in physical size*. Understanding the genetic and environmental overlap between recovery in physical size and recovery of cognitive abilities will inform healthcare providers of the extent to which genetic or environmental factors are shared between the recovery of physical size and cognitive ability in twins.

The Present Study

To fit gaps in the literature on the co-recovery of physical size and cognitive ability, I fit two types of growth models to explore the association between physical recovery (i.e., height, weight, and head circumference) and cognitive recovery. I first fit parallel process growth curve models to examine associations between each physical recovery indicator (height, weight, and head circumference) and cognitive development. I then leveraged the twin-study design to examine additive genetic, shred environmental, and nonshared environmental contributions to the associations between physical and cognitive recovery. Second, I fit a series of bivariate dual change score models to explore dynamic associations between physical and cognitive recovery.

The study design and hypotheses were preregistered (https://osf.io/g2vyx/). Given the dearth of literature examining prospective associations between physical and cognitive recovery in twins, analyses were generally exploratory in nature. As previous research suggests that individuals with a smaller physical size at birth perform more poorly on early cognitive assessments (Kohlhauser et al., 2000; Upadhyay et al., 2019), I expected the intercepts of the physical size variables to be positively correlated with the lower asymptote of cognitive ability. Additionally, as some research indicates a positive correlation between height and cognitive ability in adulthood (Keller et al., 2013), I hypothesized that the upper asymptote of height would correlate positively with the upper asymptote of cognitive ability. Finally, as previous research suggests that a faster rate of catch-up growth from early physical deficits is positively associated with cognitive development in infancy and toddlerhood (Varella & Moss, 2015), I hypothesized that the rate of height, weight, and head circumference recovery would correlate positively with the rate of cognitive recovery.

Findings from studies 1 and 2 suggest that shared environmental factors account for the majority of the variance in initial cognitive and physical recovery. Additionally, it has been hypothesized that the physical and cognitive deficits twins show in early life are attributable to prenatal experiences, which is an aspect of the shared environment (Buckler & Green, 2004; Datar & Jacknowitz, 2009). Indeed, in studies 1 and 2, I observed a positive association between gestational age and initial physical measurements and cognitive abilities. Therefore, I hypothesized that overlapping shared environmental experiences would account for initial physical size (i.e., intercept of height, weight, and head circumference) and initial cognitive abilities (i.e., lower asymptote). As additive genetics become increasingly important in explaining variance in physical

and cognitive recovery in later developmental periods, I hypothesized that the same genetic factors would account for the upper asymptotes of physical size and cognitive ability. I did not have hypotheses as to whether overlapping genetic factors, environmental factors, or a combination will account for one's inflection point of physical recovery and cognitive recovery or one's rate of physical recovery and cognitive recovery. These analyses were exploratory in nature.

Methods

Participants

I used data from the Louisville Twin Study, a longitudinal study of temperament and cognitive development in twins (Davis et al., 2019; Wilson, 1983). 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.

Over the 36-year course of the Louisville Twin Study, there were 1,770 individuals (885 pairs) listed as participating at least once. Twins missing zygosity information will not be included in analyses (n = 120). Of those remaining, 1,642 had at least one physical measurement or cognitive measurement. As is typical in twin studies of physical or cognitive development, I restricted analyses to monozygotic and same-sex dizygotic twins (n = 1,292). Finally, provided my interest in typical development, I removed individuals that had a physical measurement greater than four standard deviations above or below the population mean (n = 77). The final study sample was 1,215 (52.0% 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. Physical measurement procedures are described in detail in Wilson (1979). Birth length, weight, and head circumference measurements were obtained from birth certificates. Infant weights between 3 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. Weights were then age-standardized 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.

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 3 and 24 months. After 24 months, standing height was measured using a wall-mounted metric scale. As with weight data,

height data were converted into age-standardized z-scored based on 2000 CDC growth norms (Kuczmarski, 2000).

Cognitive Development. Over the course of the Louisville Twin Study, twins were administered a variety of age-appropriate cognitive batteries. A breakdown of each measure including sample size and means is presented in Table 2. Twins were administered the Bayley Scales of Mental Development between ages 3 and 24 months, with the majority of twins completing the first edition (Bayley, 1969) and a handful of infants completing the second edition (Bayley, 1993). At 30 months, a minority of twins completed the Bayley. However, 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 restandardization (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). At age 5, twins completed 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.

All of the cognitive scales administered over the course of the Louisville Twin Study have a 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 4.1.2 (R Core Team, 2021).

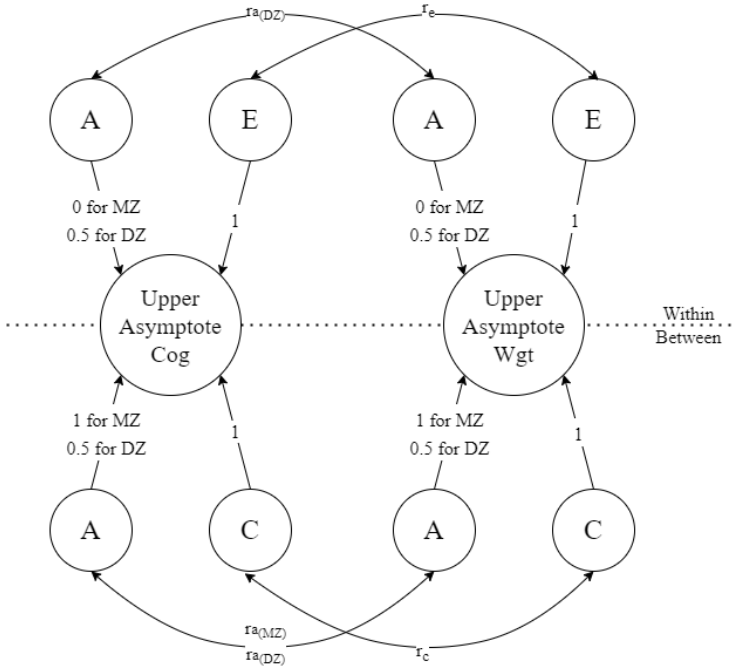
Parallel Growth in Physical and Cognitive Development. The parallel process growth curve models were guided based on the best-fitting models for height, weight, head circumference, and cognitive recovery from studies 1 and 2. Specifically, a Weibull model fit the data best for height and weight, a Morgan-Mercer-Flodin fit best for head circumference, and a Gompertz model fit best for cognitive ability. Parallel process models for each physical growth indicator (height, weight, and head circumference) and cognitive ability were fit separately. Parallel process growth curve models were fit in a multilevel framework with twins nested within families using the "twolevel" option in Mplus (Muthén & Muthén, 2017). This allowed me to calculate phenotypic correlations between the growth parameters at the within-pair and between-pair levels.

To test biometric correlations between the growth factors, I decomposed the variance of the growth parameters into additive genetic, shared environmental, and nonshared environmental components. I then included covariance paths between the biometric components (see Figure 27 for a simplified path diagram of the biometric correlations between the upper asymptote of cognitive ability and weight). Because nonshared environmental variance is only apparent within a pair, nonshared environmental covariances were included at the within-pair portion of the model.

Likewise, shared environmental covariances were included only at the between-pair portion of the model. For dizygotic twins, additive genetic covariances were included at the within-pair and between-pair portions of the model because dizygotic twins vary genetically within and between pairs. For monozygotic twins, additive genetic covariances were included at the between-pair level of the model. Additive genetic covariances for dizygotic twins were constrained to be equal across the within and between levels of the model and additive genetic covariances for monozygotic twins were constrained to be double the covariance of dizygotic twins. The covariance coefficients were standardized within Mplus and presented as correlation coefficients.

Figure 27

Path Diagram of the Biometric Correlations between Physical and Cognitive Growth Parameters



Note. $r_{a(MZ)} = 2*r_{a(DZ)}$

Examining Temporal Relationships Between Cognitive and Physical Development. I leveraged latent-change score models (McArdle, 2001) to address study questions related to the temporal relationship between the recovery of physical size and cognitive recovery. Previous research has typically specified constant growth as a linear growth parameter, but additional parameters can be entered into the model to estimate nonlinear growth (Grimm et al., 2013; Hamagami & McArdle, 2019). Factor loadings for nonlinear growth parameters in a latent change framework were derived by taking the partial derivatives of the first derivative of the target function with respect to time (Grimm et al., 2013).

A specific type of latent change score model is the dual change score model, which decomposes growth into stable, "constant" growth and proportional growth (McArdle, 2001). Proportional growth represents the deviation from constant growth over time. Positive proportional growth parameters serve to amplify the rate of growth over time whereas negative proportional growth parameters dampen the rate of growth over time. In linear dual change score models, proportional

growth adds nonlinearity to the estimated growth curve. In nonlinear models, proportional growth moderates the acceleration of growth over time (Hamagami & McArdle, 2019).

A bivariate dual change score model is a multivariate extension of the dual change score model which includes "coupling parameters" in addition to the constant and proportional growth parameters (McArdle, 2001). Coupling parameters reflect the extent to which variable X at time *t* predicts the change in growth in variable Y and time *t+1*. Applied to the current study, the coupling parameter between height and cognitive ability would reflect the extent to which a child's height at 6 months is associated with a deviation from the constant rate of growth between 6 and 9 months. Figure 28 depicts a path diagram of a linear bivariate dual change score model.

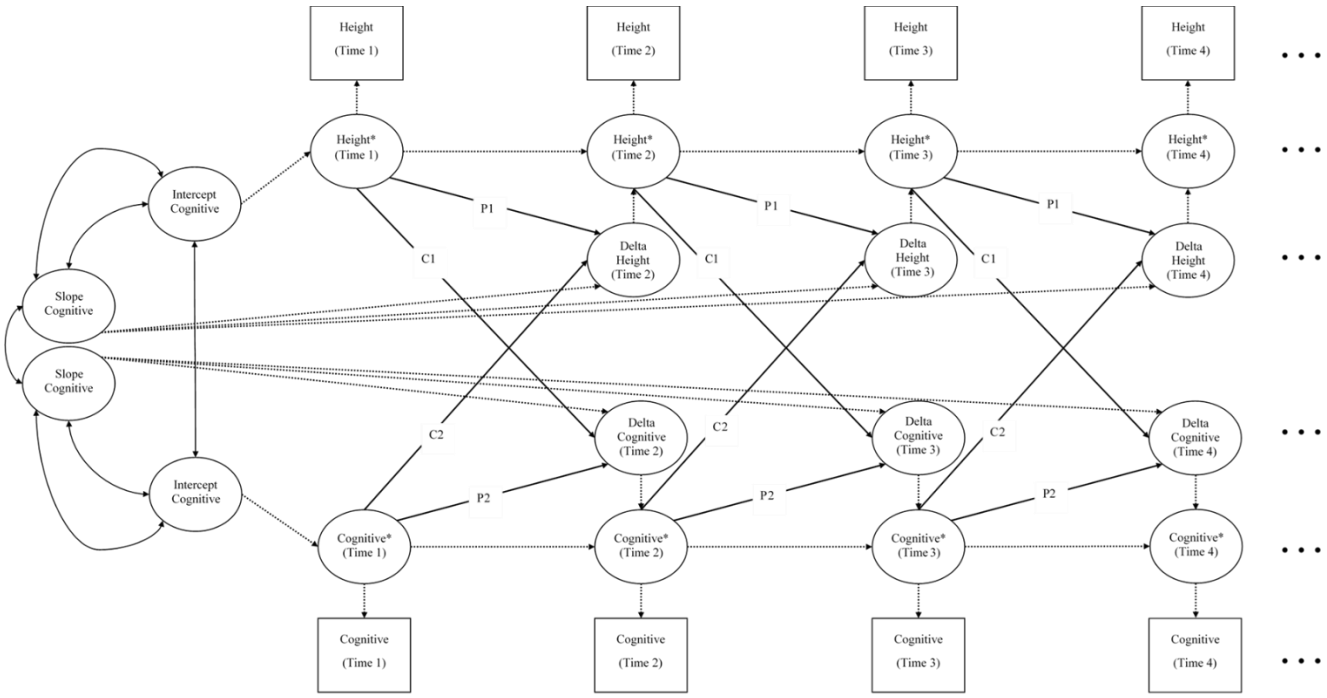


Figure 28. Generic path diagram of a linear bivariate dual change score model of height and cognitive ability. "P" refers to proportional parameters and "C" refers to coupling parameters. All solid lines are freely estimated, and dashed lines are constrained to 1.

Univariate dual change score models for each study variable (height, weight, head circumference, and cognitive ability) were fit following recommendations by Matusik and colleagues (2020). First, univariate dual change score models were fit for height, weight, head circumference, and cognitive ability. The structure of constant change was initially guided by the best-fitting growth models for each variable (i.e., Weibull for height and weight, Morgan-Mercer-Flodin for head circumference, and Gompertz for cognitive ability). However, the Weibull and Morgan-Mercer-Flodin dual change score models fit to height, weight, and head circumference did not converge. I used an exponential dual change score model to model height, weight, and head circumference. Exponential models provided an acceptable fit to the data for each growth measurement. For each variable, four models were compared: (1) dual change (i.e., constant and proportional change estimated), (2) constant change only (i.e., proportional change constrained to 0), (3) intercept only (i.e., constant change and proportional change constrained to 0), (4) no change (i.e., constant and

proportional change constrained to 0). Because models were estimated using maximum likelihood estimation with robust standard errors, Satorra-Bentler chi-square difference tests (Satorra & Bentler, 2001) were conducted comparing models 2, 3, and 4 to model 1. In cases where the Satorra-Bentler chi-square difference test resulted in a negative chi-square statistic, I calculated the strictly positive Satorra-Bentler chi-square difference statistic (Satorra & Bentler, 2010). The most parsimonious model that did not result in a significant decrease in model fit was retained as the final univariate dual change score model.

For each bivariate dual change score model four models were compared: (1) full coupling (i.e., coupling parameters from height to cognitive ability and cognitive ability to height estimated), (2) height → cognitive ability (i.e., coupling parameters from cognitive ability to height constrained to 0), (3) cognitive ability → height (i.e., coupling parameters from height to cognitive ability constrained to 0), (4) no coupling (all coupling parameters constrained to 0). Satorra-Bentler chi-square difference tests were conducted comparing models 2-4 to model 1. The most parsimonious model that did result in a significant decrease in model fit was retained as the final model.

One assumption of latent change score models is equal time intervals between measurement waves. However, in the Louisville Twin Study, waves were not equally spaced from infancy to adolescence. One strategy to deal with unequal measurement spacing in latent change score models is to include "phantom variables" to fill in waves without a measurement (Voelkle & Oud, 2014). Phantom variables are latent variables without an indicator (Rindskopf, 1984). In the Louisville Twin Study, the shortest increment of time between waves was three months. Therefore, phantom variables were included in the model to ensure constant three-month intervals between waves. Birth measurements were not included in the dual change score models to ensure that the first measurement of physical size and cognitive ability occurred at the same age (3 months).

All change score models will be fit in a structural equation modeling framework using Mplus (Muthén & Muthén, 2017).

Missing Data

Due to the longitudinal nature of the Louisville Twin Study, there were missing data on all measures. Rates of missingness for each study variable are presented in Table 26.

Table 26
Proportion of Missingness for Each Study Variable

Age	% Missing Cognitive Ability	% Missing Height	% Missing Weight	% Missing Head Circumference
Birth	[redacted]	23.1%	0.0%	57.2%
3 months	70.3%	45.1%	46.7%	45.1%
6 months	48.9%	32.2%	31.4%	32.4%
9 months	55.9%	34.3%	32.0%	34.7%
12 months	44.9%	20.4%	16.9%	20.9%
18 months	42.1%	24.6%	20.3%	25.6%
24 months	42.0%	19.9%	19.9%	26.5%
30 months	47.2%	46.6%	46.4%	50.4%
36 months	36.5%	21.2%	20.9%	27.8%

4 years	38.7%	26.2%	25.9%	[redacted]
5 years	40.5%	27.7%	27.7%	
6 years	37.7%	30.0%	30.0%	
7 years	42.9%	41.1%	41.1%	
8 years	36.1%	42.1%	42.1%	
9 years	55.2%	46.1%	46.1%	
12 years	85.1%	83.1%	83.1%	
15 years	53.8%	57.0%	57.0%	
Average	48.6%	35.8%	34.6%	35.6%

Rubin (1976) describes three patterns that may describe missing data: missing completely at random (MCAR), missing at random (MAR), and missing not at random (MNAR). Data are MCAR when missingness is unrelated to any observed variables (e.g., other variables in the study) or unobserved variables (e.g., the missing value). In the present study, data would be MCAR if missingness on IQ scores was unrelated to study covariates (e.g., SES), IQ scores from previous assessments, and the would-be IQ score from the missing timepoint. MAR occurs when missingness is related to other observed variables, but not the missing value itself. For example, if children from lower SES households are more likely to be missing cognitive scores and lower IQs are not more likely to be missing, data are considered MAR. When missingness is related to the value of the missing variable (e.g., if children with low IQ scores did not take intelligence tests at a particular wave), data are considered MNAR. There are a variety of strategies to handle missing data when data are MCAR or MAR, however, parameter estimates are likely to be biased in cases where data are MNAR (Enders, 2011a).

To explore patterns of missingness, I then fit a series of logistic regression models predicting missing cognitive scores from background variables including socioeconomic status, maternal age at birth, gestational age, sex, zygosity, and birth year, as well as cognitive scores, height, weight, and head circumference from the previous wave. Likewise, I fit a series of logistic regression models predicting the missingness of each physical growth parameter using the aforementioned background variables as well as measurements of height, weight, head circumference, and cognitive ability from the previous wave. Including previous cognitive measurements in predicting missingness of cognitive ability and physical measurements when predicting missingness of height, weight, and head circumference allowed me to approximate MNAR. Because consecutive measurements of physical size (and cognitive ability) are highly correlated, if a value at time t predicts missingness at time $t + 1$, missingness may be related to the missing value itself.

Birth year significantly predicted missing cognitive scores at 9 out of 16 assessments, missing weights at 6 out of 16 assessments, missing heights at 7 out of 16 assessments, and missing head circumferences at 3 out of 8 assessments. In general birth year was positively associated with missingness on the physical growth measures, indicating that children born later (and enrolled in the study later) were more likely to have missing scores. Family socioeconomic status was positively associated with missing cognitive scores at 3, 7, and 9 years. Socioeconomic status was not related to missingness on any physical growth measurements. Gestational age was negatively associated with missing cognitive scores at 5 years and missing cognitive, height, and weight scores at 8 years.

There was little evidence that data were MNAR. At 3 out of 16 assessments (30 months, 9 years, and 12 years) missing cognitive scores were positively related to cognitive scores at the previous assessment. Thus, there may be a slight bias towards lower cognitively performing youth at a minority of the study waves. Missing weight at 24 months was positively related to weight at 18 months, and missing weight at 12 years was negatively related to weight at 9 years. Missing height at 24 months was negatively related to height at 18 months and missing height at 7 years was positively related to height at 6 years. Missing head circumference scores were not predicted by head circumference at a previous age. I proceeded to handle missing data using full information maximum likelihood estimation (FIML), which assumes that data are MAR.

