Obstetrical and Neonatal Complications, Prematurity, and Childhood Effortful Control Development: A Longitudinal Twin Study

by

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ABSTRACT

Background: Premature infants may be at risk for lower effortful control, and subsequent lower academic achievement, peer competence, and emotional and physical wellness throughout the lifespan. However, because prematurity is related to obstetrical and neonatal complications, it is unclear what may drive the effect. Effortful control also has a strong heritable component; therefore, environmental factors during pregnancy and the neonatal period may interact with genetic factors to predict effortful control development. In this study, I aimed to dissect the influences of genetics, prematurity, and neonatal and obstetrical complications on the development of effortful control from 12 months to 10 years using a twin cohort. Methods: This study used data from the Arizona Twin Project, an ongoing longitudinal study of approximately 350 pairs of twins. Twins were primarily Hispanic/Latinx (23.8%-27.1%) and non-Hispanic/Latinx White (53.2%-57.8%), and families ranged in socioeconomic status with around one-third falling below or near the poverty line. Of the twins, 62.6% were born prematurely. Effortful control was assessed via parent report at six waves. Results: There was not a significant relationship between gestational age and effortful control regardless of whether obstetrical and neonatal complications were controlled for. Biometric twin modeling revealed that the attentional focusing subdomain of effortful control was highly heritable. Gestational age did not moderate genetic and environmental estimates. Conclusions: The findings help inform the risk assessment of prematurity and provide evidence for differing etiology of each subdomain of effortful control and the strong role of genetics in effortful control development.

DEDICATION

This document is a conglomerate of hard work and support from colleagues at Arizona State and my family and friends. I would specifically like to thank my mom, my dad, and my best friends and sisters – Andrea, Katelyn, and Erin. Finally, I would like to thank my fiancé, Eric, for his unwavering support and love.

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De-identified data are available from the study principal investigators upon reasonable request. All relevant code for this study's analyses is available at https://osf.io/qpmwc/?view_only=7b1fb9f351ee479a92e5fc2d85ba95e7.

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Obstetrical and Neonatal Complications, Prematurity, and Childhood Effortful Control Development: A Longitudinal Twin Study

Effortful control is the ability to control and focus attentional resources and inhibit behavioral responses to regulate emotions and behaviors in the service of reaching goals (Rothbart et al., 2006). Children with lower effortful control struggle to focus in school and maintain healthy relationships and habits, whereas children with higher effortful control exhibit greater academic achievement, peer competence, and emotional and physical wellness throughout the lifespan (Eisenberg et al., 2009; Obradović, 2010; Olson et al., 2017; Poehlmann et al., 2010).

Research spanning multiple fields demonstrates that premature infants may have lower effortful control when compared to full-term infants in infancy and early childhood (Cassiano et al., 2020; Consentino-Rocha et al., 2014; Lejeune et al., 2015; Reyes et al., 2020; Voigt et al., 2012), highlighting the importance of understanding the developmental predictors of effortful control in this at-risk population. Furthermore, although effortful control is highly heritable, environmental influences also play an important role in its development, and a small body of research in middle childhood suggests that the extent to which effortful control is genetically influenced may vary depending on environmental factors (Lemery-Chalfant et al., 2013). Thus, it is important to consider the prenatal environment not only as a longitudinal predictor of effortful control across early and middle childhood, but also as a potential influence on its broader genetic and environmental etiology.

I present a novel study that closely examined the links between gestational age and effortful control in a large, diverse community sample. The study used comprehensive measures of obstetrical and neonatal complications in predicting specific facets of effortful control at multiple ages. I also used a twin design to examine the environmental and genetic contributions to effortful control from infancy through middle childhood. Furthermore, I examined gestational age as a moderator of environmental and genetic contributions to effortful control at multiple ages. Overall, this study advances the literature on the relation between gestational age and effortful control in a number of novel and important ways.

Prematurity and Effortful Control

Theoretical Approach

Prematurity may be related to effortful control development because brain systems theorized to be responsible for effortful control, namely the anterior cingulate gyrus and prefrontal cortex, do not advance rapidly until late gestation (Adams-Chapman, 2006; Lean et al., 2017; Poehlmann et al., 2010). Prematurity, or factors associated with prematurity, may also introduce harmful environments such as extended exposure to painful treatments, infection, or overstimulation during critical points of pregnancy (Cassiano et al., 2017; Klein et al., 2009; Klein et al., 2013; Poehlmann et al., 2010; Voigt et al., 2014). According to fetal programming theory, exposure to such environments may cause delays or long-term deficits in physical and cognitive development (Godfrey & Barker, 2001). Furthermore, exposure to harmful environments during the neonatal stage, sensitive period of development during infants' first four

weeks postpartum, may lead to similar long-term deficits (Skuse et al., 1994). Although there are many potential mechanisms through which this may happen, the most explored mechanisms are summarized below.

When infants are born prematurely, they may experience significant physical stress, including, but not limited to invasive medical procedures, risky surgeries, respiratory distress, and inflammation from infections (Mayo Clinic, 2023). These medical risks have the potential to damage the anterior cingulate gyrus or prefrontal cortex or expose infants to more overwhelming stimuli, such as extended periods in neonatal intensive care units (NICU). In addition, premature infants often have higher rates of obstetrical complications, which can also exacerbate risk for developmental deficits (Murphy et al., 2005). This relationship may also be explained by damage to the anterior cingulate gyrus and prefrontal cortex (Murphy et al., 2005). For example, maternal smoking during pregnancy is related to prefrontal cortex development delays (Bublitz & Stroud, 2011). Premature infants may also be suddenly exposed to sensory stimuli outside of the womb at a critical period of brain development, and this exposure alters neural development of attention skills (Pineda et al., 2014).

The Current Literature

The literature on gestational age, neonatal complications, obstetrical complications, and effortful control shows an overall pattern linking lower gestational age with lower effortful control, but findings vary based on differences in measurement and sample.

First, findings differ based on the subdomain of effortful control studied. Effortful control is typically divided into attentional focusing, inhibitory control, and activation control in middle childhood studies (TMCQ; Simonds, 2006). Attentional focusing is the ability to maintain focus on a task while ignoring distracting stimuli, such as working on homework while little siblings play. Inhibitory control is the ability to suppress impulsive thoughts and reactions, such as resisting talking over others. Finally, activation control is the ability to complete an action when there is a tendency to avoid it, such as completing chores. In infancy, duration of orienting is commonly studied as a precursor to effortful control (IBQ-R; Gartstein & Rothbart, 2003). Duration of orienting is the length of time that an infant can focus on one task, such as how long an infant can play with one toy. Duration of orienting during the first year of life has been related to effortful control outcomes during preschool (Gartstein et al., 2009).

Perhaps the best evidence for the relationship between prematurity and effortful control comes from a recent meta-analysis that analyzed 22 articles on the relationship between prematurity and effortful control in infancy and early childhood and found that very premature infants (less than 32 weeks gestation) had lower levels of attentional focusing compared to full-term infants (d = 0.48), but not inhibitory control (d = 0.13), or activation control (d = -0.22; Cassiano et al., 2020).

Other studies have also found a link between prematurity and effortful control that appears to depend at least in part on the extent of prematurity (Lejeune et al., 2015; Reyes et al., 2020; Voigt et al., 2012). Typically, prematurity is divided into infants born very preterm (less than 32 weeks gestation), moderately preterm (between 32 weeks gestation

and 37 weeks gestation), and full-term infants (at least 37 weeks gestation). In comparisons between very preterm infants and full-term infants, preterm infants showed lower levels of effortful control (Cassiano et al., 2017; Voigt et al., 2012). However, when Voigt et al. (2012) compared moderately preterm infants to full-term infants and Cassiano et al. (2017) compared very preterm infants to moderately preterm infants, there were no significant differences between groups in effortful control. In a study by Olafsen et al. (2008), which included all infants born before 37 weeks, there were also no significant differences in effortful control. Taken together, these findings suggest that deficits in effortful control may be evident only in extreme cases of prematurity.

The current literature also includes studies that have utilized observational measures of effortful control, including delay of gratification tasks (Cassiano et al., 2020; Lejeune et al., 2015; Voigt et al., 2012) and parent-report measures, most commonly the Rothbart temperament questionnaires (i.e., Infant Behavior Questionnaires; Cassiano et al., 2020; Consentino-Rocha et al., 2014; Reyes et al., 2020; Voigt et al., 2012). Lower effortful control in children with prematurity is seen in studies that employed both observational tasks and parent-report measures (Consentino-Rocha et al., 2014; Lejeune et al., 2015; Voigt et al., 2012; Voigt et al., 2014). A distinct advantage of parent reports is the ease of administration which facilitates the study of larger and more representative samples. The age-appropriate Rothbart temperament questionnaires that I relied on in the current study are reliable and valid, with primary caregiver report of effortful control and observational assessments loading on the same latent factors (Sulik et al., 2010) and share genetic and environmental influences (Rea-Sandin et al., 2023). Because the majority of

studies comprising the current literature have also examined effortful control in relation to prematurity without controlling for neonatal medical risk, these studies are unable to clarify whether medical risks are a potential mechanism linking premature birth and the subsequent development of effortful control. Some studies, however, have begun to try to disentangle gestational age from neonatal medical risk.

Prematurity, Neonatal Medical Risk, and Effortful Control

When studies control for neonatal medical risk, the relationship between prematurity and effortful control is generally non-significant (Cassiano et al., 2017; Kerestes, 2005; Klein et al., 2009; Klein et al., 2013; Voigt et al., 2014), but one study has found a marginally significant relationship between gestational age and effortful control, even when controlling for neonatal medical risk (Klein et al., 2013).

Importantly, although various studies have controlled for different neonatal risks (i.e., length of stay in the NICU (Cassiano et al., 2017), number of painful procedures (Klein et al., 2009; Voigt et al., 2014), and major medical risks such as cerebral palsy (Klein et al., 2013), findings have been consistent. In fact, neonatal medical risk has been negatively related to effortful control (Poehlmann et al., 2010), suggesting a potential path from prematurity to medical risk and medical risk to lower effortful control.

Limitations in the Study of Prematurity and Effortful Control

Despite the evidence for a relation between prematurity and effortful control, current research is mainly limited to cross-sectional studies of small samples (n = ~50 premature infants; Cosentino-Rocha et al., 2014; Lejeune et al., 2015, Voigt et al., 2012) of very preterm or extremely preterm infants in infancy and early childhood (Lejeune et

al., 2015; Reyes et al., 2020; Voigt et al., 2012). This has the potential to confound results because extremely (<28 weeks) and very (<32 weeks) preterm infants likely have other neonatal medical risks, and it may be these risks, rather than preterm birth, that explain associations with effortful control. Furthermore, there are no studies which look at the link between prematurity and effortful control outcomes beyond six years old, so it is unknown if any early deficits associated with prematurity and/or medical risks continue into middle childhood.

