Examining Relations Between Parental Behaviour, Children's Characteristics, and Children's Executive Function in Early and Middle Childhood

by

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Abstract

Parental behaviour is an important predictor of executive function (EF) in early childhood and the transition to middle childhood. Yet, not all children are equally impacted by parental behaviour. This dissertation examined interactions and bidirectional relations between parental behaviours and children's characteristics (genotype and EF) on children's EF.

Emerging research suggests that parental behaviour and child genotype interact to predict EF in early childhood, birth to age 5. The first study in this dissertation systematically reviewed research on gene × parental behaviour interactions on children's EF in early childhood. Psychology and psychiatry databases were searched for potentially relevant studies. A total of 17 published peer-reviewed studies met inclusion criteria. Of the 17 studies, 13 (76%) reported at least one significant gene × parental behaviour interaction on children's EF, although only 24 of 51 (47%) interactions were significant. Studies were heterogenous in terms of sample composition; measurement of genes, parental behaviour, and EF; statistical methods; and power. This made it difficult to compare findings and evaluate the strength of the evidence for gene × parental behaviour interactions on EF. To better understand the role of gene × parental behaviour interactions in the development of children's EF, future research should aim to reduce heterogeneity by adopting more rigorous study designs and methods.

The second study in this dissertation examined (1) whether the interactions between cumulative dopaminergic genetic risk and problematic discipline and responsiveness were associated with children's EF, and (2) whether the forms of the interactions were consistent with the diathesis-stress or differential susceptibility model of gene × environment interaction. Participants were 135 36-month-old children and their mothers drawn from a prospective cohort followed longitudinally from pregnancy. Children were genotyped for dopamine active transporter 1 (*DAT1*), dopamine receptor D2 (*DRD2*), dopamine receptor D4 (*D2D4*), and

catechol-O-methyltransferase (*COMT*), and scored on the number of risk alleles they carried. Maternal problematic discipline and responsiveness were coded during a series of structured mother-child interactions. EF was operationalized as self-control and working memory/inhibitory control. Path analysis suggested there was an interaction between genetic risk and problematic discipline on self-control, but not working memory/inhibitory control. Higher problematic discipline was associated with poorer self-control for children with higher genetic risk scores. Results were consistent with the diathesis-stress model, suggesting that children at higher genetic risk may be more vulnerable to the negative effect of problematic discipline. This has implications for developmental psychopathology: Problematic discipline may increase the risk for developing behaviour problems in children at higher genetic risk via its association with poorer self-control.

Children with poorer EF are thought to elicit harsher discipline from their caregivers, with the use of harsh discipline predicting poorer EF. Children with better EF are argued to elicit more appropriate discipline techniques, such as inductive discipline, that support the development of EF. Further, parental behaviour may differentially predict child outcomes depending on children's developmental stage. Study 3 examined bidirectional relations between parental discipline and children's EF, operationalized as inhibitory control, and child age as a moderator of these relations. Participants were 136 4- to 6-year-olds and their primary caregiver. At two timepoints, separated by 12 months, children completed a battery of inhibitory control tasks and parents completed a questionnaire about their use of harsh and inductive discipline. A latent change score model did not find support for bidirectional relations between parental discipline and children's inhibitory control or for age as a moderator of these relations. Results

did not support bidirectional relations between parental discipline and children's inhibitory control during the transition to middle childhood.

This dissertation adds to the literature on the role of interactions and bidirectional relations between parental behaviour and children's characteristics in the development of EF in early childhood and the transition to middle childhood. In the first two studies, parental behaviour and child genotype interacted to predict children's EF. In the third study, there were no significant bidirectional relations between parental discipline and children's inhibitory control. These results suggest that not all children are equally impacted by parental behaviour. A more nuanced understanding of how children's characteristics shape how they are impacted by parental behaviour may allow researchers to identify which children's developing EF may be adversely impacted by or benefit from particular parental behaviours.

Preface

This dissertation is original work by Daphne Maria Vrantsidis. Ethics approval was obtained for studies two and three of this dissertation. Ethnics approval for the study in chapter 3 was obtained from the University of Nebraska-Lincoln Institutional Review Board. Ethnics approval for the study in chapter 4 (study title: *Executive Function in the Transition to School*; study ID: MS3_Pro00027228) was obtained from the University of Alberta Research Ethnics Board.

Chapter 2 was completed at the University of Alberta under the supervision of Dr. Sandra Wiebe and with the assistance of Viktoria Wuest. I developed the research question and data collection tools, collected and analyzed data, and drafted the manuscript. Viktoria Wuest collected data.

Chapter 3 was a project done in collaboration with Dr. Caron Clark (University of Nebraska-Lincoln), Dr. Lauren Wakschlag (Northwestern University), and Dr. Kimberly Andrews Espy (University of Texas at San Antonio), under the supervision of Dr. Sandra Wiebe (University of Alberta). Data were collected at the University of Nebraska-Lincoln by the Cognitive Neuroscience Laboratory under the supervision of Drs. Nicolas Chevalier, Kimberly Andrews Espy, and Sandra Wiebe. I analyzed the data and drafted the manuscript.

Chapter 4 was completed at the University of Alberta under the supervision of Dr. Sandra Wiebe. Mahsa Khoei and Naaila Ali managed the study and collected data. I collected, cleaned, and analyzed the data, and drafted the manuscript.

Dedication

SK who read all 206 pages of my “book.”

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Chapter 1

The Development of Executive Function in Early and Middle Childhood: The Contributions of Parental Behaviour and Candidate Genes

The Development of Executive Function in Early and Middle Childhood: The Contributions of Parental Behaviour and Candidate Genes

Michael and Troy are baking cookies and they have everything they need: ingredients, measuring cups, a mixer, baking sheets, and an oven. They work together measuring and pouring the flour and sugar, being extra careful to make sure they have not missed an ingredient or added one in twice. Michael really wants milk chocolate cookies, while Troy wants white chocolate and they successfully resolve the disagreement, agreeing to add both types of chocolate to the dough. They bake their cookies in the oven, remembering to set the timer so they do not forget about them and burn them. Finally, and most importantly, when Michael and Troy pull the warm, melt-in-your-mouth cookies out of the oven, they divide them up so they can save some for later.

Getting started and sticking with a task; planning; organizing time, items, and ideas; regulating emotions; and delaying gratification all belong to a set of skills called executive function (EF). EF is the set of higher-order cognitive processes necessary for goal-directed behaviour (Garon, Bryson, & Smith, 2008). Early childhood and the transition to middle childhood are important for the development of EF. EF undergoes rapid development during preschool (Clark et al., 2013; Wiebe, Sheffield, & Espy, 2012), and the transition to middle childhood marks a shift towards adult organization (Huizinga, Dolan, & van der Molen, 2006; Lehto, Juujärvi, Kooistra, & Pulkkinen, 2003). Furthermore, EF during these periods lays the foundation for the acquisition of more demanding socioemotional and cognitive competencies – for example, prosocial behaviour (K. Blair, Denham, Kochanoff, & Whipple, 2004) and math ability (C. Blair & Razza, 2007) – and is predictive of long-term outcomes like SAT scores (Mischel, Shoda, & Rodriguez, 1989). Because of the significance of early and middle childhood for the development of EF, it is important to identify factors contributing to individual

differences in emergent EF skills during these periods. Two factors linked to individual differences in EF are parental behaviour and genotype (Barnes, Dean, Nandam, O'Connell, & Bellgrove, 2011; Logue & Gould, 2014; Valcan, Davis, & Pino-Pasternak, 2017). In this dissertation, I examined interactions and bidirectional relations between parental behaviour and children's characteristics (genotype and EF) on children's EF in early childhood and the transition to middle childhood.

The Development of EF in Early and Middle Childhood

The development of EF is closely linked to the development of the prefrontal cortex (PFC) (Moriguchi & Hiraki, 2009). EF begins to emerge in the first year of life (Diamond & Goldman-Rakic, 1989; M. Johnson, 1995), with preschool being a time of substantial qualitative and quantitative development. Between the ages of 3 and 5, children transition from perseverating on the *Dimensional Change Card Sort* task, a measure of set shifting (the ability to switch between using multiple mental sets or rules), to being able to appropriately shift sets (Blakey, Visser, & Carroll, 2016). Further, between the ages of 3 and 5, gains in EF are most dramatic between the ages of 3 and 3.75 (Clark et al., 2013; Wiebe et al., 2012). On the *Go/No-Go* task, a measure of inhibitory control (the ability to inhibit an automatic response), accuracy on no-go trials improves from 47% to 74% (Wiebe et al., 2012). Accuracy and reaction time also improve from 27% to 59% and from 3.83 s to 2.86 s on the set shifting component of *Shape School*, a measure of inhibitory control and set shifting (Clark et al., 2013). During middle childhood, ages 6 to 13, accuracy on incongruent trials of the *Dots* task, a measure of inhibitory control, improves from 88% to 95%, with reaction time decreasing from 620 ms to 403 ms (Davidson, Amso, Cruess, & Diamond, 2006). Performance on set shifting tasks also improves

over this period, with children reaching accuracy and reaction times equivalent to adults between the ages of 11 and 15 (Davidson et al., 2006; Huizinga et al., 2006).

The Structure of EF

EF changes qualitatively, as well as quantitatively, in early and middle childhood. The dominant model of EF in adulthood includes three correlated but separable components: working memory/updating (the ability to hold in mind and manipulate information), inhibitory control, and set shifting (Miyake et al., 2000). EF may be less differentiated in early childhood with the transition to middle childhood marking a shift toward the three factor structure seen in adults (Huizinga et al., 2006; Lehto et al., 2003). Research on the structure of EF in preschoolers has mainly focused on the structure of working memory and inhibitory control and has found that a single-factor model fits the data as well as two-factor or more complex models in 2- to 6-year-olds (Wiebe, Espy, & Charak, 2008; Wiebe et al., 2011; Willoughby, Kupersmidt, Voegler-Lee, & Bryant, 2011). In 6- to 8-year-olds, there is support for a three-factor structure of EF, with working memory, inhibitory control, and set shifting forming separate but correlated factors (Huizinga et al., 2006; Lehto et al., 2003).

There is also empirical support for a second dimension of EF, termed self-control, that is distinct from working memory, inhibitory control, and set shifting (Wiebe et al., 2015; Willoughby et al., 2011; but see Allan & Lonigan, 2011 for an exception). Self-control is the ability to carry out goal-directed behaviour in motivationally or affectively charged circumstances (Zelazo & Carlson, 2012). Self-control is subserved by the ventral and medial parts of PFC, which are part of a broader network involving the amygdala and limbic system (Zelazo & Muller, 2012). Conversely, the ability to carry out goal-directed behaviour in decontextualized circumstances without extrinsic rewards (e.g., working memory tasks) is

subserved by the dorsolateral PFC (Zelazo & Carlson, 2012). Further, the two dimensions of EF are differentially related to developmental outcomes. Self-control predicts the development of behaviour problems, and symptoms of hyperactivity/impulsivity, but not inattention, in children with ADHD (Castellanos, Sonuga-Barke, Milham, & Tannock, 2006; Kim, Nordling, Yoon, Boldt, & Kochanska, 2013; Willoughby et al., 2011). Working memory, inhibitory control, and set shifting uniquely predict cognitive outcomes, like academic performance, and symptoms of inattention in children with ADHD (Castellanos et al., 2006; Kim et al., 2013; Willoughby et al., 2011).

Parental Behaviour and Children's EF

The associations between environmental factors and children's EF are well documented (C. Blair & Raver, 2012; Hughes & Ensor, 2009), with parental behaviour thought to be one contextual factor of particular importance for the development of EF (Hughes & Devine, 2017; Valcan et al., 2017). Multiple dimensions of parental behaviour have been shown to support or compromise EF development, with two dimensions receiving particular attention: responsiveness and discipline, sometimes termed control (Hughes & Devine, 2017; Karreman, van Tuijl, van Aken, & Deković, 2006; Valcan et al., 2017). Responsiveness captures parents' contingent responses to their child's behaviour and how well these responses match the child's behaviour, as well as parental warmth (Hill, Maskowitz, Danis, & Wakschlag, 2008). Parental discipline refers to the ways parents manage or control their children's behaviour (Hill et al., 2008; Valcan et al., 2017). Parents can use positive discipline techniques, such as monitoring or explaining expectations for appropriate behaviour; or negative discipline techniques that are coercive, inconsistent, or harsh, to manage children's behaviour (Hill et al., 2008; Kiff, Lengua, & Zalewski, 2011). More responsive parental behaviour and the use of positive discipline

techniques predict better EF, both concurrently and longitudinally, in children between the ages of 2 and 8 (C. Blair, Raver, & Berry, 2014; Hughes & Ensor, 2005; Roskam, Stievenart, Meunier, & Noël, 2014). The use of negative discipline techniques predicts poorer EF in early and middle childhood (Hughes & Devine, 2019; Lucassen et al., 2015; Roskam et al., 2014).

Why might responsiveness and discipline be associated with children's EF? Multiple theories have been proposed. First, responsiveness and discipline are thought to help regulate or dysregulate children's level of arousal (C. Blair, 2016; Hoffman, 2000). Responsive parental behaviours and positive discipline techniques provide children with an optimal environment for regulating their actions and emotions by reducing their distress and arousal. Conversely, negative discipline techniques, such as physical discipline, prolong children's experience of distress leading to overarousal. Arousal affects EF in an inverted U-shape. Children who are less aroused in distressing situations have better EF skills than children who are overaroused. This is thought to be because overarousal undercuts children's attempts at regulation, impeding their EF development.

Second, responsiveness and discipline are related to how parents teach children to regulate their behaviour in arousing or distressing situations. Parents who are responsive or use positive discipline techniques are more likely to model constructive ways of managing distress, which is related to better EF (Carr & Pike, 2012; Karreman et al., 2006). Conversely, parents who use negative discipline techniques are less likely to teach their children successful strategies to self-regulate their behaviour, which is associated with poorer EF (Eisenberg et al., 2005; Grusec & Davidov, 2010).

Third, differences in parents' responsiveness and discipline techniques are related to differences in children's language exposure. Language is important for the development of EF

(Vygotsky, 1934/2012). Compared to children whose parents are more responsive or use more positive discipline techniques, children whose parents use more negative discipline techniques are typically exposed to fewer and poorer quality language learning experiences, which negatively impacts their language development (Pungello, Iruka, Dotterer, Mills-Koonce, & Reznick, 2009; Raviv, Kessenich, & Morrison, 2004). These differences in language development predict EF abilities (Hammond, Müller, Carpendale, Bibok, & Liebermann-Finestone, 2012; Slot & von Suchodoletz, 2018).

Candidate Genes and EF

Research on candidate genes involved in EF has primarily focused on genes involved in the dopaminergic, serotonergic, norepinephrine, and cholinergic systems because these neurotransmitters modulate activity in the PFC (Logue & Gould, 2014).

The Dopaminergic System. The dopaminergic system is made up of three major pathways (Seamans & Yang, 2004). The nigrostriatal pathway begins in the substantia nigra and projects to the basal ganglia. The mesolimbic pathway begins in the ventral tegmental area and projects to the nucleus accumbens, in the striatum, and olfactory tubercle. The mesocortical pathway begins in the ventral tegmental area and projects to the PFC. Through the mesocortical pathway, dopamine modulates functioning in the PFC. Activity in the PFC depends on the continued release of dopamine, with dopamine levels impacting performance on behavioural tasks dependent on the PFC in an inverted U-shaped fashion (Robbins & Arnsten, 2009). Low levels of dopamine in the PFC are associated with decreased neural activity. As dopamine levels increase, activity in the PFC increases. However, beyond a moderate level, increases in dopamine lead to dopamine receptors becoming saturated and neural activity in the PFC

decreases, impairing performance on behavioural tasks dependent on PFC functioning, such as EF tasks.

Dopaminergic Genes. Several genes are associated with dopamine levels in the PFC and EF, including the genes coding for the dopamine transporter (DAT1), responsible for releasing dopamine into the synaptic cleft; dopamine receptors D2 (DRD2) and D4 (DRD4), which are involved in the post-synaptic uptake of dopamine; and catechol-O-methyltransferase (COMT), involved in the degradation of dopamine (Barnes et al., 2011; Logue & Gould, 2014). Reduced release into the synaptic cleft, increased post-synaptic uptake, and faster degradation of dopamine are associated with lower dopamine levels in the PFC and poorer EF.

The Serotonergic System. The serotonergic system originates in the raphe nuclei with projections extending to the PFC (Puig & Gulledge, 2011). Serotonin is thought to play a role in regulating PFC activity by inhibiting functioning of pyramidal neurons and regulating the release of GABA, making it more or less likely for neurons in the cortex to fire. Nonetheless, the exact mechanisms by which serotonin facilitates PFC activity and cognitive functioning are not fully understood. Research has primarily focused on the effects of serotonin depletion on neurocognitive outcomes: higher levels of serotonin in the PFC are associated with better working memory, inhibitory control, and set shifting, and worse self-control (Anderson, Bell, & Awh, 2012).