Results

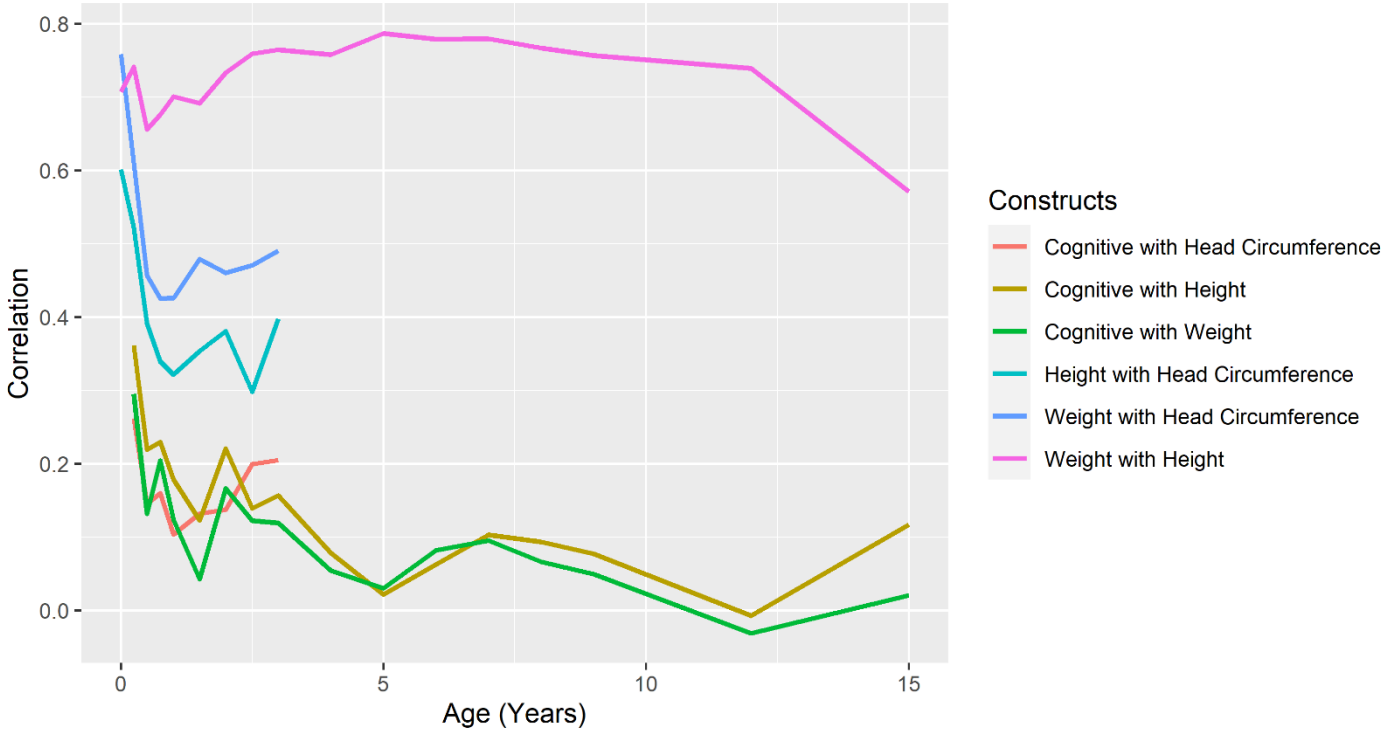
Descriptive Statistics and Intercorrelations

Detailed descriptive statics on physical size, cognitive ability scores, and study covariates are presented in studies 1 and 2.

Concurrent correlations between physical growth indicators and cognitive ability are presented in Figure 29. Weight and height were strongly concurrently correlated from infancy to adolescence (*r*'s = .57-.79). Correlations between weight and head circumference (*r* = .75) and height and head circumference (*r* = .61) were strongest at birth and remained moderate through 36 months (the final age head circumference was measured). Cognitive ability was most strongly correlated with the physical growth constructs in infancy. After 48 months, correlations between cognitive ability and height and weight remained below .1. Surprisingly, correlations between head circumference and cognitive ability were relatively weak across infancy and toddlerhood (*r*'s = .10-.26).

Figure 29

Longitudinal Correlations between Physical and Cognitive Measures



Weight and cognitive recovery. The unconditional parallel process growth curve model between weight recovery and cognitive ability had an acceptable fit to the data (X^2 = 3,922.5, df = 1,011, p < .001; TLI = .90; RMSEA = .05). Correlation coefficients between the growth parameters at the within- and between-pair level are presented in Table 27. Consistent with expectations, the intercept of weight was positively correlated with the lower asymptote of cognitive ability at both the within- and between-pair levels. Weight at birth was positively correlated with the inflection point of weight and cognitive growth, suggesting that heavier children at birth had a later inflection point. Within-pair, the rate of weight recovery was negatively correlated with the rate of cognitive recovery and positively associated with the total growth of cognitive ability. In other words, within a pair, the twin that had a faster weight recovery demonstrated a slower rate of cognitive recovery but ultimately demonstrated the largest gain in cognitive abilities.

Table 27
Phenotypic Correlations between Weight Recovery and Cognitive Recovery

	Intercept (Weight)	Upper Asymptote (Weight)	Rate (Weight)	Inflection (Weight)	Lower Asymptote (Cognitive)	Total Growth (Cognitive)	Rate (Cognitive)	Inflection (Cognitive)
Intercept (Weight)	1	**.41 [.31, .51]**	-.01 [-.13, .11]	**.16 [.03, .28]**	**.37 [.05, .69]**	-.05 [-.23, .13]	**.25 [.01, .50]**	.23 [-.08, .55]
Upper Asymptote (Weight)	.11 [-.01, .22]	1	**-.17 [-.30, -.04]**	**-.37 [-.50, -.24]**	-.29 [-.04, .62]	-.09 [-.30, .13]	**.33 [.07, .58]**	.09 [-.22, .41]
Rate (weight)	-.07 [-.20, .07]	**-.28 [-.41, -.15]**	1	.11 [-.04, .25]	-.16 [-.43, .11]	**.31 [.08, .54]**	**-.32 [-.61, -.02]**	-.22 [-.53, .09]
Inflection (Weight)	**.19 [.07, .32]**	**-.33 [-.44, -.22]**	**.14 [.01, .28]**	1	.01 [-.30, .31]	.14 [-.08, .37]	-.16 [-.47, .14]	-.02 [-.37, .34]
Lower Asymptote (Cognitive)	**.50 [.38, .63]**	**.15 [.01, .29]**	**-.17 [-.31, -.04]**	-.15 [-.30, .01]	1	.14 [-.32, .60]	.22 [-.38, .82]	0
Total Growth (Cognitive)	**-.19 [-.32, -.05]**	-.10 [-.24, .04]	.07 [-.06, .20]	**.17 [.02, .32]**	**-.64 [-.78, -.50]**	1	-.34 [-.69, .01]	-.01 [-.44, .41]
Rate (Cognitive)	.14 [-.05, .33]	.05 [-.10, .20]	-.15 [-.31, .01]	-.14 [-.33, .06]	**.41 [.16, .66]**	**-.63 [-.74, -.51]**	1	-.16 [-.86, .53]
Inflection (Cognitive)	**.13 [.00, .27]**	-.02 [-.14, .10]	**-.15 [-.28, -.03]**	-.04 [-.16, .08]	**.47 [.27, .67]**	**-.59 [-.73, -.46]**	**.62 [.40, .84]**	1

Note. 95% confidence intervals are included below the correlation coefficients. Significant correlations are bolded for clarity.

Height and cognitive recovery. The unconditional parallel process growth model between height and cognitive recovery had an acceptable fit to the data (X^2 = 3,086.8, df = 1,011, p < .001; TLI = 0.92, RMSEA = .04). Length at birth was positively correlated with the lower asymptote of cognitive abilities both within-and between-pair. The upper asymptote of height was not significantly correlated with the total change of cognitive abilities from the lower to upper asymptote. As with weight recovery, the rate of height recovery was significantly negatively

corelated with the rate of cognitive recovery within-pair. However, the confidence interval of the within-pair association between the rate of height and rate of cognitive recovery was extremely large and needs to be interpreted with caution. All correlation coefficients between height recovery and cognitive recovery are presented in Table 28.

Table 28
Phenotypic Correlations between Height Recovery and Cognitive Recovery

	Intercept (Height)	Upper Asymptote (Height)	Rate (Height)	Inflection (Height)	Lower Asymptote (Cognitive)	Total Growth (Cognitive)	Rate (Cognitive)	Inflection (Cognitive)
Intercept (Height)	1	**.53 [.44, .62]**	**-.25 [-.39, -.12]**	**.35 [.21, .49]**	**.51 [.17, .85]**	-.04 [-.25, .16]	.32 [-.03, .67]	.35 [-.01, .71]
Upper Asymptote (Height)	**.50 [.40, .59]**	1	-.10 [-.23, .04]	**-.25 [-.41, -.10]**	.10 [-.27, .46]	.08 [-.17, .32]	.11 [-.22, .44]	-.15 [-.48, .17]
Rate (Height)	**-.28 [-.39, -.17]**	-.03 [-.14, .09]	1	-.21 [-.42, .00]	-.24 [-.55, .06]	.23 [-.03, .50]	**-.52 [-.99, -.02]**	-.01 [-.37, .35]
Inflection (Height)	**.41 [.29, .52]**	**-.18 [-.30, -.07]**	**-.32 [-.45, -.20]**	1	**.43 [.02, .84]**	-.13 [-.44, .18]	.19 [-.28, .66]	**.50 [.06, .95]**
Lower Asymptote (Cognitive)	**.31 [.17, .45]**	-.07 [-.06, .20]	-.11 [-.28, .06]	.22 [.05, .38]	1	.23 [-.26, .74]	.01 [-.79, .81]	0
Total Growth (Cognitive)	-.10 [-.23, .03]	.03 [-.10, .216]	.07 [-.09, .22]	**-.18 [-.34, -.03]**	**-.59 [-.72, -.46]**	1	-.29 [-.72, .15]	.04 [-.41, .50]
Rate (Cognitive)	-.02 [-.19, .15]	-.11 [-.26, .04]	-.10 [-.29, .09]	**.23 [.02, .45]**	**.33 [.10, .55]**	**-.60 [-.72, -.49]**	1	-.33 [-.99, .58]
Inflection (Cognitive)	-.07 [-.21, .06]	-.10 [-.21, .03]	-.06 [-.20, .08]	.04 [-.11, .19]	**.37 [.19,.55]**	**-.53 [-.65, -.40]**	**.52 [.28, .76]**	1

Note. 95% confidence intervals are included below the correlation coefficients. Significant correlations are bolded for clarity.

Head circumference and cognitive recovery. As head circumference-for-age z-scores were only available from birth to 36 months, I first fit a series of growth models to determine the function that best described growth in cognitive ability between 3 and 36 months. None of the asymptotic models fit to cognitive data from 3 to 36 months converged, which is likely due to the relatively little change in cognitive ability scores over the first three years (see Figure 23, Study 2). The cubic model converged, but the variance of the cubic growth parameter as well as the covariances between the cubic growth parameter and other growth parameters had to be constrained to 0. Based on the BIC values, the quadratic growth model fit the data best (see Table 29). Moreover, chi-square difference tests revealed that the quadratic model fit better than the linear model (X^2 = 45, df = 4, p < .001) and the cubic model did not fit significantly better than the quadratic model (X^2 = 1, df = 1, p = .317).

Table 29

Model Fit Statistics – Cognitive Ability 3 to 36 Months

Fit Statistic	No Change	Linear	Quadratic	Cubic
X^2/df	680/34	160 /31	115 /27	116/26
TLI	.70	.93	.95	.95
RMSEA	.15	.07	.06	.06
AIC	36,511	35,880	35,835	35,836
BIC	36,559	35,942	35,916	35,922

The parallel process growth model of head circumference and cognitive recovery fit the data well (X^2 = 608.9, df = 232, p < .001; TLI = .96, RMSEA = .04). The intercepts of head circumference and cognitive ability were significantly positively correlated, in line with hypotheses. The slope and quadratic growth parameters of cognitive ability were not significantly correlated with any of the head circumference growth parameters. See Table 30 for all correlation coefficients.

Table 30

Phenotypic Correlations between Head Circumference Recovery and Cognitive Recovery

	Intercept (Head)	Upper Asymptote (Head)	Rate (Head)	Inflection (Head)	Intercept (Cognitive)	Slope (Cognitive)	Quadratic (Cognitive)
Intercept (Head)	1	**.48 [.34, .62]**	0	**.85 [.64, .99]**	.02 [-.40, .43]	-.15 [-.64, .35]	.20 [.20, .60]
Upper Asymptote (Head)	**.57 [.40, .74]**	1	**.30 [.06, .55]**	-.10 [-.31, .11]	.19 [-.11, .48]	-.35 [-.80, .10]	.33 [-.04, .70]
Rate (Head)	0	.05 [-.14, .24]	1	-.41 [-.65, .-.17]	.10 [-.35, .54]	-.38 [-.99, .28]	.31 [-.20, .82]
Inflection (Head)	**.26 [.03, .50]**	-.06 [-.17, .29]	.05 [-.23, .33]	1	-.08 [-.66, .50]	.41 [-.39, .99]	-.23 [-.84, .38]
Intercept (Cognitive)	**.69 [.49, .88]**	.04 [-.11, .19]	**-.23 [-.46, -.01]**	.10 [-.17, .37]	1	0	.16 [-.22, .55]
Slope (Cognitive)	-.20 [-.47, .06]	.11 [-.07, .29]	**.34 [.08, .61]**	.10 [-.21, .40]	**-.57 [-.69, -.44]**	1	**-.94 [-.99, -.82]**
Quadratic (Cognitive)	.23 [-.06, .52]	-.07 [-.28, .14]	**-.41 [-.72, -.11]**	-.10 [-.44, .24]	**.45 [.29, .63]**	**-.90 [-.94, -.86]**	1

Note. Within-pair correlations are presented above the diagonal and between-pair correlations are presented below the diagonal. 95% confidence intervals are included below the correlation coefficients. Significant correlations are bolded for clarity.

Parallel Process Growth Model – Biometric Correlations

In estimating the biometric correlations between physical recovery and cognitive recovery, numerous correlations had to be constrained to 0. This happened in cases where the estimated variance was negative and had to be constrained to 0 (as was the case for the additive genetic variance for the lower asymptote of cognitive ability). Additionally, some correlations were estimated to be greater than 1 and statistically nonsignificant (as was the case for the additive genetic correlation between the rate and inflection point of cognitive recovery). These correlations were also constrained to be 0.

Weight and cognitive recovery. There was a moderate shared environmental correlation between the intercept of weight and the lower asymptote of cognitive ability (r_c = .54), which likely reflects the common influence of gestational age and other prenatal factors on early weight and cognitive ability. The upper asymptotes of weight and cognitive ability did not share common additive genetic or shared environmental factors. However, there were common nonshared environmental factors associated with the upper asymptotes of weight and cognitive ability. There was a significant negative shared environmental correlation between the rate of weight recovery and the rate of cognitive recovery. This may reflect differences in the association between gestational age and the rate of weight and cognitive recovery. That is, children born later displayed faster cognitive recovery, but slower weight recovery.

Genetic factors associated with the upper asymptote of weight were also associated with an earlier inflection point. In exponential models, an earlier inflection point reflects more rapid early gains. Therefore, additive genetic components associated with rapid early weight gain are also associated with greater total weight gain. There was a strong nonshared environmental correlation between the intercept and upper asymptote of weight. This correlation may reflect measurement error, which is also a part of the nonshared environmental correlation. That is, children with birth weights significantly below the estimated intercept may be more likely to have an upper asymptote below the estimated upper asymptote. Additive genetic, shared environmental, and nonshared environmental correlations between weight recovery and cognitive recovery are presented in Tables 31, 32, and 33, respectively.

Table 31
Additive Genetic Correlations between Weight Recovery and Cognitive Recovery

	Intercept (Weight)	Upper Asymptote (Weight)	Rate (Weight)	Inflection (Weight)	Lower Asymptote (Cognitive)	Total Growth (Cognitive)	Rate (Cognitive)	Inflection (Cognitive)
Intercept (Weight)	1							
Upper Asymptote (Weight)	**.35 [.02, .68]**	1						
Rate (weight)	0	-.23 [-.49, .03]	1					
Inflection (Weight)	**.47 [.03, .90]**	**-.23 [-.43, -.04]**	-.11 [-.46, .23]	1				
Lower Asymptote (Cognitive)	0	0	0	0	1			
Total Growth (Cognitive)	.14 [-.29, .56]	.13 [-.06, .31]	.21 [-.15, .56]	.09 [-.20, .37]	0	1		
Rate (Cognitive)	0	0	0	0	0	0	1	
Inflection (Cognitive)	**.71 [.08, .99]**	-.20 [-.49, .09]	-.23 [-.75, .28]	.21 [-.18, .60]	0	**-.47 [-.82, -.11]**	0	1

Note. 95% confidence intervals are included below the correlation coefficients. Significant correlations are bolded for clarity. Correlations with a value of 0 were constrained to be 0.

Table 32
Shared Environmental Correlations between Weight Recovery and Cognitive Recovery

	Intercept (Weight)	Upper Asymptote (Weight)	Rate (Weight)	Inflection (Weight)	Lower Asymptote (Cognitive)	Total Growth (Cognitive)	Rate (Cognitive)	Inflection (Cognitive)
Intercept (Weight)	1							
Upper Asymptote (Weight)	.36 [-.29, .99]	1						
Rate (weight)	**-.52 [-.71, -.32]**	.03 [-.80, .85]	1					
Inflection (Weight)	**.41 [.12, .70]**	-.36 [-.92, .20]	-.29 [-.65, .06]	1				
Lower Asymptote (Cognitive)	**.53 [.36, .71]**	.38 [-.14, .90]	**-.34 [-.59, -.10]**	.16 [-.09, .41]	1			
Total Growth (Cognitive)	-.33 [-.67, .01]	-.42 [-.99, .41]	.14 [-.26, .54]	-.16 [-.51, .20]	**-.76 [-.91, -.60]**	1		
Rate (Cognitive)	.12 [-.11, .34]	-.17 [-.63, .30]	**-.35 [-.64, -.06]**	**-.35 [-.64, -.06]**	**.32 [.04, .59]**	**-.77 [-.99, -.53]**	1	
Inflection (Cognitive)	-.14 [-.43, .15]	.13 [-.45, .70]	-.19 [-.53, .14]	-.19 [-.53, .15]	**.42 [.20, .64]**	**-.60 [-.82, -.39]**	**.57 [.25, .90]**	1

Note. 95% confidence intervals are included below the correlation coefficients. Significant correlations are bolded for clarity.

Table 33
Nonshared Environmental Correlations between Weight Recovery and Cognitive Recovery

	Intercept (Weight)	Upper Asymptote (Weight)	Rate (Weight)	Inflection (Weight)	Lower Asymptote (Cognitive)	Total Growth (Cognitive)	Rate (Cognitive)	Inflection (Cognitive)
Intercept (Weight)	1							
Upper Asymptote (Weight)	**.75 [.61, .89]**	1						
Rate (weight)	**-.62 [-.77, -.47]**	**-.62 [-.78, -.47]**	1					
Inflection (Weight)	-.20 [-.81, .40]	-.24 [-.78, .30]	.55 [-.06, .99]	1				
Lower Asymptote (Cognitive)	**.48 [.09, .88]**	-.06 [-.41, .30]	.13 [-.33, .59]	.33 [-.69, .99]	1			
Total Growth (Cognitive)	-.01 [-.39, .38]	**.38 [.06, .69]**	**-.46 [-.86, -.06]**	-.59 [-.99, .26]	0	1		
Rate (Cognitive)	0	-.25 [-.99, .54]	.54 [-.82, .99]	0	-.13 [-.99, .78]	0	1	
Inflection (Cognitive)	-.03 [-.50, .44]	-.13 [-.52, .26]	.46 [-.11, .99]	.43 [-.66, .99]	**.61 [.22, .99]**	0	0	1

Note. 95% confidence intervals are included below the correlation coefficients. Significant correlations are bolded for clarity.

Height and cognitive recovery. Consistent with correlations between weight and cognitive recovery, common shared environmental factors were associated with the correlation between the intercept of height and lower asymptote of cognitive ability. There were no significant additive genetic correlations between the recovery of height and cognitive ability. As with weight, there were significant negative additive genetic correlations between the upper asymptote of height and the rate and inflection point of recovery. In other words, genetic factors associated with slower, steadier height growth were associated with being taller. Common shared environmental factors were associated with the associations between the intercept of height and the rate and inflection point of recovery. Additive genetic, shared environmental, and nonshared environmental correlations between height and cognitive recovery are presented in Tables 34, 35, and 36, respectively.