Twin Pregnancies, Risks, and Prematurity

The effects of prematurity versus the effects of neonatal and obstetrical complications are particularly relevant questions when studying twins, as over 50% of twins are born prematurely (Goldenberg et al., 2008). Twin pregnancies are automatically considered high risk, as twins are more likely than singletons to experience complications during pregnancy and birth (Rao et al., 2004), but medical risk is not the only reason that twins are more likely than singletons to be born prematurely. For example, many twins are born prior to 37 weeks gestation due to limited space available in the womb (Basso & Wilcox, 2010). Thus, twin samples are ideal for differentiating the influences of gestational age from those of neonatal and obstetrical complications, while also allowing consideration of genetic influences on effortful control development.

The Heritability of Effortful Control

Effortful control, like other aspects of temperament, is explained by a complex combination of genetics and environment (Posner & Rothbart, 2007). Twin and adoption studies converge on the finding that genetic influences play an important role in effortful

control (e.g., Auerbach et al., 2001; Ganiban et al., 2021; Lemery-Chalfant et al., 2008), and one genome-wide association study of the overlapping construct of executive function points to contributions of 129 independent genome-wide significant lead variants in 112 distinct loci (Hatoum et al, 2020).

Specifically, twin studies from early childhood to adolescence find that effortful control shows moderate to high heritability, defined as the proportion of phenotypic variance in a trait within a given sample at a given time. Studies have shown that between 49% and 79% of the variance in effortful control is attributable to broad genetic influences, with some evidence for shared environmental influences in early childhood (Fagnani et al., 2017; Gagne & Saudino, 2016; Lemery-Chalfant et al., 2008; Yamagata et al., 2005). These studies suggest that heritability may be highest in middle childhood and decrease through adolescence and young adulthood, but differences in heritability may also be due to differences in measurement, sample characteristics, or random variation. Longitudinal research following twins across developmental periods is needed to understand developmental patterns in heritability. In addition, there is some evidence of variability depending on the subdomain of effortful control studied (Gagne & Saudino, 2016; Lemery-Chalfant et al., 2008; Yamagata et al., 2005), such that inhibitory control is less heritable compared to the other domains of activation and attentional control (Yamagata et al., 2005), which may be as high as 83% heritable (Lemery-Chalfant et al., 2008). However, replication of this finding across multiple samples is still needed.

Furthermore, even strong genetic influences must be interpreted in the context of a child's environment. Genetically informed research has begun to explore the role of

gene-environment interplay in the development of effortful control (e.g., Ganiban et al., 2021; Lemery-Chalfant et al., 2013).

Gene-Environment Interactions

According to the biopsychosocial model of development, a person's outcomes are explained by a combination of their biology, psychology, and socio-environmental factors. The study of gene-environment interplay is relatively young, but there is some evidence that effortful control develops through an interaction of genetic and environmental influences (Ganiban et al., 2021; Lemery-Chalfant et al., 2013; Zhao et al., 2020).

For example, a study of twins in middle childhood examined how the heritability of effortful control differed across quality of the home environment and chaos in the home and found that although effortful control was highly heritable regardless of home environment, variance in effortful control attributed to genetic influences were highest when the home environment was highly chaotic, even after accounting for passive gene-environment correlation (Lemery-Chalfant et al., 2013). This moderation of heritability reflects a change in the variance in effortful control attributed to genetics and suggests that genetically influenced individual differences were most prominent when the environment did not facilitate the development of effortful control. In other words, when the environment was less chaotic, effortful control was more homogeneous. In highly chaotic environments, individuals varied more in their effortful control, and this variance was attributable to genetic factors.

To the extent that premature birth represents both a stressor and the loss of a protected environment during a time of rapid brain development, it may also be associated with changes in the broad genetic and environmental etiology of effortful control. However, given differences in the nature of prematurity versus home environment as a risk, it would not necessarily be expected to follow a similar pattern. For example, it may be that genetic differences become less salient in an environment that does not support the optimal development of heritable neural systems.

Other studies contribute additional evidence of a gene-environment interaction. For example, Ganiban and colleagues (2021) found that adoptive parents' laxness and over-reactive parenting interacted in complex ways with children's heritable risk as indexed by birth mother personality. For instance, adoptive parents' laxness was associated with higher effortful control for children of birth mothers high in emotion dysregulation or low in agreeableness, whereas children of birth mothers who were highly agreeable or low in emotion dysregulation showed lower effortful control when adoptive parents were more lax. Thus, the implications of children's genetically-influenced predispositions differed depending on the environment, but, in this case, according to a pattern more consistent with a goodness-of-fit perspective than unilateral associations with risk or resilience. Zhao et al. (2020) utilized a molecular genetic design (n = 1531) and found that the MAOA gene interacted with parental acceptance in boys such that boys with the MAOA gene were more sensitive to parental acceptance and thus had higher effortful control when their parents were more accepting. These studies could indicate that other prenatal and neonatal medical risk factors might also interact with

genetics, so it is important to consider gene-environment interactions when examining effortful control development.

As with most gene-environment interaction research, however, the literature is sparse. Although there is limited evidence that gene-environment interactions play a role in effortful control development, no study has specifically considered the interactions between genetics and prematurity.

Research Questions and Hypotheses

The purpose of this study was to address limitations in the existing literature by answering three questions: (1) Does gestational age predict effortful control over the course of childhood, and does gestational age predict effortful control above and beyond obstetrical and neonatal complications? (2) What are the genetic and environmental influences on effortful control throughout childhood? (3) Are the genetic and environmental influences on effortful control development moderated by gestational age?

Based on prior literature (Cassiano et al., 2020), I hypothesized that prematurity would be related to the attentional focusing subdomain of effortful control, but not the subdomains of inhibitory control or activation control in early childhood and middle childhood when not controlling for neonatal complications or obstetrical complications. Because there is evidence that prematurity effects are really due to medical risk (Cassiano et al., 2017; Kerestes, 2005; Klein et al., 2009; Klein et al., 2013; Voigt et al., 2014), I also hypothesized that gestational age would not predict effortful control development above and beyond neonatal medical risk in early childhood or middle childhood when controlling for neonatal complications and obstetrical complications.

Based on previous findings from cross-sectional twin studies (Fagnani et al., 2017; Gagne & Saudino, 2016; Lemery-Chalfant et al., 2008; Yamagata et al., 2005), I hypothesized that effortful control would have moderately high heritability estimates in early childhood and high heritability estimates in middle childhood. Because there may be differences in the heritability of effortful control based on subdomain, I examined the heritability of each domain of effortful control separately in this study. I also examined the heritability of effortful control separately in early childhood and middle childhood as there are differences in heritability estimates longitudinally.

I did not have a formal hypothesis about gene-environment interactions because this area of research is mainly exploratory, but I speculated that prematurity would decrease the heritability of effortful control because environmental factors become more salient for premature infants. All hypotheses were preregistered on OSF; see https://osf.io/qpmwc/?view_only=7b1fb9f351ee479a92e5fc2d85ba95e7

Method

Participants

The Arizona Twin Project is an ongoing longitudinal study designed to assess risk and resilience. Families were originally recruited from birth records in collaboration with the Vital Records Office from the Arizona Department of Health Services between 2007 and 2008. The sample size ranged from 636 twins at 12 months to 780 twins at age 9-11 years. The twins were first assessed at 12 months (52.5% female, 29.7% monozygotic [MZ], 36.9% same-sex dizygotic [ssDZ], and 33.4% other sex DZ [osDZ]) and were followed across eight additional waves from 30 months to 13-14 years of age. Beginning

with the fourth wave of data collection when the twins were 7-9 years of age, the initial sample was re-contacted, and new families from the same birth cohort were recruited from parents of twins' groups and online postings. A summary of the sample size at each wave along with the descriptive statistics for each wave are presented in Table 1. Detailed information on retention rates for the early childhood sample can be found in earlier publications (Lemery-Chalfant et al., 2013; Lemery-Chalfant et al., 2019). HIPAA consent forms to collect birth records were initially sent to families at 30 months, and then again at the launch of the 9th wave when twins were approximately 14 years old for new families or families with missing data. Birth records were obtained for a total of 474 participants (both mothers and twins) from 159 families.

The sample was racially and ethnically diverse, with primarily Hispanic/Latinx (ranging from 23.8%-27.1%) and non-Hispanic/Latinx White (ranging from 53.2%-57.8%) participants, and the remainder being Asian American (1.9%-4.8%), Black or African American (2.1%-3.3%%), Native American (2.7%-3.9%), or multiracial or other (1.5%-3.6%). Families were socioeconomically diverse, with a substantial proportion having income-to-needs ratios that fell below (6.52-13.36%) or near (21.38-24.22%) the poverty line, and the remainder categorized as lower middle class (15.59-22.83%), middle class (16.30-20.00%), and upper middle-to-upper class (20.67-33.22%). Parental education ranged from less than a high school degree to a professional degree, with mean education of a college degree. In terms of prematurity, 37.8% of the twin sample was born full-term (at least 37 weeks gestation), 44% was late preterm (34-37 weeks

gestation), 10.5% was moderately preterm (32-34 weeks gestation), 7.7% was very preterm (25.5-32), and 0.4% was extremely preterm (25 weeks gestation or less).

Twenty-two twins from 14 families were excluded (not included in the reported sample size or demographics) because of developmental or cognitive disabilities that interfered with their ability to complete study procedures. There were no other exclusions. While there was no pre-designated endpoint for participant recruitment, the project's goal was to include a minimum of 300 participants in each wave of data collection in order to have adequate power for multivariate twin analyses not conducted in this manuscript. Participants were contacted separately to participate in the birth records portion of the project, so there was no established minimum participation goal.

Power analyses conducted in G*Power (Faul et al., 2007) revealed that 55 participants were necessary for sufficiently powered regression analyses with a power of 0.80 assuming a medium effect size (R^2 change for a single predictor = .15), and 395 participants were needed to detect a small effect (R^2 change = .02) at a power of .80. The sample was well over this threshold, so I continued with analyses.