Serotonergic Genes. Research on the association between candidate genes involved in serotonergic function and EF has primarily focused on polymorphisms of the gene coding for the serotonin transporter protein (5-HTTLPR), responsible for transporting serotonin from the synaptic cleft to the presynaptic neuron (Barnes et al., 2011; Logue & Gould, 2014). Fewer transporter proteins are associated with less efficient removal of serotonin from the synaptic

cleft, resulting in higher concentrations of serotonin, which differentially predicts performance on working memory, inhibitory control, and set shifting tasks versus self-control tasks (Anderson et al., 2012; Canli & Lesch, 2007).

The Norepinephrine System. The norepinephrine system originates in the locus coeruleus and lateral tegmental field (Ramos & Arnsten, 2007). The PFC receives most of its norepinephrine projections from the locus coeruleus. The PFC is also one of the few higher cortical regions that provides input to the locus coeruleus. Norepinephrine modulates activity in the PFC in a fashion similar to dopamine: moderate levels of norepinephrine increase PFC activity, while low and high levels of norepinephrine impair PFC functioning, thus impairing EF.

Norepinephrine Genes. The gene that codes for the enzyme dopamine β-hydroxylase (DβH), which catalyzes the conversion of dopamine to norepinephrine, has been identified as a candidate gene involved in EF (Barnes et al., 2011). Lower DβH activity is associated with less dopamine-to-noradrenaline conversion, higher dopamine levels, and poorer EF.

The Cholinergic System. The cholinergic system originates in the nucleus basalis of Mynert in the basal forebrain and has diffuse projections throughout the brain (Logue & Gould, 2014). There are two classes of cholinergic receptors, nicotinic and muscarinic, with nicotinic receptors being more clearly associated with EF. Nicotinic receptors indirectly impact EF via their effects on neurotransmitter levels in the PFC. More acetylcholine in the PFC leads to greater dopamine and norepinephrine release. Further, acetylcholine modulates activity in the ventral tegmental area, locus coeruleus, and raphe nuclei which alters the amount of dopamine, norepinephrine, and serotonin released in the PFC. These changes in neurotransmitter levels in the PFC are associated with differences in EF task performance.

Cholinergic Genes. The primary gene involved in cholinergic functioning that is associated with EF is *CHRNA4*, which codes for the cholinergic receptor nicotinic alpha 4 (Logue & Gould, 2014). This receptor is involved in the uptake of acetylcholine in the PFC. Greater uptake of acetylcholine is associated with lower acetylcholine levels in the PFC, and poorer EF.

Models of Gene × Environment Interaction

A gene × environment interaction is a genetic difference in susceptibility to an environmental exposure (Moffitt, Caspi, & Rutter, 2005). Candidate genes related to EF are associated with individual differences in sensitivity to environmental factors, particularly parental behaviour (Bakermans-Kranenburg & Van Ijzendoorn, 2011; Van Ijzendoorn, Belsky, & Bakermans-Kranenburg, 2012). Candidate genes and parental behaviour interact to predict the development of behaviour problems in children (for meta-analyses see Kim-Cohen et al., 2006; A. Taylor & Kim-Cohen, 2007). Deficits in EF play a role in the etiology of behaviour problems (Nigg & Casey, 2005); therefore, candidate genes and parental behaviour are thought to interact to predict children's EF.

There are two prevailing models of the form of gene × environment interaction: diathesis-stress (Monroe & Simons, 1991) and differential susceptibility (J. Belsky et al., 2009; J. Belsky & Pluess, 2009). The diathesis-stress model (Monroe & Simons, 1991) proposes that certain genotypes confer vulnerability to the negative effects of negative environmental factors. In contrast, the differential susceptibility model (J. Belsky et al., 2009; J. Belsky & Pluess, 2009) proposes that particular genotypes confer increased sensitivity to both positive and negative environmental factors, rather than vulnerability to negative factors.

Transactional Models of Child Development

Transactional models of child development (Lerner & Busch-Rossnagel, 1981; Sameroff, 1975; Scarr & McCartney, 1983) emphasize the role of the child in co-creating their development. Children are not "tabulae rasae" where parents, and the larger environment, unilaterally influence their development. Rather, children play an active role in influencing their development. In particular, children's innate characteristics are argued to elicit particular responses from their environment (Sameroff, 1975; Scarr & McCartney, 1983). For example, a longitudinal cross-lagged twin study of 2- to 4-year-olds found that genetic differences in children's cognitive ability predict the quality of cognitive stimulation they receive from their parents, such that children with higher cognitive abilities elicit higher quality cognitive stimulation from their parents (Tucker-Drob & Harden, 2012). Further, the influence of the child on their environment and the environment on the child are interdependent and change as a function of their mutual influence on one another, such that there is a continual and progressive interplay between children and their environment (Sameroff, 1975). In the previous example, parent's provision of cognitive stimulation also predicted children's later cognitive ability (Tucker-Drob & Harden, 2012), suggesting that the parent-child dyad mutually influence each other and the nature of their relationship changes as a result of this mutual influence.

The Proposed Research

In the three studies comprising this dissertation, I explored interactions and bidirectional relations between parental behaviour and children's characteristics (genotype and EF) on children's EF in early and middle childhood. The first study in this dissertation consisted of a systematic review of studies examining gene × parental behaviour interactions on the development of EF from birth to age 5. Although EF is moderately to highly heritable, candidate

gene studies report conflicting findings and many findings have failed to replicate (Klaus, Butler, Curtis, Bridle, & Pennington, 2019). Gene × environment interactions may help explain the heterogeneous findings from candidate gene studies (Ioannidis, Trikalinos, & Khoury, 2006). Thus, there has been a rise in the number of studies looking at the joint contributions of candidate genes and environmental factors, particularly parental behaviour, on the development of children's EF. However, findings from these studies are mixed and the literature has yet to be systematically evaluated. Therefore, the aim of this study was to systematically synthesize findings from studies examining whether candidate genes and parental behaviour interact to predict the development of EF in early childhood.

The second study in this dissertation examined (1) whether the interactions between cumulative dopaminergic genetic risk and parental behaviour (problematic discipline and responsiveness) were associated with children's EF, and (2) whether the forms of the interactions were consistent with the diathesis-stress (Monroe & Simons, 1991) or differential susceptibility model (J. Belsky et al., 2009; J. Belsky & Pluess, 2009). Previous research has examined whether individual candidate genes involved in dopaminergic functioning interact with parental behaviour to predict children's EF (Vrantsidis, Wuest, & Wiebe, under review). However, individual genes account for minimal variability in EF (Fossella et al., 2002). In addition, capturing the full range of positive and negative environmental exposures is necessary to be able to test questions about the form of gene × environment interactions (J. Belsky & Pluess, 2009). This study adopted a polygenic approach, examining interactions between the cumulative effect of genes involved in dopamine transport, reuptake, and catabolism, and two domains of parental behaviour (problematic discipline and responsiveness) meant to capture both negative and positive parental behaviours, on children's EF.

The third study in this dissertation examined (1) bidirectional relations between parents' harsh and inductive discipline and children's inhibitory control, one component of EF, among 4- to 7-year-olds, and (2) child age as a moderator of these relations. While the relation between parental behaviour and children's EF is well established (Valcan et al., 2017), few studies have examined whether children's EF predicts parental behaviour and the transactional nature of this relation. Children with poorer EF are thought to elicit more harsh discipline from their caregivers, which, in turn, predicts poorer EF (J. Belsky, Pasco Fearon, & Bell, 2007; Choe, Olson, & Sameroff, 2013; Eisenberg, Taylor, Widaman, & Spinrad, 2015). Conversely, children with better EF are argued to elicit more appropriate discipline techniques (e.g., inductive discipline) from their caregivers, which are associated with better EF in early and middle childhood (Karreman, van Tuijl, van Aken, & Dekovic, 2008; Roskam et al., 2014). Finally, the behaviours children elicit from their parents changes depending on their developmental stage (Collins, Madsen, & Susman-Stillman, 2002), suggesting children's age may moderate bidirectional relations between parental discipline and children's EF. This study examined these possibilities.

Chapter 2

The Effects of Candidate Gene × Parental Behaviour Interactions on Executive Function in Early Childhood: A Systematic Review

The Effects of Candidate Gene × Parental Behaviour Interactions on Executive Function in Early Childhood: A Systematic Review

Executive function (EF) is a set of cognitive processes necessary for carrying out goal-directed behaviours in situations involving cognitive, motivational, or affective load (Garon et al., 2008; Zelazo & Carlson, 2012). These cognitive processes include, but are not limited to, working memory, inhibitory control, cognitive flexibility, delay of gratification, frustration suppression, and compliance (Carlson, 2005; Diamond, 2013; Garon et al., 2008; Zelazo & Carlson, 2012; Zelazo & Cunningham, 2007). EF is a proposed endophenotype for externalizing behaviour problems (Castellanos & Tannock, 2002; Doyle et al., 2005; Matthys, Vanderschuren, & Schutter, 2013). For example, deficits in working memory are associated with inattentive-type ADHD (Castellanos & Tannock, 2002), while deficits in delay of gratification, frustration suppression, and compliance are associated with the hyperactive/impulsive-type of ADHD and oppositional defiant disorder (Castellanos & Tannock, 2002; Willoughby et al., 2011). By age 5, EF plays a role in the etiology of behaviours problems (Calkins & Fox, 2002; Chang, Olson, Sameroff, & Sexton, 2011; Eisenberg et al., 2015). Therefore, it is important to identify factors contributing to individual differences in emergent EF skills during early childhood, birth to age 5.

Gene × environment interactions, genetic differences in the susceptibility to environmental exposures, are argued to play a role in the development of EF (J. Li & Roberts, 2017; Moffitt et al., 2005). Candidate genes thought to be associated with EF, such as *DRD4*, are associated with individual differences in sensitivity to environmental factors (Bakermans-Kranenburg & Van Ijzendoorn, 2011; Van Ijzendoorn, Belsky, & Bakermans-Kranenburg, 2012). Parent's behaviour is one particularly salient aspect of children's home environment

known to predict EF (for a meta-analysis see Valcan et al., 2017). Candidate genes associated with EF and parental behaviours interact to predict the development of externalizing behaviour problems (for meta-analyses see Byrd & Manuck, 2014; Kim-Cohen et al., 2006; A. Taylor & Kim-Cohen, 2007), suggesting that candidate genes and parental behaviour may interact to predict EF. Examining interactions between child genotype and parental behaviour on the development of EF in early childhood is a growing research area. Yet, the variety of candidate genes and parental behaviours examined, as well as contradictory findings, make it difficult for researchers to follow and evaluate the accumulating evidence on the subject. Therefore, we systematically and comprehensively reviewed studies on gene × parental behaviour interactions on children's EF from birth to age 5.

Candidate Genes Associated with EF

Research on candidate genes associated with EF has focused primarily on genes involved in the catecholaminergic, serotonergic, and cholinergic systems because these neurotransmitters modulate activity in the prefrontal cortex (PFC), which subserves EF (for reviews see Barnes, Dean, Nandam, O'Connell, & Bellgrove, 2011; Logue & Gould, 2014). Dopamine and norepinephrine levels in the PFC impact performance on EF tasks in an inverted U-shaped fashion, such that low and high levels of dopamine and norepinephrine are associated with poorer EF (Ramos & Arnsten, 2007). The most widely researched candidate genes involved in catecholaminergic functioning are the genes coding for catechol-O-methyltransferase (*COMT*; a protein involved in the degradation of dopamine and norepinephrine), dopamine transporter (*DAT1*; responsible for releasing dopamine into the synaptic cleft), dopamine β-hydroxylase (*DβH*; which catalyzes the conversion of dopamine to norepinephrine), dopa decarboxylase (*DDC*; involved in catalyzing the conversion of L-DOPA to dopamine), dopamine receptors D2

and D4 (*DRD2* and *DRD4*; involved in the post-synaptic uptake of dopamine), and monoamine oxidase A (*MAOA*; which breaks down dopamine) (Barnes et al., 2011; Derringer et al., 2010; Logue & Gould, 2014).

Serotonin plays a role in regulating PFC activity, although the exact mechanisms by which serotonin facilitates PFC activity and cognitive functioning are not fully understood (Puig & Gulledge, 2011). Lower serotonin in the PFC is linked to poorer EF (Robbins, 2000). Research on candidate genes involved in serotonergic functioning has linked *SLC6A4*, which codes for the serotonin transporter (responsible for transporting serotonin from the synaptic cleft to the presynaptic neuron); *HTR1A* and *HTR2A,* which code for serotonin receptors 1A and 2A (involved in the post-synaptic uptake of serotonin); and *TPH2*, which codes for tryptophan hydroxylase (involved in the synthesis of serotonin), to EF (Barnes et al., 2011; Logue & Gould, 2014).

Choline is indirectly linked to EF via the effect of acetylcholine on neurotransmitter levels in the PFC (Logue & Gould, 2014). Acetylcholine directly and indirectly (via regulation of activity in the ventral tegmental area, locus coeruleus, and raphe nuclei) modulates PFC activity by altering the amount of dopamine, norepinephrine, and serotonin released into the cortex, which impacts EF performance. Genes involved in the cholinergic system that are associated with EF include *CHRNA4* and *CHRNA5*, which code for cholinergic receptors nicotinic alpha 4 and 5 (involved in the post-synaptic uptake of acetylcholine); and *SLC5A7*, which codes for the choline transporter (involved in transporting choline to cholinergic terminals) (English et al., 2009; Fernandes, Hoyle, Dempster, Schalkwyk, & Collier, 2006; Logue & Gould, 2014).

Parental Behaviour and Children's EF

Parental behaviour is a crucial proximal factor linked to the development of children's EF (Valcan et al., 2017). Two categories of parental behaviour, socioemotional and instructional, independently predict children's EF (Hughes & Devine, 2017; Valcan et al., 2017). Socioemotional behaviours are parental behaviours that are concerned with establishing, monitoring, or maintaining the parent-child relationship. These include positive behaviours like sensitivity, warmth, and engagement. Positive behaviours are associated with better EF, both concurrently and longitudinally, between the ages of 2 and 6 (C. Blair et al., 2014; Hughes & Ensor, 2005; Li-Grining, 2007). Socioemotional behaviours can also be negative; for example, harsh, controlling, inconsistent, and intrusive behaviours. Negative behaviours are associated with poorer EF in early childhood (C. Blair et al., 2011; Cuevas et al., 2014; Rochette & Bernier, 2014). Instructional behaviours are parental behaviours related to instructing, problem-solving, or completing a task, such as scaffolding. Instructional behaviours are associated with better EF in 2- to 5-year-olds (Bibok, Carpendale, & Müller, 2009; Hammond et al., 2012; Hughes & Devine, 2019).

The Present Review

The aim of this systematic review was to comprehensively review studies on gene × parental behaviour interactions on EF in early childhood, from birth to age 5. We also wanted to identify specific candidate genes and parental behaviours that may be risk or protective factors for EF development.

Methods

Registration

This review was registered with the International Prospective Register of Systematic Reviews PROSPERO network (http://www.crd.york.ac.uk/prospero/), registration no. CRD42019147088. The PRISMA statement for transparently reporting systematic reviews and meta-analyses was followed (Moher et al., 2015).

Search Strategy

The search strategy was developed in consultation with systematic review and psychology subject librarians at the University of Alberta. The full search strategy is provided in Appendix A. On August 7th, 2019, the search strategy was run through OVID PsycINFO, Ovid MEDLINE(R) ALL, OVID EMBASE, Web of Science BIOSIS Citation Index, Web of Science Core Collection, and Elsevier Scopus. No study design, date, or language limits were imposed on the search. To limit results to published peer-reviewed articles, publication type limits were applied to PsycINFO, EMBASE, and Web of Science Core Collection to remove dissertations and conference abstracts. Reference lists and citations of included articles and relevant reviews were checked for additional relevant studies. Five key informants, identified through the included studies, were contacted via e-mail and asked to list the five most important papers they were aware of that related to this systematic review. Two key informants responded to the request. Searches were re-run before final data analysis (December 4th, 2019) to retrieve any new studies for inclusion.