Table 34
Additive Genetic Correlations between Height Recovery and Cognitive Recovery

	Intercept (Height)	Upper Asymptote (Height)	Rate (Height)	Inflection (Height)	Lower Asymptote (Cognitive)	Total Growth (Cognitive)	Rate (Cognitive)	Inflection (Cognitive)
Intercept (Height)	1							
Upper Asymptote (Height)	**.35 [.04, .67]**	1						
Rate (Height)	0	-.23 [-.49, .02]	1					
Inflection (Height)	**.48 [.06, .90]**	**-.23 [-.42, -.03]**	-.12 [-.46, .23]	1				
Lower Asymptote (Cognitive)	0	0	0	0	1			
Total Growth (Cognitive)	.16 [-.22, .53]	.12 [-.07, .32]	.28 [-.06, .63]	-.01 [-.21, .20]	0	1		
Rate (Cognitive)	0	-.09 [-.69, .52]	-.72 [-.99, .50]	.84 [-.99, .24]	0	**-.60 [-.99, -.05]**	1	
Inflection (Cognitive)	**.66 [.05, .99]**	-.20 [-.49, .10]	-.13 [-.39, .12]	.16 [-.69, .38]	0	**-.43 [-.80, -.06]**	-.02 [-.84, .80]	1

Note. 95% confidence intervals are included below the correlation coefficients. Significant correlations are bolded for clarity.

Table 35
Shared Environmental Correlations between Height Recovery and Cognitive Recovery

	Intercept (Height)	Upper Asymptote (Height)	Rate (Height)	Inflection (Height)	Lower Asymptote (Cognitive)	Total Growth (Cognitive)	Rate (Cognitive)	Inflection (Cognitive)
Intercept (Height)	1							
Upper Asymptote (Height)	.37 [-.29, .99]	1						
Rate (Height)	**-.51 [-.71, -.32]**	.02 [-.81, .85]	1					
Inflection (Height)	**.40 [.11, .70]**	-.35 [-.91, .21]	-.30 [-.65, .06]	1				
Lower Asymptote (Cognitive)	**.53 [.35, .71]**	-.38 [-.99, .47]	**-.32 [-.56, -.08]**	.16 [-.09, .41]	1			
Total Growth (Cognitive)	-.25 [.61, .10]	-.29 [-.99, .86]	-.04 [-.53, .45]	-.13 [-.51, .26]	**-.81 [-.99, -.64]**	1		
Rate (Cognitive)	-.18 [-.68, .32]	-.29 [-.99, .86]	.15 [-.43, .73]	-.08 [-.64, .47]	.38 [-.03, .78]	**-.63 [-.97, -.28]**	1	
Inflection (Cognitive)	-.13 [-.42, .15]	.13 [-.44, .70]	-.22 [-.55, .12]	-.02 [-.27, .23]	**.41 [.19, .64]**	**-.66 [-.91, -.41]**	**.74 [.13, .99]**	1

Note. 95% confidence intervals are included below the correlation coefficients. Significant correlations are bolded for clarity.

Table 36
Nonshared Environmental Correlations between Height Recovery and Cognitive Recovery

	Intercept (Height)	Upper Asymptote (Height)	Rate (Height)	Inflection (Height)	Lower Asymptote (Cognitive)	Total Growth (Cognitive)	Rate (Cognitive)	Inflection (Cognitive)
Intercept (Height)	1							
Upper Asymptote (Height)	**.75 [.61, .89]**	1						
Rate (Height)	**-.62 [-.77, -.47]**	**-.63 [-.78, -.47]**	1					
Inflection (Height)	-.20 [-.80, .39]	-.25 [-.78, .29]	.57 [-.04, .99]	1				
Lower Asymptote (Cognitive)	**.60 [.21, .99]**	.00 [-.35, .35]	.01 [-.45, .46]	.49 [-.53, .99]	1			
Total Growth (Cognitive)	-.08 [-.48, .32]	**.34 [.02, .67]**	**-.42 [-.82, -.02]**	-.69 [-.99, .14]	0	1		
Rate (Cognitive)	0	.24 [-.99, .52]	.71 [-.87, .99]	0	-.00 [-.76, .76]	0	1	
Inflection (Cognitive)	.07 [-.43, .57]	-.10 [-.51, .31]	.37 [-.23, .97]	.61 [-.56, .99]	**.58 [.16, .99]**	0	0	1

Note. 95% confidence intervals are included below the correlation coefficients. Significant correlations are bolded for clarity.

Head circumference and cognitive recovery. There was a significant shared environmental correlation between the head circumference intercept and the intercept of cognitive ability. There were no significant additive genetic, shared environmental, or nonshared environmental correlations between the growth parameters for head circumference and the linear or quadratic growth of cognitive ability. Common additive genetic factors were associated with the rate of head circumference and the upper asymptote of head circumference. That is genetic factors related to quicker head circumference growth were related to larger head size. Additive genetic, shared environmental, and nonshared environmental correlations between head circumference recovery and cognitive recovery are presented in Tables 37, 38, and 39, respectively.

Table 37
Additive Genetic Correlations between Head Circumference Recovery and Cognitive Recovery

	Intercept (Head)	Upper Asymptote (Head)	Rate (Head)	Inflection (Head)	Intercept (Cognitive)	Slope (Cognitive)	Quadratic (Cognitive)
Intercept (Head)	1						
Upper Asymptote (Head)	0	1					
Rate (Head)	0	**.36 [.23, .50]**	1				
Inflection (Head)	0	0	0	1			
Intercept (Cognitive)	0	.07 [-.06, .21]	.11 [-.06, .27]	0	1		
Slope (Cognitive)	0	0	0	0	0	1	
Quadratic (Cognitive)	0	0	0	0	0	0	1

Note. 95% confidence intervals are included below the correlation coefficients. Significant correlations are bolded for clarity.

Table 38
Shared Environmental Correlations between Head Circumference Recovery and Cognitive Recovery

	Intercept (Head)	Upper Asymptote (Head)	Rate (Head)	Inflection (Head)	Intercept (Cognitive)	Slope (Cognitive)	Quadratic (Cognitive)
Intercept (Head)	1						
Upper Asymptote (Head)	0	1					
Rate (Head)	0	0	1				
Inflection (Head)	**.37 [.19, .56]**	0	0	1			
Intercept (Cognitive)	**.65 [.47, .83]**	0	0	.05 [-.19, .30]	1		
Slope (Cognitive)	-.04 [-.22, .14]	0	0	.11 [-.09, .33]	**-.42 [-.59, -.24]**	1	
Quadratic (Cognitive)	0	0	0	0	0	0	1

Note. 95% confidence intervals are included below the correlation coefficients. Significant correlations are bolded for clarity.

Table 39
Nonshared Environmental Correlations between Head Circumference Recovery and Cognitive Recovery

	Intercept (Head)	Upper Asymptote (Head)	Rate (Head)	Inflection (Head)	Intercept (Cognitive)	Slope (Cognitive)	Quadratic (Cognitive)
Intercept (Head)	1						
Upper Asymptote (Head)	**.50 [.33, .68]**	1					
Rate (Head)	**-.57 [-.72, -.43]**	-.08 [-.30, .14]	1				
Inflection (Head)	**.41 [.20, .62]**	-.28 [-.56, .00]	0	1			
Intercept (Cognitive)	0	0	0	0	1		
Slope (Cognitive)	-.10 [-.63, .43]	0	-.20 [-.99, .68]	0	0	1	
Quadratic (Cognitive)	0	0	0	0	0	0	1

Note. 95% confidence intervals are included below the correlation coefficients. Significant correlations are bolded for clarity.

Dynamic Growth: Weight and Cognitive Recovery

Univariate dual change score. A univariate Gompertz dual change score model fit to the cognitive ability data from 3 months to 15 years fit the data acceptably (X^2 = 586.8, df = 123, p < .001; TLI = .92; RMSEA = .06). The dual change model fit the data better than the constant change, proportional change, and no change models (see Table 40). The univariate exponential dual change score model fit to the weight data had a suboptimal fit (X^2 = 1,773.9, df = 128, p = .001; TLI = .87; RMSEA = .10). However, the dual change score model fit the data better than the constant change, proportional change, and no change models (see Table 41). Model fit was substantially improved by adding covariances between consecutive residuals (X^2 = 910.0, df = 113, p < .001; TLI = .93; RMSEA = .08).

Table 40
Model Comparison – Univariate Dual Change Score Model: Cognitive Ability

Model	X^2	df	Satorra-Bentler X^2	Δ df	p-value
Dual Change Score	586.8	123	-	-	-
Constant Growth	606.4	124	17.3	1	< .001
Proportional Growth	3,243.8	135	3087.8	8	< .001
No Growth	3181.9	136	2275.2	9	< .001

Table 41
Model Comparison – Univariate Dual Change Score Model: Weight

Model	X^2	df	Satorra-Bentler X^2	Δ df	p-value
Dual Change Score	1,773.9	128	-	-	-
Constant Growth	1,780.7	129	10.5	1	.001
Proportional Growth	7609.3	135	10,740.6	7	< .001
No Growth	7.105.4	136	2691.1	8	< .001

Bivariate dual change score. Fit statistics for the full coupling, weight to cognitive, cognitive to weight, and no coupling models are presented in Table 42. The no-coupling bivariate dual change score model did not fit significantly worse than the full coupling model. A no-coupling model is equivalent to the parallel process growth curve models fit previously and suggests that dynamic feedback between changes in weight and changes in cognitive ability are not important aspects of cognitive or weight recovery from infancy to adolescence. In other words, change in the rate of cognitive recovery from infancy to adolescence is not informed by changes in weight recovery, and vice versa. Parameter estimates for the no-coupling bivariate dual change score model between weight and cognitive recovery are presented in Table 43. The estimated trajectories of physical and cognitive recovery are depicted in Figure 30.

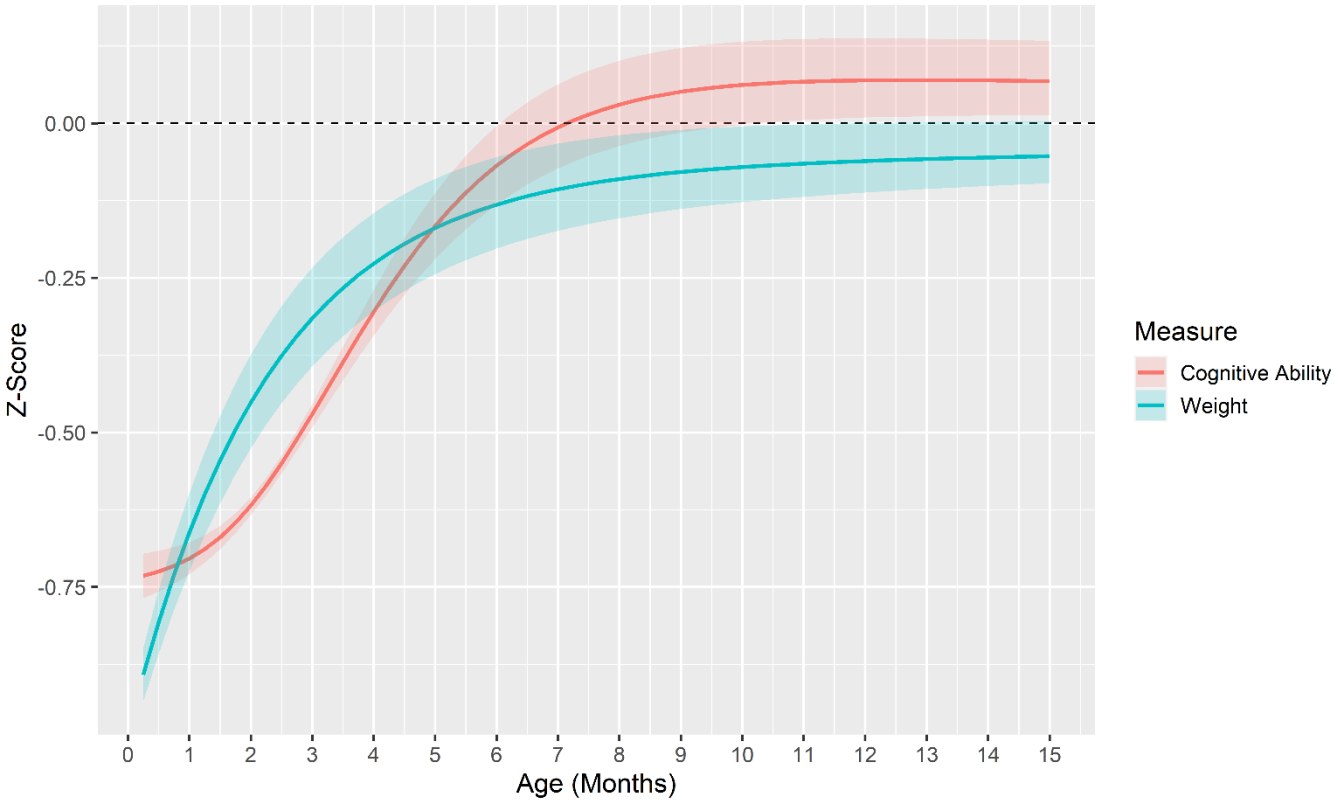
Table 42
Model Comparison – Bivariate Dual Change Score Model: Weight & Cognitive Ability

Model	X^2	df	Satorra-Bentler X^2	Δ df	*p*-value
Full Coupling	1928.0	476	-	-	-
Weight → Cog	1930.6	477	3.0	1	.086
Cog → Weight	1,927.1	477	0.0	1	.999
No Coupling	1929.1	478	3.5	2	.175

Table 43
Parameter Estimates from the Bivariate Dual change Score Model between Weight and Cognitive Ability

Parameter	*B*	95% C.I.
Constant Growth		
Intercept Weight	-0.89	[-0.98, -0.81]
Asymptote Weight	0.80	[0.68, 0.92]
Rate Weight	0.11	[0.10, 0.12]
Intercept Cog	-0.73	[-0.80, -0.66]
Asymptote Cog	0.74	[0.62, 0.86]
Rate Cog	0.14	[0.13, 0.16]
Inflection Cog	13.0	[12.2, 13.9]
Proportional Growth		
Proportional Head	-0.01	[-0.01, -0.00]
Proportional Cog	-0.01	[-0.01, -0.00]
Coupling Parameters		
Head → Cog	0	
Cog → Head	0	

Figure 30
Predicted Trajectories of Weight and Cognitive Recovery



Correlations between the constant growth parameters in the no-coupling bivariate dual change score model are extremely similar to the correlations observed in the phenotypic parallel process growth model between weight and cognitive ability (see Table 44). The intercept of weight was significantly positively correlated with the intercept of cognitive ability. The rate of weight recovery was negatively correlated with the rate of cognitive recovery but positively correlated with the upper asymptote of cognitive recovery. That is, children who recovered more quickly in terms of weight had a slower rate of cognitive recovery but ultimately had higher cognitive scores.

Table 44
Correlations between Constant Growth Parameters: Weight and Cognitive Ability

	Intercept Weight	Asymptote Weight	Rate Weight	Intercept Cog	Asymptote Cog	Rate Cog	Inflection Cog
Intercept Weight	1						
Asymptote Weight	**-.35 [-.48, -.22]**	1					
Rate Weight	**-.10 [-.20, -.01]**	**-.19 [-.35, -.04]**	1				
Intercept Cog	**.26 [.15, .38]**	-.10 [-.25, .05]	**-.22 [-.35, -.08]**	1			
Asymptote Cog	-.07 [-.20, .07]	-.03 [-.17, .12]	**.20 [.05, .34]**	**-.35 [-.50, -.21]**	1		
Rate Cog	.03 [-.12, .18]	.05 [-.11, .22]	**-.22 [-.38, -.06]**	**.19 [.01, .37]**	**-.69 [-.80, -.58]**	1	
Inflection Cog	-.07 [-.19, .05]	.05 [-.09, .19]	-.11 [-.24, .01]	**.34 [.19, .48]**	**-.51 [-.62, -.41]**	**.57 [.41, .73]**	1

Note. 95% confidence intervals are included below the correlation coefficients. Significant correlations are bolded for clarity.

Dynamic Growth: Height and Cognitive Recovery

Univariate dual change score. The exponential dual change score model fit the data significantly better than the constant growth model, proportional growth model, and no change model (see Table 45). This indicates that the shape of height recovery was best described by an exponential curve with a negative feedback parameter, which served to dampen the rate of growth around the upper asymptote.

Table 45
Model Comparison – Univariate Dual Change Score Model: Height

Model	X^2	df	Satorra-Bentler X^2	Δ df	*p*-value
Dual Change Score	1,337.0	128	-	-	-
Constant Growth	1,361.4	129	15.7	1	< .001
Proportional Growth	4,988.7	135	934.1	7	< .001
No Growth	4,912.7	136	933.2	8	< .001

Bivariate dual change score model. The no-coupling bivariate dual change score model of cognitive and height recovery did not fit significantly worse than the full coupling model (see Table 46). The no coupling model fit the data acceptably (X^2 = 2,493.1, df = 493, p < .001; TLI = .91, RMSEA = .06). As with the dynamic associations between weight recovery and cognitive recovery, the no coupling model suggests that there is no reciprocal feedback between height recovery and cognitive recovery from infancy to adolescence. Table 47 contains the parameter estimates for the bivariate dual change score model of height and cognitive recovery. Predicted trajectories of height recovery and cognitive recovery are presented in Figure 31. Correlations between the constant growth parameters are presented in Table 48.