Attrition Analyses

Attrition analyses were estimated to compare demographics and effortful control scales at each wave of data collection and between the sample with birth record data and the full sample. There were no differences on SES, age, sex, or effortful control measures for families who had birth record data versus those who did not have birth record data. However, there were some differences in the families who participated at each wave. Participants who did not participate at the 5 year wave had lower SES at the 30 month

wave compared to those who did not participate, M_{diff} = -0.366, *SE* = 0.092, *t*(301) = -3.964, *95% CI* [-0.55, -0.18], $p < .001$. Participants who did not participate at the 8 year wave had lower SES at the 5 year wave compared to those who continued participation, M_{diff} = -0.387, *SE* = 0.151, *t*(184) = -2.570, *95% CI* [-0.68, -0.09], $p = .011$. Finally, participants who did not participate at the 10 year wave had lower attentional focusing at the 9 year wave compared to those who were retained, M_{diff} = -0.442, *SE* = 0.190, *t*(355) = -2.327, *95% CI* [-0.82, -0.07], $p = .021$. There were no other differences on SES, age, sex, or effortful control measures with the sample from wave to wave. All sample sizes are reported in the regression tables.

Covid-19 Pandemic

The onset of the COVID-19 pandemic occurred during the 10-year-old wave of data collection, resulting in 162 twins (21.89%) at Wave 6 and their families participating virtually after quarantine was declared in the state of Arizona. There were no significant differences on any demographic variable or effortful control scale based on whether the family participated in these waves prior to the onset of the COVID-19 pandemic or during the pandemic (Murillo et al., under review).

Procedure

The twins' primary caregiver (>95.8% mothers) was contacted via telephone or email at 12 months, 30 months, and 5 years to either complete a phone interview with a trained research assistant or fill out online surveys including the Zygosity Questionnaire and temperament questionnaires. At ages 8, 9, and 10, primary caregivers (>93.3% mothers) completed temperament questionnaires during 2-3 hour home visits or online

(see Lemery-Chalfant et al., 2013; Lemery-Chalfant et al., 2019 for details, including information on procedures and measures not considered in this study). The twin's biological mother and the twin's primary caregiver were also mailed HIPAA consent forms to grant access to the biological mother's obstetrical and birth records and the twins' birth records. After HIPAA consent was received, hospitals were contacted to retrieve medical information. Institutional Review Board approval was obtained, including written informed consent from primary caregivers and verbal assent from children. Families were compensated for all components of the study.

Measures

Gestational Age

Prematurity was determined by the child's gestational age, a calculation of the first day of the pregnant person's last menstrual period to the day of the child's birth, obtained from the biological mother and twins' birth records when available or taken from the Zygosity Questionnaire otherwise. The sample included a wide range of gestational ages, with 460 full-term infants (at least 37 weeks gestation). The sample was 37.8% full term (at least 37 weeks), 44% late preterm (24-37 weeks), 10.5% moderately preterm 32-34 weeks), 7.7% very preterm (25.5 – 32 weeks), and 0.4% extremely preterm (25 weeks or less).

Neonatal Medical Risk

To assess neonatal medical risk, birth records were coded by two different undergraduate research assistants trained to identify 21 complications based on a coding protocol. After coding the records individually, the two research assistants met to resolve

any potential discrepancies. Complications were selected from the Neonatal Complications Scale (Littman & Parmalee, 1974) and the Neonatal Morbidity Scale (Minde et al.,1983), which are well-established measures of common complications at birth, with examples including “received resuscitation”, “surgery other than circumcision”, “bradycardia”, and “respiratory distress syndrome.” After reliably coding the birth records, all complications were summed to create a composite score, with a potential score of 0-26. For a full list of the risk variables included in the composite measure, see Supplemental Materials.

Obstetrical Complications

To assess obstetrical complications, the birth records were also coded for 49 risk variables experienced during gestation using the Obstetrical Complications Scale (Littman & Parmalee, 1974), a well-established measure of common complications during pregnancy. Examples of obstetrical complications include whether or not the biological mother experienced risk factors during gestation, such as “smoking during pregnancy” or “pre-eclampsia.” After coding, a composite score was created, with a potential score of 0-53 for obstetrical complications by summing the risk variables identified in the birth records. For a full list of the risk variables included in the composite measure, see Supplemental Materials.

Effortful Control

To assess twins’ effortful control and its infant precursor, duration of orienting, I employed Rothbart and colleagues’ well-established, age-appropriate temperament questionnaires, which have shown good construct and convergent validity (Ellis &

Rothbart, 2001; Gartstein & Rothbart, 2003; Kozlowski et al., 2022; Rothbart et al., 2001). Specifically, at twin age 12 months (Wave 1), primary caregivers reported on twins' duration of orienting (12 items; Cronbach's alpha=.83) on a Likert scale from 1 (*never*) to 5 (*always*) using the Infant Behavior Questionnaire-Revised (IBQ-R; Gartstein & Rothbart, 2003). At 30 months (Wave 2) and 5 years (Wave 3), effortful control was assessed using the primary caregiver-report attentional focusing (6 items; Cronbach's alphas=.71-.73) and inhibitory control (6 items; Cronbach's alphas = .67-.74) subscales of the Children's Behavior Questionnaire - Short Form (CBQ-SF; Putnam & Rothbart, 2001), with all items answered on a 7-point Likert scale from 1 (*extremely untrue*) to 7 (*extremely true*). In middle to late childhood (Waves 4, 5, 6), primary caregivers reported on twins' activation control (15 items; Cronbach's alphas = .77-.82; waves 4 and 6 only), attentional focusing (7 items; Cronbach's alphas = .76-.90), and inhibitory control (8 items; Cronbach's alphas = .58-.68), using the Temperament in Middle Childhood Questionnaire (TMCQ; Simonds, 2007; waves 4 and 5) and Early Adolescent Temperament Questionnaire (EATQ; Ellis & Rothbart, 2001; wave 6), with all items asked on a Likert scale from 1 (*almost always untrue*) to 5 (*almost always true*).

Zygosity

In this sample, zygosity was determined through the 32-item caregiver-report Zygosity Questionnaire for Young Twins (Goldsmith, 1991), a comprehensive list of questions given to parents that ask about the physical similarities and differences between twins, which has approximately 95% agreement with zygosity determined by genotyping (Forget-Dubois et al., 2003). When zygosity was difficult to determine, medical records,

expert ratings based on pictures taken at most waves of data collection, and genotyping (for three twin pairs) were also used.

Demographics

Demographic covariates included the sex assigned at birth (0 = Male, 1 = Female) of the twins, twin age at each wave of data collection, and socioeconomic status at each wave of data collection, defined as a standardized mean composite of income-to-needs ratio, primary caregiver education, and other caregiver education. I did not expect findings to differ by racial or ethnic status (Li-Grining, 2007; Valiente et al., 2008), so racial or ethnic status were not included as covariates.

Data Analysis

Research Question 1

To examine the relationship between prematurity and effortful control, I first estimated separate cluster robust standard error regression models, which adjusted the standard error to account for within-cluster dependence (twins clustered within families), with gestational age as the independent variable and each subdomain of effortful control as dependent variables at each age. I used the lavaan package in R (v0.6-7; Rosseel, 2012) to complete these analyses. Because effortful control can also vary by age, sex, and family socioeconomic status (Kochanska et al., 2000), these variables were included as control variables in each model. To answer the main question of whether prematurity predicted effortful control above and beyond neonatal medical risk, I then included the neonatal medical risk score and the obstetrical complications variables as additional

controls in a second set of cluster robust standard error regression models. Full information maximum likelihood was used which makes use of all available data.

Research Question 2

To examine the extent to which individual differences in each subdomain of effortful control (attentional focusing, inhibitory control, activation control) are explained by genetic influences at each age (12 months, 30 months, 5 years, 8 years, 9 years, 10 years), I used the quantitative genetic ACE model, a multi-group structural equation model that uses differences in the phenotypic resemblance of MZ and DZ twin pairs to parse phenotypic variance into additive genetic (A), common/shared environment (C), and non-shared environment (E) components (Neale & Cardon, 2013). The A component includes all factors that increase the resemblance of MZ twins (who share approximately 100% of their DNA) relative to DZ twins (who share approximately 50%). Because MZ twins share approximately 100% of their DNA and DZ twins share 50% on average, the A component of the ACE model is fixed to a correlation of 1.0 for MZ twin pairs and .50 for DZ twin pairs. The C component includes factors that increase the MZ and DZ cross-twin resemblance to the same degree and is fixed to a correlation of 1.0 for both groups. Finally, nongenetic factors that reduce the resemblance between twin pairs, including both nonshared environmental influences and measurement error, are accounted for by the E component, which is uncorrelated between twins. Heritability (h^2) is the proportion of total phenotypic variance explained by additive genetic factors.

When MZ cross-twin correlations were more than twice as high as DZ cross-twin correlations, I used an alternate ADE (A = additive genetic, D = dominant genetic, E =

non-shared environment) model to account for interactions between alleles. In this model, the D component is correlated 1.0 between MZ twins and .25 between DZ twins because DZ twins inherit the same alleles at a locus 25% of the time (Neale & Cardon, 2013), and broad sense heritability (H^2) is estimated as the proportion of the phenotypic variance explained by additive and dominant genetic influences together.

After the full ACE/ADE models were fit, the significance of A and C/D paths were tested by dropping them from the model and comparing the fit of the reduced nested model to the full model using the -2 log likelihood chi-square test of fit, although E was always retained in the model because it includes measurement error.

Research Question 3

To investigate whether the genetic and environmental influences on effortful control were moderated in early childhood and middle childhood by gestational age, I used a moderated ACE/ADE twin model (Purcell, 2002). Moderated ACE/ADE twin models parse phenotypic variance into genetic and environmental components in the same way as ACE/ADE twin models do, but they also consider how a moderating factor can change how the variance is attributed by allowing the moderator to affect the paths from each latent A, C/D, or E factor to the phenotype (see Figure 1). As with univariate ACE/ADE models, the significance of paths can be tested using the -2 log likelihood chi-square test of fit. I tested the effect of the moderator on each path in turn, and only attempted to drop A or C/D variance from the model if moderation of that component was found to be non-significant. In addition, I included the moderator gestational age in the means model as a predictor of effortful control, essentially controlling for the main

effect association before parsing the independent, residual variance into A, C/D, and E components. Including the moderator in the means model controls for gene-environment correlation between the moderator and the outcome (Purcell, 2002). The subsample of 186 twin pairs at age five was not large enough to support testing the moderation of ACE estimates, so models were not estimated at this age.