Inclusion Criteria

To be included in the systematic review, articles had to be published, peer reviewed studies that satisfied the a priori population, intervention, comparator, and outcome (PICO)

criteria (Schardt, Adams, Owens, Keitz, & Fontelo, 2007). The population was healthy (i.e., no diagnosed genetic, psychological, or neurological disorder) children who completed an EF assessment at least once between birth and age 5 (mean age < 6 years) or, when age was not available, before beginning Grade 1. For longitudinal and intervention studies, this criterion applied to participants' baseline age. Interventions (exposures) were gene × environment interaction studies where: (1) children were genotyped for at least one candidate gene, (2) a direct measure of parents' behaviour was available, and (3) researchers tested for at least one gene × parental behaviour interaction. There was no comparator. The outcome was EF, defined as a set of higher-order cognitive processes necessary for carrying out goal-directed behaviour in situations involving cognitive, motivational, or affective load (Garon et al., 2008; Zelazo & Carlson, 2012). For studies to be included in the review, EF had to be operationalized as a construct separate from other domains (e.g., studies using an aggregate measure of EF and aggression would be ineligible). Studies published in languages other than English were included if the article could be translated with Google Translate (Balk et al., 2012).

Study Selection

The number of studies screened at each stage of the selection process and reasons for exclusion are shown in Figure 2-1. Search records were imported into RefWorks where duplicate records were removed. Two independent reviewers scanned the titles and abstracts of all potentially relevant articles. For all articles meeting initial screening criteria, full text copies were obtained. If at least one reviewer included an article, the article was obtained for further review. The same two independent reviewers screened the full text articles against the inclusion criteria. Inclusion discrepancies were resolved by discussions between reviewers, or by a third reviewer, if required.

Data Collection

Data were extracted by one of the reviewers and verified by the second reviewer. Information on publication year, study design, population, measurement of exposures and outcomes, statistical methods, and results were extracted and recorded in Microsoft Excel. Results of included studies were deemed significant at a $p < .05$.

Quality and Risk of Bias Assessment

Study quality and risk of bias were assessed by two independent reviewers. Because no established risk of bias assessment for gene × environment interaction studies exists, we used a quality checklist derived from the Strengthening the Reporting of Genetic Association Studies (STREGA; Little et al., 2009) and Strengthening the Reporting of Observational Studies in Epidemiology (STROBE; von Elm et al., 2014) checklists to evaluate the methodological quality of included studies. This checklist has been used in meta-analyses of gene × environment interactions (Hosang, Shiles, Tansey, McGuffin, & Uher, 2014; Karg, Burmeister, Shedden, & Sen, 2011). The quality criteria were (1) clear statements of objectives and hypotheses, (2) clear eligibility criteria for study participants, (3) clear definitions of all variables, (4) replicability of statistical methods, (5) assessment of Hardy-Weinberg equilibrium, (6) assessment of ethnicity, (7) addressed the problem of mixed ethnicities statistically (if applicable), (8) sufficient descriptive data (age, sex, and ethnicity), (9) statement of genotype frequencies, (10) sample in Hardy-Weinberg equilibrium, and (11) consideration of population stratification. Items were rated as either "+" (present), "-" (absent), or "not applicable". In accordance with current guidelines (Higgins & Green, 2019), the studies were not weighted by quality scores or excluded based on low scores. Discrepancies between reviewers were resolved by discussion among

reviewers, or by a third reviewer, if required. Inter-rater reliability was high (mean κ = .85; mean exact agreement = 98%).

Results

Study Characteristics

Seventeen studies were included in the review. An overview of their characteristics is provided in Table 2-1. The studies involved a total of 3137 participants. The sample sizes for individual studies ranged from 45 to 436 participants (*Mdn* = 150). Four studies used samples from one longitudinal cohort recruited to investigate children's socioemotional development (Y. Li et al., 2016; Sulik et al., 2012, 2015; Z. Taylor et al., 2014) and three studies used samples from one longitudinal cohort recruited to investigate the development of temperament (Sheese, Rothbart, Voelker, & Posner, 2012; Sheese, Voelker, Rothbart, & Posner, 2007; Voelker, Sheese, Rothbart, & Posner, 2009). All 17 studies used an observational study design. Three studies were prospective cohorts (Kok et al., 2013; Pickles et al., 2013; Quan et al., 2017), eight were longitudinal (Augustine, Leerkes, Smolen, & Calkins, 2018; Davies & Cicchetti, 2014; Kochanska, Philibert, & Barry, 2009; Y. Li et al., 2016; Sheese et al., 2012; Sulik et al., 2012, 2015; Z. Taylor et al., 2014), and six were cross-sectional (Montirosso et al., 2015; Sheese et al., 2007; Smith, Kryski, Sheikh, Singh, & Hayden, 2013; Smith et al., 2012; Voelker et al., 2009; Zhang, Chen, Deng, & Lu, 2014). Studies spanned almost the entire age range covered by the systematic review. Child age at first EF assessment ranged from 4 to 48 months (*Mdn* = 24 months). Studies were conducted in six countries (USA: *n* = 11, England: *n* = 1, Italy: *n* = 1, Netherlands: *n* = 1, China: *n* = 1, Singapore: *n* = 1, and not reported: *n* = 1). All studies were published between 2007 and 2020 and five were published since 2015.

Gene × Parental Behaviour Interactions

Results are summarized in Table 2-2 and described below in four sections organized by candidate gene. Genes involved in catecholaminergic functioning are presented first, followed by genes involved in serotonergic, cholinergic, and oxytocinergic functioning.

Catecholaminergic Genes. Twelve studies tested for interactions between candidate genes involved in catecholaminergic functioning (*DRD4*: $n = 5$, *COMT*: $n = 4$, *MAOA*: $n = 2$, *DRD2*: $n = 1$, and *DAT1*: $n = 1$) and parental behaviour (parenting quality: $n = 5$, maternal sensitivity: $n = 4$, positive parenting: $n = 2$; negative parenting: $n = 2$, positive discipline: $n = 1$, and negative discipline: $n = 1$) on EF (inhibitory control: $n = 6$, frustration suppression: $n = 2$, compliance: $n = 2$, executive attention: $n = 2$, and attention focusing: $n = 1$). Nine of twelve studies (75%) reported at least one significant interaction between a catecholaminergic gene and parental behaviour on EF. Overall, 14 of 31 (45%) gene × parental behaviour interactions were significant.

DRD4. Five studies tested for an interaction between *DRD4* and parental behaviour on children's EF and results were conflicting. In a longitudinal study of 18- to 48-month-olds, the interaction between *DRD4* and parenting quality only predicted inhibitory control (the ability to inhibit a prepotent or automatic response in favor of a subdominant response; Rothbart & Rueda, 2005) after 36-months of age (Sheese et al., 2012, 2007). Among 36- to 48-month-old children, parenting quality was associated with better inhibitory control among children with at least one 7-repeat allele (Sheese et al., 2012). Results of a cross-sectional study of 36- to 47-month-olds were similar (Smith et al., 2013). Compared to children without a 7-repeat allele, children with at least one 7-repeat had better inhibitory control when positive parenting was high. *DRD4* did not moderate the relation between negative parenting and inhibitory control. Importantly, a direct

replication found the opposite pattern of results: negative parenting was associated with poorer inhibitory control in 36- to 48-month-olds with at least one 7-repeat allele and was not associated with inhibitory control in children without a 7-repeat allele (Smith et al., 2012). *DRD4* did not moderate the relation between positive parenting and inhibitory control.

A prospective cohort study (Kok et al., 2013) examined interactions between *DRD4* and positive and negative discipline on children's compliance, the ability to comply with a directive in the face of temptation (e.g., withholding eating candies during the *Snack Delay Task*; Wiebe et al., 2015; Zelazo & Carlson, 2012). *DRD4* did not significantly interact with either discipline measure to predict children's compliance at 36-months.

COMT. Across four studies there was conflicting evidence for an interaction between *COMT* and parental behaviour on children's EF. There was support for an interaction between the *COMT* Val(108/158)Met polymorphism and parents' positive discipline, but not negative discipline, on children's compliance at 36 months (Kok et al., 2013). The association between positive discipline and compliance was strongest for children with two Met alleles of the Val(108/158)Met polymorphism and weakest for children with two Val alleles.

There was also support for an interaction between *COMT* and parenting quality on children's inhibitory control at 42 months, but not attention focusing (a component of inhibitory control involving the ability to maintain attention on a stimulus; Derryberry & Rothbart, 1988), or growth in either construct between 42 and 84 months (Sulik et al., 2015). Children were assigned to either the ValGG haplotype present or absent group based on whether they had at least one copy of the Val allele of Val(108/158)Met, one copy of the G allele of the 3'UTR VNTR, and one copy of the G allele of the Intron 1 SNP. The interaction between haplotype and parenting quality differed by sex. For girls with the ValGG haplotype, higher quality parenting

was associated with better inhibitory control. For boys without the ValGG haplotype, parenting quality was positively associated with inhibitory control.

In a sample of 45 18- to 21-month-olds, parenting quality was associated with two of three indices of executive attention (the voluntary focusing and shifting of attention; Eisenberg, Smith, Sadovsky, & Spinrad, 2004) – total anticipatory looks and correct anticipatory looks, but not incorrect anticipatory looks – for children with two *COMT* haplotypes associated with lower pain sensitivity (Voelker et al., 2009). However, when the Val(108/158)Met polymorphism was examined individually, Val(108/158)Met interacted with parenting quality to predict total anticipator looks and incorrect anticipatory looks, but not correct anticipatory looks. A larger prospective cohort study (n = 209) did not find an interaction between the Val(108/158)Met polymorphism and maternal sensitivity on two measures of executive attention, derived from the Visual Expectation Task, in 6-month-olds (Quan et al., 2017).

MAOA. Two studies tested for interactions between *MAOA* and maternal sensitivity on frustration suppression, the ability to use top-down processes to regulate affect (e.g., regulating negative affect when receiving an undesirable gift; Carlson, 2005; Zelazo & Carlson, 2012; Zelazo & Cunningham, 2007). Both studies reported significant interactions between *MAOA* and maternal sensitivity on children's frustration suppression, although the pattern of results differed across studies. An epidemiological prospective cohort found sex differences in the interaction in 14-month-olds (Pickles et al., 2013). For girls, having two high expression (3.5- or 4-repeats) alleles of the uVNTR polymorphism was associated with poorer frustration suppression when maternal sensitivity was high. For boys, having one low expression allele (3- or 5-repeats) was associated with better frustration suppression when maternal sensitivity was high. Conversely, a cross-sectional study of 6-month-old children found that, compared to girls with two copies of

the high expression (4-repeat) allele, girls with two copies of the low expression (3-repeat) allele had poorer frustration suppression when maternal sensitivity was low (Zhang et al., 2014). For boys, the interaction between *MAOA* and maternal sensitivity on frustration suppression was not significant.

DAT1. One study tested for an interaction between *DAT1* and parenting quality on inhibitory control. Using the same data as in Sulik et al. (2015), Y. Li et al. (2016) tested for interactions between *DAT1* and parenting quality on inhibitory control at 30 months and growth in inhibitory control between 30 and 50 months. Planned contrasts tested for interactions between parenting quality and three non-orthogonal haplotypes: Intron 8/Intron 13 haplotype, Intron 12/3'UTR haplotype, and Intron 8/3'UTR haplotype. For the Intron 8/Intron 13 haplotype, children were assigned to the AG present or absent group based on whether they had at least one copy of the A allele of Intron 8 and one copy of the G allele of Intron 13. For the Intron 8/3'UTR VNTR haplotype, children were assigned to the A10 present or absent group based on whether they had at least one copy of the A allele of Intron 8 and one copy of the 10-repeat allele of the 3'UTR VNTR. For the Intron 13/3'UTR VNTR haplotype, children were assigned to the G10 present or absent group based on whether they had at least one copy of the G allele of Intron 13 and one copy of the 10-repeat allele of the 3'UTR VNTR. For children with the AG haplotype, A10 haplotype, and without the G10 haplotype, parenting quality was associated with inhibitory control at 30 months, but not growth in inhibitory control.

DRD2. One study tested for an interaction between *DRD2* and maternal sensitivity on inhibitory control. *DRD2* did not interact with an aggregate measure of early maternal sensitivity (assessed at 6 and 12 months) to predict children's compliance at 24 months (Augustine et al., 2018).

Serotonergic Genes. Six studies tested for an interaction between *SLC6A4* (n = 6) and parental behaviour (parenting quality: n = 2, attachment style: n = 1, maternal sensitivity: n = 1, maternal social engagement: n = 1, and maternal unresponsiveness: n = 1) on EF (frustration suppression: n = 3, inhibitory control: n = 1, compliance: n = 1, and cognitive flexibility: n = 1). Four studies (66%) reported at least one significant interaction. In total, 9 of 17 (53%) interactions were significant.

A longitudinal study found that the 5-HTTLPR polymorphism of *SLC6A4* moderated the effect of attachment style at 15 months on a composite measure of inhibitory control between 25 and 52 months, such that attachment style was only associated with inhibitory control for children with at least one short allele (Kochanska et al., 2009).

There was support for an interaction between *SLC6A4* and parenting quality on children's noncompliance. Sulik et al. (2012) assigned children to one of three haplotype groups: S10, comprising children with at least one short allele of the 5-HTTLPR polymorphism and one 10-repeat allele of the STin2 VNTR polymorphism; S12, comprising children with at least one short allele of 5-HTTLPR and one 12-repeat allele of STin2; and L10-L12, comprising children with two long alleles of 5-HTTLPR and at least one 10- or 12-repeat allele of STin2. Parenting quality was negatively associated with (1) noncompliance at 18 months for children with the L10-L12 haplotype and (2) growth in noncompliance, between 18 and 24 months, for children with the S10 haplotype. For the individual polymorphsisms, parenting quality was negatively associated with noncompliance for children with a short allele of 5-HTTLPR. The interactions between STin2 and parenting quality on baseline noncompliance and growth in noncompliance were not significant.

A cross-sectional study of 4-month-olds found that increased maternal social engagement was associated with fewer displays of frustration and better frustration suppression for infants with at least one short allele of the 5-HTTLPR polymorphism (Montirosso et al., 2015). A second cross-sectional study, of 6-month-olds, found that the effect was sex-specific: for girls, the presence of two long alleles of 5-HTTLPR was associated with better frustration suppression when maternal sensitivity was low (Zhang et al., 2014). For boys, there was no significant interaction between 5-HTTLPR and maternal sensitivity on frustration suppression. However, a longitudinal study did not find a significant interaction between 5-HTTLPR and maternal unresponsiveness at 24 months on children's frustration suppression at 36 months (Davies & Cicchetti, 2014).

One study tested for interactions between *SLC6A4* haplotypes and parenting quality on children's cognitive flexibility, the ability to modify thoughts and behaviours in response to changing circumstances, such as goals or environmental factors (Diamond, 2013), using the same data and haplotype groups as in Sulik et al. (2012) (Z. Taylor et al., 2014). *SLC6A4* haplotypes-and parenting quality did not significantly interact to predict cognitive flexibility at 18 months or growth in cognitive flexibility between 18 and 84 months.

Cholinergic Genes. One study tested for an interaction between *CHRNA4*, rs1044396 polymorphism, and maternal sensitivity on two measures of executive attention, derived from performance on a Visual Expectation Task, in 6-month-olds (Quan et al., 2017). The interaction between *CHRNA4* and maternal sensitivity on either executive attention measure was not significant.

Oxytocinergic Genes. One study tested for an interaction between *OXTR*, which codes for the oxytocin receptor, and maternal sensitivity on children's compliance. Low maternal

sensitivity at 6 months predicted poorer compliance at 24 months for children without an A allele of the rs53576 polymorphism compared to children with at least one A allele (Augustine et al., 2018).

Risk of Bias

Study quality and risk of bias is presented in Table 2-3. In accordance with current guidelines (Higgins & Green, 2019), the individual studies were not assigned quality scores. More than 60% of studies included objectives or hypotheses (n = 17), eligibility criteria (n = 11), sufficient information to replicate statistical methods (n = 16), reported descriptive data (age, gender, and ethnicity; n = 13), reported genotype frequencies (n = 13), assessed ethnicity (n = 15), assessed Hardy-Weinberg equilibrium (n = 14), and addressed mixed ethnicities statistically (n = 8 of 13). Less than 50% of studies defined variables (n = 6) or considered the impact of population stratification on their results (n = 8).

Discussion

The aim of this review was to systematically examine evidence for gene × parental behaviour interactions on children's EF between birth and age 5. Supplementary aims of this review were to identify candidate genes and parental behaviours that confer risk or resilience for EF development. Overall, the majority of studies found evidence for interactions between candidate genes and parental behaviour: 13 of 17 studies (76%) found at least one significant gene × parental behaviour interaction on children's EF, although less than half of interactions were significant (24 of 51; 47%). Further, significant interactions were reported in 9 of 12 (75%) studies examining catecholaminergic genes, 4 of 6 (67%) studies examining serotonergic genes, 0 of 1 (0%) study examining cholinergic genes, and 1 of 1 (100%) study examining oxytocinergic genes. Nonetheless, heterogeneity across studies and a lack of direct replications

made it difficult to make cross-study comparisons and evaluate the strength of the evidence. In particular, we were unable to draw conclusions about the strength of the evidence for gene × parental behaviour interactions on EF nor about the importance of specific candidate genes and parental behaviours. Before firm conclusions can be made, the limitations in the literature, particularly regarding the lack of consistency in operationalization and measurement of EF and parental behaviour, need to be addressed.