Table 46
Model Comparison – Bivariate Dual Change Score Model

Model	X^2	df	Satorra-Bentler X^2	Δ df	p-value
Full Coupling	2,494.7	491	-	-	-
Height → Cog	2,493.6	492	2.2	1	.140
Cog → Height	2,494.1	492	0.1	1	.999
No Coupling	2,493.1	493	2.9	2	.233

Table 47
Parameter Estimates from the Bivariate Dual change Score Model between Height and Cognitive Ability

Parameter	*B*	95% C.I.
Constant Growth		
Intercept Weight	-0.89	[-0.98, -0.81]
Asymptote Weight	0.80	[0.68, 0.92]
Rate Weight	0.11	[0.10, 0.12]
Intercept Cog	-0.73	[-0.80, -0.66]
Asymptote Cog	0.74	[0.62, 0.86]
Rate Cog	0.14	[0.13, 0.16]
Inflection Cog	13.0	[12.2, 13.9]
Proportional Growth		
Proportional Head	-0.01	[-0.01, -0.00]
Proportional Cog	-0.01	[-0.01, -0.00]
Coupling Parameters		
Head → Cog	0	
Cog → Head	0	

Figure 31

Predicted Trajectories of Height and Cognitive Recovery

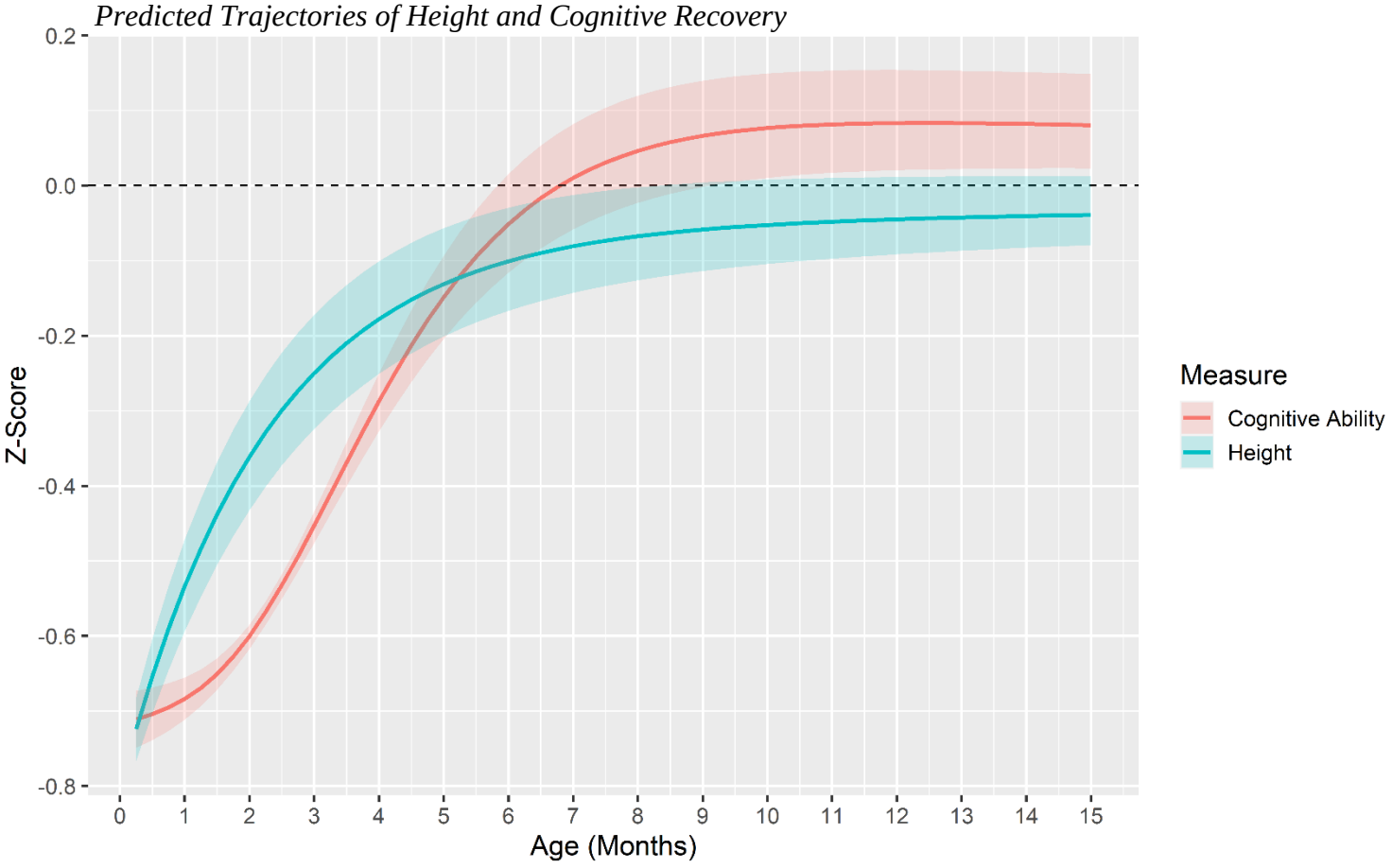


Table 48

Correlations between Constant Growth Parameters: Height and Cognitive Ability

	Intercept Height	Asymptote Height	Rate Height	Intercept Cog	Asymptote Cog	Rate Cog	Inflection Cog
Intercept Height	1						
Asymptote Height	**-.31 [-.44, -.18]**	1					
Rate Height	**-.40 [-.49, -.30]**	-.01 [-.14, .12]	1				
Intercept Cog	**.28 [.16, .40]**	**-.14 [-.27, -.01]**	-.11 [-.25, .03]	1			
Asymptote Cog	.02 [-.11, .16]	.04 [-.08, .16]	.04 [-.08, .17]	**-.35 [-.49, -.21]**	1		
Rate Cog	-.08 [-.23, .07]	-.04 [-.19, .12]	-.09 [-.25, .07]	**.17 [.00, .35]**	**-.69 [-.80, -.58]**	1	
Inflection Cog	-.04 [-.15, .08]	-.07 [-.18, .05]	-.06 [-.18, .07]	**.32 [.17, .47]**	**-.51 [-.61, -.41]**	**.57 [.41, .73]**	1

Note. 95% confidence intervals are included below the correlation coefficients. Significant correlations are bolded for clarity.

Dynamic Growth: Head Circumference and Cognitive Recovery

Univariate dual change score model. A dual change score model fit to head circumference recovery fit the data acceptably (X^2 = 247.4, df = 33, p< .001; TLI = .96; RMSEA = .08). The dual change score model fit significantly better than the constant growth, proportional growth, and no growth models (see Table 49).

Table 49
Model Comparison – Univariate Dual Change Score Model: Head Circumference

Model	X^2	df	Satorra-Bentler X^2	Δ df	p-value
Dual Change Score	247.4	33	-	-	-
Constant Growth	274.9	34	31.9	1	< .001
Proportional Growth	4214.6	40	4,149.5	7	< .001
No Growth	3893.9	41	2,667.6	8	< .001

I tested a series of latent change scores models to determine which best described the growth of cognitive ability from 3 to 36 months. A quadratic dual change score model fit significantly worse than a linear dual change score model (X^2 = 11.9, df=4, p .018). Due to the negative proportional change score parameters, the linear dual change score model describes cognitive ability that increases at a declining rate. The dual change score model fit to cognitive ability fit significantly better than constant change, proportional change, and no change models (see Table 50).

Table 50
Model Comparison –Dual Change Score Model: Cognitive Ability (3-36 months)

Model	X^2	df	Satorra-Bentler X^2	Δ df	p-value
Dual Change Score	241.1	37	-	-	-
Constant Growth	269.1	38	28.0	1	< .001
Proportional Growth	252.0	38	10.9	1	< .001
No Growth	287.9	39	46.8	2	< .001

Bivariate dual change score model. The bivariate dual change score model with full coupling fit the data well (X^2 = 602.6, df = 128, p < .001; TLI = .95, RMSEA = .06). The full coupling model fit the data better than models containing only coupling parameters from head circumference to cognitive ability, only coupling parameters from cognitive ability to head circumference, and no coupling parameters (see Table 51). Parameter estimates from the model are presented in Table 52 and correlations between the constant growth parameters are presented in Table 53. The negative proportional growth parameters for head circumference and cognitive ability suggests that the rate of recovery slows over time. The negative coupling parameter between head circumference and cognitive ability suggests that having a larger head circumference is related to a subsequent decline in the rate of growth of cognitive ability. Likewise, the negative coupling parameter between

cognitive ability and head circumference suggests that higher cognitive scores are associated with a subsequent decline in the rate of growth of head circumference.

Thinking about head circumference and cognitive ability as a dynamic system, children who are closer to their upper asymptote of head circumference are also slowing down in their cognitive recovery. Alternatively, children who are farther from their upper asymptote of head circumference growth have greater room to grow cognitively. Figure 32 depicts predicted trajectories of head circumference recovery and cognitive development from 3 to 36 months. It should be noted that there is relatively little recovery of cognitive ability during this time period, and cognitive recovery accelerates rapidly starting in toddlerhood (see Figure 23, study 2).

Table 51
Model Comparison – Bivariate Dual Change Score Model

Model	X^2	df	Satorra-Bentler X^2	Δ df	*p*-value
Full Coupling	602.6	128	-	-	-
Head → Cog	623.1	129	4.0	1	.029
Cog → Head	607.4	129	14.4	1	< .001
No Coupling	627.0	130	19.3	2	< .001

Table 52
Parameter Estimates from the Bivariate Dual change Score Model between Head Circumference and Cognitive Ability

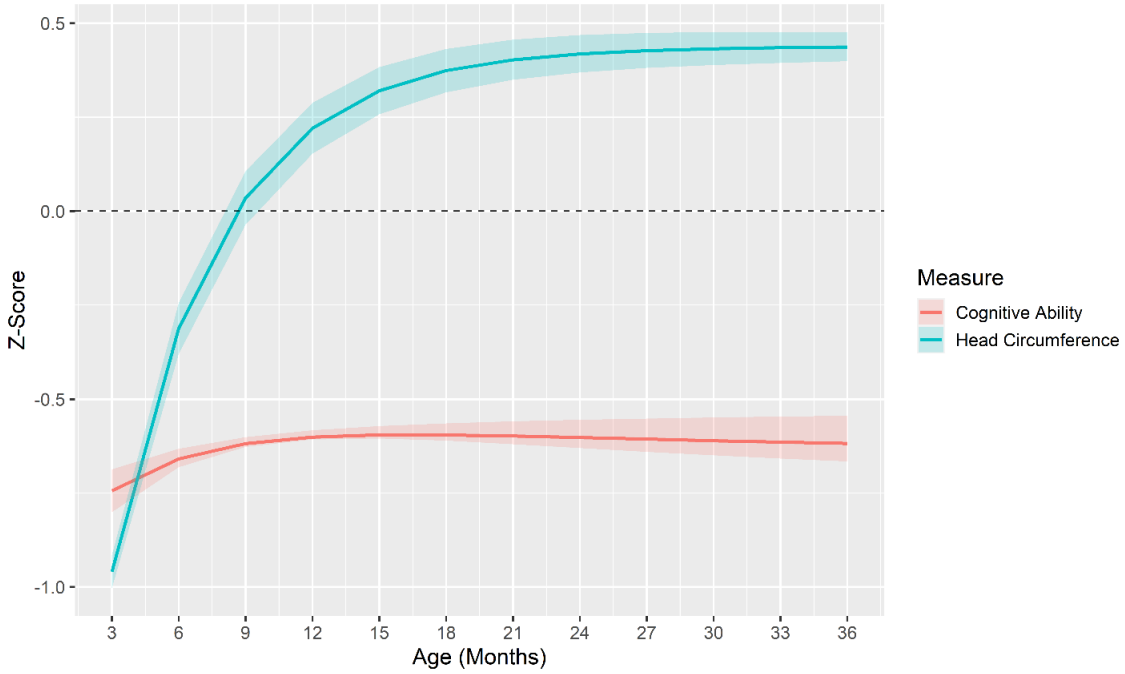
Parameter	*B*	95% C.I.
Constant Growth		
Intercept Head	-0.96	[-1.04, -0.85]
Total Growth Head	1.84	[1.22, 1.47]
Rate Head	0.62	[0.58, 0.67]
Intercept Cog	-0.74	[-0.85, -0.63]
Slope Cog	-0.06	[-0.10, -0.03]
Proportional Growth		
Proportional Head	-0.02	[-0.03, -0.02]
Proportional Cog	-0.13	[-0.18, -0.09]
Coupling Parameters		
Head → Cog	-0.05	[-0.10, -0.00]
Cog → Head	-0.02	[-0.02, -0.01]

Table 53
Correlations between Constant Growth Parameters: Head Circumference and Cognitive Ability

	Intercept Head	Asymptote Head	Rate Head	Intercept Cog	Slope Cog
Intercept Head	1				
Asymptote Head	-.07 [-.21, .07]	1			
Rate Head	-.08 [-.23, .07]	-.16 [-.00, .33]	1		
Intercept Cog	**.29** **[.20, .40]**	**-.28** **[-.43, -.14]**	-.04 [-.19, .11]	1	
Slope Cog	**.39** **[.22, .57]**	**.22** **[.06, .37]**	-.14 [-.28, .01]	-.04 [-.18, .10]	1

Note. 95% confidence intervals are included below the correlation coefficients. Significant correlations are bolded for clarity.

Figure 32
Predicted Trajectories of Head Circumference and Cognitive Recovery



Post-Hoc: Physical Recovery and Mental Development across the First Two Years of Life

Dual change score models fit to height and cognitive recovery and weight and cognitive recovery revealed little in the way of dynamic relationships between physical and cognitive recovery. This may have been due to the long timespan of the models (infancy to adolescence) much of which was defined by an upper asymptote where there was little to no change. Additionally, in dual change score models, the proportional change parameters are constrained to be equal over time. The relatively small windows between waves (3-month intervals) in addition to long upper asymptotes may have obscured some of the dynamic relationship between physical and cognitive recovery during developmental periods with accelerated physical and cognitive development. That is, because the proportional change and coupling parameters are constrained to be equal over time in dual change score models (McArdle, 2001), small proportional growth or coupling at the upper asymptotes may have dampened proportional growth and coupling estimates.

I conducted exploratory analyses by fitting dual change score models to physical recovery (i.e., height, weight, and head circumference) and mental development from 3 to 24 months as measured by raw Bayley scores. In contrast to standardized cognitive scores, which reflect performance relative to the population mean, raw cognitive scores reflect the number of items a child "passes" at each assessment. Because raw scores build upon "easier" items that a child would have passed at an earlier assessment, raw scores almost always increase rapidly over time (Bayley, 1969). As raw scores are not comparable across test versions due to different items, I restricted the supplementary analyses to Bayley first edition scores, which is the version of the Bayley that the majority of the infants in the Louisville Twin Study completed. Tasks on the first edition of the Bayley include responding appropriately to environmental stimuli, manipulating basic objects, and basic language production.

For height, weight, head circumference, and mental development, univariate quadratic dual change score models fit better than linear dual change score models. In all cases, a dual change score model fit better than constant change, proportional change, and no change models.

Weight and mental development. A dual change score model with coupling parameters from mental development to weight did not fit the data significantly worse than a model with full coupling (see Table 54). This model had an acceptable fit to the data (X^2 = 470.9, df = 58, p < .001; TLI = .90, RMSEA = .08). Contrary to expectations, the negative coupling parameter from mental development to weight suggests that higher mental development scores are related to a subsequent slowdown in the rate of weight gain. The intercept of weight was positively correlated with the intercept of mental development and the slopes of weight gain and mental development. In other words, heavier children at 3 months had higher mental development scores and initially had more rapid gains in their weight and mental development. Figure 33 depicts the trajectories of mental development for children 1 SD above and below the sample mean in terms of weight and trajectories of weight for children 1 SD above and below the sample mean in terms of Bayley scores. Heavier children at 3 months maintain a consistent advantage over lighter children. By 24 months, there is about a 4-point difference in raw scores for children 1 SD above and below the weight mean, which translates to about 6 standardized points. The negative coupling relationship between mental development and weight recovery appears to reflect an initial decline in weight for children with the highest mental development scores, followed by a steeper increase.

Parameters estimated for the dual change score are presented in Table 55 and correlations between constant growth parameters are presented in Table 56.

Table 54
Model Comparison – Bivariate Dual Change Score Model

Model	X^2	df	Satorra-Bentler X^2	Δ df	*p*-value
Full Coupling	475.5	57	-	-	-
Weight → MD	549.2	58	38.6	1	< .001
MD → Weight	470.9	58	0.2	1	.689
No Coupling	557.1	59	56.1	2	< .001

Note. MD stands for mental development as measured by raw Bayley Mental Development scores.

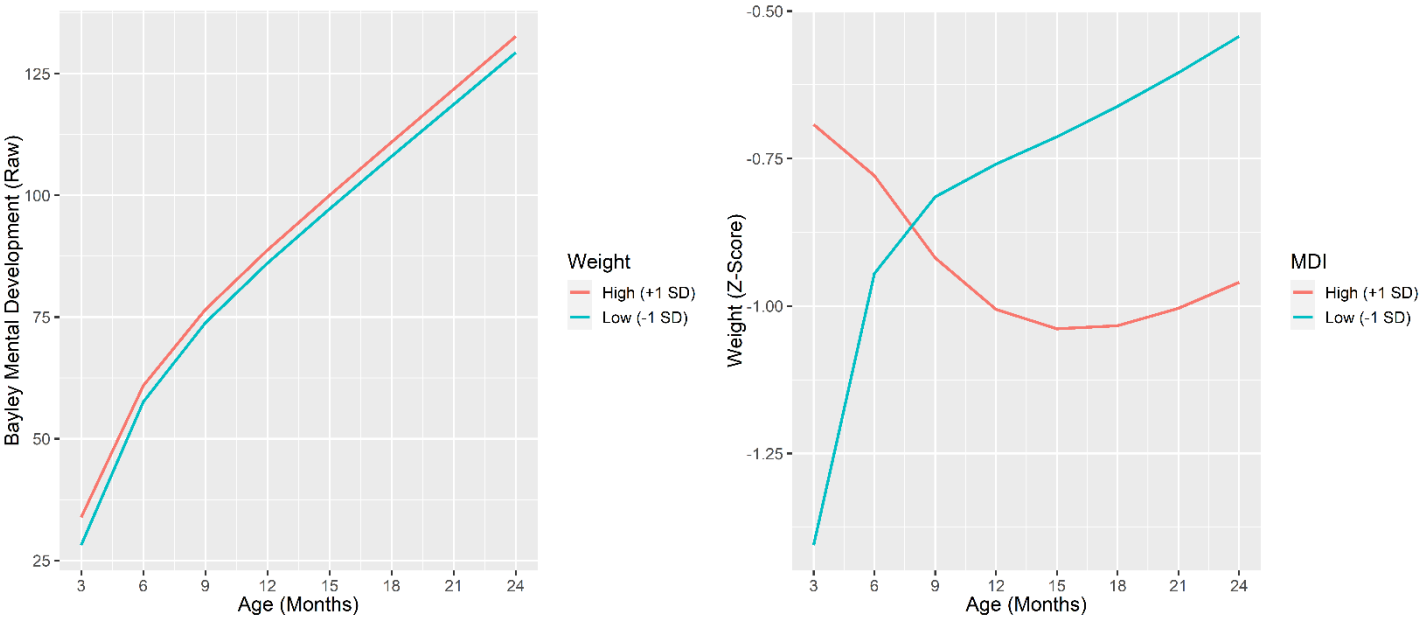
Table 55
Parameter Estimates from the Bivariate Dual Change Score Model between Weight and Bayley Mental Development

Parameter	*B*	95% C.I.
Constant Growth		
Intercept Weight	-1.05	[-1.12, -0.97]
Slope Weight	0.05	[-0.14, 0.23]
Quadratic Weight	0.06	[0.04, 0.07]
Intercept MD	30.99	[-0.85, -0.63]
Slope MD	53.88	[52.15, 55.61]
Quadratic MD	3.97	[3.48, 4.09]
Proportional Growth		
Proportional Weight	-0.43	[-0.56, -0.29]
Proportional Cog	-0.71	[-0.75, -0.67]
Coupling Parameters		
Weight → MD	0	
MD → Weight	-0.01	[-0.01, -0.01]

Note. MD stands for mental development as measured by raw Bayley Mental Development scores.

Figure 33

Predicted Trajectories of Mental Development and Weight Recovery



Note. On the left, predicted Bayley Mental Development trajectories are presented for infants scoring 1 SD above and below the sample mean on weight at 3 months. On the right are predicted head circumference trajectories for infants scoring 1SD above and below the sample mean on the Bayley at 3 months.

Table 56

Correlations between Constant Growth Parameters: Weight and Mental Development

	Intercept Weight	Slope Weight	Quadratic Weight	Intercept MD	Slope MD	Quadratic MD
Intercept Weight	1					
Slope Weight	**.62 [.49, .75]**	1				
Quadratic Weight	**-.22 [-.34, -.10]**	**-.54 [-.64, -.44]**	1			
Intercept MD	**.38 [.26, .51]**	-.12 [-.03, .28]	-.03 [-.22, .16]	1		
Slope MD	**.26 [.11, .41]**	**.18 [.00, .35]**	-.18 [-.37, .02]	**.68 [.48, .88]**	1	
Quadratic MD	.07 [-.08, .22]	.05 [-.11, .21]	.16 [-.02, .34]	-.11 [-.30, .08]	**-.41 [-.59, -.23]**	1

Note. 95% confidence intervals are included below the correlation coefficients. Significant correlations are bolded for clarity. MD stands for mental development as measured by raw Bayley Mental Development scores.

Height and mental development. A model with coupling parameters from only height to mental development did not fit the data worse than a model with full coupling (see Table 57). This model fit the data well (X^2 = 321.4, df = 58, p < .001; TLI = .94, RMSEA = .06). The positive coupling parameter between height and mental development suggests that taller heights are associated with an increase in the subsequent rate of mental development growth. The slope of height recovery was negatively correlated with the slope of mental development. Likewise, the quadratic growth of height recovery was negatively correlated with the quadratic growth of mental development. Thus, children who grew taller faster had a slower average rate of mental development but as children grew taller their rate of mental development increased. Parameter estimates for the dual change score are presented in Table 58 and correlations between constant growth parameters are presented in Table 59. The predicted trajectories of weight recovery and mental development from 3 to 24 months are presented in Figure 34.

Table 57
Parameter Estimates from the Bivariate Dual Change Score Model between Height and Bayley Mental Development

Parameter	*B*	95% C.I.
Constant Growth		
Intercept Height	-1.35	[-1.44, -1.26]
Slope Height	-0.22	[-0.34, -0.11]
Quadratic Height	0.00	[-0.01, 0.01]
Intercept MD	31.04	[-0.85, -0.63]
Slope MD	88.08	[63.76, 112.41]
Quadratic MD	5.73	[4.49, 6.97]
Proportional Growth		
Proportional Height	-0.58	[-0.68, -0.48]
Proportional Cog	-1.11	[-1.39, -0.84]
Coupling Parameters		
Height → MD	16.79	[4.31, 29.27]
MD → Height	0	

Note. MD stands for mental development as measured by raw Bayley Mental Development scores.