Supplementary Analysis

To more precisely replicate prior research which has analyzed the links between prematurity and effortful control by comparing extremely preterm or very preterm infants to full-term infants (Lejeune et al., 2015; Reyes et al., 2020; Voigt et al., 2012), I estimated additional cluster robust standard error regression models using dichotomized gestational age as the independent variable controlling for age, sex, and socioeconomic status. The gestational age variable was dichotomized into very preterm infants (<32 weeks gestation, n = 90) and full-term infants (>37 weeks gestation, n = 460). Very preterm infants were coded as 1. Full-term infants were coded as 0. This analysis significantly reduced the sample size, but it was included to provide a basis for comparison to prior literature.

Transparency and Openness

I report the sample size and how it was determined, all exclusions, attrition, and all manipulations, measures, and analyses. I follow JARS guidelines (Kazak, 2018). Data were analyzed using SPSS Version 28 and the R packages OpenMx Version 2.21.1 for twin analyses (Boker et al., 2011), and lavaan Version 0.6-16 (Rosseel, 2012) for cluster robust standard error regression analyses in R Version 4.2.3.

Prior publications with this sample have examined effortful control at one age in relation to outcomes such as sleep or school achievement, or examined their genetic and environmental underpinnings (Clifford et al., 2020; Miadich et al., 2022; Rea-Sandin et al., 2023; Valiente et al., 2021). This is the first study using these data to examine effortful control as an outcome of gestational age and to examine each subdomain of effortful control across early and middle childhood.

Sample scripts used to conduct cluster robust standard error regressions, ACE twin models, and moderated ACE twin models are available at https://osf.io/qpmwc/?view_only=7b1fb9f351ee479a92e5fc2d85ba95e7. Deidentified data are available from study principal investigators upon reasonable request. The effortful control questionnaires used in this study are not mine to disseminate, but can be obtained free of charge by completing a request form on Dr. Rothbart's website [https://research.bowdoin.edu/rothbart-temperament-questionnaires/request-forms/], or by contacting Dr. Putnam over email [sputnam@bowdoin.edu] or postal mail [Department of Psychology, Bowdoin College, 6900 College Station, Brunswick, ME 04011].

Results

Preliminary Analysis

Descriptive statistics are presented in Table 1, and intercorrelations are presented in Table 2. All variables used in regressions were assessed for skewness, kurtosis, and normality, and it was determined that no variables required transformation (skewness < +/-2.00, kurtosis < +/-7.00). Consistent with prior literature, lower gestational age was related to higher obstetrical complications and neonatal complications. Contrary to my

hypothesis, gestational age was not significantly correlated with effortful control. Additionally, girls had significantly higher effortful control for all effortful control measures except duration of orienting at 12 months. Family SES was positively correlated with all effortful control measures except for duration of orienting at 12 months and activation control at age 10.

Research Question 1

Regressions with Gestational Age Measured Continuously

Contrary to my hypothesis, cluster robust standard error regressions (presented in Table 3) showed no evidence that gestational age was significantly related to effortful control outcomes when gestational age was measured continuously and while controlling for age, sex, and SES. The null association remained when controlling for neonatal complications and obstetrical complications and there were no significant relationships between neonatal complications or obstetrical complications and effortful control.

Research Question 2

ACE/ADE Models

I fit ACE/ADE models to examine genetic and environmental effects on each subdomain of effortful control at 12 months, 30 months, 5 years, 8 years, 9 years, and 10 years, beginning with the full model and then dropping A and C/D to find the most parsimonious model. Identical twins were more than twice as similar as fraternal twins on attentional focusing at all waves, so ADE models were run for attentional focusing at each wave. For each analysis, the most parsimonious model is presented in bold in Table 5.

Heritability estimates ranged from 7% (duration of orienting at 12 months) to 88% (attentional focusing at 10 years). At all waves, additive genetics were significant sources of variance in effortful control outcomes, but there was variation in estimates for each subdomain of effortful control. Consistent with my hypotheses, attentional focusing was consistently more heritable compared to activation control and inhibitory control, and heritability estimates were higher in middle childhood than early childhood (64% - 88%). ADE or AE models fit best for attentional focusing, whereas inhibitory control and activation control were best explained by either ACE or AE models. Inhibitory control and activation control were moderately heritable. At later waves, inhibitory control and activation control had slightly higher heritability. Duration of orienting had the lowest heritability estimates and highest shared environment estimates compared to other subdomains of effortful control.

Research Question 3

Moderated ACE/ADE Models

Overall, there was no consistent evidence for gestational age as a moderator of ACE estimates on effortful control outcomes, with results presented in Table 6. Dropping the association between the moderator and the mean did not result in a significant loss of fit for any model, and thus, I used the simpler full models without this association estimated when testing the significance of other paths. Dropping moderation of ACE/ADE path estimates did not result in significantly worse fit for any model except activation control at age 8. For age 8 activation control, dropping moderation of the A, C, or E paths resulted in significant loss of fit (see Table 6), offering some suggestion that

gestational age broadly moderates the genetic and environmental etiology of this subdomain at this age. However, the inconsistency of this finding with other models and its lack of strong theoretical support suggests it should be interpreted with caution. Based on these results, gestational age was not a significant moderator of additive genetic, dominant genetic, shared environment, or unique environmental influence.

Supplementary Analysis

To replicate analyses conducted in the existing literature, I dichotomized the gestational age variable to compare very preterm infants (<32 weeks gestation) to full-term infants (>37 weeks gestation). Contrary to my hypothesis, I found no evidence that full-term infants had significantly higher effortful control compared to very preterm infants, whether or not I contolled for neonatal medical risk and obstetrical complications. Results controlling for neonatal medical risk and obstetrical complications are presented in the supplementary materials.

Discussion

The goal of the present study was to improve understanding of the etiology of effortful control by examining prematurity, neonatal complications, obstetrical complications, and genetic influences. By using longitudinal data from a community twin sample, diverse in race, ethnicity, and SES, the study added to understanding of the relation between gestational age and childhood effortful control from infancy to middle childhood. Contrary to my hypothesis, gestational age was not significantly related to effortful control outcomes, and very preterm infants did not have significantly different effortful control compared to full-term infants at any age that was assessed. These results

held when controlling for neonatal medical risk and obstetrical complications. In line with my predictions and past cross-sectional research, results showed that effortful control was moderately to highly heritable across early and middle childhood. This supports the possibility that the attentional focusing subdomain is particularly highly heritable and that there are higher estimates of heritability of effortful control in middle childhood compared to infancy. Lastly, gestational age was not a consistent moderator of genetic or environmental influences on effortful control.

Gestational Age and Effortful Control

Contrary to my hypothesis, when I did not control for neonatal medical risk and obstetrical complications, gestational age did not predict effortful control, and when I compared very preterm infants to full-term infants, I also did not see significant differences. The majority of past literature finds that there is some relationship between gestational age and effortful control, without controlling for medical risk, particularly with the subdomain of attentional focusing (Cassiano et al., 2020; Lejeune et al., 2015; Reyes et al., 2020). However, some studies have also reported null findings (Olafsen et al., 2008; Voigt et al., 2012).

Differences in findings may be due to differences in samples. This sample was a large, community twin sample in the United States, whereas studies that have found significant findings were predominantly smaller and comprises singleton samples of infants recruited from hospitals outside of the United States (Lejeune et al., 2015; Voigt et al., 2012; Reyes et al., 2020).

A notable strength of this study was that it was the first, to my knowledge, to study the relationship between gestational age and effortful control using a twin sample. Twin premature births can be caused by medical risks, but they can also be caused by limited space in the womb. Singleton premature births are more likely to be caused by medical risk (Basso & Wilcox, 2010). In the present sample, there was a high percentage of premature births (62.6%), but the majority of participants had a low number of obstetrical complications (M = 6.74 complications) and neonatal complications (M = 2.36 complications). Thus, the sample was different than most samples in existing literature which are typically clinical samples of premature infants who have high rates of obstetrical and neonatal complications, in addition to low gestational age.

For example, Consentino-Rocha and colleagues (2014) and Lejuene and colleagues (2015) assessed singleton infants who had been admitted to the hospital after birth due to medical complications. Using a twin study allowed me to assess many infants who were born prematurely but did not have significant medical risks, thus decoupling gestational age and medical risk and improving the generalizability of the findings. Future studies should attempt to replicate these findings in other twin samples, especially given the strength of the genetic component.

Discrepancies in findings could also be explained by different definitions of prematurity. Some researchers define "premature" as any infant who is born at less than 37 weeks gestation (Voigt et al., 2014). Others define prematurity as infants who are born weighing less than 2000 grams (Olafsen, et al., 2008). Still others argue that infants can be either less than 37 weeks gestation or less than 2000 grams to be considered premature

(Reyes et al., 2020), or that infants must meet both qualifications to be considered premature (Voigt et al., 2012). The present study assessed gestational age as a continuous variable and as a dichotomous variable to compare very preterm infants to full term infants. However, some studies that found that prematurity predicts effortful control included birthweight in their definition of prematurity (Reyes et al., 2020; Voigt et al., 2012). There is some evidence that low birthweight is related to lower effortful control (Poehlmann et al., 2010), so the exclusion of birth weight from the definition of prematurity in this study may explain differences in outcomes. Future research should consider gestational age and birth weight separately to clarify the concept of prematurity and relations with effortful control.

Finally, age differences in the present sample may also partially explain the differences in findings. Consistent with literature, I did not find significant links with gestational age and duration of orienting at 12 months (Olafsen et al., 2008). This may be because effortful control does not emerge until around 1 year. Past literature has found, however, that there is a significant relationship between lower gestational age and lower effortful control at 24 months (Lejeune et al., 2015; Voigt et al., 2012), whereas I did not find a relationship at 30 months. It is possible that gestational age may lead to temporary deficits in effortful control which emerge by 24 months, but by 30 months, premature infants catch up to full-term infants. This idea is backed by some evidence, as Poehlmann et al. (2010) found that higher neonatal medical risk predicts lower effortful control at 24 months, but not 36 months. As this is the only study that has examined effortful control beyond early childhood, there is a clear need for more research which examines effortful

control beyond early childhood and in smaller increments throughout early childhood to further examine this idea.

Of course, the findings may also support the conclusion that there is not a true relationship between gestational age and effortful control. Because my results showed narrow confidence intervals (see Table 3), there is some support for this idea. However, due to the discrepancies in the literature based on measurement and sample, it ultimately cannot be concluded whether lower gestational age is or is not a risk factor for lower effortful control.