Limitations of the Research on Gene × Parental Behaviour Interactions and EF

The fundamental limitation that impacted our ability to synthesize the literature was heterogeneity in the measurement of EF and parental behaviour. Gene × environment correlations, insufficient statistical power, and inconsistences in the reporting of results also limited our ability to make cross-study comparisons and evaluate the strength of the evidence.

It is unclear to what extent the measures of EF across studies assess the same constructs or map onto the same underlying neurophysiology. If the phenotypes in genetic studies do not share the same underlying biology, genetic effects may be masked and interpretation of how genes are related to the phenotype in question may be confounded by differences in the measurement of the phenotype and underlying neurobiology (Kendler, 2013). This is a known issue with candidate gene research on EF. For example, a recent meta-analysis found no significant associations between candidate genes (*ANKK1* and *DRD2*) and EF, with sensitivity analyses suggesting that the null findings were due to heterogeneity in the measurement of EF across studies (Klaus et al., 2019).

There were three main concerns regarding heterogeneity in the operationalization and measurement of EF. First, there is a longstanding debate about the structure of EF: some researchers consider EF to be a unitary construct (Baddeley, 1986; Norman & Shallice, 1986);

others adopt a three-factor model comprised of inhibitory control, working memory/updating, and cognitive flexibility (Friedman & Miyake, 2017; Miyake et al., 2000); and others further break down these factors into more fine-grained constructs (e.g., dividing inhibitory control into response inhibition, interference control, and resisting temptation) (Diamond, 2013). The studies included in this review conceptualized EF differently. For example, all but one study (Sulik et al., 2015) treated inhibitory control as a unitary construct. These discrepancies may contribute to inconsistent findings across studies and hamper interpretation of results. There is an argument to be made for more fine-grained measurement of EF. Decomposing EF measures into their constituent components could allow researchers to determine which components are most impacted by gene × parental behaviour interactions and evaluate the biological plausibility of proposed pathways linking gene × parental behaviour interactions to EF (Caspi & Moffitt, 2006). It also reduces heterogeneity in the operationalization of EF, allowing for valid cross-study comparisons (Klaus et al., 2019).

Second, individual EF tasks are often unreliable measures of EF because task performance reflects both EF abilities and the basic abilities required to complete the task (e.g., motor skills). This issue is commonly referred to as the task impurity problem (Miyake et al., 2000). Most of the reviewed studies (12 of 17; 71%) used a single task or questionnaire to assess EF. Using multiple measures of each EF construct allows for the creation of a latent factor or composite score (Snyder, Miyake, & Hankin, 2015). This would improve construct reliability and the ability to detect gene × parental behaviour interactions on EF (McArdle & Prescott, 2010; Snyder et al., 2015).

Third, the studies included in this review differed in their measurement of EF. For example, the six studies that tested for a gene × parental behaviour interaction on inhibitory

control used four different measures. In particular, half of the studies used a questionnaire (e.g., the *Children's Behavior Questionnaire*; see Sheese et al., 2012; Sulik et al., 2015) and half used a battery of tasks (e.g., *Tower of Patience* and *Snack Delay*; see Smith et al., 2013, 2012). Questionnaires and task-based measures of EF are thought to measure different constructs because correlations between the two types of measures tend to be low in magnitude (Toplak, West, & Stanovich, 2013). Questionnaires are thought to capture behaviours in real-world situations, while tasks are thought to capture specific neurocognitive processes (Snyder et al., 2015). This suggests that studies may not have assessed the same construct or used measures that map onto the same underlying neurophysiology. Consistency in the measurement of EF, and its subcomponents, is necessary to reduce heterogeneity in the measurement of EF across studies. As a first step, conducting direct replications rather than indirect replications would increase the number of studies using similar measures of EF (Klaus et al., 2019).

There was heterogeneity in the measurement of parental behaviour, both in terms of constructs assessed and reliability of measures. All 17 studies examined socioemotional parental behaviours, that is behaviours concerned with the quality of the parent-child relationship. Nonetheless, across studies, nine different socioemotional parental behavioural constructs were assessed (e.g., seven studies examined parenting quality). In addition, only 6 of 17 studies clearly defined their construct. For example, two of the seven studies that defined parenting quality defined it as accepting, supportive, and responsive behaviours toward the child (Y. Li et al., 2016) or as sensitive parenting, in contrast to intrusive, overprotective, or harsh parenting (Z. Taylor et al., 2014). Therefore, even when studies purported to examine the same construct, it is not clear that constructs were defined in the same way. Novel and indirect replications are four times more likely than direct replications to report significant results (Duncan & Keller, 2011).

More direct replications, defining and operationalizing parental behaviours in the same way, are needed to help identify false positives. Studies also differed in the quality of their parental behaviour measures. Only eight studies (47%) provided evidence for reliability. Of these, Cronbach's alphas ranged from .66 (Smith et al., 2012) to .82 (Davies & Cicchetti, 2014; Smith et al., 2012). Low Cronbach's alpha reduces power (Wong, Day, Luan, Chan, & Wareham, 2003). Maximizing the reliability of measures of parental behaviour (e.g., by using measures with proven reliability and empirical precedent) can increase power to detect significant gene × parental behaviour interactions on EF (Wong, Day, Luan, & Wareham, 2004).

Gene × environment interactions are confounded by the presence of gene × environment correlations, correlations between genotype or allele frequency and the environmental factor under study (Manuck & McCaffery, 2014; Rutter & Dodge, 2011). Only 8 of 17 studies reported bivariate correlations between candidate genes and parental behaviour. Further, one study (Kochanska et al., 2009) reported a significant gene × parental behaviour correlation but did not account for this correlation in the analyses. Therefore, it is unclear to what extent gene × environment correlations account for the observed interactions. Multiple statistical approaches to test and control for the effects of gene × environment correlations when examining gene × environment interactions have been published (see Dick et al., 2015). Controlling for any pre-existing relations between candidate genes and environmental factors would strengthen the evidence for gene × parental behaviour interactions on children's EF.

Most studies were underpowered to detect significant gene × parental behaviour interactions on children's EF. Only 1 of 17 studies (Y. Li et al., 2016) conducted a power analysis. Low statistical power likely resulted in failures to detect significant effects and overestimates of effect sizes among studies reporting significant results (Button et al., 2013).

Assuming three predictors and an alpha of .05, 80% power to detect a gene × environment interaction with a large effect size (r^2 = .05) requires an estimated sample size of 2185 participants (Pasman, Verweij, & Vink, 2019). The largest sample size among included studies was 436 participants. Statistical power depends on the size of the smallest group in an analysis, so it is maximized when major and minor allele frequencies and exposure rates are 50% (Caspi, Hariri, Holmes, Uher, & Moffitt, 2010). Selective sampling of individuals with minor allele frequencies or rare environmental exposures can improve power while reducing the sample size necessary to detect an effect by as much as 70% (Boks et al., 2007). Likewise, increasing the reliability of EF and parental behaviour measures can offset the detrimental impact of small sample sizes on power (Wong et al., 2004). Finally, meta-analysis would also help address power issues. However, there were at most six studies examining the same gene (*SLC6A4*), and at least 10 studies are required for a meta-analysis (Higgins et al., 2019).

We had to focus our review on statistically significant findings rather than on the magnitude of effects as only 6 of 17 studies reported standardized effect sizes. Furthermore, information on the precision of estimates was not always available: three studies reported standard errors for all analyses, seven studies partially reported standard errors, and one study reported 95% confidence intervals. Additionally, only one study reported results without covariates making it difficult to evaluate the robustness of findings. There are multiple guidelines and recommendations for improving methodological quality and transparent reporting in gene × environment interaction studies (e.g., Dick et al., 2015; Dunn et al., 2011; Little et al., 2009; NCI-NHGRI Working Group on Replication in Association Studies, 2007). More complete reporting of statistics, including standardized effect sizes, null findings, exact p values, sample

sizes per test, and results without covariates, is necessary to help increase confidence in findings and allow researchers to interpret and compare results across studies.

Future Directions

This systematic review highlights several directions for future research. One avenue for future research involves strengthening the evidence for gene × parental behaviour interactions on EF via the use of more rigorous study methods and designs, and direct replication. A second direction is to address gaps in the literature and theoretical questions related to the role of gene × parental behaviour interactions in the development of EF. This review identified important gaps in the literature, including a lack of research on theoretically important genes, parental behaviours, and EF components, and the generalizability of findings. It is also unclear whether there is longitudinal change in how genes and parental behaviour interact to influence children's EF and whether genes confer vulnerability or differential susceptibility to environmental factors.

Improving the methodological rigor of studies will build confidence in the strength and quality of the evidence base. All of the studies in this review examined single candidate genes. Individual genes account for minimal variability in EF (Barnett, Scoriels, & Munafò, 2008) and are unlikely to produce statically significant findings, especially when sample sizes are small (Dick et al., 2015). Given that multiple genes are involved in EF, the use of cumulative genetic risk or polygenic risk scores can address this shortcoming, increasing power to detect significant associations. A cumulative genetic risk score is a sum score of the number of risk alleles of polymorphisms of candidate genes an individual carries (J. Belsky & Beaver, 2011). A polygenic risk score is a weighted sum score of the number of risk alleles where the weights are effect sizes from genome-wide association studies (GWAS) (Duncan et al., 2019). Both can be used to test for gene × environment interactions, although polygenic risk scores yield more consistent

findings than cumulative genetic risk scores (Pasman et al., 2019). Finally, Genome-Environment-Wide Interaction Studies (GEWIS) is an emerging methodology that uses GWAS to test for gene × environment interactions in a hypothesis-free manner (Thomas, 2010). The selection of genes in candidate gene research is theory-driven, based on biologically plausible pathways to the outcome of interest (Moffitt, Caspi, & Rutter, 2006). This has the advantage of prespecifying a clear biological basis for the role of genes in EF. However, researchers' ability to identify genes related to outcomes using a theory-driven approach is poor (Kendler, 2013). Thus, the GEWIS approach is likely to aid in the identification of important genes linked to both environmental sensitivity and EF. GEWIS have recently been used to identify gene × trauma exposure interactions involved in alcohol misuse (Hawn et al., 2018; Polimanti et al., 2018) suggesting they have potential to inform research on gene × parental behaviour interactions on children's EF.

To date, all studies on gene × parental behaviour interactions on children's EF have used an observational study design. Observational study designs are typical of the field. Nonetheless, they provide low quality evidence for gene × environment interactions (GRADE Working Group, 2004; Higgins & Green, 2019). This is because they cannot rule out the effects of plausible confounders, such as the presence of a gene × environment correlation, as they are not an experimental study design. Randomized control trials (RCTs) provide the highest quality, causal, evidence for gene × environment interactions because they eliminate the effects of plausible confounders through the use of random assignment and experimental manipulation. RCTs have been used to demonstrate that parenting interventions are more effective at decreasing externalizing behaviour for children with risk alleles of candidate genes, such as *DRD4* (Bakermans-Kranenburg, van IJzendoorn, Pijlman, Mesman, & Juffer, 2008; Chhangur,

Weeland, & Belsky, 2017). The use of experimental study designs would provide causal evidence that the impact of parental behaviour on children's EF is dependent on children's genotype.

This review identified only one direct replication attempt (Smith et al., 2013). Direct replications of existing findings, particularly for under-researched constructs, such as *DAT1*, *OXTR*, cognitive flexibility, and attention focusing are needed. Further, pre-registration of studies can also help distinguish between replication attempts and exploratory research, and reduce publication bias (Munafò et al., 2017). Studies can be preregistered on websites such as Open Science Framework (http://osf.io/), AsPredicted (http://AsPredicted.org/), and AEA Registry (http://socialscienceregistry.org). Direct replications using independent samples and pre-registration would allow researchers to substantiate initial reports of gene × parental behaviour interactions, building evidence for specific gene × parental behaviour interactions on specific components of EF.

Several theoretically relevant candidate genes, parental behaviours, and EF components have yet to be studied. Future research should examine key candidate genes involved in catecholaminergic, serotonergic, and cholinergic neurotransmission, including *CHRNA5, DβH*, *HTR1A*, *HTR2A*, and *TPH2* (Barnes et al., 2011; Logue & Gould, 2014), as potential mediators of the effects of parental behaviour on children's EF. In addition, stress response systems (e.g., HPA axis) are theorized to confer differential susceptibility to parental behaviour (Boyce & Ellis, 2005; Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2011). As stress is linked to EF, genes involved in HPA axis functioning like *NR3C1* (glucocorticoid receptor gene) and *CRHR1* (corticotropin-releasing hormone receptor gene) may moderate the effects of parental behaviour on EF (J. Belsky et al., 2015; Blair, 2010; van Ijzendoorn & Bakermans-

Kranenburg, 2014). Furthermore, two categories of parental behaviour, socioemotional and instrumental, are associated with children's EF (Hughes & Devine, 2017). Candidate genes may moderate the association between instrumental behaviours, like autonomy support, and children's EF (Bernier, Carlson, & Whipple, 2010). Finally, unexamined components of EF, such as working memory (the ability to hold in mind and manipulate information; Snyder et al., 2015) and common EF (a higher-order general factor that accounts for the covariation among individual EF components; Snyder et al., 2015), may be impacted by gene × parental behaviour interactions. In addition, it is possible that gene × parental behaviour interactions impact common EF rather than specific EF components like inhibitory control (Friedman & Miyake, 2017).

There was preliminary evidence for ethnic differences in gene × parental behaviour interactions on children's EF. The short allele of the 5-HTTLPR polymorphism of *SCL6A4* was the risk allele in predominantly European American and Han samples (Kochanska et al., 2009; Zhang et al., 2014), while the long allele was the risk allele in an African American sample (Davies & Cicchetti, 2014). Further, parental behaviours vary across ethnic groups. For example, African American parents tend to be higher in control and intrusion than European American parents (Tamis-LeMonda, Briggs, McClowry, & Snow, 2008). But, for African American parents, higher intrusiveness may not be associated with poorer EF in children; while for European American parents, higher intrusiveness is associated with poorer EF (Cuevas et al., 2014; Rhoades, Greenberg, Lanza, & Blair, 2011). More research investigating ethnic differences in gene × parental behaviour interactions on EF using representative, diverse samples is needed.

How genes and parental behaviour interact to influence children's EF may vary over development (Hariri & Holmes, 2006). For example, COMT activity increases over the course of early childhood, peaking in adulthood (Tunbridge, Weickert, Kleinman, & Herman, 2007). Emerging evidence suggests that the association between Val and Met alleles and cognitive outcomes changes as COMT enzyme activity increases (Dumontheil et al., 2011). Similarly, positive socioemotional parental behaviours, like sensitivity, may be particularly important for children's EF development before 36 months of age (Bernier, Carlson, Deschênes, & Matte-Gagné, 2012; C. Blair et al., 2014). The use of longitudinal designs with repeated measurement of parental behaviour and EF would allow researchers to examine developmental change.

Two competing theories of the form of gene × environment interactions have been proposed: diathesis-stress (Monroe & Simons, 1991) and differential susceptibility (J. Belsky et al., 2009; J. Belsky & Pluess, 2009). The diathesis stress model (Monroe & Simons, 1991) proposes that individuals with certain alleles of candidate genes are more vulnerable to negative developmental outcomes when exposed to negative environmental factors. In contrast, the differential susceptibility model (J. Belsky et al., 2009; J. Belsky & Pluess, 2009) proposes that these alleles confer increased sensitive to environmental factors, in a "for better-and-for-worse" fashion, such that individuals with these alleles have their functioning disproportionately undermined and enhanced by negative and positive environmental factors. Recommendations for distinguishing between these two theories have been published elsewhere (see J. Belsky et al., 2013; Roisman et al., 2012; Widaman et al., 2013). In the present systematic review, only 7 of 14 studies reporting significant interactions tested competing theories of gene × parental behaviour interactions. Four studies supported the diathesis-stress model (Augustine et al., 2018; Y. Li et al., 2016; Smith et al., 2013, 2012) and three supported the differential susceptibility model (Kok

et al., 2013; Sulik et al., 2012, 2015). Thus, evidence is mixed as to whether genes confer vulnerability or differential susceptibility to environmental factors.