Table 58
Model Comparison – Bivariate Dual Change Score Model

Model	X^2	df	Satorra-Bentler X^2	Δ df	*p*-value
Full Coupling	347.1	57	-	-	-
Height → MD	321.4	58	0.0	1	.948
MD → Height	470.9	58	9.0	1	.003
No Coupling	363.2	59	13.1	2	.001

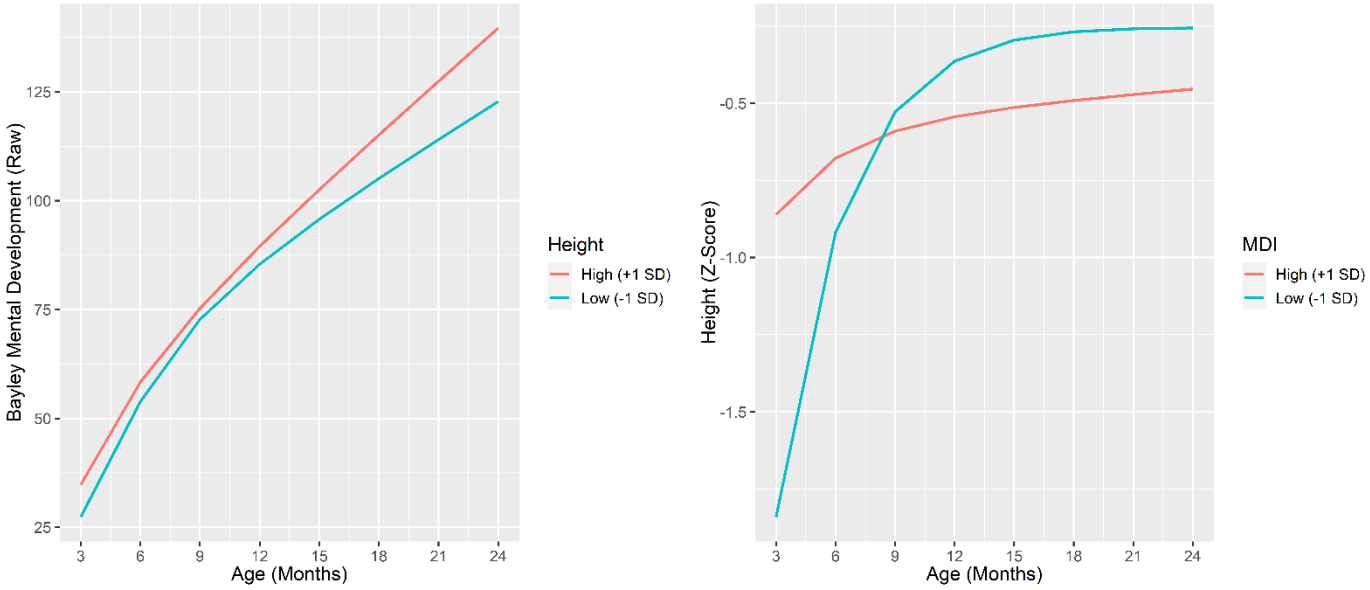
Note. MD stands for mental development as measured by raw Bayley Mental Development scores.

Table 59
Correlations between Constant Growth Parameters: Height and Mental Development

	Intercept Height	Slope Height	Quadratic Height	Intercept MD	Slope MD	Quadratic MD
Intercept Height	1					
Slope Height	**.82 [.72, .92]**	1				
Quadratic Height	**-.31 [-.43, -.21]**	**-.34 [-.47, -.21]**	1			
Intercept MD	**.49 [.37, .60]**	**.18 [.04, .32]**	-.17 [-.35, .02]	1		
Slope MD	**-.89 [-.99, -.79]**	**-.89 [-.96, -.81]**	**.40 [.29, .50]**	**-.18 [-.35, -.01]**	1	
Quadratic MD	**.47 [.36, .58]**	**.17 [.02, .31]**	**-.54 [-.79, -.30]**	**.29 [.08, .51]**	**-.48 [-.57, -.39]**	1

Note. 95% confidence intervals are included below the correlation coefficients. Significant correlations are bolded for clarity. MD stands for mental development as measured by raw Bayley Mental Development scores.

Figure 34
Predicted Trajectories of Mental Development and Height Recovery



Note. On the left, predicted Bayley Mental Development trajectories are presented for infants scoring 1 SD above and below the sample mean at 3 months. On the right are predicted head circumference trajectories for infants scoring 1SD above and below the sample mean on the Bayley at 3 months.

Head circumference and mental development. A bivariate dual change score model with only coupling parameters from head circumference to mental development did not fit significantly worse than a model with full coupling (see Table 60). This model fit the data well (X^2 = 288.3, df = 57, p < .001; TLI = .95, RMSEA = .06). The coupling parameter from head circumference to mental development was positive, which suggests that larger head circumference measurements were associated with a subsequent increase in the rate of growth of mental development. However, the constant slope of head circumference was strongly negatively correlated with the constant slope of mental development. In quadratic growth models, the slope refers to the instantaneous rate of growth when time equals 0. This may reflect the lagged relationship between growth in head circumference (and underlying neurological development) and growth in early cognitive abilities. That is, a steep instantaneous rate of head circumference growth may correspond with a relatively flat rate of initial cognitive growth as neurological gains take time to translate into observable cognitive abilities. All parameter estimates from the bivariate dual change score are presented in Table 61 and correlations between constant growth parameters are presented in Table 62. Estimated trajectories of head circumference recovery and mental development from 3 to 24 months are presented in Figure 34.

Table 60
Model Comparison – Bivariate Dual Change Score Model

Model	X^2	df	Satorra-Bentler X^2	Δ df	p-value
Full Coupling	288.3	57	-	-	-
Head → MD	289.0	58	2.3	1	.129
MD → Head	403.8	58	15.2	1	< .001
No Coupling	399.6	59	121.6	2	< .001

Note. MD stands for mental development as measured by raw Bayley Mental Development scores.

Table 61
Parameter Estimates from the Bivariate Dual Change Score Model between Head Circumference and Bayley Mental Development

Parameter	*B*	95% C.I.
Constant Growth		
Intercept Head	-1.03	[-1.11, -0.95]
Slope Head	0.24	[0.20, 0.29]
Quadratic Head	-0.01	[-0.01, -0.01]
Intercept MD	30.89	[29.85, 31.93]
Slope MD	72.42	[65.55, 79.29]
Quadratic MD	5.06	[4.52, 5.60]
Proportional Growth		
Proportional Head	-0.41	[-0.45, -0.367]
Proportional Cog	-1.00	[-1.11, -0.89]
Coupling Parameters		
Head → MD	9.64	[6.09, 13.20]
MD → Head	0	

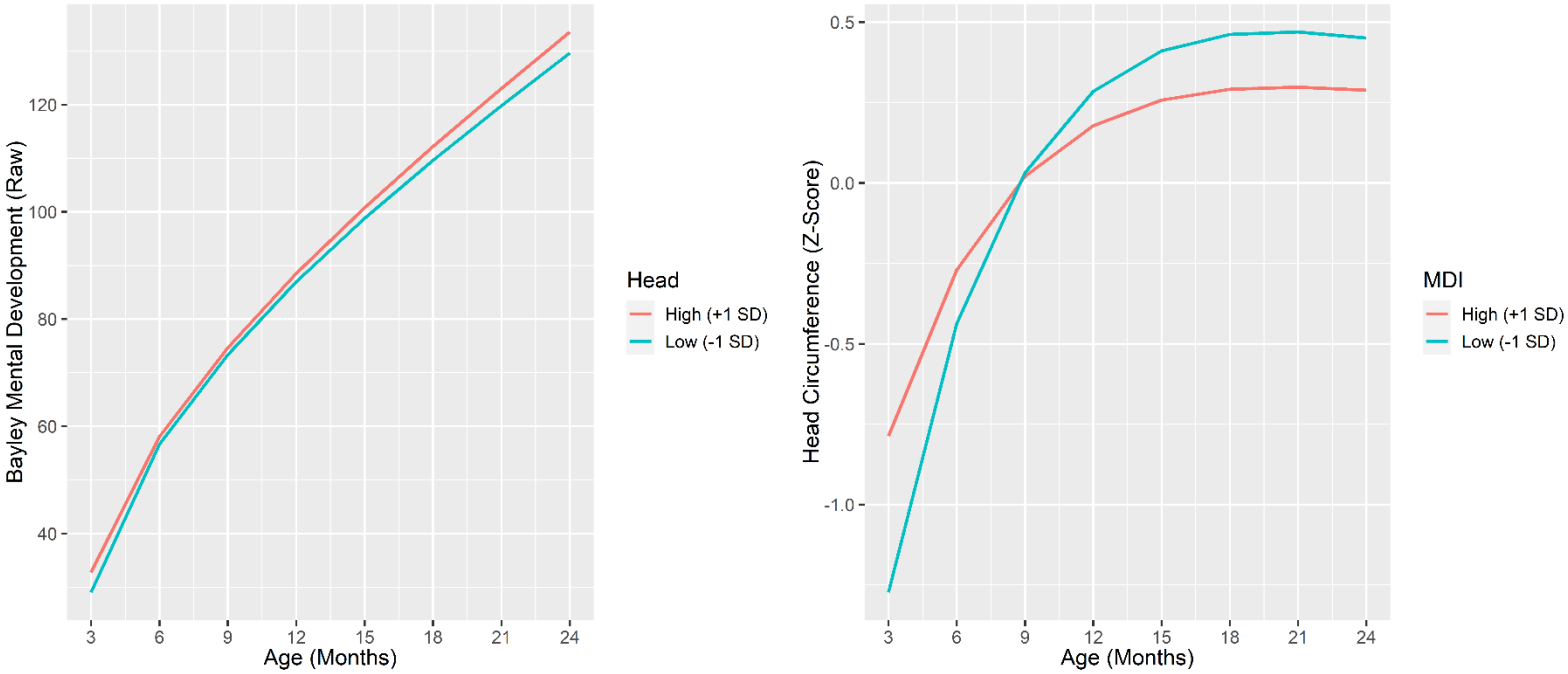
Note. MD stands for mental development as measured by raw Bayley Mental Development scores.

Table 62
Correlations between Constant Growth Parameters: Head Circumference and Mental Development

	Intercept Head	Slope Head	Quadratic Head	Intercept MD	Slope MD	Quadratic MD
Intercept Head	1					
Slope Head	**.75 [.68, .81]**	1				
Quadratic Head	-.15 [-.33, .02]	**-.40 [-.55, -.25]**	1			
Intercept MD	**.26 [.10, .42]**	-.13 [-.29, .04]	**.24 [.04, .45]**	1		
Slope MD	**-.86 [-.91, -.82]**	**-.84 [-.90, -.78]**	**.33 [.16, .50]**	.12 [-.04, .28]	1	
Quadratic MD	**.29 [.18, .39]**	-.10 [-.23, .03]	-.20 [-.43, .01]	.11 [-.07, .27]	**-.31 [-.41, -.20]**	1

Note. 95% confidence intervals are included below the correlation coefficients. Significant correlations are bolded for clarity. MD stands for mental development as measured by raw Bayley Mental Development scores.

Figure 34
Predicted Trajectories of Mental Development and Head Circumference Recovery



Note. On the left, predicted Bayley Mental Development trajectories are presented for infants scoring 1 SD above and below the sample mean at 3 months. On the right are predicted head circumference trajectories for infants scoring 1SD above and below the sample mean on the Bayley at 3 months.

Discussion

Using prospective data from the Louisville Twin Study spanning birth to adolescence, I tested a series of growth models to explore the longitudinal relationship between physical and cognitive recovery 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, 1974a; Wilson, 1974b; Wilson, 1979). Moreover, early deficits in physical size are a risk factor for poor neurocognitive development (Cooke, 2006; Upadhyay et al., 2019). I sought to fill gaps in extant research by exploring the process of co-recovery of physical size in a community sample of twins followed from infancy to adolescence using two models of co-development: parallel process growth and dynamic growth.

In general, findings indicate a moderate to strong relationship between physical size and cognitive ability in infancy and toddlerhood. However, at later developmental stages, physical size was not indicative of cognitive performance. Therefore, physical size relative to the population mean appears to be an important indicator of cognitive wellbeing in infant and toddler twins. The relationship between growth in physical size and growth in cognitive abilities was inconsistent across physical growth measurements (height, weight, or head circumference) and depended on the type of model (parallel or dynamic) and developmental span considered.

Parallel Development & Biometric Contributions

Consistent with study hypotheses and previous research (Cheong et al., 2008; Kohlhauser et al., 2000; Kuklina et al., 2006), the lower asymptote of cognitive recovery correlated positively with the intercepts of height, weight, and head circumference. Correlations between initial levels of height and weight and early cognitive abilities were significant at both with within- and between-pair levels. Between-pair correlations indicate that across pairs, being longer, heavier, and having a larger head circumference at birth was associated with having a higher lower asymptote of cognitive abilities. Significant within-pair correlations indicate that, 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 within-pair correlations provide robust evidence that weight and length at birth are important indicators of early cognitive development. Shared environmental factors accounted for the majority of the early association between physical size at birth and cognitive ability in infancy. This is likely due to the strong association between gestational age and early height, weight, head circumference, and cognitive ability (as observed in Studies 1 and 2). 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.

Within-pair, individual differences in birth weight were positively associated with individual differences in the rate of cognitive recovery. A similar pattern was observed at a trend level between the intercept of height and rate of cognitive recovery. Thus, within a pair of twins, the larger twin at birth demonstrated a faster rate of cognitive recovery. Using data from the National Longitudinal Study of Youth 1979, Cheadle and Goosby (2010) observed a positive association between birth weight and the rate of academic achievement growth from 5 to 14 years. As the majority of physical and cognitive recovery in the Louisville Twin sample occurs before age 5 (see Studies 1 and 2), the present study extends associations between birth weight and cognitive development to earlier developmental stages. Feldman and Otto (1997) raised the idea that more

phenotypically similar twins elicit more similar environmental experiences from their parents. Therefore, infant twins (regardless of zygosity) who are highly discrepant in their physical size may elicit different parenting strategies, which could lead to differences in the rate of cognitive recovery. Additionally, the larger twin may have an initial advantage in terms of their ability to explore their environment, leading to differences in the rate of recovery (Flensborg-Madsen & Mortensen, 2017).

Contrary to hypotheses, the rate of cognitive recovery was not significantly related to the rate of height or weight recovery between-pair. Moreover, within pair, the rate of cognitive recovery correlated negatively with the rate of both height and weight recovery. This suggests that the twin with the faster rate of height and weight recovery had a slower rate of cognitive recovery. It should be noted that the confidence intervals around the association between the rate of height recovery and cognitive recovery were extremely large and ranged in magnitude from .99 to .01. Therefore, the within-pair association between the rate of height and cognitive recovery should be interpreted with caution. Regarding weight, within-pair differences in the rate of weight recovery were positively associated with the upper asymptote of cognitive recovery. Children who recovered faster in terms of weight had a slower, steadier cognitive recovery, and ultimately ended up with higher cognitive scores.

The biometric correlations between the rate of cognitive recovery and the rate of weight recovery were incongruent with the phenotypic correlations. That is, the phenotypic correlation between the rate of weight recovery and the rate of cognitive recovery was significant only at the within-pair level (the between-pair correlation was significant at a trend level). However, there was a significant shared environmental correlation between the rate of weight recovery and the rate of cognitive recovery. In the initial model, the additive genetic variance of the rate of cognitive recovery was negative and had to be constrained to 0. Constraining the additive genetic variance to 0 may have allowed for more shared environmental variance to be estimated, resulting in a significant shared environmental correlation. In Study 1, I found that gestational age was negatively related to the rate of weight recovery, and in Study 2 I found gestational age to be positively related to the rate of cognitive recovery. Therefore, the negative shared environmental correlation between the rate of weight recovery and the rate of cognitive recovery may reflect the differential associations between gestational age and weight and cognitive recovery.

Phenotypically, the rate of head circumference recovery correlated positively with the slope of cognitive recovery but negatively with the quadratic growth of cognitive recovery, between pair. In other words, children who recovered more quickly in terms of head circumference had a steeper initial rate of cognitive recovery, but a slower overall rate of growth. The negative association between the rate of head circumference recovery and quadratic cognitive recovery may reflect a plateauing of cognitive abilities following early gains in head circumference. It is worth noting that the parallel process models of head circumference and cognitive recovery were restricted to 36 months as the CDC does not provide tables to calculate standardized head circumference measurements beyond 36 months. In the study sample, there was relatively little cognitive recovery by 36 months, and therefore analyses are limited in their ability to elucidate associations between head circumference recovery and cognitive recovery into early childhood when cognitive recovery is most rapid. The biometric models did not yield much insight into the genetic or environmental contributions to the co-development of head circumference and cognitive ability as numerous

variances and covariances had to be constrained to 0. Within-pair, the variances of the intercept, slope, and quadratic growth parameters were not significant, which may have prohibited me from identifying significant nonshared environmental or additive genetic variance components.

Associations between the upper asymptotes of height and cognitive recovery and weight and cognitive recovery were nonsignificant. Therefore, physical size in childhood does not appear to be an important indicator of childhood cognitive abilities. 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. As humans continue growing well beyond age 15 (Kuczmarski, 2002), the estimated upper asymptotes are not based on the final heights or weights of the twins, which may account for the null associations between the upper asymptotes. In the present study, there were low rates of extreme deficits in physical size after toddlerhood (see Figure 6, Study 1). Therefore, the significant cognitive deficits associated with deficits in physical size may be 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.

Dynamic Recovery of Physical Size and Cognitive Ability

Dynamic models of the co-recovery of physical and cognitive recovery spanning infancy to adolescence suggest that there is not a dynamic relationship between physical recovery and cognitive recovery. All coupling parameters could be constrained to 0 without a loss of model fit, indicating that the more parsimonious parallel process growth model was adequate to describe physical and cognitive recovery from infancy to adolescence. The absence of a dynamic relationship between height and weight recovery and cognitive recovery may reflect the extended developmental period assessed as well as the small increments of time between measurements. As twins made most of their physical and cognitive recovery by early childhood, the majority of the developmental span included in the models was characterized by an upper asymptote where individuals exhibited relatively little proportional or constant growth. This may contribute to dampened estimates of coupling between the growth parameters as coupling parameter estimates are averaged over time.

Models restricted to an earlier developmental period and shorter developmental span revealed evidence of a dynamic relationship between physical and cognitive recovery. For example, there was a bidirectional slowing down of the relationship between head circumference recovery and standardized cognitive development from 3 to 36 months. A dynamic slowing down relationship suggests that as twins approached their upper asymptote of head circumference, their rate of cognitive recovery also slowed. Likewise, as twins made gains in their early cognitive abilities, their head circumference recovery slowed down. This dynamic pattern reflects a system that is slowing down, which is consistent with expectations for a model of asymptotic growth. That is, early cognitive development would be expected to slow down as the rate of head circumference growth slows down around the upper asymptote. The bidirectional relationship between head circumference recovery and early cognitive development needs to be interpreted in light of the fact that twins displayed relatively little cognitive recovery over the first 36 months (see Table 20, Study 2). Therefore, the slowing down of cognitive recovery does not reflect twins approaching

their upper asymptote of cognitive ability, but rather a slowing down of modest cognitive gains across infancy and early toddlerhood. Extending the bidirectional relationship between head circumference recovery to later developmental stages may yield insight into the bidirectional relationship between head circumference recovery and cognitive recovery across a developmental span than encompasses larger gains in cognitive abilities. However, the CDC does not provide a means to calculate head circumference z-scores beyond 36 months.