Gestational Age and Effortful Control, Controlling for Medical Risk

I found no relationship between gestational age and effortful control with or without controlling for obstetrical and neonatal medical risk. Thus, the findings do not clarify the important issue of whether medical risk accounts for the association between lower gestational age and lower effortful control seen in the literature.

Interestingly, in this study, I found no evidence that composite measures of neonatal and obstetrical complications were related to effortful control. While this may suggest that effortful control was not predicted by neonatal medical risk and obstetrical complications, it is important to remember that the current sample had relatively low rates of medical complications. Furthermore, although well-established measures of neonatal medical risk and obstetrical complications were used, which helped to assess an infant's overall medical risk, these measures could not capture all possible medical risks and did not allow for pinpointing of specific complications that may predict effortful control. Poehlmann et al. (2010) used a similar composite measure of medical risk, but

they did find that higher medical risk predicted lower effortful control. The neonatal medical risk measure in the present study included every item in their measure with the exception of "*gastroesophageal reflux*," "*apnea monitor at NICU discharge*," and "*NICU stay of more than 30 days*," so these items could be relevant for predicting effortful control. Other studies have also found that infants who stay in the NICU may have lower effortful control (Consentino-Rocha et al., 2014; Klein et al., 2009; Lejeune et al., 2015), so future research should investigate if there is a relationship between time spent in the NICU and effortful control.

Although based on my findings, I cannot conclude that prematurity and/or obstetrical and neonatal complications are risk factors for effortful control, this was a well-powered, community-based study which can help to inform public health decisions around prematurity. Prematurity is often associated with negative outcomes, and due to the complexity of isolating gestational age from obstetrical or neonatal complications, the causal relationships remain unclear. As a result, gestational age may be implicated for correlated negative outcomes, but not have a causal influence. Thus, the burden of prematurity may be overstated, and resources potentially allocated incorrectly. Because there was no compelling evidence that gestational age was related to effortful control in this large, longitudinal, diverse study and there is division in the literature, public health officials should be cautious when drawing causal paths between gestational age and effortful control. On a broader level, officials should also consider that prematurity may not be as strong of a risk factor as it once seemed. Through improving understanding of

the risks associated with prematurity, research in this field contributes to efforts to improve outcomes for children born prematurely.

The Heritability of Effortful Control

Many studies have established effortful control as a moderately heritable trait, but very few studies have examined each subdomain of effortful control separately and throughout infancy and childhood. To my knowledge, this study is the first to have examined each subdomain of effortful control in infancy, early childhood, and middle childhood. Consistent with the small existing literature, I found evidence that attentional focusing is the most highly heritable subdomain. The heritability estimates of attentional focusing at 9 years (87%) and 10 years (88%) were consistent with heritability estimates in past literature. Most closely related, Lemery-Chalfant et al. (2008) found that attentional focusing at 8 years was 83% heritable compared to general effortful control which was 68% heritable at 5 years and 79% heritable at 8 years. In a sample of adults, Yamagata et al. (2005) found that attentional focusing was 45% heritable compared to activation control which was 39% heritable and inhibitory control which was 32% heritable in adulthood. Furthermore, my results combined with Yamagata et al. (2005) improve developmental understanding of heritability estimates with higher heritability estimates in middle childhood compared to early childhood. It remains an open question if heritability estimates will be higher in adolescence in this sample, or if heritability estimates will be lower after middle childhood. Although the heritability estimates were higher later in development, I cannot rule out the effect of a measurement change. Because genetics seem to play a particularly salient role in the development of effortful

control, especially in the development of attentional focusing, evaluation of genetic influences should be included in future research to accurately assess the etiology of effortful control. Based on the present sample, there is also evidence for examining each subdomain of effortful control separately as there is evidence for different genetic etiologies.

This study was also the first, to my knowledge, to assess gestational age as a moderator of genetic and environmental influence estimates of effortful control. This was an exploratory analysis, and I found negligible evidence for moderation. With sufficient sample sizes, future studies should also assess neonatal complications and obstetrical complications as moderators of these estimates.

Strengths, Limitations, and Conclusions

This study filled important gaps in the literature by examining the genetic and environmental predictors of each subdomain of effortful control at 12 months, 30 months, 5 years, 8 years, 9 years, and 10 years. Through the longitudinal sample of twins and the extensive coding of birth records, the study uniquely contributed to the understanding of effortful control development by disentangling genetic and environmental influences and gestational age from neonatal complications and obstetrical complications. Furthermore, the racially, ethnically, and socioeconomically diverse sample improved the generalizability of prematurity and effortful control research.

This study could have been improved by greater consistency in the longitudinal data. Because the project introduced new twins at different ages and there was attrition at each age, the sample size at age five was limited, preventing evaluation of moderated

models at this age. The 22 twins that were dropped from the study due to various cognitive difficulties may have also represented some of the more extreme cases of low effortful control, although this is likely not true for all participants. In addition, the study lacked specific subdomains of effortful control at some waves, limiting ability to observe the subdomains longitudinally. I also used age-appropriate assessments of each subdomain longitudinally to accommodate changes in effortful control based on age, so I could not make conclusions about change over time. Furthermore, parents reported effortful control at all ages. The study could have benefitted from a child report, teacher report, or observational measure of effortful control.

I found that those who dropped out at the 10 year wave had worse attentional focusing at the 9 year wave compared to those who did not drop out. As a consequence of this dropout, I may have had less variability in my effortful control measures at the 10 year wave.

In the genetic analyses, I estimated both ACE and ADE models. Although the ADE models allow for more accurate modeling of additive and dominant genetics, it is impossible to estimate C, or shared environment with these models due to limited degrees of freedom. Thus, the ADE models did not capture any shared environmental variance. This does not indicate that there is no shared environment contribution to attentional focusing, however.

In sum, the study contributed uniquely to existing literature on prematurity and effortful control development through utilizing a twin sample, examining genetics, and extending findings beyond early childhood. In the present sample, gestational age was not

related to attentional focusing, inhibitory control, or activation control at any age regardless of whether I controlled for obstetrical complications and neonatal complications. Thus, I cannot conclude that gestational age is a risk for lower effortful control. I also found that attentional focusing is highly heritable, particularly in middle childhood, emphasizing the continuing need to study the etiology of each subdomain of effortful control separately and to study how genetics and environment contribute to effortful control development.

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APPENDIX A

TABLES AND FIGURES

Table 1

Descriptive Statistics for Study Variables

	n	*M*	*SD*	median	min	max	range	skew	kurtosis	se
Gestational Age	1,123	35.48	2.68	36.00	23.50	40.50	17.00	-1.22	1.89	0.08
Obstetrical Complications	315	6.74	3.82	7.00	0.00	16.00	16.00	-0.11	-0.57	0.22
Neonatal Medical Risk	316	2.36	3.21	1.00	0.00	16.00	16.00	1.72	2.50	0.18
Age at 12 month visit	636	12.69	1.37	12.26	8.08	24.77	16.69	3.02	19.83	0.05
Age at 30 month visit	526	31.92	0.25	2.57	1.66	3.83	2.17	1.60	4.93	0.01
Age at 5 year visit	381	5.19	0.26	5.20	4.41	5.70	1.29	-0.33	-0.33	0.01
Age at 8 year visit	700	8.43	0.68	8.42	6.96	9.97	3.01	-0.18	-0.40	0.03
Age at 9 year visit	800	9.72	0.94	9.60	7.70	12.09	4.39	0.32	-0.03	0.03
Age at 10 year visit	780	10.88	1.13	10.69	8.41	14.80	6.39	0.37	0.18	0.04
SES at 12 month visit	600	-0.04	0.86	-0.11	-2.04	1.89	3.93	0.07	-0.78	0.04
SES at 30 month visit	606	-0.01	0.81	0.00	-1.90	1.54	3.44	0.01	-0.78	0.03
SES at 5 year visit	372	-0.01	0.80	0.01	-1.86	1.61	3.47	-0.02	-0.72	0.04
SES at 8 year visit	674	-0.02	0.81	-0.10	-1.72	3.34	5.06	0.53	0.39	0.03
SES at 9 year visit	713	-0.01	0.78	-0.09	-1.53	3.33	4.86	0.58	0.53	0.03
SES at 10 year visit	742	-0.02	0.80	-0.08	-1.83	3.08	4.91	0.41	0.07	0.03
Duration of Orienting 12 mo visit	562	2.97	0.68	3.00	1.17	4.91	3.74	-0.12	0.09	0.03
Inhibitory Control 30 mo visit	501	4.52	0.97	4.60	1.33	7.00	5.67	-0.29	0.29	0.04
Attentional Focusing 30 mo visit	503	4.80	1.02	4.83	1.33	7.00	5.67	-0.41	0.06	0.05
Attentional Focusing 5 year visit	372	5.08	1.02	5.17	1.33	7.00	5.67	-0.58	0.43	0.05
Inhibitory Control 5 year visit	372	5.06	1.02	5.00	1.67	7.00	5.33	-0.34	-0.07	0.05
Activation Control 8 year visit	641	3.44	0.53	3.43	1.33	4.80	3.47	-0.12	-0.11	0.02
Attentional Focusing 8 year visit	641	3.29	0.93	3.43	1.00	4.86	3.86	-0.36	-0.57	0.04
Inhibitory Control 8 year visit	641	3.18	0.59	3.25	1.25	4.63	3.38	-0.26	-0.23	0.02
Attentional Focusing 9 year visit	715	3.29	0.95	3.43	1.00	5.00	4.00	-0.37	-0.50	0.04
Inhibitory Control 9 year visit	715	3.23	0.59	3.25	1.13	4.75	3.62	-0.24	0.37	0.02
Activation Control 10 year visit	722	3.24	0.71	3.29	1.29	5.00	3.71	-0.11	-0.19	0.03
Attentional Focusing 10 year visit	722	3.34	0.70	3.33	1.00	5.00	4.00	-0.23	-0.20	0.03
Inhibitory Control 10 year visit	722	3.66	0.61	3.60	1.80	5.00	3.20	-0.19	-0.39	0.02
Very Preterm Infants										
Very Preterm	90 (8.01%)									
Full-term	460 (40.96%)									
Zygosity at 10 Year										
Monozygotic	350 (29.49%)									
Same Sex Dizygotic	452 (38.08%)									
Opposite Sex Dizygotic	385 (32.43%)									

Note. Very preterm infants are infants born at less than 32 weeks gestational age.