Conclusion

The development of EF is driven by both genetic and environmental factors (J. Li & Roberts, 2017). In particular, candidate genes involved in neurotransmission, such as *DRD4* and *SLC6A4*, may interact with parental behaviours to predict children's EF (Kochanska et al., 2009; Smith et al., 2012). The aim of this review was to systematically examine and evaluate the evidence for gene × parental behaviour interactions on EF in early childhood. There was broad support for an association between gene × parental behaviour interactions and children's EF: 13 of 17 studies (76%) reporting at least one significant gene × parental behaviour interaction, although only 24 of 51 (47%) examined interactions that were significant. However, heterogeneity among studies meant we were unable to evaluate the strength of the evidence for gene × parental behaviour interactions on EF. This points to a need for additional research replicating existing findings; more rigorous statistical tests and reporting of gene × parental behaviour interactions; and moving beyond a single candidate gene approach. Perhaps most important is the need for greater consistency in the measurement of EF and parental behaviours. Even with improvements in genotyping and analytic methods, future research is unlikely to fair much better than past research without addressing issues in the conceptualization and measurement of the environment and phenotypes. Without this, future research is unlikely to advance research on the role of gene × parental behaviour interactions in the development of children's EF. Addressing these issues has the potential to inform research on gene × environment interactions on children's EF and the biopsychosocial mechanisms underlying the development of EF.

Figure 2-1. Flow diagram of the identification, screening, eligibility, and inclusion of studies.

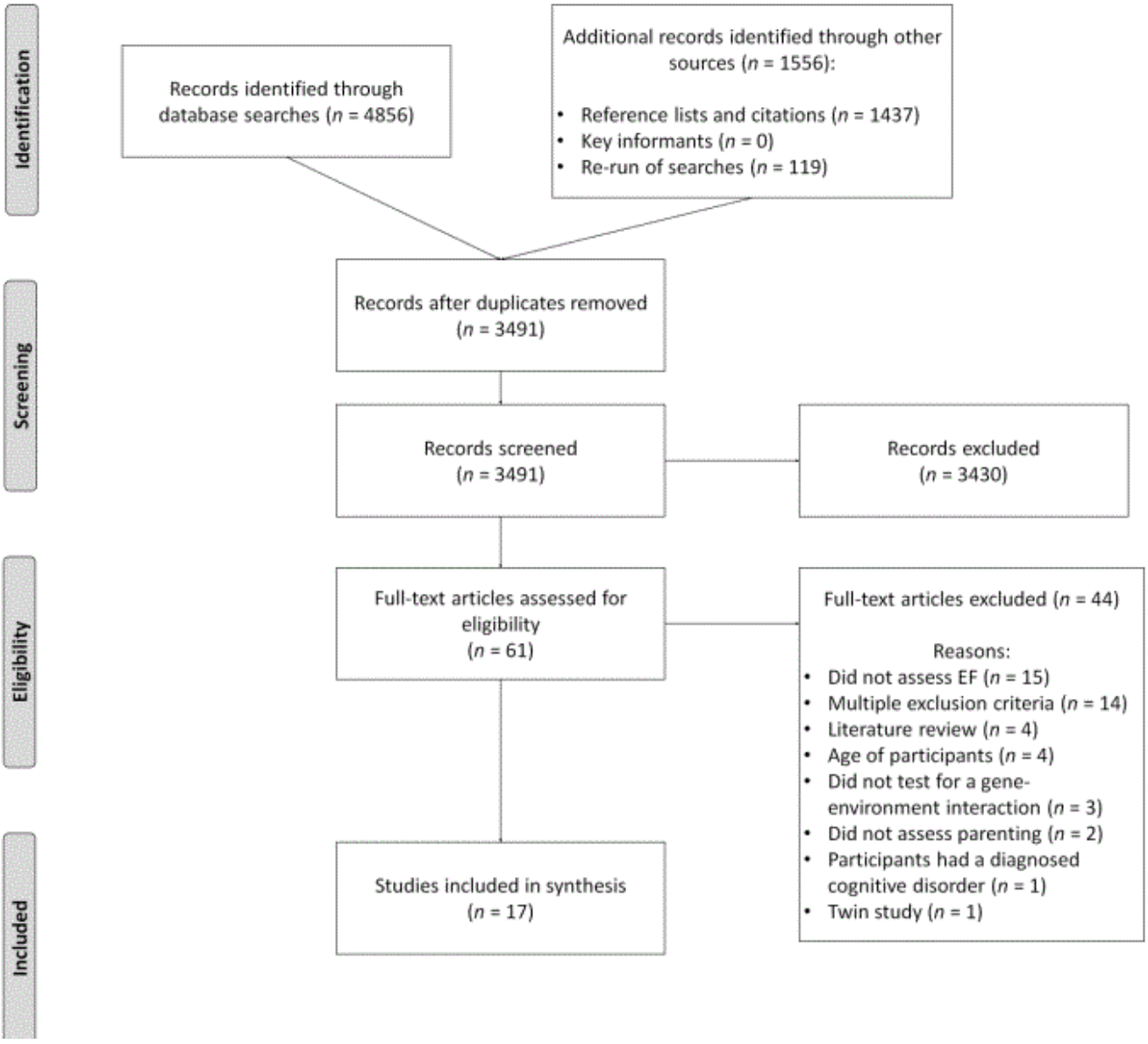


Table 2-1. *Characteristics of studies included in the systematic review*

Study	Study design	Country	*N* (sex)	Predominant ethnicity[a]	Candidate gene	Child age at genotyping (months)	Parental behaviour constructs (method of assessment)	Child age at parental behaviour assessment (months)	EF constructs (method of assessment)	Child age at EF assessment (months)
Augustine et al. (2018)	Longitudinal; observational	United States	186 (48% male)	Mixed[b]	*DRD2*, *OXTR*	24	Maternal sensitivity (clean up task; observation)	6	Compliance (clean-up task; observation)	24
Davies & Cicchetti (2014)	Longitudinal; observational	United States	201 (46% male)	African American	*SLC6A4*	24	Maternal unresponsiveness; (Iowa Family Interaction Rating Scale and Adult-Adolescent Parenting Inventory; maternal report)	24	Frustration suppression (Simulated Phone Argument Task; observation)	36
Kochanska et al. (2009)	Longitudinal; observational	United States	88 (50% male)	European American	*SLC6A4*	52	Attachment style (Strange Situation; observation)	15	Inhibitory control (28 inhibitory control and delay of gratification tasks; behavioural tasks)	25, 38, 52
Kok et al. (2013)	Prospective cohort; observational	Netherlands	436 (51% male)	Dutch	*COMT*, *DRD4*	Birth	Maternal positive and negative discipline (Erickson Scale for Supportive Presence; observation)	36	Compliance (disciplinary context; observation)	36
Y. Li et al. (2016)	Longitudinal; observational	United States	145[c] (54% male)	European American	*DAT1*	72	Parenting quality (free play, challenging teaching task, and clean-up task; observation)	30	Inhibitory control (Rabbit and Turtle Task, Gift Bag/Box Task, and Dinky Toys Task; behavioural tasks)	30, 42, 54
Montirosso et al. (2015)	Cross-sectional; observational	Italy	73 (45% male)	Italian	*SLC6A4*	4	Maternal social engagement (normal play	--	Frustration suppression (negative	--

							episode; observation)		emotionality, reactivity and recovery coded during the Face-to-Face Still-Face Paradigm; observation)	
Pickles et al. (2013)	Prospective epidemiological cohort; observational	England	150 (61% male)	English	*MAOA*	14	Maternal sensitivity (play activity; observation)	29 weeks	Frustration suppression (Infant Behavioral Questionnaire Revised; maternal report)	14
Quan et al. (2017)	Prospective cohort; observational	Singapore	209 (43% male)	Chinese	*CHRNA4*, *COMT*	Birth	Maternal sensitivity (mini-Maternal Behavior Q-Set; observation)	6	Executive attention (Visual Expectation Task; behavioural task)	6
Sheese et al. (2012)	Longitudinal; observational	United States	52[d] (38% male)	European American	*DRD4*	18-21	Parenting quality (free-play procedure; observation)	18-21	Inhibitory control (Children's Behavior Questionnaire; parent report)	36-48
Sheese et al. (2007)	Cross-sectional; observational	United States	45[d] (64% male)	European American	*DRD4*	18-21	Parenting quality (free-play procedure; observation)	--	Inhibitory control (Early Childhood Behavior Questionnaire; parent report)	--
Smith et al. (2013)	Cross-sectional; observational	Not reported	407 (49% male)	European	*DRD4*	36-47	Positive and negative parenting (Teaching Tasks coding manual and Qualitative Ratings for Parent-Child Interactions scale; observation)	--	Inhibitory control (Tower of Patience Task, Snack Delay Task; behavioural tasks)	--
Smith et al. (2012)	Cross-sectional; observational	United States	382 (53% male)	European American	*DRD4*	36-48	Positive and negative parenting (Teaching Tasks battery; observation)	--	Inhibitory control (Tower of Patience Task, Snack Delay Task; behavioural tasks)	--

Sulik et al. (2012)	Longitudinal; observational	United States	138[c] (52% male)	European American	*SLC6A4*	72	Parenting quality (free play, challenging teaching task, and clean-up task; observation)	18	Noncompliance (Infant-Toddler Social and Emotional Assessment; maternal report)	18, 30, 42, 54
Sulik et al. (2015)	Longitudinal; observational	United States	146[c] (54% male)	European American	*COMT*	72	Parenting quality (free play, challenging teaching task, and clean-up task; observation)	18	Inhibitory control and attention focusing (Children's Behavior Questionnaire; maternal report)	42, 54, 72, 84
Z. Taylor et al. (2014)	Longitudinal; observational	United States	153[c] (54% male)	European American	*SLC6A4*	72	Parenting quality (free play, challenging teaching task, and clean-up task; observation)	18	Cognitive flexibility (Q-Sort; maternal report)	18, 30, 42, 54, 72, 84
Voelker et al. (2009)	Cross-sectional; observational	United States	45[d] (62% male)	European American	*COMT*	18-21	Parenting quality (free-play procedure; observation)	--	Executive attention (Visual Sequence Task; behavioural task)	--
Zhang et al. (2014)	Cross-sectional; observational	China	281 (54% male)	Han	*MAOA*, *SLC6A4*	6	Maternal sensitivity (free play session; observation)	--	Frustration suppression (duration of looks away from a negative stimulus; observation)	--

Note. [a] Predominant > 50%. [b] No predominant ethnic group. Largest ethnic group was European American (48% of the sample). [c] Studies used samples from the same longitudinal cohort. [d] Studies used samples from the same longitudinal cohort.

Table 2-2. *Summary of study results*

EF outcome	Study (year)	Candidate gene	Polymorphism (risk allele)	Parental behaviour constructs	Results	Main findings
Inhibitory control	Sheese et al. (2012)	*DRD4*	exon 3 VNTR (7-repeat)	Parenting quality	+	*DRD4* × parenting quality interaction (β = 0.47, p = .01). Parenting quality associated with inhibitory control for the 7-repeat present group (β = .57, t(46) = 2.46, p = .02) but not the 7-repeat absent group (β = -.21, t(46) = -1.19, p = .24).
Inhibitory control	Sheese et al. (2007)	*DRD4*	exon 3 VNTR (7-repeat)	Parenting quality	−	No interaction (F(1, 39) = .01, p = .94).
Inhibitory control	Smith et al. (2013)	*DRD4*	exon 3 VNTR (7-repeat)	Positive parenting	+	*DRD4* × positive parenting interaction (b = .05, p < .05). Positive parenting associated with inhibitory control for the 7-repeat present group (b = .04, SE = .02, p = .04) but not the 7-repeat absent group (b = -.01, SE = .01, p = .49) at values of positive parenting below -.71.
Inhibitory control		*DRD4*	exon 3 VNTR (7-repeat)	Negative parenting	−	No interaction (b = .08, p > .05).
Inhibitory control	Smith et al. (2012)	*DRD4*	exon 3 VNTR (7-repeat)	Positive parenting	−	No interaction (B = -.01, p > .05).
Inhibitory control		*DRD4*	exon 3 VNTR (7-repeat)	Negative parenting	+	*DRD4* × negative parenting interaction (B = .41, p < .05). Negative parenting was associated with poorer inhibitory control for the 7-repeat present group (b = .53, SE = .16, p < .01), but not the 7-repeat absent group (b = .09, SE = .12, p = .43) at values of negative parenting above .09 (t(370) = 1.97, p < .05).
Compliance	Kok et al. (2013)	*DRD4*	exon 3 VNTR (7-repeat)	Positive discipline	−	No interaction.
Compliance		*DRD4*	exon 3 VNTR (7-repeat)	Negative discipline	−	No interaction.
Compliance		*COMT*	Val(108/158)Met (none)	Positive discipline	+	*COMT* × positive discipline interaction (B = -.09, β = -.11, SE = .04, t = -2.46, p < .05). The strongest association between positive discipline and compliance was for the Met/Met group (r = .51, p < .01),

						then the Met/Val (r = .29, p < .01) and Val/Val groups (r = .16, p = .10) at values of positive discipline below .00 and above .98.
Compliance		*COMT*	Val(108/158)Met (none)	Negative discipline	−	No interaction.
Inhibitory control (intercept[c])	Sulik et al. (2015)	*COMT*	Val(108/158)Met (Val), 3'UTR VNTR (G), Intron 1 SNP (G)	Parenting quality	+	*COMT* Val(108/158)Met/3'UTR/Intron 1 haplotype × parenting quality × child sex interaction (t = -2.23, p < .05, pseudo r^2 = 25.1%). For girls, parenting quality was associated with inhibitory control for the ValGG haplotype present group but not the ValGG haplotype absent group. For boys, parenting quality was associated with inhibitory control for the ValGG haplotype absent group and not the ValGG haplotype present group. Results were the same for the Val(108/158)Met (b = -.66, t = -2.75, p < .01), 3'UTR (t = -2.75, p < .01, r^2 = 20.1%), and Intron 1 (t = -2.63, p < .01, pseudo r^2 = 20.9%) polymorphisms.
Inhibitory control (slope[c])		*COMT*	Val(108/158)Met (Val), 3'UTR VNTR (G), Intron 1 SNP (G)	Parenting quality	−	No interactions involving the *COMT* haplotypes or polymorphisms and parenting quality.
Attention focusing (intercept[c])		*COMT*	Val(108/158)Met (Val), 3'UTR VNTR (G), Intron 1 SNP (G)	Parenting quality	−	No interactions between *COMT* Val(108/158)Met/3'UTR/Intron 1 haplotype and parenting quality. No interactions between *COMT* polymorphisms and parenting quality.
Attention focusing (slope[c])		*COMT*	Val(108/158)Met (Val), 3'UTR VNTR (G), Intron 1 SNP (G)	Parenting quality	−	No interaction between *COMT* Val(108/158)Met/3'UTR/Intron 1 haplotype and parenting quality. No interactions between *COMT* polymorphisms and parenting quality.
Executive attention (total anticipatory looks)	Voelker et al. (2009)	*COMT*	Val(108/158)Met (Val), rs6269 (G), rs4633 (C), rs4818 (G)	Parenting quality	+	*COMT* Val(108/158)Met /rs6269/rs4633/rs4818 haplotype × parenting quality interaction (F(1, 31) = 8.93, partial η^2 = .22, p < .01). Parenting quality associated with total anticipatory

						looks for the low pain sensitivity haplotype group. Val(108/158)Met × parenting quality interaction (F(1, 39) = 6.71, partial η^2 = .15, p = .01). Parenting quality associated with total anticipatory looks for the Val present group but not Val absent group.
Executive attention (correct anticipatory looks)		*COMT*	Val(108/158)Met (Val), rs6269 (G), rs4633 (C), rs4818 (G)	Parenting quality	+	*COMT* Val(108/158)Met /rs6269/rs4633/rs4818 haplotype × parenting quality interaction (F(1, 31) = 6.27, partial η^2 = .27, p = .02). No Val(108/158)Met × parenting quality interaction (p = .06).
Executive attention (incorrect anticipatory looks)		*COMT*	Val(108/158)Met (Val), rs6269 (G), rs4633 (C), rs4818 (G)	Parenting quality	−	Marginally significant Val(108/158)Met /rs6269/rs4633/rs4818 haplotype × parenting quality interaction (F(1, 31) = 4.02, partial η^2 = .12, p = .054). Val(108/158)Met × parenting quality interaction (p = .03).
Executive attention (entire pattern phase of Visual Expectation Task)	Quan et al. (2017)	*COMT*	Val(108/158)Met (Val)	Maternal sensitivity	−	No interaction (F(2, 189) = .62, partial η^2 = .007, p = .54).
Executive attention (middle third of pattern phase)		*COMT*	Val(108/158)Met (Val)	Maternal sensitivity	−	No interaction (F(2, 181) = .55, partial η^2 = .006, p = .58).
Frustration suppression	Pickles et al. (2013)	*MAOA*	uVNTR (low expression = 3- or 5-repeat)	Maternal sensitivity	+[a]	*MAOA* × maternal sensitivity interaction (weighted F(1, 143) = 9.19, weighted standardized coefficient = .52, p < .05, 95% CI [0.18, 0.86]). The values of the slope for the interaction differed for boys (low activity = -.33, 11% variance explained; high activity = -.15, 2% variance explained) and girls (low activity group = .02, 0% variance explained; high activity group = .72, 50% variance explained).
Frustration	Zhang et al.	*MAOA*	uVNTR (low	Maternal sensitivity	+	For girls, *MAOA* × maternal sensitivity