Across all physical size indicators, there appears to be a lag between the peak rate of physical recovery and the peak rate of cognitive recovery. In all cases, physical recovery asymptotes before cognitive recovery (see Figures 30, 31, and 32). Height, weight, and head circumference are all accelerating towards their upper asymptote during a time when there is little growth in cognitive abilities. Cognitive recovery begins in earnest after physical recovery begins to slow down. The timing difference in the growth patterns of physical and cognitive recovery may make it difficult to detect dynamic relationships between the two constructs if one construct is accelerating in growth while the other is decelerating. Rather than a direct, dynamic relationship between physical and cognitive recovery across childhood, physical recovery may lead to cognitive gains indirectly. For example, recovery of height and weight may more immediately afford twins a greater ability to explore their environments (Flensborg-Madsen & Mortensen, 2017). Through increased environmental exploration and interaction with people and objects, twins may then experience cognitive gains. Likewise, recovery of head circumference is thought to be related to cognitive recovery through increases in brain size and neural development (Bartholomeusz et al., 2002). However, it may take time for advances in neural development to translate into observable skills (Estrada et al., 2019). Thus, growth in physical size may provide infants with the anatomical framework necessary to make gains in their cognitive abilities.

Post-hoc analyses conducted focusing on early physical recovery and *raw mental development* provided evidence of a unidirectional relationship between growth in head circumference and height and growth in early cognitive abilities. Growth of raw mental development describes the total accumulation of cognitive skills across the first years of life. Increases in head circumference were associated with increases in the subsequent rate of mental development. Likewise, increases in height recovery were associated with subsequent increases in the rate of mental development. For both height and head circumference, there was a divergence in the predicted trajectories for children who were larger at baseline relative to smaller children; larger children at 3 months exhibited a steeper rate of growth in early mental abilities. Thus, twins who are longer and have larger head circumferences in early infancy continue to build upon their early cognitive advantage relative to smaller infant twins across the first years of life.

Contrary to expectations, there was a negative unidirectional relationship between cognitive ability to weight. That is, higher cognitive scores were associated with a *decline* in the rate of subsequent weight. In the weight and mental development model, the expected trajectories of weight are highly variable based on the initial cognitive score. Trajectories range from initial declines in weight relative to the population mean for children with high initial scores to rapid growth in weight for children with low mental development scores (see Figure 33). There is no reason to expect that having high mental development scores would lead to twins falling further behind the population mean in terms of weight. Among the constant growth parameters, there was a positive correlation between the intercepts of weight and mental development as well as the slopes of

weight and mental development. Therefore, twins who were born heavier had higher initial mental development scores, and twins who gained weight more rapidly demonstrated a more rapid increase in their early mental abilities. The negative coupling parameter may reflect a slowing down of weight recovery among children who were initially heavier and who made larger initial gains in both their weight and cognitive abilities.

Limitations

As outlined in studies 1 and 2, the Louisville Twin sample is a predominantly White, American sample. 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 countries. Rates of extreme deficits in height, weight, and head circumference were very low in the present sample; less than 5% of children in the Louisville Twin Study were stunted, microcephalic, or clinically underweight after age 2. Children born in developing countries with higher rates of malnutrition and infectious diseases may experience higher rates of stunting and wasting[7], and microcephaly (Morris et al., 2021; Ssentongo et al., 2021). Higher rates of stunting, wasting, or microcephaly in childhood may lead to greater variability in the upper asymptotes of physical size and a stronger relationship between physical size and cognitive ability at later developmental stages.

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. By fitting growth models to different cognitive tests within wave and across waves, I 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 me from testing measurement invariance across assessments. However, wave-to-wave correlations between different tests were generally high (see Figure 22, Study 2). Moreover, the use of standardized cognitive scores provided a common reference point to understand a child's abilities relative to the population mean.

Although bivariate dual-change score models provide important information regarding the development of a system, they cannot determine *causal* relationships between the variables in the system. Relatedly, bivariate dual change score models do not yield insight into *how* physical and cognitive development are related to one another over time. There may be several mechanisms by which increases in height and head circumference lead to increases in early mental development (e.g., increased ability to explore the environment). Future research is necessary to explore the mechanisms underlying the association between early height or head circumference and mental development.

Finally, the reference group I used to generate standardized head circumference measurements limited the developmental span I was able to include in the growth models of head circumference. I selected CDC norms (Kuczmarski, 2000) based on the historical overlap with the Louisville Twin Study and overlap in the country of origin (CDC norms are based on an American sample). However, the CDC does not provide standardized growth charts for head circumference beyond 3 years. Consequently, I was restricted to testing parallel and dynamic growth between head circumference and cognitive ability during a developmental period with relatively little cognitive growth. Using other reference groups (e.g., World Health Organization norms) would provide head

[7] Wasting indicates having a weight less than two standard deviations below the average person at your height.

circumference norms to 5 years, but norms would be based on a more diverse reference group which may not necessarily correspond to children in the Louisville Twin Study.

Conclusion

Findings suggest that there is a moderate to large 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 a faster rate of cognitive recovery. Together findings from study 3 highlight the importance of physical size at birth, and in particular birth weight, in early cognitive development. Height, weight, and head circumference at birth were related to cognitive abilities in infancy through shared environmental factors. The shared environmental association between physical size and birth and cognitive functioning in infancy is likely due to perinatal experiences, including gestational age. Therefore, interventions designed to promote complete gestation and healthy prenatal growth in mothers expecting twins may benefit physical size and birth and early cognitive development.

However, prematurity appears to be relatively normative in twin pregnancies (Giuffrè et al., 2012), 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, weight, and head circumference recovery postnatally may encourage early cognitive development. Within pair, a faster rate of weight recovery was associated with higher overall cognitive scores. Moreover, early gains in height and head circumference were associated with an acceleration of the rate of early mental development. Protein- and calcium-enriched nutritional interventions for premature, undersized infants have been found to promote lean body mass gain and healthy bone development (Rigo & Senterre, 2006). Such postnatal interventions may have downstream benefits for emerging cognitive development.

General Discussion

Findings from the three studies yield important insight into the process by which twins recover from early physical and cognitive deficits. This dissertation is the first study to leverage growth curve models to explore the latent rate and shape of physical and cognitive recovery in twins from infancy to adolescence. Although twins displayed deficits in early height, weight, head circumference, and cognitive ability ranging from 0.9 to 1.5 SD below the population mean, the average twin recovered to the population mean physically and cognitively by middle childhood. Therefore, although it is common for twins to be significantly undersized in infancy and perform poorly on early cognitive assessments, most twins recover completely from early physical and cognitive deficits. Although this dissertation focused on physical and cognitive recovery in a sample of twins, findings have implications for our understanding of physical and cognitive development in singletons exposed to perinatal stressors common in twin pregnancies (e.g., premature birth, nutrient deficiencies, maternal health complications).

Physical and cognitive recovery in twins was best described using nonlinear, asymptotic growth models (as opposed to polynomial growth models), which suggests that twins sustain the physical and cognitive gains that they make towards the population mean. In other words, physical growth and the accumulation of early cognitive abilities are *faster* in twins relative to singletons in infancy and toddlerhood. Once physical and cognitive recovery in twins has reached an upper asymptote

in middle childhood, the rate of physical growth and cognitive development is similar in twins and singletons. Temporally, physical recovery preceded cognitive recovery, suggesting that the typical twin begins making advances toward the population mean physically before cognitive recovery accelerates. During the first year of life, twins made massive gains relative toward the population mean in terms of height, weight, and head circumference, but make minimal cognitive gains. Alternatively, in toddlerhood, cognitive recovery begins accelerating whereas physical recovery begins decelerating.

Findings related to typical trajectories of physical and cognitive recovery in twins have relevance for pediatricians, clinicians, and other healthcare providers working with populations of children born prematurely or at low birth weight. Specifically, healthcare providers should focus on promoting healthy physical development over the first year. Physically undersized children not demonstrating substantial physical recovery over the first year may be at an elevated likelihood to remain undersized beyond infancy which may elevate the risk for poor early neurocognitive development (Ghods et al., 2011; Upadhyay et al., 2019). On the other hand, poor cognitive performance among twins or other children born prematurely appears to be relatively normative in infancy and early toddlerhood. Clinicians should account for typical deficits in early cognitive performance when assessing twins for early developmental delays (e.g., global developmental delay) or estimating the prognosis of a twin meeting the criteria for an early developmental delay. For example, provided the enormous cognitive gains observed in twins across toddlerhood and early childhood, being a twin may be an indicator of a positive prognosis among children diagnosed with an early developmental delay, such as global developmental delay.

This dissertation makes additional contributions to the existing literature by exploring the nuances in the relationship between twinning, gestational age, and birth weight. Gestational age and birth weight are strongly correlated (Oken et al., 2003), and twin pregnancies are at an elevated risk for both prematurity and low birth weight (Giuffrè et al., 2012). Therefore, it is challenging to disentangle the relative contributions of gestational age, birth size, and twinning to developmental outcomes. Consistent with expectations and previous research (Livshits et al., 2000; Villar et al., 2014), gestational age emerged as an important environmental correlate to early length, weight, and head circumference. However, full-term twins were still significantly below population norms in terms of early physical growth measurements. Likewise, gestational age was positively related to early cognitive abilities controlling for birth weight, which is consistent with previous research (Datar & Jacknowitz, 2009). Yet, full-term twins' early cognitive abilities were still a half standard deviation below population norms. Thus, twin pregnancies appear to confer additional risk for deficits in early physical and cognitive development beyond the increased risk associated with low birth weight and premature birth. Although beyond the scope of the present dissertation, incorporating non-twin siblings into the twin analyses would provide a means to further disentangle the relationship between gestational age, birth weight, and twining.

When working with children born prematurely, it is common practice to use growth norms or standardized cognitive norms that are corrected for gestational age (Aylward, 2020; Babson & Benda, 1976). Although correcting for gestational age may reduce some of the discrepancies between premature and full-term twins in terms of their early physical size and cognitive abilities, norms corrected for gestational age may still inflate estimates of physical and cognitive deficits in twins. Generating physical and cognitive development standards corrected for both gestational age

and twinning may be most helpful in determining which twins are at the greatest risk for poor physical and cognitive development. Importantly, gestational age was not significantly related to physical or cognitive development long-term, suggesting that the negative effects of prematurity on child development may be constrained to infancy and toddlerhood.

Findings from Studies 1 and 2 also extend our understanding of how socioeconomic deprivation contributes to physical and cognitive development across childhood. Children reared in the poorest homes displayed a concerning pattern of weight gain characterized by a longer, steadier growth which led to them overshooting the population mean for weight in adolescence. Although children born into poverty were also taller than the population mean on average by adolescence, their estimated weight-for-age z-scores were higher than their height-for-age z-scores in adolescence, raising concerns about their risk for obesity. Additionally, children reared in low-SES homes were delayed in making cognitive gains until late toddlerhood and ultimately displayed lower cognitive scores than their more affluent peers. In the Louisville Twin Study, family SES was measured at birth. Therefore, findings from Studies 1 and 2 reflect the pervasive association between early environmental deprivation and long-term developmental outcomes.

There was evidence of a moderate to strong association between early physical size and cognitive abilities, which was largely accounted for by common shared environmental factors. Gestational experiences, which reflect the earliest shared environmental experiences, likely account for the early association between physical size and cognitive abilities. Beyond infancy, there was little evidence that physical and cognitive development unfold as parallel processes. The strength of the associations between physical size and cognitive ability attenuated over time, and the upper asymptotes of height and weight were unrelated to the total cognitive growth. There was some evidence that faster physical recovery was associated with slower cognitive recovery, which may indicate a cost of early physical recovery. That is, quicker physical growth in infancy may consume energy that may otherwise be used to build foundational cognitive abilities. Alternatively, in S-shaped growth models, a slower rate of recovery indicates a longer period of growth. As physical recovery was found to precede cognitive recovery, children who recover more rapidly physically may begin recovering earlier cognitively and have slower, steadier cognitive development.

In infancy, there was evidence of a dynamic relationship between physical size and raw cognitive development. Larger heights and head circumference measurements (but not weights) were associated with an increase in the rate of raw cognitive growth. In previous work, early catch-up growth in height and head circumference have been stronger predictors of future cognitive ability than early weight catch-up (Adair et al., 2013; Poveda et al., 2021; Pongcharoen et al., 2012; Scharf et al., 2016). Findings from this study extend previous work by showing that children build off of early advantages in height and head circumference and further diverge from children who are physically smaller at birth across infancy.

In sum, children are, on average, resilient following early biological disadvantage. Despite displaying substantial deficits in height, weight, head circumference, and cognitive ability in infancy, the average twin caught up to the population mean. Moreover, twins born prematurely also caught up physically and cognitively both to the population mean and their full-term twin peers. Exposure to early bioenvironmental adversity appears to impact early development globally as evidenced by the large initial physical and cognitive deficits as well as the moderate shared

environmental correlations between early physical size and cognitive ability. In most twin pregnancies, early environmental stressors are removed following birth and become distal experiences as twins age. As twins age, postnatal environmental experiences (e.g., the home environment) and genetics have increasing room to operate in explaining different developmental trajectories.

References

Adair, L. S., Fall, C. H., Osmond, C., Stein, A. D., Martorell, R., Ramirez-Zea, M., Sachdev, H. S., Dahly, D. L., Bas, I., Norris, S. A., Micklesfield, L., Hallal, P., Victora, C. G., & COHORTS Group (2013). Associations of linear growth and relative weight gain during early life with adult health and human capital in countries of low and middle income: findings from five birth cohort studies. *The Lancet*, *382*(9891), 525-534.

Albertsson-Wikland, K., & Karlberg, J. (1997). Postnatal growth of children born small for gestational age. *Acta Paediatrica*, *86*(S423), 193-195.

Aylward, G. P. (2020). Is it correct to correct for prematurity? Theoretic analysis of the Bayley-4 normative data. *Journal of Developmental & Behavioral Pediatrics*, *41*(2), 128-133.

Aylward, G. P., Pfeiffer, S. I., Wright, A., & Verhulst, S. J. (1989). Outcome studies of low birth weight infants published in the last decade: a metaanalysis. *The Journal of Pediatrics*, *115*(4), 515-520.

Antoniou, E. E., Fowler, T., Thiery, E., Southwood, T. R., Van Gestel, S., Jacobs, N., Vlietinck, R., van Os, J., Rijsdijk, F. V., Derom, C., & Zeegers, M. P. (2013). Intrauterine environment and cognitive development in young twins. *Journal of Developmental Origins of Health and Disease*, *4*(6), 513-521.

Ashworth, A., Morris, S. S., & Lira, P. I. (1997). Postnatal growth patterns of full-term low birth weight infants in Northeast Brazil are related to socioeconomic status. The Journal of Nutrition, 127(10), 1950-1956.

Batista, R. F., Silva, A. A., Barbieri, M. A., Simões, V. M., & Bettiol, H. (2012). Factors associated with height catch-up and catch-down growth among schoolchildren. *PloS one*, *7*(3), e32903.

Bailey, Z. D., Krieger, N., Agénor, M., Graves, J., Linos, N., & Bassett, M. T. (2017). Structural racism and health inequities in the USA: evidence and interventions. *The Lancet*, *389*(10077), 1453-1463.

Bartholomeusz, H. H., Courchesne, E., & Karns, C. M. (2002). Relationship between head circumference and brain volume in healthy normal toddlers, children, and adults. *Neuropediatrics, 33*(05), 239-241.

Batty, G. D., Deary, I. J., & Gottfredson, L. S. (2007). Premorbid (early life) IQ and later mortality risk: systematic review. *Annals of epidemiology*, *17*(4), 278-288.

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, C. R., & Turkheimer, E. (2013). Phenotype–environment correlations in longitudinal twin models. *Development and Psychopathology*, *25*(1), 7-16.

Beam, C.R., Turkheimer, E., Finkel, D., Levine, M., Zandi, E., Guterbock, T, Giangrande, E.J., Ryan, L., Pasquenza, N, & Davis, D.W. (2020). Midlife study of the Louisville twins: Connecting cognitive development to biological and cognitive aging. *Behavior Genetics, 50*(2), 73-83. https://doi.org/10.1007/s10519-019-09983-6

Beauchamp, J. P., Cesarini, D., Johannesson, M., Lindqvist, E., & Apicella, C. (2011). On the sources of the height–intelligence correlation: New insights from a bivariate ACE model with assortative mating. *Behavior Genetics*, *41*(2), 242-252.

Bélanger, S. A., & Caron, J. (2018). Evaluation of the child with global developmental delay and intellectual disability. *Paediatrics & Child Health, 23*(6), 403-410.

Bishop, E. G., Cherny, S. S., Corley, R., Plomin, R., DeFries, J. C., & Hewitt, J. K. (2003). Development genetic analysis of general cognitive ability from 1 to 12 years in a sample of adoptees, biological siblings, and twins. *Intelligence*, *31*(1), 31-49. https://doi.org/10.1016/S0160-2896(02)00112-5

Bitler, M., Bartfeld, J., Gundersen, C., Smeeding, T., & Ziliak, J. (2015). The health and nutrition effects of SNAP. *SNAP matters: How food stamps affect health and well-being*, 134-60.

Blickstein, I. (2004). Is it normal for multiples to be smaller than singletons?. *Best practice & research Clinical Obstetrics & Gynaecology*, *18*(4), 613-623.

Bode, M. M., D'Eugenio, D. B., Mettelman, B. B., & Gross, S. J. (2014). Predictive validity of the Bayley, at 2 years for intelligence quotient at 4 years in preterm infants. *Journal of Developmental & Behavioral Pediatrics*, *35*(9), 570-575. http://doi.org/10.1097/DBP.0000000000000110

Bradley, R. H., Caldwell, B. M., Rock, S. L., Ramey, C. T., Barnard, K. E., Gray, C., Gottfried, A. W., Siegel, L., & Johnson, D. L. (1989). Home environment and cognitive development in the first 3 years of life: A collaborative study involving six sites and three ethnic groups in North America. *Developmental Psychology*, *25*(2), 217-235. https://doi.org/10.1037/0012-1649.25.2.217

Brandt, I., Sticker, E. J., & Lentze, M. J. (2003). Catch-up growth of head circumference of very low birth weight, small for gestational age preterm infants and mental development to adulthood. *The Journal of Pediatrics*, *142*(5), 463-470.

Broekman, B. F., Chan, Y. H., Chong, Y. S., Quek, S. C., Fung, D., Low, Y. L., Ooi, Y. P., Gluckman, P. D., Meaney, M. J., Wong, T. Y., & Saw, S. M. (2009). The influence of birth size on intelligence in healthy children. *Pediatrics*, *123*(6), e1011-e1016.

Buckler, J. M. H., & Green, M. (2004). A comparison of the early growth of twins and singletons. *Annals of Human Biology*, *31*(3), 311-332.

Burchinal, M., & Appelbaum, M. I. (1991). Estimating individual developmental functions: Methods and their assumptions. *Child Development*, *62*(1), 23-43.

Calvin, C. M., Deary, I. J., Fenton, C., Roberts, B. A., Der, G., Leckenby, N., & Batty, G. D. (2011). Intelligence in youth and all-cause-mortality: systematic review with meta-analysis. *International journal of epidemiology*, *40*(3), 626-644.

Cameron, C. E., Grimm, K. J., Steele, J. S., Castro-Schilo, L., & Grissmer, D. W. (2015). Nonlinear Gompertz curve models of achievement gaps in mathematics and reading. *Journal of Educational Psychology*, *107*(3), 789.

Casale, D., Desmond, C., & Richter, L. M. (2020). Catch-up growth in height and cognitive function: why definitions matter. *Economics & Human Biology*, *37*, 100853.

Ceci, S. J., & Williams, W. M. (1997). Schooling, intelligence, and income. *American Psychologist*, *52*(10), 1051.

Cheong, J. L., Hunt, R. W., Anderson, P. J., Howard, K., Thompson, D. K., Wang, H. X., Bear, M. J., Inder, T. E., & Doyle, L. W. (2008). Head growth in preterm infants: correlation with magnetic resonance imaging and neurodevelopmental outcome. *Pediatrics*, *121*(6), e1534-e1540.

Cheadle, J. E., & Goosby, B. J. (2010). Birth weight, cognitive development, and life chances: A comparison of siblings from childhood into early adulthood. *Social Science Research*, *39*(4), 570-584.