Table 2

Zero Order Correlations and Descriptive Statistics for Study Variables

	Variable	*M*	*SD*	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1.	Obstetrical Complic.	6.74	3.82																
2.	Neonatal Medical Risk	2.36	3.21	.16* [.05, .26]															
3.	Gestational Age	35.48	2.68	-.14* [-.25, -.03]	-.55** [-.63, -.47]														
4.	Duration of Orienting 12 mo visit	2.97	0.68	.05 [-.07, .17]	-.03 [-.15, .09]	-.01 [-.10, .07]													
5.	Inhibitory Control 30 mo visit	4.52	0.97	-.02 [-.14, .11]	-.01 [-.14, .12]	-.01 [-.10, .07]	.12* [.02, .21]												
6.	Attentional Focusing 30 mo visit	4.80	1.02	.04 [-.09, .17]	-.03 [-.15, .10]	-.03 [-.12, .06]	.20** [.11, .29]	.44** [.37, .51]											
7.	Attentional Focusing 5yr visit	5.08	1.02	-.07 [-.21, .08]	-.06 [-.20, .08]	.07 [-.03, .17]	.22** [.11, .32]	.34** [.24, .43]	.45** [.36, .53]										
8.	Inhibitory Control 5yr visit	5.06	1.02	-.12 [-.26, .03]	-.05 [-.19, .10]	.04 [-.06, .14]	.12* [.01, .22]	.50** [.41, .57]	.46** [.37, .54]	.56** [.48, .62]									
9.	Activation Control 8yr visit	3.44	0.53	-.05 [-.18, .08]	.11 [-.02, .24]	.03 [-.05, .11]	.14** [.04, .24]	.34** [.24, .42]	.17** [.07, .26]	.33** [.23, .43]	.45** [.36, .54]								
10.	Attentional Focusing 8yr visit	3.29	0.93	-.02 [-.15, .11]	-.06 [-.19, .07]	.06 [-.02, .14]	.02 [-.08, .12]	.23** [.13, .32]	.20** [.10, .30]	.44** [.34, .53]	.47** [.37, .55]	.43** [.36, .49]							
11.	Inhibitory Control 8yr visit	3.18	0.59	-.02 [-.15, .11]	.01 [-.12, .14]	.05 [-.03, .13]	.09 [-.01, .19]	.43** [.35, .51]	.31** [.21, .40]	.45** [.35, .54]	.51** [.42, .59]	.46** [.40, .52]	.58** [.53, .63]						
12.	Attentional Focusing 9yr visit	3.29	0.95	-.03 [-.18, .11]	-.04 [-.18, .11]	.03 [-.05, .10]	-.00 [-.11, .11]	.28** [.17, .38]	.25** [.14, .36]	.44** [.34, .53]	.51** [.41, .59]	.35** [.27, .42]	.78** [.74, .81]	.56** [.49, .62]					
13.	Inhibitory Control 9yr visit	3.23	0.59	.00 [-.14, .15]	.12 [-.02, .26]	.06 [-.02, .13]	.01 [-.10, .12]	.35** [.25, .45]	.23** [.12, .34]	.33** [.22, .44]	.49** [.39, .58]	.37** [.29, .44]	.51** [.44, .57]	.71** [.67, .75]	.57** [.52, .62]				
14.	Activation Control 10yr visit	3.24	0.71	.09 [-.05, .23]	.04 [-.10, .18]	-.05 [-.12, .03]	.09 [-.02, .20]	.31** [.20, .41]	.21** [.10, .32]	.39** [.28, .48]	.47** [.37, .56]	.48** [.41, .54]	.49** [.42, .56]	.43** [.36, .50]	.53** [.47, .58]	.45** [.38, .51]			
15.	Attentional Focusing 10yr visit	3.34	0.70	-.03 [-.17, .12]	.03 [-.11, .18]	.02 [-.06, .09]	.05 [-.06, .16]	.28** [.17, .38]	.32** [.22, .42]	.46** [.36, .55]	.45** [.35, .54]	.36** [.28, .44]	.60** [.54, .65]	.50** [.42, .56]	.62** [.56, .66]	.49** [.43, .55]	.60** [.56, .65]		
16.	Inhibitory Control 10yr visit	3.66	0.61	-.05 [-.19, .09]	.07 [-.07, .21]	.02 [-.15, -.01]	-.06 [-.17, .05]	.30** [.20, .40]	.27** [.16, .37]	.30** [.18, .40]	.40** [.30, .50]	.29** [.21, .37]	.43** [.35, .50]	.53** [.46, .59]	.46** [.39, .52]	.54** [.49, .60]	.46** [.40, .51]	.54** [.48, .59]	
17.	Hispanic	0.26	0.44	-.02 [-.16, .12]	-.00 [-.14, .14]	.08* [.01, .16]	-.02 [-.12, .09]	-.08 [-.19, .03]	-.02 [-.13, .09]	-.00 [-.12, .12]	.02 [-.10, .14]	-.01 [-.10, .08]	-.10* [-.18, -.01]	.02 [-.07, .11]	-.02 [-.10, .06]	.02 [-.06, .10]	-.01 [-.09, .06]	.06 [-.01, .13]	-.01 [-.09, .06]

Note. M and *SD* are used to represent mean and standard deviation, respectively. * indicates $p < .05$. ** indicates $p < .01$. 95% *CIs* are shown in brackets.

Table 3
Regressions of Gestational Age Measured Continuously on Effortful Control

	Duration of Orienting 12mo visit	Attentional Focusing 30mo visit	Inhibitory Control 30mo visit	Attentional Focusing 5yr visit	Inhibitory Control 5yr visit	Activation Control 8yr visit	Attentional Focusing 8yr visit	Inhibitory Control 8yr visit	Attentional Focusing 9yr visit	Inhibitory Control 9yr visit	Activation Control 10 yr visit	Attentional Focusing 10 yr visit	Inhibitory Control 10 yr visit
MODEL WITHOUT CONTROLLING FOR OBSTETRICAL COMPLICATIONS AND NEONATAL MEDICAL RISK													
Gestational Age	-.01 [-.04 – .02]	-.01 [-.06, .04]	-.00 [-.04, .04]	.02 [-.03, .07]	.01 [-.05, .06]	.01 [-.02, .03]	.03 [-.01, .06]	.01 [-.01, .04]	.01 [-.02, .04]	.01 [-.01, .03]	-.01 [-.04, .01]	.00 [-.02, .03]	.00 [-.02, .03]
Age at wave	.12*** [.06, .19]	.07 [-.34, .48]	.12 [-.44, .68]	.18 [-.28, .63]	.21 [-.29, .71]	.03 [-.05, .11]	.14* [.02, .25]	.08* [.00, .16]	.05 [-.04, .13]	.02 [-.04, .08]	-.01 [-.06, .05]	.02 [-.03, .08]	.01 [-.04, .06]
Sex	.06 [-.08, .20]	.22* [.03, .41]	.22* [.03, .41]	.25* [.03, .47]	.42*** [.19, .65]	.04 [-.06, .13]	.32*** [.17, .46]	.27*** [.17, .37]	.40*** [.24, .56]	.29*** [.19, .39]	.23*** [.11, .25]	.20*** [.09, .31]	.12* [.01, .22]
SES at wave	-.03 [-.12, .07]	.17* [.03, .31]	.19* [.04, .35]	.19* [.04, .33]	.18 [.00, .37]	.13*** [.06, .20]	.19*** [.09, .28]	.13*** [.06, .20]	.11 [-.00, .22]	.08* [.003, .15]	.06 [-.03, .14]	.06 [-.02, .14]	.10* [.02, .18]
R^2	.06	.03	.04	..04	.07	.04	.07	.09	.06	.08	.03	.03	.03
n	588	470	470	361	361	652	652	652	677	677	694	694	694
MODEL CONTROLLING FOR OBSTETRICAL COMPLICATIONS AND NEONATAL MEDICAL RISK													
Gestational Age	.01 [-.03, .05]	-.04 [-.13, .05]	-.01 [-.07, .05]	.02 [-.05, .08]	-.06 [-.14, .03]	-.01 [-.05, .03]	-.01 [-.08, .06]	-.01 [-.06, .04]	-.01 [-.08, .07]	-.02 [-.07, .03]	-.05 [-.11, .00]	-.03 [-.08, .02]	-.02 [-.06, .03]
Age at wave	.12* [.02, .22]	.28 [-.45, 1.02]	.80 [-.05, 1.65]	.03 [-.73, .79]	.13 [-.67, .92]	-.04 [-.21, .14]	.11 [-.11, .33]	-.04 [-.17, .10]	.13 [-.10, .35]	.13* [.004, .25]	.02 [-.10, .13]	.10 [-.03, .22]	.06 [-.05, .17]
Sex	.05 [-.17, .26]	.21 [-.08, 50]	.36** [.13, .60]	.16 [-.13, .45]	.45* [.10, .81]	.12 [-.04, .28]	.31* [.07, .56]	.31*** [.15, .47]	.35* [.06, .64]	.34*** [.15, .53]	.36** [.12, .59]	.24* [.04, .45]	.18 [-.03, .38]
SES at wave	-.03 [-.18, .11]	.21 [-.01, .43]	.11 [-.11, .34]	.15 [-.07, .38]	.13 [-.17, .42]	.12* [.01, .23]	.15 [-.02, .31]	.10 [-.03, .23]	-.05 [-.27, .17]	.08 [-.10, .25]	.10 [-.08, .26]	.07 [-.09, .23]	.12 [-.06, .30]
Obstetrical Complic.	.00 [-.03, .04]	.03 [-.01, .07]	.00 [-.04, .04]	-.01 [-.05, .03]	-.03 [-.08, .02]	-.01 [-.03, .01]	-.00 [-.04, .04]	-.01 [-.03, .02]	-.01 [-.06, .04]	-.01 [-.04, .02]	.02 [-.02, .05]	-.01 [-.04, .03]	-.01 [-.04, .02]
Neonatal Medical Risk	-.01 [-.04, .03]	-.02 [-.08, .04]	.00 [-.05, .06]	-.00 [-.06, .06]	-.01 [-.08, .06]	.02 [-.01, .05]	-.02 [-.07, .03]	-.00 [-.05, .04]	-.01 [-.07, .04]	.01 [-.03, .06]	-.01 [-.05, .04]	.00 [-.04, .04]	.02 [-.02, .05]
R^2	.04	.04	.08	.03	.08	.07	.06	.09	.05	.13	.11	.07	.07
n	269	231	231	178	178	234	234	234	189	189	193	193	193

Note. $^{*}p<.05$ $^{**}p<.01$ $^{***}p<0.001$. Each cell contains β and *95% Confidence Intervals*. Each column shows a separate regression. The sample size is the number of cases for which there is data on at least one variable included in analysis.