suppression	(2014)		expression = 3-repeat)			interaction ($F(2, 119) = 8.64$, $p < .01$). The low expression group had worse frustration suppression than the high expression group when maternal sensitivity was low ($F(2,125) = 8.99$, $p < .01$). For boys, no interaction.
Inhibitory control (intercept[c])	Y. Li et al. (2016)	*DAT1*	Intron 13 SNP (G), Intron 8 SNP (A), 3'UTR VNTR (10-repeat)	Parenting quality	+	Intron 8/Intron 13 haplotype × parenting quality interaction ($b = -7.20$, $t = -2.28$, $p < .05$). Parenting quality associated with inhibitory control for children without the AG haplotype ($b = 8.64$, $t = 4.45$, $p < .01$) at values of parenting quality ≤ -.43 points (.48 SD) below the mean. Intron 13/3'UTR haplotype × parenting quality interaction ($b = -7.09$, $t = -2.32$, $p < .05$). Parenting quality associated with inhibitory control for children without the G10-repeat haplotype ($b = 9.22$, $t = 4.36$, $p < .01$) at values of parenting quality ≤ -.59 points (.55 SD) below the mean. Intron 8/3'UTR haplotype × parenting quality interaction ($b = -7.89$, $t = -2.55$, $p < .05$). Parenting quality associated with inhibitory control for children with the A10-repeat haplotype ($b = -.10$, $t = -2.21$, $p < .05$) but not without this haplotype ($b = 1.39$, $t = .58$, p = ns) at values of parenting quality ≤ -.46 points (.51 SD) below the mean. No interactions between individual polymorphisms and parenting quality.
Inhibitory control (slope[c])		*DAT1*	Intron 13 SNP (G), Intron 8 SNP (A), 3'UTR VNTR (10-repeat)	Parenting quality	−	No interactions between *DAT1* haplotypes or polymorphisms and parenting quality.
Compliance	Augustine et al. (2018)	*DRD2*	Taq1A (A1)	Maternal sensitivity	−	No interaction ($B = .97$, $\beta = .13$, $SE = .61$, $p > .05$).
Inhibitory control	Kochanska et al. (2009)	*SLC6A4*	5-HTTLPR (short)	Attachment style	+	*SLC6A4* × attachment style interaction ($F = 4.14$, $\beta = -.40$, $p < .05$). Insecurely attached children with ss or sl genotypes

						had worse inhibitory control (b = 1.18, SE = .50, p < .02) than children with the ll genotype (b = 0.41, SE = 1.56, p = ns).
Noncompliance (intercept[c])	Sulik et al. (2012)	*SLC6A4*	5-HTTLPR (short), STin2 VNTR (10-repeat)	Parenting quality	+	*SLC6A4* 5-HTTLPR/STin2 haplotype × parenting quality interaction (bs = .38 and .22, ts = 3.19 and 2.38, ps < .01 and .05). Parenting quality was negatively associated with noncompliance for children in the long 10- or 12-repeat haplotype group (b = -.31, t = -4.72, p < .01), but not the short/10-repeat or short/12-repeat haplotype groups (bs = .08 and -.09, ts = .75 and -1.37, ps = .45 and .17), at values of parenting quality ≥ 1.10 points above (1.55 SD) and ≤ .99 points (1.40 SD) below the mean for the short/12-repeat haplotype and ≥ .03 points (.04 SD) above and ≤ 1.50 points (2.12 SD) below the mean for the short/10-repeat haplotype. Results were the same for 5-HTTLPR × parenting quality (b = -.26, t = -3.02, p < .01) interaction but not STin2 × parenting quality ($\chi^2(4)$ = 1.2, p = .88) interaction.
Noncompliance (slope[c])		*SLC6A4*	5-HTTLPR (short), STin2 VNTR (10-repeat)	Parenting quality	+	*SLC6A4* 5-HTTLPR/STin2 haplotype × parenting quality interaction (bs = .11 and .14, ts = 1.90 and 2.53, ps < .10 and .05). Parenting quality negatively associated with growth in noncompliance for children in the short/10-repeat haplotype group (b = -.10, t = -2.21, p < .05), but not the short/12-repeat or long/10- or 12-repeat haplotype groups (bs = .00 and .04, ts = .12 and 1.24, ps < .90 and .22), at values of parenting quality > .69 points (.98 SD) above the mean for the short/12-repeat haplotype group and ≥ .54 points (.76 SD) above and ≤ -1.68 points (2.37 SD) below the mean for the long/10- or 12-repeat haplotype group.

						No 5-HTTLPR × parenting quality or STin2 × parenting quality interactions.
Frustration suppression (reactivity)	Montirosso et al. (2015)	*SLC6A4*	5-HTTLPR (short)	Maternal social engagement	+	*SLC6A4* × maternal social engagement interaction (B = -.33, SE = .18, η^2 = .08, t = -2.13, p = .04). Maternal social engagement negatively associated with reactivity for the ss and sl groups but not the ll group (B = -.61, t = -3.78, p = .001, 37% of variance explained).
Frustration suppression (recovery)		*SLC6A4*	5-HTTLPR (short)	Maternal social engagement	+	*SLC6A4* × maternal social engagement interaction (B = -.49, η^2 = .08, SE = .24, t = -2.02, p = .05). Maternal social engagement negatively associated with negative emotionality during recovery for the ss and sl groups but not ll group (B = -.53, t = -3.06, p = .01, 28% of variance explained).
Frustration suppression	Zhang et al. (2014)	*SLC6A4*	5-HTTLPR (short)	Maternal sensitivity	+	For girls, *SLC6A4* × maternal sensitivity interaction (F(1, 119) = 5.01, p = .02). ll group had better frustration suppression than the ss and sl groups when maternal sensitivity was low (F(1, 125) = 11.85, p = .001). For boys, no interaction (F(1, 143) = 1.95, p = .17).
Frustration suppression	Davies & Cicchetti (2014)	*SLC6A4*	5-HTTLPR (none)	Maternal unresponsiveness	–[b]	No interaction (β = -.08; p > .05).
Cognitive flexibility (intercept[c])	Z. Taylor et al. (2014)	*SLC6A4*	5-HTTLPR (short), STin2 VNTR (10-repeat)	Parenting quality	–	No *SLC6A4* 5-HTTLPR/STin2 haplotype × parenting quality (bs = -29, -.19, and .10; ts = -1.07, -.68, -.55; ps = ns) interactions. No *SLC6A4* 5-HTTLPR × parenting quality or STin2 × parenting quality interactions.
Cognitive flexibility (slope[c])		*SLC6A4*	5-HTTLPR (short), STin2 VNTR (10-repeat)	Parenting quality	–	No *SLC6A4* 5-HTTLPR/STin2 haplotype × parenting quality interaction (bs = -.10, -.29, and .18; ts = -.56, -1.07, and .68; ps = ns). No *SLC6A4* 5-HTTLPR × parenting quality or STin2 × parenting quality interactions.

Executive attention (entire pattern phase)	Quan et al. (2017)	*CHRNA4*	rs1044396 (T)	Maternal sensitivity	−	No interaction ($F(2, 189) = 1.76$, partial $\eta^2 = .018$, $p = .18$).
Executive attention (middle third of pattern phase)		*CHRNA4*	rs1044396 (T)	Maternal sensitivity	−	No interaction ($F(2, 181) = .10$, partial $\eta^2 = .001$, $p = .91$).
Compliance	Augustine et al. (2018)	*OXTR*	rs53576 (A)	Maternal sensitivity	+	*OXTR* × maternal sensitivity interaction ($B = 1.18$, $\beta = .21$, $SE = .58$, $p < .05$). The GG group had poorer compliance than the AA and AG groups at values of maternal sensitivity below 2.28.

Note. [a] + Interaction significant ($p < .05$). [b] – Interaction not significant ($p > .05$). [c] Growth model.

Table 2-3. *Quality assessment of studies*

	Introduction	Methods						Results			Discussion
Study	1. Objectives and hypotheses clearly stated	2. Clear eligibility criteria for participants	3. Clear definition of all variables	4. Statistical methods replicable	5. Assessment of HWE	6. Assessment of ethnicity	7. Mixed ethnicities addressed statistically	8. Sufficient descriptive data (age, gender, and ethnicity)	9. Genotype frequencies stated	10. Sample in HWE	11. Consider of population stratification
Augustine et al. (2018)	+	+	+	+	+	+	+	+	-	-	+
Davies & Cicchetti (2014)	+	+	+	+	+	+	+	+	+	+	+
Kochanska et al. (2009)	+	-	-	+	+	+	-	+	+	+	-
Kok et al. (2013)	+	+	-	+	+	+	Not applicable	+	+	+	+
Y. Li et al. (2016)	+	+	+	+	+	+	+	+	+	+	+
Montirosso et al. (2015)	+	+	-	+	+	-	Not applicable	-	+	+	-
Pickles et al. (2013)	+	+	-	+	+	+	-	-	+	+	-
Quan et al. (2017)	+	+	+	+	+	+	+	+	+	+	+
Sheese et al. (2012)	+	-	-	+	-	-	-	-	-	Not applicable	-
Sheese et al. (2007)	+	-	-	+	-	+	-	+	-	Not applicable	-
Smith et al. (2013)	+	+	-	+	+	+	+	-	+	-	-
Smith et al. (2012)	+	-	-	+	+	+	Not applicable	+	+	-	+
Sulik et al. (2012)	+	+	-	+	+	+	+	+	-	+	+
Sulik et al. (2015)	+	+	-	+	+	+	+	+	+	+	-
Z. Taylor et al. (2014)	+	-	+	+	+	+	+	+	+	+	+
Voelker et al. (2009)	+	-	-	+	-	+	-	+	+	Not applicable	-
Zhang et al. (2014)	+	+	+	-	+	+	Not applicable	+	+	+	-

Chapter 3

Interplay of Cumulative Dopaminergic Genetic Risk and Parental Behaviour is Associated with Executive Function in Early Childhood: Support for the Diathesis-Stress Model

Interplay of Cumulative Dopaminergic Genetic Risk and Parental Behaviour is Associated with Executive Function in Early Childhood: Support for the Diathesis-Stress Model

Early childhood is a period of substantial, rapid development in executive function (EF), the set of higher-order cognitive processes necessary for carrying out goal-directed behaviour in situations involving affective, motivational, or cognitive load (Garon et al., 2008; Wiebe et al., 2012; Zelazo & Carlson, 2012). During early childhood, EF lays the foundation for the acquisition of more demanding cognitive and socioemotional competencies, like math ability (C. Blair & Razza, 2007) and prosocial behaviour (K. Blair, Denham, Kochanoff, & Whipple, 2004), and emerges as a risk factor for the development of externalizing behaviour problems (Chang, Olson, Sameroff, & Sexton, 2011; Oh, Greenberg, Willoughby, & The Family Life Project Key Investigators, 2020). Because of the importance of early EF skills for cognitive and psychosocial functioning across childhood and into adulthood (Moffitt, Poulton, & Caspi, 2013), it is important to identify factors contributing to the development of EF. Dopamine genotype is associated with individual differences in EF (Barnes et al., 2011; Logue & Gould, 2014). Children's EF is also shaped by environmental factors like parental behaviour (for a meta-analysis see Valcan et al., 2017). Some children are theorized to be more impacted by parental behaviour than others (Pluess & Belsky, 2010b, 2010a). Genes involved in the dopaminergic system (e.g., *DRD2* and *DRD4*) are thought to confer increased sensitivity to environmental influences (Bakermans-Kranenburg & van IJzendoorn, 2011; J. Belsky & Pluess, 2009). This suggests that genes involved in dopaminergic functioning may interact with parental behaviour to predict EF in preschoolers. This study examined whether the interaction between cumulative dopaminergic genetic risk and parental behaviour was associated with children's EF at 36 months.

Dopaminergic Genes and EF

Genes involved in dopaminergic functioning, including dopamine active transporter 1 (*DAT1*), dopamine receptor D2 (*DRD2*), dopamine receptor D4 (*D2D4*), and catechol-O-methyltransferase (*COMT*), are associated with individual differences in EF (for reviews see Barnes et al., 2011; Logue & Gould, 2014). For example, the Met allele of *COMT* is associated with better performance on tasks assessing working memory (the ability to hold in mind and manipulate information) and inhibitory control (the ability to inhibit a prepotent or automatic response), termed WMIC, in children (Diamond, Briand, Fossella, & Gehlbach, 2004; Dumontheil, Kilford, & Blakemore, 2020). This is because the Met allele of *COMT* is associated with increased dopamine availability (Tunbridge, Harrison, & Weinberger, 2006). Dopamine levels in the prefrontal cortex impact performance on EF tasks in an inverted U-shaped fashion, such that low and high levels of dopamine are associated with poorer EF (Robbins & Arnsten, 2009). Further, dopaminergic genes confer individual differences in sensitivity to environmental factors (Bakermans-Kranenburg & van IJzendoorn, 2011; van IJzendoorn & Bakermans-Kranenburg, 2014), suggesting that dopaminergic genes and parental behaviour interact to affect EF. The present study adopted a polygenic perspective and considered whether the interaction between the cumulative effect of four dopaminergic genes, *DAT1*, *DRD2*, *DRD4*, and *COMT*, and parental behaviour was associated with children's EF.

In addition to being involved in EF, dopamine is involved in motivational, decision-making, and reward processes (Gatzke-Kopp, 2011; Yacubian & Büchel, 2009). Both basal dopamine levels and responsivity may be associated with sensitivity to the environment. Lower basal dopamine levels are associated with an increased dopamine response to agonists like alcohol and nicotine (Cosgrove et al., 2014; Urban et al., 2010). A greater dopamine release in

response to stimulation is indicative of increased sensitivity to rewards and punishers in the environment (Yacubian & Büchel, 2009). Genes involved in the dopaminergic system affect how quickly dopamine moves into the synapse, is reabsorbed, and is degraded (Chen et al., 2004; Fuke et al., 2001; Seamans & Yang, 2004), which is theorized to influence sensitivity to environmental factors (Chhangur et al., 2017).

DAT1 controls the amount and duration of extracellular dopamine levels by removing dopamine from the synapse (Fuke et al., 2001). The *DAT1* gene is located on chromosome 5 at 5p15.3. It has a polymorphic 40-base pair (bp) variable number of tandem repeats (VNTR). The bp is repeated between 3 and 13 times, with 9 and 10 being the most common number of repeats in the population. Compared to the 9-repeat allele, the 10-repeat allele is linked to increased gene expression and transporter density. Higher transporter density is associated with faster removal of dopamine from the synapse, leading to lower dopamine levels (Yang et al., 2007). The 10-repeat allele is associated with increased sensitivity to the environment and poorer EF (Bakermans-Kranenburg & van IJzendoorn, 2011; Cornish et al., 2005).

Dopamine signaling is not only dependent on the availability of dopamine in the synapse but also on the efficiency of the dopamine receptors. DRD2 and DRD4 are involved in the uptake of dopamine from the synapse (Seamans & Yang, 2004). The *DRD2* gene is located on chromosome 11 at q22-q23 (Pohjalainen et al., 1998). The Taq1A polymorphism is located on the 3’ untranslated region (UTR) of the gene. Taq1A has two alleles, A1 and A2. A1 is less common in the population. Compared to the A2 allele, the A1 allele is associated with reduced gene expression and receptor density which predict reduced dopamine availability in the striatum and prefrontal cortex. Reduced dopamine availability is associated with increased sensitivity to environmental factors and poorer EF (Bakermans-Kranenburg & van IJzendoorn, 2011; Wiebe et

al., 2009). The *DRD4* gene is located on chromosome 11 at 11p15.5 (Seamans & Yang, 2004). The polymorphic repeat sequence in exon 3 of the *DRD4* gene contains a 48-bp VNTR (Schoots & van Tol, 2003). The number of VNTRs ranges from 2 to 10, with 4 and 7 repeats most common in the population. The number of repeats affects mRNA transcription, such that the 7-repeat allele leads to reduced mRNA transcription. Reduced mRNA transcription is associated with a decrease in receptors and binding affinity, which translates into reduced dopamine availability. The 7-repeat allele is associated with increased sensitivity to environmental factors and poorer EF (Bakermans-Kranenburg & van IJzendoorn, 2011; Eisenegger et al., 2010; Froehlich et al., 2007).

COMT is the main enzyme responsible for the degradation of dopamine (Chen et al., 2004). The *COMT* gene is located on chromosome 22 at position 11.21. A single nucleotide polymorphism (SNP) that codes for the substitution of Valine (Val) by Methionine (Met) at codon 158 influences activity of the enzyme. The Val allele leads to 3 to 4 times more COMT activity than the Met allele. This results in faster catabolism of extracellular dopamine and lower dopamine availability. The Val allele has been linked to poorer EF and increased sensitivity to environmental influence (J. Belsky & Pluess, 2009; Tunbridge et al., 2006).