Cherny, S. S., Fulker, D. W., Emde, R. N., Robinson, J., Corley, R. P., Reznick, J. S., Plomin, R. & DeFries, J. C. (1994). A developmental-genetic analysis of continuity and change in the Bayley Mental Development Index from 14 to 24 months: The MacArthur Longitudinal Twin Study. *Psychological Science*, *5*(6), 354-360. https://doi.org/10.1111/j.1467-9280.1994.tb00285.x

Cooke, R. W. (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.

Corcoran, M. (2008). Childhood Poverty, Race and Equal Opportunity. In S.B. Newman. *Educating the Other America*, pp 35-51.

Datar, A., & Jacknowitz, 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. https://doi.org/10.1007/s10995-009-0461-6

Davis, D. W., 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. https://doi.org/10.1017/thg.2019.37

Davis, O. S., Haworth, C. M., & Plomin, R. (2009). Dramatic increase in heritability of cognitive development from early to middle childhood: An 8-year longitudinal study of 8,700 pairs of twins. *Psychological Science*, *20*(10), 1301-1308. https://doi.org/10.1111%2Fj.1467-9280.2009.02433.x

Deater-Deckard, K., Mullineaux, P. Y., Beekman, C., Petrill, S. A., Schatschneider, C., & Thompson, L. A. (2009). Conduct problems, IQ, and household chaos: A longitudinal multi-informant study. *Journal of Child Psychology and Psychiatry*, *50*(10), 1301-1308. https://doi.org/10.1111/j.1469-7610.2009.02108.x

Demerath, E. W., Choh, A. C., Czerwinski, S. A., Lee, M., Sun, S. S., Chumlea, W. C., ... & Siervogel, R. M. (2007). Genetic and environmental influences on infant weight and weight change: the Fels Longitudinal Study. *American Journal of Human Biology*, *19*(5), 692-702.

Devlin, B., Daniels, M., & Roeder, K. (1997). The heritability of IQ. *Nature*, *388*(6641), 468-471. https://doi.org/10.1038/41319

Dicker, D., Nguyen, G., Abate, D., Abate, K. H., Abay, S. M., Abbafati, C., Abbasi, N., Abbastabar, H., Abd-Allah, F., Abdela, J., Abdelalim, A., Abdel-Rahman, O., Abdi, A., Abdollahpour, I., Abdulkader, R. S., Abdurahman, A. A., Abebe, H. T., Abebe, M., Abebe, Z., … & Abebo, T. A. (2018). Global, regional, and national age-sex-specific mortality and life expectancy, 1950–2017: A systematic analysis for the Global Burden of Disease Study 2017. *The Lancet*, *392*(10159), 1684–1735. https://doi.org/10.1016/s0140-6736(18)31891-9

Drewnowski, A., & Specter, S. E. (2004). Poverty and obesity: the role of energy density and energy costs. *The American Journal of Clinical Nutrition*, *79*(1), 6-16.

Dumas, J. E., Nissley, J., Nordstrom, A., Smith, E. P., Prinz, R. J., & Levine, D. W. (2005). Home chaos: Sociodemographic, parenting, interactional, and child correlates. *Journal of Clinical Child and Adolescent Psychology*, *34*(1), 93-104. https://doi.org/10.1207/s15374424jccp3401_9

Edmonds, C. J., Isaacs, E. B., Cole, T. J., Rogers, M. H., Lanigan, J., Singhal, A., ... & Lucas, A. (2010). The effect of intrauterine growth on verbal IQ scores in childhood: a study of monozygotic twins. *Pediatrics*, *126*(5), e1095-e1101.

Enders, C. K. (2011a). Analyzing longitudinal data with missing values. *Rehabilitation Psychology, 56*(4), 267–288. https://doi.org/10.1037/a0025579

Enders, C. K. (2011b). Missing not at random models for latent growth curve analyses. *Psychological Methods*, *16*(1), 1-16.

Enders, C. K. (2013). Dealing with missing data in developmental research. *Child Development Perspectives, 7*(1), 27-31.

Estourgie-van Burk, G. F., Bartels, M., Boomsma, D. I., & Delemarre-van de Waal, H. A. (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.

Estourgie-van Burk, G. F., Bartels, M., Hoekstra, R. A., Polderman, T. J., Delemarre-van de Waal, H. A., & Boomsma, D. I. (2009). A twin study of cognitive costs of low birth weight and catch-up growth. *The Journal of Pediatrics*, *154*(1), 29-32.

Estourgie-van Burk, G. F., Bartels, M., Van Beijsterveldt, T. C., Delemarre-van de Waal, H. A., & Boomsma, D. I. (2006). Body size in five-year-old twins: heritability and comparison to singleton standards. *Twin Research and Human Genetics*, *9*(5), 646-655.

Estrada, E., Ferrer, E., Román, F. J., Karama, S., & Colom, R. (2019). Time-lagged associations between cognitive and cortical development from childhood to early adulthood. *Developmental Psychology*, *55*(6), 1338.

Farrington, D. P. (2005). Childhood origins of antisocial behavior. *Clinical Psychology & Psychotherapy: An International Journal of Theory & Practice*, *12*(3), 177-190.

Fattal-Valevski, A., Toledano-Alhadef, 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.

Feldman, M. W., & Otto, S. P. (1997). Twin studies, heritability, and intelligence. *Science, 278*(5342), 1383-1387.

Fink, G., & Rockers, P. C. (2014). Childhood growth, schooling, and cognitive development: further evidence from the Young Lives study. *The American Journal of Clinical Nutrition*, *100*(1), 182-188.

Finkel, D., Davis, D. W., Turkheimer, E., & Dickens, W. T. (2015). Applying biometric growth curve models to developmental synchronies in cognitive development: The Louisville twin study. *Behavior Genetics*, *45*(6), 600-609. https://doi.org/10.1007/s10519-015-9747-1.

Flegal, K. M., Graubard, B. I., Williamson, D. F., & Gail, M. H. (2005). Excess deaths associated with underweight, overweight, and obesity. *JAMA*, *293*(15), 1861-1867.

Flensborg-Madsen, T., & Mortensen, E. L. (2017). Predictors of motor developmental milestones during the first year of life. *European Journal of Pediatrics*, *176*(1), 109-119.

Ford, G. W., Doyle, L. W., Davis, N. M., & Callanan, C. (2000). Very low birth weight and growth into adolescence. *Archives of Pediatrics & Adolescent Medicine*, *154*(8), 778-784.

Freides, D. (1972). Review of the Stanford-Binet intelligence scale, third revision. *The seventh mental measurements yearbook*, *1*, 772-773.

Fujikura, T., & Froehlich, L. A. (1974). Mental and motor development in monozygotic co-twins with dissimilar birth weights. *Pediatrics*, *53*(6), 884-889.

Gale, C. R., O'Callaghan, F. J., Godfrey, K. M., Law, C. M., & Martyn, C. N. (2004). Critical periods of brain growth and cognitive function in children. *Brain*, *127*(2), 321-329.

Gardner, J. M., Grantham-McGregor, S. M., Chang, S. M., & Powell, C. A. (1990). Dietary intake and observed activity of stunted and non-stunted children in Kingston, Jamaica. Part II: Observed activity. *European Journal of Clinical Nutrition*, *44*(8), 585-593.

Gärling , T., Evans, G. W. (1991). *Environment, cognition, and action: An integrated approach*. Oxford University Press on Demand.

Giangrande, E. J., Beam, C. R., Carroll, S., Matthews, L. J., Davis, D. W., Finkel, D., & Turkheimer, E. (2019). Multivariate analysis of the scarr-rowe interaction across middle childhood and early adolescence. *Intelligence*, *77*, 101400. https://doi.org/10.1016/j.intell.2019.101400

Giuffrè, M., Piro, E., & Corsello, G. (2012). Prematurity and twinning. *The Journal of Maternal-Fetal & Neonatal Medicine*, *25*(sup3), 6-10. https://doi.org/10.3109/14767058.2012.712350

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.

Goldenberg, R. L., Culhane, J. F., Iams, J. D., & Romero, R. (2008). Epidemiology and causes of preterm birth. *The Lancet*, *371*(9606), 75-84.

Graham, J. W. (2003). Adding missing-data-relevant variables to FIML-based structural equation models. *Structural Equation Modeling*, *10*(1), 80-100.

Grimm, K. J., & Ram, N. (2009). Nonlinear growth models in M plus and SAS. *Structural Equation Modeling*, *16*(4), 676-701. https://doi.org/10.1080%2F10705510903206055

Grimm, K. J., Ram, N., & Hamagami, F. (2011). Nonlinear growth curves in developmental research. *Child Development*, *82*(5), 1357-1371. https://doi.org/10.1111/j.1467-8624.2011.01630.x

Grimm, K. J., & Widaman, K. F. (2010). Residual structures in latent growth curve modeling. *Structural Equation Modeling*, *17*(3), 424-442.

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. https://doi.org/10.1080/00273171.2012.755111

Hack, M., Schluchter, M., Cartar, L., Rahman, M., Cuttler, L., & Borawski, E. (2003). Growth of very low birth weight infants to age 20 years. *Pediatrics*, *112*(1), e30-e38.

Hails, K. A., & Shaw, D. S. (2019). Associations between boys' early childhood exposure to family and neighborhood poverty and body mass index in early adolescence. *Journal of Pediatric Psychology*, *44*(9), 1009-1018.

Hamagami, F., & McArdle, J. J. (2018). Latent change score models with curvilinear constant bases. In *Longitudinal Multivariate Psychology* (pp. 80-108). Routledge.

Hauspie, R. C., Bergman, P., Bielicki, T., & Susanne, C. (1994). Genetic variance in the pattern of the growth curve for height: a longitudinal analysis of male twins. *Annals of Human Biology*, *21*(4), 347-362.

He, X. J., Dai, R. X., Tian, C. Q., & Hu, C. L. (2021). Neurodevelopmental outcome at 1 year in offspring of women with gestational diabetes mellitus. *Gynecological Endocrinology, 37*(1), 88-92. https://doi.org/10.1080/09513590.2020.1754785

Heinonen, K., Räikkönen, K., Pesonen, A. K., Kajantie, E., Andersson, S., Eriksson, J. G., Niemela, A., Vartia, A., Vartia, T., Peltola, J., & Lano, A. (2008). Prenatal and postnatal

growth and cognitive abilities at 56 months of age: a longitudinal study of infants born at term. *Pediatrics*, *121*(5), e1325-e1333.

Hiersch, L., Okby, R., Freeman, H., Rosen, H., Nevo, O., Barrett, J., & Melamed, N. (2020). Differences in fetal growth patterns between twins and singletons. *The Journal of Maternal-Fetal & Neonatal Medicine*, *33*(15), 2546-2555.

Hjelmborg, J. V., Fagnani, C., Silventoinen, K., McGue, M., Korkeila, M., Christensen, K., Rissanen, A., & Kaprio, J. (2008). Genetic influences on growth traits of BMI: a longitudinal study of adult twins. *Obesity*, *16*(4), 847-852.

Hollingshead, A. B. (1975). *Four factor index of social status*. Unpublished manuscript. New Haven, CT: Department of Sociology, Yale University.

Honzik, M. P., Macfarlane, J. W., & Allen, L. (1948). The stability of mental test performance between two and eighteen years. *The Journal of Experimental Education*, *17*(2), 309-324. https://doi.org/10.1080/00220973.1948.11010388

Horta, B. L., Victora, C. G., de Mola, C. L., Quevedo, L., Pinheiro, R. T., Gigante, D. P., Santos Motta, J. V., & Barros, F. C. (2017). Associations of linear growth and relative weight gain in early life with human capital at 30 years of age. *The Journal of Pediatrics*, *182*, 85-91.

Itabashi, K., Mishina, J., Tada, H., Sakurai, M., Nanri, Y., & Hirohata, Y. (2007). Longitudinal follow-up of height up to five years of age in infants born preterm small for gestational age; comparison to full-term small for gestational age infants. *Early Human Development*, *83*(5), 327-333.

Jelenkovic, A., Sund, R., Yokoyama, Y., Latvala, A., Sugawara, M., Tanaka, M., Matsumoto, S., Freitas, D. L., Maia, J. A., Knafo-Noam, A., Mankuta, D., Abramson, L., Ji, F., Ning, F., Pang, Z., Rebato, E., Saudino, K. J., Cutler, T. L., Hopper, J. L., ... & Silventoinen, K. (2020). Genetic and environmental influences on human height from infancy through adulthood at different levels of parental education. *Scientific Reports*, *10*(1), 1-11.

Jeng, S. F., Yau, K. I. T., Liao, H. F., Chen, L. C., & Chen, P. S. (2000). Prognostic factors for walking attainment in very low-birthweight preterm infants. *Early Human Development*, *59*(3), 159-173.

Jensen, R. B., Juul, A., Larsen, T., Mortensen, E. L., & Greisen, G. (2015). Cognitive ability in adolescents born small for gestational age: associations with fetal growth velocity, head circumference and postnatal growth. *Early Human Development*, *91*(12), 755-760.

Johnson, L., Llewellyn, C. H., Van Jaarsveld, C. H., Cole, T. J., & Wardle, J. (2011). Genetic and environmental influences on infant growth: prospective analysis of the Gemini twin birth cohort. *PLoS One*, *6*(5), e19918.

Johnson, S., & Marlow, N. (2006). Developmental screen or developmental testing? *Early Human Development*, *82*(3), 173-183. https://doi.org/10.1016/j.earlhumdev.2006.01.008

Kainz, K., Willoughby, M. T., Vernon-Feagans, L., Burchinal, M. R., & Family Life Project Investigators. (2012). Modeling family economic conditions and young children's development in rural United States: Implications for poverty research. *Journal of Family and Economic Issues*, *33*(4), 410-420. https://doi.org/10.1007/s10834-012-9287-2

Kelishadi, R., Haghdoost, A. A., Jamshidi, F., Aliramezany, M., & Moosazadeh, M. (2015). Low birthweight or rapid catch-up growth: which is more associated with cardiovascular disease and its risk factors in later life? A systematic review and cryptanalysis. *Paediatrics and International Child Health*, *35*(2), 110-123.

Keller, M. C., Garver-Apgar, C. E., Wright, M. J., Martin, N. G., Corley, R. P., Stallings, M. C., Hewitt, J. K., & Zietsch, B. P. (2013). The genetic correlation between height and IQ: shared genes or assortative mating?. *PLoS Genetics*, *9*(4), e1003451.

Kerr-Wilson, C. O., Mackay, D. F., Smith, G. C. S., & Pell, J. P. (2012). Meta-analysis of the association between preterm delivery and intelligence. *Journal of Public Health*, *34*(2), 209-216. https://doi.org/10.1093/pubmed/fdr024

Khan, Amira M., Bianca Carducci, and Zulfiqar A. Bhutta. "Low Birth Weight and Small for Gestational Age in the Context of 1,000 Days." *The Biology of the First 1,000 Days*. CRC Press, 2017. 171-188.

Klebanov, P. K., Brooks-Gunn, J., McCarton, C., & McCormick, M. C. (1998). The contribution of neighborhood and family income to developmental test scores over the first three years of life. *Child Development*, *69*(5), 1420-1436. https://doi.org/10.2307/1132275

Klebanov, P. K., Evans, G. W., & Brooks-Gunn, J. (2014). Poverty, ethnicity, and risk of obesity among low birth weight infants. *Journal of Applied Developmental Psychology*, *35*(3), 245-253.

Kline, J., Stein, Z., & Susser, M. (1989). Fetal growth and birth weight: I. Indices, patterns and risk factors. In B. MacMahon (Series Ed.), *Monographs in epidemiology and biostatistics: Vol. 14. Conception to birth: Epidemiology of prenatal development* (pp. 208–218). New York: Oxford University Press.

Koeppen-Schomerus, G., Eley, T. C., Wolke, D., Gringras, P., & Plomin, R. (2000). The interaction of prematurity with genetic and environmental influences on cognitive development in twins. *The Journal of Pediatrics*, *137*(4), 527-533. https://doi.org/10.1067/mpd.2000.108445

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.

Kowaleski-Jones, L., & Duncan, G. J. (2002). Effects of participation in the WIC program on birthweight: evidence from the national longitudinal survey of youth. *American Journal of Public Health*, *92*(5), 799-804.

Kozieł, S. M. (1998). Effect of disparities in birth weight on differences in postnatal growth of monozygotic and dizygotic twins. *Annals of Human Biology*, *25*(2), 159-168.

Kuczmarski, R. J. (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.

Kuklina, E. V., Ramakrishnan, U., Stein, A. D., Barnhart, H. H., & Martorell, R. (2006). Early childhood growth and development in rural Guatemala. *Early Human Development*, *82*(7), 425-433.

Laar, M. E., Marquis, G. S., Lartey, A., & Gray-Donald, K. (2018). Reliability of length measurements collected by community nurses and health volunteers in rural growth monitoring and promotion services. *BMC Health Services Research*, *18*(1), 1-10.

Lee, H., Andrew, M., Gebremariam, A., Lumeng, J. C., & Lee, J. M. (2014). Longitudinal associations between poverty and obesity from birth through adolescence. *American Journal of Public Health*, *104*(5), e70-e76.

Li, C., Zeng, L., Wang, D., Allen, S., Jaffar, S., Zhou, J., Chen, T., Watson, V., & Yan, H. (2019). Growth in early life and physical and intellectual development at school age: a cohort study. *British Journal of Nutrition, 121*(8), 866-876.

Liao, A. W., Brizot, M. D. L., Kang, H. J., Assunçao, R. A., & Zugaib, M. (2012). Longitudinal reference ranges for fetal ultrasound biometry in twin pregnancies. *Clinics, 67*(5), 451-455.

Livshits, G., Peter, I., Vainder, M., & Hauspie, R. (2000). Genetic analysis of growth curve parameters of body weight, height and head circumference. *Annals of Human Biology, 27*(3), 299-312.

Liu, Q., Yu, C., Gao, W., Cao, W., Lyu, J., Wang, S., Pang, Z., Cong, L., Dong, Z., Wu, F., Wang, H., Wu, X., Jiang, G., Wang, B., & Li, L. (2015). Genetic and environmental effects on weight, height, and BMI under 18 years in a Chinese population-based twin sample. *Twin Research and Human Genetics, 18*(5), 571-580.

Llewellyn, C. H., Van Jaarsveld, C. H., Johnson, L., Carnell, S., & Wardle, J. (2010). Nature and nurture in infant appetite: analysis of the Gemini twin birth cohort. *The American Journal of Clinical Nutrition, 91*(5), 1172-1179.

Lovasi, G. S., Schwartz-Soicher, O., Quinn, J. W., Berger, D. K., Neckerman, K. M., Jaslow, R., Lee, K. K. & Rundle, A. (2013). Neighborhood safety and green space as predictors of obesity among preschool children from low-income families in New York City. *Preventive Medicine, 57*(3), 189-193.

Luke, B., & Brown, M. B. (2007). Elevated risks of pregnancy complications and adverse outcomes with increasing maternal age. *Human Reproduction, 22*(5), 1264-1272. https://doi.org/10.1093/humrep/del522

Luke, B., & Keith, L. G. (1992). The contribution of singletons, twins and triplets to low birth weight, infant mortality and handicap in the United States. *The Journal of Reproductive Medicine, 37*(8), 661-666.

Lundgren, E. M., Cnattingius, S., Jonsson, B., & Tuvemo, T. (2001). Intellectual and psychological performance in males born small for gestational age with and without catch-up growth. *Pediatric Research, 50*(1), 91-96.

Mahanta, D. J., & Borah, M. (2014). Parameter Estimation of Weibull Growth Models in Forestry. *International Journal of Mathematics Trends and Technology, 8*(3), 157-163. http://dx.doi.org/10.14445/22315373/IJMTT-V8P521

Mangin, K. S., Horwood, L. J., & Woodward, L. J. (2017). Cognitive development trajectories of very preterm and typically developing children. *Child Development, 88*(1), 282-298. https://doi.org/10.1111/cdev.12585

Marcoulides, K. M. (2019). Reliability estimation in longitudinal studies using latent growth curve modeling. *Measurement: Interdisciplinary Research and Perspectives, 17*(2), 67-77.