Table 4

Twin Intraclass Correlations

	MZ	DZ
Duration of Orienting 12mo visit	.98	.93
Attentional Focusing 30mo visit	.68	.28
Inhibitory Control 30mo visit	.69	.51
Attentional Focusing 5yr visit	.86	.24
Inhibitory Control 5yr visit	.95	.62
Activation Control 8yr visit	.79	.61
Attentional Focusing 8yr visit	.70	.18
Inhibitory Control 8yr visit	.79	.53
Attentional Focusing 9yr visit	.82	.20
Inhibitory Control 9yr visit	.87	.44
Activation Control 10yr visit	.80	.53
Attentional Focusing 10yr visit	.86	.39
Inhibitory Control 10yr visit	.95	.74

MZ = Monozygotic Twins, DZ = Dizygotic Twins

Table 5

ACE Parameter Estimates

	Model	-2LL	df	△X^2	△df	p	AIC	A	C (or D)	E
Duration of Orienting 12mo visit	**ACE**	**475.13**	**554**	—	—	—	**-632.87**	**.07**	**.90**	**.03**
	AE	707.52	555	232.39	1	<.001	-402.48			
	CE	493.97	555	18.85	1	<.001	-616.03	—		
	E	1137.01	556	661.88	2	<.001	25.01			
Attentional Focusing 30mo visit	ADE	1351.16	487	—	—	—	377.16	.44	.22	.35
	AE	**1351.7**	**488**	**0.54**	**1**	**0.46**	**375.7**	**.64**		**.36**
	E	1404.74	489	53.58	2	<.001	426.74			
Inhibitory Control 30mo visit	ACE	1280.55	496	—	—	—	288.55	.57	.20	.23
	AE	**1283.63**	**497**	**3.09**	**1**	**0.08**	**289.63**	**.66**		**.34**
	CE	1293.94	497	13.39	1	<.001	299.94			
	E	1382.76	498	102.21	2		386.76			
Attentional Focusing 5yr visit	**ADE**	**961.94**	**359**	—	—	—	**243.94**	**.15**	**.68**	**.17**
	AE	966.54	360	4.60	1	0.03	246.54			
	E	1030.78	361	68.84	2	<.001	308.78			
Inhibitory Control 5yr visit	**ACE**	**863.49**	**359**	—	—	—	**145.49**	**.67**	**.27**	**.05**
	AE	868.76	360	5.27	1	0.02	148.76			
	CE	909.98	360	46.49	1	<.001	189.98			
	E	1028.78	361	165.29	2	<.001	306.78			
Activation Control 8yr visit	**ACE**	**815.72**	**637**	—	—	—	**-458.28**	**.34**	**.42**	**.20**
	AE	834.03	638	18.3	1	<.001	-441.97			
	CE	828.76	638	13.03	1	<.001	-447.24			
	E	1010.31	639	194.59	2	<.001	-267.69			
Attentional Focusing 8yr visit	**ADE**	**1621.61**	**637**	—	—	—	**347.61**	**<.001**	**.76**	**.24**
	AE	1693.91	638	72.3	1	<.001	417.91			
	E	1693.91	639	72.3	2	<.001	415.91			
Inhibitory Control 8yr visit	**ACE**	**943.55**	**637**	—	—	—	**-330.45**	**.62**	**.21**	**.18**
	AE	947.81	638	4.26	1	0.04	-328.19			
	CE	971.89	638	28.33	1	<.001	-304.11			
	E	110.29	639	166.73	2	<.001	-167.71			
Attentional Focusing 9yr visit	**ADE**	**1782.63**	**711**	—	—	—	**360.63**	**<.001**	**.86**	**.14**
	AE	1916.42	712	133.79	1	<.001	492.42			
	E	1916.42	713	133.79	2	<.001	490.42			
Inhibitory Control 9yr visit	ACE	1012.55	711	—	—	—	-409.45	.92	<.001	.08
	AE	**1012.55**	**712**	**0**	**1**	**1**	**-411.45**	**.77**		**.23**
	CE	1228.49	712	215.94	1	<.001	-195.51			
	E	1228.49	713	215.94	2	<.001	-197.51			
Activation Control 10yr visit	ACE	1332.41	718	—	—	—	-103.59	.68	.17	.15
	AE	**1335.85**	**719**	**3.44**	**1**	**0.06**	**-102.15**	**.71**		**.29**
	CE	1375.13	719	42.73	1	<.001	-62.87			
	E	1531.55	720	199.14	2	<.001	91.55			
Attentional Focusing 10yr visit	ADE	1343.56	718	—	—	—	-92.44	.60	.29	.12
	AE	**1345.67**	**719**	**2.11**	**1**	**0.15**	**-92.33**	**.88**		**.12**
	E	1528.72	720	185.17	2	<.001	88.72			
Inhibitory Control 10yr visit	**ACE**	**868.77**	**718**	**—**	**—**	**—**	**-567.23**	**.44**	**.52**	**.04**
	AE	916.11	719	47.33	1	<.001	-567.23			
	CE	958.69	719	89.92	1	<.001	-479.31			
	E	1325.2	720	456.43	2	<.001	-114.43			

Note. A,C, and E are standardized squared parameter estimates for additive genetic, common environment, and nonshared environment factors, respectively. D is a standardized squared parameter estimate for dominant genetic factors. The most parsimonious model is indicated in bold. 2LL= -2 log likelihood; df=degrees of freedom; Δ =change; AIC=Akaike's information criterion.

Table 6

ACE Parameter Estimates Moderated by Gestational Age

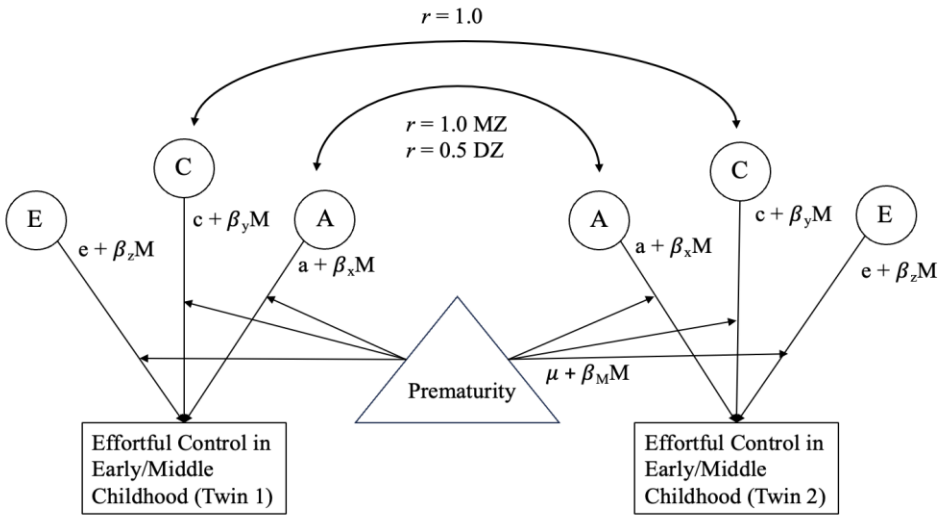
	Model	-2LL	df	△X^2	△df	p	AIC	A	C (or D)	E
Duration of Orienting 12mo visit	ACE – Full Mod.	472.31	549	—	—	—	-625.69	.08	.88	.04
	No Mod.	**474.24**	**552**	**1.93**	**3**	**0.59**	**-629.76**	**.07**	**.90**	**.02**
Attentional Focusing 30mo visit	ADE – Full Mod.	1343.57	482	—	—	—	379.57	.59	.03	.38
	No Mod.	**1344.55**	**485**	**0.98**	**3**	**0.81**	**374.55**	**.41**	**.24**	**.35**
Inhibitory Control 30mo visit	ACE – Full Mod.	1266.78	489	—	—	—	288.78			
	No Mod.	**1270.14**	**492**	**3.36**	**3**	**0.34**	**286.14**	**.45**	**.27**	**.28**
Activation Control 8yr visit	**ACE – Full Mod.**	**772.92**	**612**	—	—	—	**-451.08**	**.62**	**.27**	**.10**
	No Mod.	782.07	615	9.15	3	0.03	-447.93			
	No E Mod.	787.70	613	14.78	1	<.001	-438.30			
	No C Mod.	796.29	613	23.37	1	<.001	-429.71			
	No A Mod.	796.71	614	23.79	2	<.001	-431.29			
Attentional Focusing 8yr visit	ADE – Full Mod.	1560.69	612	—	—	—	336.69			
	No Mod.	**1562.01**	**615**	**1.32**	**3**	**0.72**	**332.01**	**.00**	**.64**	**.36**
Inhibitory Control 8yr visit	ACE -Full Mod.	911.10	612	—	—	—	-312.9			
	No Mod.	**914.17**	**615**	**3.06**	**3**	**0.38**	**-315.83**	**.61**	**.21**	**.18**
Attentional Focusing 9yr visit	ADE – Full Mod.	1677.28	662	—	—	—	353.28			
	No Mod.	**1680.70**	**665**	**3.41**	**3**	**0.33**	**350.70**	**.00**	**.86**	**.14**
Inhibitory Control 9yr visit	ACE – Full Mod.	959.62	662	—	—	—	-364.38			
	ACE – No Mod.	**961.06**	**665**	**1.44**	**3**	**0.70**	**-368.94**	**.92**	**.00**	**.08**
Activation Control 10yr visit	ACE – Full Mod.	1242.65	669	—	—	—	-95.35			
	ACE – No Mod.	**1250.06**	**672**	**7.41**	**3**	**0.06**	**-93.94**	**.69**	**.17**	**.14**
Attentional Focusing 10yr visit	ADE – Full Mod.	1249.55	669	—	—	—	-88.45			
	ADE – No Mod.	**1250.51**	**672**	**0.97**	**3**	**0.81**	**-93.49**	**.58**	**.30**	**.12**
Inhibitory Control 10yr visit	ACE – Full Mod.	816.47	669	—	—	—	-521.53			
	ACE – No Mod.	**808.55**	**672**	**-7.92**	**3**	**1.00**	**-535.45**	**.43**	**.53**	**.04**

Note. A,C, and E are standardized squared parameter estimates for additive genetic, common environment, and nonshared environment factors, respectively. D is a standardized squared parameter estimate for dominant genetic factors. The most parsimonious model is indicated in bold. 2LL= -2 log likelihood; df=degrees of freedom; **Δ** =change; AIC=Akaike's information criterion. Due to insufficient sample size at age 5, models were not estimated for this wave.