Parental Behaviour and Child EF

Problematic discipline is a robust predictor of poorer psychosocial and cognitive outcomes in children, including mental health problems, aggression, and EF (Gershoff, 2002; Shaw, Bell, & Gilliom, 2000; Valcan et al., 2017). It is marked by coercive and inconsistent parental behaviours, including over-controlling behaviour, hostility, negative affect, and the use of punitive discipline techniques (Hill et al., 2008). More problematic discipline is associated

with poorer EF in 3-year-olds (Houck & Lecuyer-Maus, 2004; Valcan et al., 2017), although few studies have examined the relation between problematic discipline and self-control.

A second dimension of parental behaviour, responsiveness, has also been linked to the development of children's EF (Hughes & Devine, 2017). Responsive parental behaviours reflect warm, sensitive, and contingent responses to children's behaviour; emotional availability; and the match between a parent's response and child's behaviour (Hill et al., 2008). More responsive parental behaviour is associated with better EF in early childhood (Li-Grining, 2007; Razza & Raymond, 2013; Valcan et al., 2017). Research examining the relation between responsiveness and self-control is limited because most research has focused on WMIC.

Models of Gene × Environment Interaction

While growing evidence suggests dopamine genotype and parental behaviour interact to predict children's EF, less is known about the form of this interaction (Vrantsidis, Wuest, et al., under review). Two competing models of gene × environment interaction have been proposed: diathesis-stress (Monroe & Simons, 1991) and differential susceptibility (J. Belsky et al., 2009; J. Belsky & Pluess, 2009). The diathesis-stress model (Monroe & Simons, 1991) proposes that certain genotypes confer vulnerability to the negative effects of negative environmental factors. This model predicts that children at higher genetic risk will have poorer EF than children at lower genetic risk when exposed to negative environmental factors (e.g., problematic discipline). There is some support for this model. Lower quality parenting at 30 months is associated with poorer effortful control, a construct closely related to EF, among 30-month-olds with risk haplotypes of *DAT1* (Y. Li et al., 2016). In addition, for 36-month-olds with at least one 7-repeat allele of *DRD4*, more negative parenting is associated with poorer effortful control (Smith et al., 2012).

In contrast, the differential susceptibility model (J. Belsky et al., 2009; J. Belsky & Pluess, 2009) proposes that particular genotypes confer increased sensitivity to both positive and negative environmental factors, rather than vulnerability to negative factors. This model predicts that children at higher genetic risk will have better EF than children at lower genetic risk when exposed to positive environmental factors (e.g., responsiveness) and worse EF when exposed to negative environmental factors (e.g., problematic discipline). In support of this model, J. Belsky and Beaver (2011) found that a measure of cumulative genetic risk that included *DAT1*, *DRD2*, *DRD4*, *MAOA* and *SLC6A4* moderated the association between parenting quality and self-regulation in adolescent males in a "for-better or for-worse" manner. Males with more risk alleles had poorer self-regulation when exposed to lower quality parenting and better self-regulation when exposed to higher quality parenting than males with fewer risk alleles. There is also converging evidence that *COMT* moderates the relations between maternal positive discipline and parenting quality and 36-month-olds' compliance and effortful control in a fashion consistent with the differential susceptibility model (Kok et al., 2013; Sulik et al., 2015).

The Present Study

This study examined whether the interactions between cumulative dopaminergic genetic risk and parental behaviour were associated with children's EF at 36 months and whether the forms of the interactions were consistent with the diathesis-stress or differential susceptibility model. We hypothesized that the interactions between genetic risk and both problematic discipline and responsiveness would be associated with children's EF. If results supported the diathesis-stress model, we expected children at higher genetic risk to have poorer EF than children at lower genetic risk when parents used more problematic discipline or were less responsive. If results supported the differential susceptibility model, in addition to having poorer

EF in higher-risk contexts, we expected children at higher genetic risk to have better EF when parents were more responsive. To test these hypotheses, we used data from the Midwestern Infant Development Study (MIDS), a prospective longitudinal study, which included observational measures of parental behaviour and behavioural assessments of EF when children were 36 months old.

Methods

Participants

Mother-child dyads were drawn from a follow-up of the MIDS completed when children were 36 months old (N = 135; 63 girls, 72 boys; M_{age} = 3 years 6 days, SD = 99 days; 55% exposed to prenatal tobacco). Mothers were prospectively recruited during pregnancy at two Midwestern study sites (Carbondale, Illinois; and Lincoln, Nebraska) to study the effects of prenatal tobacco exposure on cognitive development (Espy et al., 2011; Wiebe et al., 2015; Wiebe, Fang, Johnson, James, & Espy, 2014). Because of funding constraints, the 36-month follow-up only included children at the Lincoln, Nebraska site. Prior to participating in the study, mothers provided written, informed consent. Excluded from the cohort were mothers who reported illegal drug use (except for occasional marijuana use) or binge drinking. Infants born preterm (< 35 weeks gestational age) or with birth complications known to affect developmental outcomes (e.g., neonatal seizures) were excluded. Dyads included in this study did not significantly differ from excluded dyads in terms of ethnicity, prenatal tobacco exposure status, maternal education, or child sex. Family income ranged from under $10,000 to over $100,000 ($Mdn$ = $30,000). Parents completed between 11 and 18 years of formal education (M = 14.03). Children's ethnic backgrounds are European American (n = 76), African American (n = 32), and Hispanic or Latinx American (n = 27). Indigenous (n = 2) and mixed-ethnicity (n = 5) children

were excluded from this study because the sample sizes were not large enough to test for Hardy-Weinberg equilibrium.

Procedure

The 36-month follow-up was conducted at a developmental laboratory at the University of Nebraska-Lincoln. Children were individually tested by a trained research assistant over three sessions separated by approximately one week. After the completion of each session, children received a small toy. After completing all three sessions mothers received a gift card. In the first session, children completed the *Disruptive Behaviour Diagnostic Observation Schedule* (DB-DOS) (Wakschlag et al., 2008). The DB-DOS is a standardized clinical observation designed to differentiate between clinically salient patterns of behavioural dysregulation and normative misbehaviour in early childhood (Wakschlag et al., 2008). In the second and third sessions, children completed a battery of EF tasks. A saliva sample was also collected for subsequent genotyping. EF tasks were administered in a fixed order to ensure that potential carry-over effects across tasks would be consistent across participants. Adherence to experimental protocols was maintained by reviewing session video recordings and regular team meetings.

Measures

Genotyping. Children were genotyped for the 3'UTR VNTR polymorphism of *DAT1*, the Taq1A polymorphism of *DRD2*, the exon 3 VNTR polymorphism of *DRD4*, and the Val(108/158)Met polymorphism *COMT.* Trained research assistants collected saliva samples from participants using the DNA Genotek Oragene Self-Collection Kits (Ottawa, Canada). DNA was extracted and quantified with Quanti-iT Pico Green dsDNA assay (Thermo Fisher; Waltham, MA). Following polymerase chain reaction, products were separated on a 3730 Genetic Analyzer (Wakschlag et al., 2010). If genotyping was unsuccessful (e.g., the

experimenter was unable to collect a saliva sample due to child noncompliance), a second saliva sample was collected from children at the 5-year follow-up and genotyping was redone.

For *DAT1*, *DRD2*, *DRD4*, and *COMT* allele frequencies and results of testing for Hardy-Weinberg equilibrium are presented separately for each ethnic group and the whole sample in Table 3-1. If all cells had more than five participants, Hardy-Weinberg equilibrium was tested for using a χ^2 test; otherwise, an exact test was used (Wigginton, Cutler, & Abecasis, 2005). For each gene, the individual ethnic groups and whole sample were in Hardy-Weinberg equilibrium.

To create the measure of cumulative dopaminergic genetic risk, we first tested whether each candidate gene was correlated with the measures of parental behaviour using bivariate correlations. None of the candidate genes were correlated with parental behaviour (rs = -.11 – .16, ps > .05); therefore, all four genes were used in the creation of the genetic risk score. Each gene was dummy-coded based on the presence or absence of the allele associated with increased sensitivity to the environment. The risk alleles were: (1) the 10-repeat allele of the 3'UTR VNTR polymorphism of *DAT1*, (2) the A1 allele of the Taq1A polymorphism of *DRD2*, (3) the 7-repeat allele of the exon 3 VNTR polymorphism of *DRD4*, and (4) the Val allele of the Val(108/158)Met polymorphism of *COMT*. The dummy codes were summed to form a risk score ranging from 0 to 4 (D. Belsky & Israel, 2014; Jolicoeur-Martineau, Wazana, et al., 2019).

Parental behaviour. Mother-child dyads were videotaped completing the parent context of the DB-DOS and maternal behaviour was coded offline. The parent context was designed to "press" for children's behavioural dysregulation in four ecologically salient situations: child compliance during a clean up task, completing a challenging puzzle task, mother's withdrawal of attention, and free play (Hill et al., 2008). Each situation lasted 5 minutes. Before starting the context, the procedure was explained to the mother and she was given flip cards with

instructions. Transitions between tasks were marked by the ringing of a bell. Mothers were encouraged to act as they normally would at home.

Parental behaviour was assessed using a structured observational measure of parental behaviour, the *Parenting Clinical Observation Schedule* (P-COS; Hill et al., 2008). The P-COS is designed to assess problematic and competent parental behaviours. It is a validated observational measure of parental behaviour: Scores on the P-COS are correlated with self-report measures of parenting, such as *Coping with Children's Negative Emotions* (rs = -.31 – .19, ps < .05) (Hill et al., 2008).

Two coders, trained to reliability (> 80% exact agreement on each item) by a master coder involved in the development of the coding scheme, completed all coding. All three coders double coded 20% of the videos. Ongoing reliability was maintained through regular meetings. Disagreements between coders were resolved by consensus. Inter-rater reliability was high (problematic discipline: mean κ = .88 – .91, exact agreement: 97 – 98%; responsiveness: mean κ = .86, exact agreement: 95%). Coders watched each video twice before assigning final codes. Mothers' behaviour was coded globally, with codes capturing mothers' behaviours across all four situations. Each item was rated on a 4-point scale ranging from 0 (*no evidence of the behaviour*) to 3 (*high levels of the behaviour*).

The measure of problematic discipline consisted of the summed score of seven items: hostile behaviour, verbally aggressive discipline, physical discipline, power struggles, emotional misattunement, intensity of angry/irritable affect, and pervasiveness of angry/irritable affect. Because scores on the intensity of angry/irritable affect and pervasiveness of angry/irritable affect items were highly correlated (r = .86), scores on these items were averaged together to

form one item (Hill et al., 2008). Internal consistency for the six remaining items that constituted the problematic discipline measure was adequate (ordinal ω total = .71).

The measure of responsiveness consisted of the summed score of the scaffolding, responsiveness to positive behaviours, warm affection, positive engagement, labelling, intensity of positive affect, and pervasiveness of positive affect items. Labelling was dropped from the measure because of poor loading on internal consistency analyses (corrected item-total r = .15) (Hill et al., 2008). Internal consistency for the six items in the responsiveness measure was good (ordinal ω total = .80).

Executive Function. Children completed a battery of seven EF tasks meant to assess self-control and WMIC (Wiebe et al., 2015). Administration, psychometric properties, scoring, and validation of the EF battery are described in more detail elsewhere (Wiebe et al., 2015, 2011).

Two tasks assessed children's ability to regulate their behaviour in situations where rewards were highly salient. *Goody Shelf* was administered as part of the DB-DOS (Wakschlag et al., 2008). In *Goody Shelf*, an experimenter unveiled an appealing set of toys on a small shelf. During a 5-minute delay, where the experimenter completed paperwork, children were instructed to sit at a table and were given crayons and paper to draw on. Children were told they could look at, but not touch, the toys during the delay. Each instance of toy touching was coded for intensity on a scale from 1 (brief touches) to 3 (sustained touches where the child was resistant to examiner prompts to stop touching the toys). The dependent measure was a summary score representing child noncompliance.

In *Snack Delay*, children were instructed to keep their hands on a placemat marked with two handprints and stand still in front of M&M candies placed under a transparent cup for 4

minutes. Two dependent measures were created: a summary score representing child compliance during the delay, and a measure of task success. To create the summary score, children's behaviour was scored in 5-second intervals and summed across all intervals until either the child ate the snack or the task ended. Children received up to 3 points for standing still, keeping their hands on the mat, and remaining silent. The measure of task success was coded as 0 (at the snack during the delay) or 1 (did not eat the snack during the delay).

Five tasks assessed children's abilities to hold in mind and manipulate information and inhibit a prepotent response. In *Delayed Alternation,* a food reward was hidden in one of two locations and children had to pick the correct location of the reward. The experimenter changed the location of the reward after each correct trial. Trials were separated by a 10-second delay that required children to hold the previously rewarded location in working memory. The dependent measure was the proportion of correct responses.

For the *Nebraska Barnyard* task, children listened to sequences of animal names. Next, they pressed coloured buttons on a touch screen that corresponded to the correct sequence of animal names. The dependent measure was a summary score calculated by summing the proportion of correct trials at each sequence length.

In the *Big-Little Stroop* task, children were shown small pictures of everyday objects, embedded within larger pictures that matched (congruent trials) or mismatched (incongruent trials) the smaller pictures. Children were asked to name the smaller, embedded pictures. The dependent measure was the proportion of correct responses on incongruent trials.

In *Preschool Go/No-Go,* children were told to press a button on a button box to catch coloured fish (75% of trials) but to withhold pressing the button when a shark appeared (25% of

trials). The dependent measure was d-prime (*d'*), the standardized difference between the hit rate and false alarm rate (Stanislaw & Todorov, 1999).

The inhibit condition of *Shape School* required children to name the colour of a cartoon shape character when the character had a happy face and to remain silent when the character had a sad face (inhibit trials). The dependent measure was the proportion of correct responses on inhibit trials.

Confirmatory factor analysis supported a two-factor measurement model for EF consisting of self-control and WMIC (see Wiebe et al., 2015). A two-factor model fit the data well, $\chi^2(19) = 32.52$, $p = .03$, $RMSEA = .07$, $CFI = .93$, $SRMR = .06$. It provided a better model fit than a one-factor model ($\Delta\chi^2(1) = 37.88$, $p < .01$) and was more parsimonious than a three-factor model (self-control, WM, and IC) model ($\Delta\chi^2(2) = 1.71$, $p = .43$). *Goody Shelf* and *Snack Delay* loaded on a self-control factor. All self-control factor loadings were statistically significant and standardized factor loadings ranged from .43 to .97. *Delayed Alternation, Nebraska Barnyard, Big-Little Stroop*, *Preschool Go/No-Go,* and *Shape School*-inhibit condition loaded on a WMIC factor. The five factor loadings were statistically significant and standardized factor loadings ranged from .42 to .56. Factor scores for the self-control and WMIC latent factors were used as the measures of EF.

Covariates. Child ethnicity, prenatal tobacco exposure status, maternal psychological distress, household socioeconomic status, and child sex were included as covariates. Ethnicity is associated with the distribution of alleles in the population (Dick et al., 2015). Child ethnicity was coded using a set of dummy codes with European American ethnicity serving as the reference. Prenatal tobacco exposure status was included as a covariate because mothers who smoked during pregnancy were overenrolled in the MIDS cohort. At two points during

pregnancy and shortly after their child's birth, mothers completed timeline-follow-back interviews about daily smoking. Maternal urine and infant meconium were assayed for cotinine, a metabolite of nicotine, to verify tobacco exposure status (Espy et al., 2011). Prenatal tobacco exposure status was dummy coded as 0 (absent) or 1 (present). Maternal psychological distress, household socioeconomic status, and child sex are sometimes correlated with parental behaviour and EF (Carlson & Wang, 2007; Linver, Brooks-Gunn, & Kohen, 2002; Vrantsidis, Clark, Chevalier, Espy, & Wiebe, 2020). The Global Severity Index of the *Brief Symptom Inventory* (BSI; Derogatis, 1993) was used as the measure of psychological distress. After mothers completed the BSI, scores on each item were summed and divided by the number of questions answered to create the Global Severity Index. To minimize the number of participants lost due to missingness on exogenous variables, missing scores (n = 1) were replaced with Global Severity Index scores from the 6-month follow-up. Socioeconomic status was indexed using parental education. Mothers reported on each parent or significant other's highest educational degree. For single-parent households, mother's highest degree was used. For two-parent households, the highest degree in the household was used.