Marceau, K., McMaster, M. T., Smith, T. F., Daams, J. G., van Beijsterveldt, C. E., Boomsma, D. I., & Knopik, V. S. (2016). The prenatal environment in twin studies: a review on chorionicity. *Behavior Genetics, 46*(3), 286-303.

Martin, A., Connelly, A., Bland, R. M., & Reilly, J. J. (2017). Health impact of catch-up growth in low-birth weight infants: systematic review, evidence appraisal, and meta-analysis. *Maternal & Child Nutrition, 13*(1).

Martin, J. A., Hamilton, B. E., Osterman, M. J., & Driscoll, A. K. (2021). Births: Final Data for 2019. *National Vital Statistics Reports: From the Centers for Disease Control and*

Prevention, National Center for Health Statistics, National Vital Statistics System, 70(2), 1-51.

Martin, A., Razza, R. A., & Brooks-Gunn, J. (2012). Specifying the links between household chaos and preschool children's development. *Early Child Development and Care*, *182*(10), 1247-1263. https://dx.doi.org/10.1080%2F03004430.2011.605522

Matheny Jr, A. P., Wachs, T. D., Ludwig, J. L., & Phillips, K. (1995). Bringing order out of chaos: Psychometric characteristics of the confusion, hubbub, and order scale. *Journal of Applied Developmental Psychology*, *16*(3), 429-444. https://doi.org/10.1016/0193-3973(95)90028-4

Matusik, J. G., Hollenbeck, J. R., & Mitchell, R. L. (2020). Latent Change Score Models for the Study of Development and Dynamics in Organizational Research. *Organizational Research Methods*, 1094428120963788.

McArdle, J. J. (2001). A latent difference score approach to longitudinal dynamic structural analyses. In R. Cudeck, S. duToit, & D. Sorbom (Eds.), Structural equation modeling: Present and future (pp. 342-380). Lincolnwood, I: Scientific Software International.

McArdle, J. J., & Prescott, C. A. (2005). Mixed-effects variance components models for biometric family analyses. *Behavior Genetics*, *35*(5), 631-652. https://doi.org/10.1007/s10519-005-2868-1

McCarthy, D. (1972). *Manual for the McCarthy Scales of Children's Abilities.* New York: Psychological Corporation.

McNeish, D., & Dumas, D. (2017). Nonlinear growth models as measurement models: A second-order growth curve model for measuring potential. *Multivariate Behavioral Research*, *52*(1), 61-85.

Messinger, D. S., Bauer, C. R., Das, A., Seifer, R., Lester, B. M., Lagasse, L. L., Wright, L. L., Shankaran S., Bada, H. S., Smeriglio, V. L., Langer, J. C., Beeghly, M., & Poole, W. K. (2004). The maternal lifestyle study: cognitive, motor, and behavioral outcomes of cocaine-exposed and opiate-exposed infants through three years of age. *Pediatrics, 113(6)*, 1677-1685. https://doi.org/10.1542/peds.113.6.1677

Mook-Kanamori, D. O., Van Beijsterveldt, C. E., Steegers, E. A., Aulchenko, Y. S., Raat, H., Hofman, A., Eilers, P. H., Boomsma, D. I., & Jaddoe, V. W. (2012). Heritability estimates of body size in fetal life and early childhood. *PLoS One, 7*(7), e39901.

Morag, I., Stern Levkovitz, O., Siman-Tov, M., Frisch, M., Pinhas-Hamiel, O., & Strauss, T. (2018). Postnatal growth disadvantage of the small for gestational age preterm twins. *Nutrients*, *10*(4), 476-486.

Morgan, P. H., Mercer, L. P., & Flodin, N. W. (1975). General model for nutritional responses of higher organisms. *Proceedings of the National Academy of Sciences, 72*(11), 4327-4331. https://doi.org/10.1073/pnas.72.11.4327

Morris, J., Orioli, I. M., Benavides-Lara, A., de la Paz Barboza-Arguello, M., Tapia, M. A. C., de França, G. V. A., Groisman, B., Holguin, J., Hurtado-Villa, P. M., Ramirez, M. I., Mellado, C., Pardo, R., Bucardo, D. M. P., Rodriguez, C., Zarante, I., Limb, E. & Dolk, H. (2021). Prevalence of microcephaly: the Latin American Network of Congenital Malformations 2010–2017. *BMJ Paediatrics Open*, *5*(1).

Muthén, L. K., & Muthén, B. O. (2017). Mplus. Statistical analysis with latent variables. User's guide, 8.

Niklasson, A., Engström, E., Hård, A. L., Wikland, K. A., & Hellström, A. (2003). Growth in very preterm children: a longitudinal study. *Pediatric Research*, *54*(6), 899-905.

Oken, E., Kleinman, K. P., Rich-Edwards, J., & Gillman, M. W. (2003). A nearly continuous measure of birth weight for gestational age using a United States national reference. *BMC Pediatrics*, *3*(1), 1-10.

Peterson, M., Warf, B. C., & Schiff, S. J. (2018). Normative human brain volume growth. *Journal of Neurosurgery: Pediatrics*, *21*(5), 478-485.

Petrill, S. A., Pike, A., Price, T., & Plomin, R. (2004). Chaos in the home and socioeconomic status are associated with cognitive development in early childhood: Environmental mediators identified in a genetic design. *Intelligence*, *32*(5), 445-460. https://doi.org/10.1016/j.intell.2004.06.010

Phillips, K., & Matheny, A. P. (1990). Quantitative genetic analysis of longitudinal trends in height: preliminary results from the Louisville Twin Study. *Acta Geneticae Medicae et Gemellologiae: Twin Research*, *39*(2), 143-163.

Pongcharoen, T., Ramakrishnan, U., DiGirolamo, A. M., 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.

Poveda, N. E., Hartwig, F. P., Victora, C. G., Adair, L. S., Barros, F. C., Bhargava, S. K., Horta, B. L., Lee, N. R., Martorell, R., Mazariegos, M., Menees, A. M. B., Norris, S. A., Richter, L. M., Sachdev, H. S., Stein, A., Wehrmeister, F. C., Stein, A. D., & COHORTS Group. (2021). Patterns of Growth in Childhood in Relation to Adult Schooling Attainment and Intelligence Quotient in 6 Birth Cohorts in Low-and Middle-Income Countries: Evidence from the Consortium of Health-Oriented Research in Transitioning Societies (COHORTS). *The Journal of Nutrition*, *151*(8):2342-2352.

Price, T. S., Eley, T. C., Dale, P. S., Stevenson, J., Sandino, K., & Plomin, R. (2000). Genetic and environmental covariation between verbal and nonverbal cognitive development in infancy. *Child Development*, *71*(4), 948-959. https://doi.org/10.1111/1467-8624.00201

Ramey, C. T., Campbell, F. A., & Nicholson, J. E. (1973). The predictive power of the Bayley Scales of Infant Development and the Stanford-Binet Intelligence Test in a relatively constant environment. *Child Development*, 790-795. https://doi.org/10.2307/1127725

Ratkowsky, D. (1983). *Nonlinear regression modelling*. Marcel Dekker, New York.

Rigo, J., & Senterre, J. (2006). Nutritional needs of premature infants: current issues. *The Journal of pediatrics*, *149*(5), S80-S88.

Rindskopf, D. (1984). Using phantom and imaginary latent variables to parameterize constraints in linear structural models. Psychometrika, 49, 37–47. doi:10.1007/bf02294204

Robinson, B. F., & Mervis, C. B. (1998). Disentangling early language development: Modeling lexical and grammatical acquisition using and extension of case-study methodology. *Developmental Psychology*, *34*(2), 363.

Roh, L., Braun, J., Chiolero, A., Bopp, M., Rohrmann, S., & Faeh, D. (2014). Mortality risk associated with underweight: a census-linked cohort of 31,578 individuals with up to 32 years of follow-up. *BMC Public Health*, *14*(1), 1-9.

Ross, G. S., Krauss, A. N., & Perlman, J. M. (2012). Physical growth and cognitive abilities in concordant versus discordant birth weight twins at three years old. *Early Human Development*, *88*(9), 753-756.

Rubin, D. B. (1976). Inference and missing data. *Biometrika*, *63*(3), 581-592.

Rutter, M., & Sroufe, L. A. (2000). Developmental psychopathology: Concepts and challenges. *Development and Psychopathology*, *12*(3), 265-296.

Satorra, A., & Bentler, P. M. (2001). A scaled difference chi-square test statistic for moment structure analysis. *Psychometrika*, *66*(4), 507-514.

Satorra, A., & Bentler, P. M. (2010). Ensuring positiveness of the scaled difference chi-square test statistic. *Psychometrika*, *75*(2), 243-248.

Scharf, R. J., Rogawski, E. T., Murray-Kolb, L. E., Maphula, A., Svensen, E., Tofail, F., Rasheed, M., Abreu, C., Vasquez, A. O., Shrestha, R., Pendergast, L., Mduma, E., Koshy, B., Conaway, M. R., Platts-Mills, J. A., Guerrant, R. L., & DeBoer, M. D. (2018). Early childhood growth and cognitive outcomes: Findings from the MAL-ED study. *Maternal & Child Nutrition*, *14*(3), e12584. https://doi.org/10.1111/mcn.12584

Scharf, R. J., Stroustrup, A., Conaway, M. R., & DeBoer, M. D. (2016). Growth and development in children born very low birthweight. *Archives of Disease in Childhood-Fetal and Neonatal Edition*, *101*(5), F433-F438.

Schneider, W., Niklas, F., & Schmiedeler, S. (2014). Intellectual development from early childhood to early adulthood: The impact of early IQ differences on stability and change over time. *Learning and Individual Differences*, *32*, 156-162. https://doi.org/10.1016/j.lindif.2014.02.001

Schulte, S., Wölfle, J., Schreiner, F., Stoffel-Wagner, B., Peter, M., Bartmann, P., & Gohlke, B. (2016). Birthweight differences in monozygotic twins influence pubertal maturation and near final height. *The Journal of Pediatrics*, *170*, 288-294.

Shah-Kulkarni, S., Lee, S., Jeong, K. S., Hong, Y. C., Park, H., Ha, M., Kim, Y., & Ha, E. H. (2020). Prenatal exposure to mixtures of heavy metals and neurodevelopment in infants at 6 months. *Environmental Research*, *182*, 109122. https://doi.org/10.1016/j.envres.2020.109122

Shenkin, S. D., Starr, J. M., & Deary, I. J. (2004). Birth weight and cognitive ability in childhood: a systematic review. *Psychological Bulletin*, *130*(6), 989-1013.

Siegel, L. S. (1981). Infant tests as predictors of cognitive and language development at two years. *Child Development*, *52*(2), 545-557. https://psycnet.apa.org/doi/10.2307/1129173

Silventoinen, K., Iacono, W. G., 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.

Silventoinen, K., Kaprio, J., & Lahelma, E. (2000a). Genetic and environmental contributions to the association between body height and educational attainment: a study of adult Finnish twins. *Behavior Genetics*, *30*(6), 477-485.

Silventoinen, K., Kaprio, J., Lahelma, E., & Koskenvuo, M. (2000b). Relative effect of genetic and environmental factors on body height: differences across birth cohorts among Finnish men and women. *American Journal of Public Health*, *90*(4), 627.

Silventoinen, K., Pitkäniemi, J., Latvala, A., Kaprio, J., & Yokoyama, Y. (2014). Association between physical and motor development in childhood: a longitudinal study of Japanese twins. *Twin Research and Human Genetics*, *17*(3), 192-198.

Silventoinen, K., Posthuma, D., Van Beijsterveldt, T., Bartels, M., & Boomsma, D. I. (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.

Silventoinen, K., Sammalisto, S., Perola, M., Boomsma, D. I., Cornes, B. K., Davis, C., Dunkel, L., de Lange, M., Harris, J. R., Hjelmborg, J. V. B., Luciano, M., Martin, N. G., Mortensen, J., Nistico, L., Pedersen, N. L., Skytthe, A., Spector, T. D., Stazzi, M. A.,

Willemsen, G., & Kaprio, J. (2003). Heritability of adult body height: a comparative study of twin cohorts in eight countries. *Twin Research and Human Genetics*, *6*(5), 399-408.

Simpson, L. L., & Society for Maternal-Fetal Medicine (SMFM. (2013). Twin-twin transfusion syndrome. *American Journal of Obstetrics and Gynecology*, *208*(1), 3-18.

Singer, J. D., Willett, J. B., & Willett, J. B. (2003). *Applied longitudinal data analysis: Modeling change and event occurrence*. Oxford university press.

Smit, D. J., Luciano, M., Bartels, M., Van Beijsterveldt, C. E., Wright, M. J., Hansell, N. K., Brunner, H. G., Estourgie-van Burk, G. F., de Gues, E. J. C., Martin, N. G., & Boomsma, D. I. (2010). Heritability of head size in Dutch and Australian twin families at ages 0–50 years. *Twin Research and Human Genetics*, *13*(4), 370-380.

Ssentongo, P., Ssentongo, A. E., Ba, D. M., Ericson, J. E., Na, M., Gao, X., Fronterre, C., Chinchilli, V. M., & Schiff, S. J. (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.

Stevenson, D. K., Verter, J., Fanaroff, A. A., Oh, W., Ehrenkranz, R. A., Shankaran, S., Donovan, E. F., Wright, L. L., Lemons, J. A., Tyson, J. E., Korones, S. B., Bauer, C. R., Stoll, B. J., & Papile, L. A. (2000). Sex differences in outcomes of very low birthweight infants: the newborn male disadvantage. *Archives of Disease in Childhood-Fetal and Neonatal Edition*, *83*(3), F182-F185.

Sudfeld, C. R., McCoy, D. C., Danaei, G., Fink, G., Ezzati, M., Andrews, K. G., & Fawzi, W. W. (2015). Linear growth and child development in low-and middle-income countries: a meta-analysis. *Pediatrics*, *135*(5), e1266-e1275.

Sundet, J. M., Tambs, K., Harris, J. R., Magnus, P., & Torjussen, T. M. (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.

Swamy, R. S., McConachie, H., Ng, J., Rankin, J., Korada, M., Sturgiss, S., & Embleton, N. D. (2018). Cognitive outcome in childhood of birth weight discordant monochorionic twins: the long-term effects of fetal growth restriction. *Archives of Disease in Childhood-Fetal and Neonatal Edition*, *103*(6), F512-F516.

Tariq, M. M., Iqbal, F., Eyduran, E., Bajwa, M. A., Huma, Z. E., & Waheed, A. (2013). Comparison of non-linear functions to describe the growth in Mengali sheep breed of Balochistan. *Pakistan Journal of Zoology*, *45*(3), 661-665.

Testa, M., Quigley, B. M., & Eiden, R. D. (2003). The effects of prenatal alcohol exposure on infant mental development: a meta-analytical review. *Alcohol and Alcoholism*, *38*(4), 295-304. https://doi.org/10.1093/alcalc/agg087

Tucker-Drob, E. M., & Briley, D. A. (2014). Continuity of genetic and environmental influences on cognition across the life span: a meta-analysis of longitudinal twin and adoption studies. *Psychological Bulletin*, *140*(4), 949-979. https://doi.org/10.1037%2Fa0035893

Tucker-Drob, E. M., Rhemtulla, M., Harden, K. P., Turkheimer, E., & Fask, D. (2011). Emergence of a gene× socioeconomic status interaction on infant mental ability between 10 months and 2 years. *Psychological Science*, *22*(1), 125-133.

United Nations Children's Fund (2021). *UNICEF/WHO/World Bank joint child malnutrition estimates (global and regional)*. https://data.unicef.org/wp-

content/uploads/2021/04/Joint-Malnutrition-Estimates-Regional-and-Global-Estimates-April-2021.xlsx

Upadhyay, R. P., Naik, G., Choudhary, T. S., Chowdhury, R., Taneja, S., Bhandari, N., Martines, J. C., Bahl, R., & Bhan, M. K. (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.

Van Dommelen, P., De Gunst, M., Van Der Vaart, A., Van Buuren, S., & Boomsma, D. (2008). Growth references for height, weight and body mass index of twins aged 0–2.5 years. *Acta Paediatrica*, *97*(8), 1099-1104.

Varella, M. H., & Moss, W. J. (2015). Early growth patterns are associated with intelligence quotient scores in children born small-for-gestational age. *Early Human Development*, *91*(8), 491-497.

van Baal, C. G., & Boomsma, D. I. (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.

van Dommelen, P., De Gunst, M. C., Van Der Vaart, A. W., & Boomsma, D. I. (2004). Genetic study of the height and weight process during infancy. *Twin Research and Human Genetics*, *7*(6), 607-616.

Villar, J., Ismail, L. C., Victora, C. G., Ohuma, E. O., Bertino, E., Altman, D. G., ... & Kennedy, S. H. (2014). International standards for newborn weight, length, and head circumference by gestational age and sex: the Newborn Cross-Sectional Study of the INTERGROWTH-21st Project. *The Lancet*, *384*(9946), 857-868.

Voelkle, M. C., & Oud, J. H. (2015). Relating latent change score and continuous time models. *Structural Equation Modeling: A Multidisciplinary Journal*, *22*(3), 366-381.

von Stumm, S., & Plomin, R. (2015). Socioeconomic status and the growth of intelligence from infancy through adolescence. *Intelligence*, *48*, 30-36. https://doi.org/10.1016/j.intell.2014.10.002

Wadhwa, P. D., Sandman, C. A., Porto, M., Dunkel-Schetter, C., & Garite, T. J. (1993). The association between prenatal stress and infant birth weight and gestational age at birth: a prospective investigation. *American Journal of Obstetrics and Gynecology*, *169*(4), 858-865. https://doi.org/10.1016/0002-9378(93)90016-c

Webbink, D., Posthuma, D., Boomsma, D. I., de Geus, E. J., & Visscher, P. M. (2008). Do twins have lower cognitive ability than singletons?. *Intelligence*, *36*(6), 539-547. https://doi.org/10.1016/j.intell.2007.12.002

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.

Wen, Y., Liu, K., Liu, H., Cao, H., Mao, H., Dong, X., & Yin, Z. (2019). Comparison of nine growth curve models to describe growth of partridges (Alectoris chukar). *Journal of Applied Animal Research. 47*(1), 195-200.

Wijlaars, L. P., Johnson, L., van Jaarsveld, C. H., & Wardle, J. (2011). Socioeconomic status and weight gain in early infancy. *International Journal of Obesity*, *35*(7), 963-970.

Wilson, R. S. (1972). Twins: Early mental development. *Science*, *175*(4024), 914-917.

Wilson, R. S. (1974a). Twins: Mental development in the preschool years. *Developmental Psychology, 10*(4), 580–588. https://doi.org/10.1037/h0036596

Wilson, R. S. (1974b). Twins: Measures of birth size at different gestational ages. *Annals of Human Biology*, *1*(1), 57-64.

Wilson, R. S. (1975). Twins: Patterns of cognitive development as measured on the Wechsler Preschool and Primary Scale of Intelligence. *Developmental Psychology*, *11*(2), 126.

Wilson, R. S. (1979). Twin growth: initial deficit, recovery, and trends in concordance from birth to nine years. *Annals of Human Biology, 6*(3), 205-220. https://doi.org/10.1080/03014467900007212

Wilson, R. S. (1983). The Louisville twin study: Developmental synchronies in behavior. *Child Development, 54*(2), 298-316. https://psycnet.apa.org/doi/10.2307/1129693

Wolfinger, R. (1993). Covariance structure selection in general mixed models. *Communications in Statistics-Simulation and Computation*, *22*(4), 1079-1106.

Yang, W., Kelly, T., & He, J. (2007). Genetic epidemiology of obesity. *Epidemiologic Reviews*, *29*(1), 49-61.

Yu, H., McCoach, D. B., Gottfried, A. W., & Gottfried, A. E. (2018). Stability of intelligence from infancy through adolescence: An autoregressive latent variable model. *Intelligence, 69*, 8-15. https://doi.org/10.1016/j.intell.2018.03.011

Yule, W., Gold, R. D., & Busch, C. (1982). Long-term predictive validity of the WPPSI: An 11-year follow-up study. *Personality and Individual Differences*, *3*(1), 65-71. https://doi.org/10.1016/0191-8869(82)90075-7