Figure 1

ACE Twin Model for Effortful Control in Early Childhood and Middle Childhood:

Moderated by Prematurity



Note. Moderated heritability model that allows for the moderation of one family-level phenotype (i.e., prematurity) on an individual-level phenotype (i.e., effortful control). A = additive genetic variance, C = shared environmental variance, E = nonshared environmental variance, M = moderator, MZ = monozygotic twins, DZ = dizygotic twins. Equations next to each path represent the linear relationship between the path coefficient and the moderator (i.e., prematurity). An interaction between the path coefficient and the moderator is represented when β_x is significantly different from zero (Purcell, 2002).

APPENDIX B

NEONATAL AND OBSTETRICAL COMPLICATION MEASURES

Items Included in the Neonatal Medical Risk Scale (Littman & Paralee, 1974; Minde et al., 1983)

Note: Each of these items was measured separately for each twin

1. Received resuscitation
2. No stable respiration within 6 minutes of birth
3. 5-min Apgar score between 0-6
4. 1-min Apgar score between 0-6
5. Small or large size for gestational age
6. Neonatal heart rate of <100 or >160
7. Neonatal respiratory rate of <30 or >80
8. Infection at birth
9. Noninfectious illness at birth
10. Temperature disturbance
11. No nipple feeding within 48 hours
12. Surgery (other than circumcision)
13. No urination within 24 hours
14. No defecation within 24 hours
15. Twin-to-twin transfusion
16. Bradycardia
17. Tachypnea (rapid respiration, >80 bpm)
18. Apnea (note: 1 = aminophylline/caffeine required, 2 = requiring CPAP, 3 = requiring ventilation)
19. Respiratory distress syndrome (note: 1 = extra oxygen, 2 = requiring CPAP, 3 = requiring ventilation)
20. Hyperbilirubinemia (note: 1 = jaundice needing phototherapy, 2 = exchange transfusion)
21. Nil per os >12 hours per day

Items Included in the Obstetrical Complications Scale (Littman & Paralee, 1974)

1. Premature birth history
2. Stillbirths in history
3. Prolonged unwanted sterility
4. Fertility drugs used with the twin pregnancy
5. Less than 12 months since last pregnancy
6. No prenatal care
7. Smoking during pregnancy
8. Drinking during pregnancy
9. Chronic drug use during pregnancy
10. Cephalopelvic disproportion
11. Bleeding during pregnancy
12. Edema or pre-eclampsia
13. Magnesium sulfate given for preterm labor
14. Bedrest during pregnancy
15. Infections or acute medical problems during pregnancy
16. Drugs given to mother during pregnancy

17. Maternal chronic illness or disease
18. Blood pressure during pregnancy over 140/90
19. Rh antagonism/blood group incompatibility
20. Albuminuria
21. Hospitalized during pregnancy for vomiting/hyperemesis
22. Hospitalized for vomiting/hyperemesis for multiple trimesters during pregnancy
23. Hgb level at the end of pregnancy <10 or hemocrit <30
24. Anesthetic drugs given during labor and delivery
25. Stimulations of contractions (oxytocin and Pitocin)
26. Duration of 1st stage of labor (onset of contractions to full dilation) of less than 3 hours or greater than 20
27. Preterm labor
28. Ruptured membranes longer than 12 hours prior to delivery
29. Stained amniotic fluid (note: 1 = staining for one twin, 2 = staining for both twins)
30. Meconium staining anywhere other than amniotic fluid (note; 1 = staining for one twin, 2 = staining for both twins)
31. Episiotomy
32. Abnormal placental connections
33. Placenta previa or abrupted placenta
34. Fetal heart rate during 1st and 2nd stage labor for twin A < 100 or >160
35. Fetal heart rate during 1st and 2nd stage labor for twin B < 100 or >160
36. Mode of delivery other than vaginal for Twin A (Note: 1 = C-Section, 2 = Vacuum Extraction)
37. Mode of delivery other than vaginal for Twin B (Note: 1 = C-Section, 2 = Vacuum Extraction)
38. Forceps used for delivery for Twin A
39. Forceps used for delivery for Twin B
40. Fetal Presentation for Delivery other than vertex or cephalic for Twin A
41. Fetal Presentation for Delivery other than vertex or cephalic for Twin B
42. Cord around neck for Twin A
43. Cord around neck for Twin B
44. Cord prolapse for Twin A
45. Cord prolapse for Twin B
46. Abnormal cord insertion for Twin A
47. Abnormal cord insertion for Twin B
48. Placental Infarction for Twin A
49. Placental Infarction for Twin B

APPENDIX C

CATEGORICAL GESTATIONAL AGE SUPPLEMENTARY TABLE

Regressions of Gestational Age Measured Categorically on Effortful Control

	Duration of Orienting 12mo visit	Attentional Focusing 30mo visit	Inhibitory Control 30mo visit	Attentional Focusing 5yr visit	Inhibitory Control 5yr visit	Activation Control 8yr visit	Attentional Focusing 8yr visit	Inhibitory Control 8yr visit	Attentional Focusing 9yr visit	Inhibitory Control 9yr visit	Activation Control 10 yr visit	Attentional Focusing 10 yr visit	Inhibitory Control 10 yr visit
MODEL WITHOUT CONTROLLING FOR OBSTETRICAL COMPLICATIONS AND NEONATAL MEDICAL RISK													
Very Preterm	.12 [-.18, .42]	-.09 [-.47, .40]	-.21 [-.62, .20]	-.45 [-.97, .07]	-.45 [-.99, .09]	-.04 [-.28, .21]	-.28 [-.57, .00]	-.05 [-.30, .20]	-.12 [-.41, .18]	-.03 [-.22, .17]	.08 [-.14, .29]	-.08 [-.32, .16]	.01 [-.24, .25]
Age at wave	.14** [.05, .23]	.05 [-.53, .64]	-.51 [-1.05, .02]	-.01 [-.69, .67]	-.23 [-.95, .50]	.06 [-.07, .19]	.11 [-.06, .29]	.08 [-.03, .20]	.06 [-.06, .17]	.03 [-.05, .10]	-.02 [-.09, .05]	.04 [-.03, .11]	-.00 [-.07, .06]
Sex	-.03 [-.21, .15]	.41** [.13, .69]	.46*** [.22, .69]	.20 [-.11, .50]	.48** [.16, .79]	.06 [-.09, .22]	.36** [.14, .59]	.29*** [.14, .44]	.42*** [.21, .64]	.32*** [.18, .46]	.28** [.10, .46]	.21* [.05, .37]	.13 [-.02, .28]
SES at wave	-.08 [-.22, .06]	.16 [-.06, .37]	.19 [-.03, .40]	.17 [-.05, .39]	.14 [-.09, .37]	.12* [.01, .24]	.04 [-.11, .19]	.06 [-.06, .18]	-.11 [-.27, .06]	-.01 [-.11, .10]	-.10 [-.23, .02]	-.05 [-.17, .07]	-.00 [-.12, .11]
R^2	.09	.06	.09	.06	.09	.04	.06	.08	.06	.07	.05	.03	.01
n	269	225	225	159	159	298	298	298	344	344	339	339	339
MODEL CONTROLLING FOR OBSTETRICAL COMPLICATIONS AND NEONATAL MEDICAL RISK													
Very Preterm	.04 [-.38, .46]	-.08 [-1.10, .94]	-.22 [-.94, .49]	-.43 [-1.14, .29]	.44 [-.52, 1.40]	.20 [-.22, .62]	-.44 [-1.08, .20]	.13 [-.46, .73]	.11 [-.48, .70]	.39 [-.02, .80]	.49* [.002, 1.00]	.18 [-.23, .60]	.22 [-.18, .61]
Age at wave	.16* [.003, .32]	.28 [-1.42, 1.98]	.66 [-.26, 1.59]	-.00 [-1.06, 1.05]	-.51 [-1.71, .70]	.06 [-.26, .38]	.25 [-.05, .56]	.03 [-.15, .20]	.02 [-.32, .36]	.09 [-.08, .26]	-.11 [-.31, .08]	.09 [-.11, .30]	.02 [-.21, .24]
Sex	-.09 [-.36, .19]	.36 [-.09, .81]	.50** [.14, .87]	.17 [-.23, .58]	.52* [.05, .99]	.20 [-.03, .43]	.27 [-.11, .64]	.34** [.10, .58]	.32 [-.06, .69]	.36** [.13, .59]	.62** [.25, .99]	.33* [.01, .65]	.29* [.01, .58]
SES at wave	-.03 [-.28, .21]	.32 [-.07, .70]	.04 [-.29, .37]	.26 [-.14, .66]	.04 [-.31, .39]	.07 [-.11, .24]	.18 [-.08, .43]	-.02 [-.25, .21]	-.29* [-.54, -.04]	-.11 [-.30, .07]	-.001 [-.20, .20]	-.03 [-.25, .20]	-.10 [-.36, .16]
Obstetrical Complic.	-.01 [-.05, .04]	.06 [-.07, .70]	-.02 [-.06, .03]	.01 [-.05, .08]	-.02 [-.11, .06]	.01 [-.03, .05]	.02 [-.05, .08]	.02 [-.03, .06]	-.02 [-.08, .06]	.02 [-.02, .05]	.01 [-.08, .06]	-.01 [-.08, .07]	-.01 [-.07, .05]
Neonatal Medical Risk	-.00 [-.05, .05]	.02 [-.09, .12]	.01 [-.07, .08]	.07 [-.00, .14]	-.03 [-.13, .07]	.02 [-.02, .07]	.04 [-.03, .11]	.02 [-.05, .08]	.02 [-.06, .09]	.02 [-.03, .07]	-.01 [-.08, .07]	.03 [-.04, .10]	.03 [-.02, .08]
R^2	.08	.10	.11	.10	.10	.11	.10	.15	.09	.30	.19	.13	.12
n	126	110	110	76	76	111	111	111	89	89	90	90	90

Note. $^{*}p<.05$ $^{**}p<.01$ $^{***}p<0.001$. Each cell contains β and *95% Confidence Intervals*. Each column shows a separate regression. The sample size is the number of cases for which there is data on at least one variable included in analysis. Gestational age was dichotomized into very preterm (less than 32 weeks gestation) and full-term (at least 37 weeks)