Analytic Strategy

All dependent variables were examined for outliers and non-normality. In total, 5% of the data were missing, ranging from < 1% (maternal psychological distress) to 31% (*Shape School*). Full information maximum likelihood estimation (FIML) using an expectation maximization algorithm was used to address missing data on endogenous variables. FIML assumes that data are missing at random. To test whether missing data on endogenous variables were related to demographic characteristics, a series of logistic regression models were computed. Missingness

was unrelated to ethnicity, prenatal tobacco exposure status, maternal education, or child sex (*p*s > .05).

Before testing for moderation, a path model was established using MPlus 8.1 (Muthén & Muthén, 2012). First, predictors were mean-centered and product terms representing the interactions between cumulative dopaminergic genetic risk and parental behaviour (genetic risk × problematic discipline and genetic risk × responsiveness) were calculated. Following Keller's (2014) recommendations on controlling for potential confounders in gene × environment interaction research, product terms representing the two-way interactions between each covariate and genetic risk and parental behaviour were calculated. Next, a path model was tested by adding directional paths from the measures of genetic risk, parental behaviour, interaction terms, and covariates to EF.

Moderation was tested using a series of path models. Paths for each genetic risk × parental behaviour interaction term were sequentially constrained to zero. A chi-square difference ($\Delta\chi^2$) test was used to compare the constrained model to the unconstrained model (Kline, 2015). Moderation was supported if constraining the path to zero resulted in a significant chi-square difference test ($p < .05$). For significant tests, the less constrained model was retained; otherwise, the more parsimonious model was favored.

Significant interactions were probed following Roisman et al.'s (2012) recommendations. First, we graphed significant interactions. Values of problematic discipline and responsiveness did not range two standard deviations above and below the sample mean (Roisman et al., 2012). Therefore, we plotted observed data using a cut-off of two standard deviations, as applicable. Second, a regions of significance analysis, using the Johnson-Neyman technique (P. Johnson & Fay, 1950), was used to test whether potential interactions supported the diathesis-stress or

differential susceptibility model. A regions of significance analysis identifies the values of parental behaviour at which differences in EF due to genetic risk are significant (Hayes, 2018). Third, the Proportion of Interaction (POI) was computed (Fraley, 2012). The POI compares the area between the regression lines to the right of the crossover point to the area to the left. A POI closer to 0% or 100% supports the diathesis-stress model because the likelihood of benefiting from, or being adversely affected by, an environmental factor is much greater than the alternative. A POI closer to 50% (between 40% and 60%) supports the differential susceptibility model because the likelihood of either outcome is almost equivalent.

DRD2 was correlated with ethnicity (Latinx American: $r = .22$, $p = .01$; African American: $r = .03$, $p = .77$). Therefore, we ran all analyses (1) separately for European American participants as they were the largest ethnic group; and (2) using the whole sample but excluding Latinx American participants. Results for both analyses are reported in Appendix B. The results were similar to the results for the analyses including European American, African American, and Latinx American participants; therefore, we included all three groups in the analyses and controlled for ethnicity.

Results

Descriptive Statistics

Descriptive statistics for all variables used in the analyses are presented in Table 3-2, and correlations are presented in Table 3-3. Cumulative dopaminergic genetic risk and parental behaviour measures were not significantly correlated. Problematic discipline and responsiveness were moderately correlated. The self-control and WMIC factor scores were strongly correlated. EF factor scores were generally not significantly correlated with genetic risk or the parental behaviour measures. Correlations among the covariates were typically not significant, as were

correlations between the covariates and main predictors. Correlations between EF factor scores and covariates were generally not significant or small in magnitude.

Path Analysis

To test whether the interactions between cumulative dopaminergic genetic risk and parental behaviour were associated with EF, self-control and WMIC were regressed on genetic risk scores, problematic discipline, responsiveness, interaction terms, and covariates. For both self-control and WMIC, the genetic risk × problematic discipline and genetic risk × responsiveness interaction terms were tested for moderation by comparing a model estimating the effect of the interaction term to a model constraining the path to zero. If the chi-square difference test was significant, the path was retained. If the test was not significant, the most parsimonious model was retained. For self-control, the genetic risk × problematic discipline path was retained in the final model ($\Delta\chi^2(1) = 8.18$, $p < .01$); while the path from genetic risk × responsiveness was dropped ($\Delta\chi^2(1) = .44$, $p = .51$). For WMIC, the path from genetic risk × problematic discipline was retained ($\Delta\chi^2(1) = 5.02$, $p = .03$), and the path from genetic risk × responsiveness was dropped ($\Delta\chi^2(1) = 2.78$, $p = .10$).

The final model is presented in Figure 3-1. The model was just identified, meaning it had the same number of free parameters and observations (i.e., the model had zero degrees of freedom); therefore, model fit statistics could not be calculated (Kline, 2015). Complete results are presented in Appendix C. The final model accounted for 40% of the variability in self-control. Cumulative dopaminergic genetic risk predicted self-control, but this main effect was qualified by an interaction with problematic discipline. Of the covariates, prenatal tobacco exposure and higher maternal psychological distress were associated with poorer self-control. Boys had poorer self-control than girls, although this association was qualified by an interaction

with responsiveness. When responsiveness was low, boys had poorer self-control relative to girls. The interaction between responsiveness and prenatal tobacco exposure status on self-control was marginally significant. Child ethnicity and parental education were not associated with self-control, nor were any of the remaining interactions between the covariates and main predictors.

To determine at which values of problematic discipline significant differences in the association with self-control emerged between children at different levels of genetic risk, we tested for regions of significance. Results are presented in Figure 3-2. Consistent with the diathesis-stress model, there were significant differences in self-control for children at low (25^{th} percentile) versus moderate (50^{th} percentile) genetic risk at values of problematic discipline above .54 (.52 standard deviations below the mean) and high (75^{th} percentile) genetic risk at values of problematic discipline above .34 (.33 standard deviations below the mean). There were significant differences in self-control for children at moderate (50^{th} percentile) versus high (75^{th} percentile) genetic risk at values of problematic discipline above .52 (.50 standard deviations below the mean). Higher problematic discipline was associated with poorer self-control for children at higher genetic risk relative to children at lower genetic risk. The crossover points for the interaction fell outside of two standard deviations; therefore, it was not possible to calculate the POI index.

The final model accounted for 27% of the variability in WMIC. Although the parameter estimate for the cumulative dopaminergic genetic risk × problematic discipline interaction was marginally significant according to the Wald test, removing the interaction resulted in significantly poorer model fit ($\Delta\chi^2(1) = 5.02$, $p = .03$). We probed this interaction using regions of significance analysis. When problematic discipline was higher, children with high genetic risk

scores tended to have poorer WMIC than children with moderate risk scores, who had poorer WMIC than children with low risk scores, but the upper and lower bounds for all three groups overlapped. Genetic risk, problematic discipline, and responsiveness were not associated with WMIC. Of the covariates, higher maternal psychological distress was associated with poorer WMIC. The interaction between genetic risk and prenatal tobacco exposure status was also significant. Children at low genetic risk tended to have better WMIC in the absence of prenatal tobacco exposure relative to children at moderate and high genetic risk, although the upper and lower bounds for all three groups overlapped. African American children had poorer WMIC, although this association was marginal. Prenatal tobacco exposure status, parental education, and sex were not significantly associated with WMIC. None of the remaining interactions between the covariates and genetic risk or parental behaviour were associated with WMIC.

Discussion

This study was the first to examine whether the interactions between cumulative dopaminergic genetic risk and parental behaviour (problematic discipline and responsiveness) were associated with children's EF (self-control and WMIC) in early childhood in a manner consistent with the diathesis-stress or differential susceptibility model. To achieve this aim, we adopted a multi-faceted approach, examining pathways involving multiple dimensions of parental behaviour and EF, using state-of-the-art, direct assessment methods. We hypothesized that the interactions between genetic risk and parental behaviour would be associated with children's EF. We found partial support for this hypothesis: higher problematic discipline was associated with poorer self-control for children at higher genetic risk relative to children at lower genetic risk. This result supported the diathesis-stress model as genetic risk was associated with self-control when problematic discipline was high. Contrary to the differential susceptibility

model, genetic risk was not associated with self-control when responsiveness was high. The interaction between cumulative dopaminergic genetic risk and problematic discipline was also associated with WMIC, but follow-up tests probing the interaction were inconclusive; therefore, the interaction was not interpretable. The interaction between genetic risk and responsiveness was not associated with EF.

Results were consistent with the diathesis-stress model rather than the differential susceptibility model of gene × environment interaction: Only higher problematic discipline was associated with poorer self-control for children at higher genetic risk relative to children at lower genetic risk. For responsiveness, children's self-control did not significantly differ by level of genetic risk. This pattern of findings suggests that cumulative dopaminergic genetic risk confers vulnerability for the development of poorer self-control, the motivational dimension of EF, in the context of a negative environmental factor. This has potential implications for understanding pathways to the development of externalizing behaviour problems. Deficits in EF, particularly self-control, play a role in the etiology of behaviour problems (Chang et al., 2011; Oh et al., 2020). Further, problematic discipline is theorized to increase the risk of developing behaviour problems in children with reduced mesocortical dopamine activity (Beauchaine, Gatzke-Kopp, & Mead, 2007). Therefore, the present finding suggests that problematic discipline may be a risk factor for the development of behaviour problems in children at genetic risk for lower dopaminergic activity via the impact of problematic discipline on self-control. There is preliminary support for this pathway in middle childhood and adolescence (Davies, Pearson, Cicchetti, Martin, & Cummings, 2019; Thibodeau, Cicchetti, & Rogosch, 2015). More work examining this pathway in early childhood is needed. Finally, the results of this study suggest that interventions aimed at improving self-control in children at risk for reduced dopaminergic

activity, particularly by reducing the use of problematic discipline strategies, could potentially buffer against the development of behaviour problems (Beauchaine & Gatzke-Kopp, 2012).

Previous research on interactions between dopamine genotype and parental behaviour on children's EF has primarily focused on self-control (Vrantsidis, Wuest, et al., under review), but this study included WMIC, as well. The interaction between genetic risk and problematic discipline was associated with WMIC. However, a regions of significance analysis found that the upper and lower bounds of the genetic risk groups overlapped at all values of problematic discipline. Therefore, tests for an interaction between genetic risk and problematic discipline on WMIC were inconclusive. The discrepant findings for WMIC and self-control may be due to the age of the participants. Perseveration and impulsive responding are prevalent in preschoolers (Carlson, 2005). Therefore, the association between problematic discipline and WMIC, compared to self-control, may have been masked, but could emerge as the trajectories of children at different levels of genetic risk diverge with development. Additional research is necessary to better understand the relation between gene × parental behaviour interactions and WMIC in early childhood.

The interaction between genetic risk and responsiveness on EF was not significant. This result runs counter to previous work suggesting individual dopaminergic candidate genes (e.g., *COMT*) and responsiveness interact to predict children's EF in early childhood (Kok et al., 2013; Pickles et al., 2013). It is possible that aspects of responsiveness, unmeasured in the present study, such as parent's supportive presence or engagement, are important for the development of EF in the context of dopaminergic genetic risk (Kok et al., 2013; Smith et al., 2013). In addition, the P-COS was developed for use with children at-risk for the development of behaviour problems (Hill et al., 2008). The responsiveness measure was meant to distinguish between

"good enough" and clinically concerning parental behaviours. It is possible that the measure was not sensitive to variations in responsiveness that distinguish between "good enough" parents, potentially limiting our ability to detect significant main or interaction effects involving responsiveness.

This study's results should be interpreted within the context of the study's strengths and limitations. The key strengths of this study were the use of a polygenic genetic risk score; developmentally sensitive, multi-dimensional, direct assessment methods of both parental behaviour and EF; and rigorous statistical methods to control for potential confounders, particularly ethnicity. First, this study used a cumulative dopaminergic genetic risk score to assess dopamine genotype. Most studies have examined one or two genes involved in dopaminergic functioning as potential moderators of the relations between parental behaviour and children's EF (Vrantsidis, Wuest, et al., under review). Individual genes account for minimal variability in EF (Fossella et al., 2002) and single candidate gene studies are unlikely to produce statistically significant results, especially when sample sizes are small (Dick et al., 2015). The use of a genetic risk score that included genes involved in dopamine transport, reuptake, and catabolism allowed us to model functioning of the dopaminergic system more broadly. This likely increased power to detect a significant gene × parental behaviour interaction (Pasman et al., 2019). Second, the inclusion of measures of problematic discipline and responsiveness meant this study captured both positive and negative environmental exposures (J. Belsky & Pluess, 2009). This meant the present study was well positioned to test competing models of gene × environment interaction. Third, the use of factor scores, based on a latent variable approach to the measurement of EF, likely improved the reliability of the EF measures. Performance on individual EF tasks reflects variations in both EF abilities and the basic abilities required to

complete the tasks (e.g., motor abilities), often making individual tasks unreliable measures of EF (Miyake et al., 2000). The use of a latent variable approach resulted in a model of EF with good fit to the data, likely improving our ability to detect any potential associations between genetic risk and parental behaviour on EF. Fourth, this study adopted more rigorous controls for covariates and population stratification than is typical in gene × environment interaction research (Dick et al., 2015; Keller, 2014). In addition to including the main effects of the covariates, all genetic risk × covariate and parental behaviour × covariate interaction terms were included in the analyses. Including the interaction terms controlled for the effects of the covariates (e.g., ethnicity) on the gene × parental behaviour interactions. In addition, because the sample was ethnically heterogeneous, sensitivity analyses were conducted to further rule out population stratification as a possible confounder.

This study had several limitations that are important to note. First, this study was cross-sectional. As a result, we were unable to disentangle the direction of the relation between parental behaviour and children's EF. It is possible that children's EF mediates the relation between their genotype and parental behaviour (Hayden et al., 2013; Kryski, Smith, Sheikh, Singh, & Hayden, 2014). Nonetheless, results of randomized controlled trials provide additional support for an interaction between children's genotype and parental behaviour on children's EF: Parenting interventions are more impactful for children at higher genetic risk relative to children at lower genetic risk (for a meta-analysis see van IJzendoorn & Bakermans-Kranenburg, 2014). Second, the sample size of the current study was relatively small for a genetic association study. It is worth noting that this study's sample size ($N = 135$) is close to the sample sizes of previous research examining gene × parental behaviour interactions on children's EF, which typically include approximately 150 participants (Vrantsidis, Wuest, et al., under review). A larger sample

size would increase power; thereby, increasing the precision of estimates. Replication of the present findings using study designs with increased power is needed. Third, the three indicators of self-control came from two tasks that were similar in task demands, including engaging in less appealing alternative behaviours, delaying gratification, and suppressing approach behaviours (Wiebe et al., 2015). Consequently, it was not possible to identify which aspects of self-control were impacted by the interaction between genetic risk and problematic discipline. Future research separating the relative contributions of emotion, delay, and reward to self-control in early childhood would enable a more nuanced examination of gene × parental behaviour interactions on self-control.

Results of the present study were consistent with the diathesis-stress model of gene × environment interaction. Higher problematic discipline was associated with poorer self-control for children at higher genetic risk relative to children at lower genetic risk. Thus, negative parental behaviours may be an important risk factor for the development of poorer self-control in at-risk children. Poorer EF is a risk factor for the development of behaviour problems (Chang et al., 2011; Oh et al., 2020). Identifying pathways from problematic discipline to externalizing behaviour problems in children at genetic risk for decreased dopaminergic functioning via self-control may help with the development of interventions aimed at improving EF and decreasing the risk for the development of behaviour problems in at-risk children.

Table 3-1. *Allele frequencies and Hardy-Weinberg equilibrium (HWE) for candidate genes*

Gene	Major allele homozygote *N*	Heterozygote *N*	Minor allele homozygote *N*	HWE	*p*
DAT1	0 copies	1 copy	2 copies		
10-repeat VNTR					
European American	3	34	39	$D = 2.26$	.25
African American	4	10	18	$D = -1.47$	.20
Latinx American	2	12	13	$D = .37$	1.00
Total	9	56	70	$\chi^2(1) = .24$	.62
DRD2 Taq1A	GG	GA	AA		
European American	51	24	1	$D = 1.22$	.68
African American	18	12	2	$D = .00$	1.00
Latinx American	10	13	4	$D = .08$	1.00
Total	79	49	7	$\chi^2(1) = .82$	.37
DRD4	0 copies	1 copy	2 copies		
7-repeat VNTR					
European American	50	22	4	$D = -1.04$	.37
African American	18	13	1	$D = .76$	.65
Latinx American	17	7	3	$D = -1.44$	.13
Total	85	42	8	$\chi^2(1) = .03$	.87
COMT	Val/Val	Val/Met	Met/Met		
Val(108/158)Met					
European American	18	35	23	$\chi^2(1) = .43$	.51
African American	13	13	6	$\chi^2(1) = .69$	.41
Latinx American	7	13	7	$\chi^2(1) = .04$	.85
Total	38	61	36	$\chi^2(1) = .1.25$	.26

Table 3-2. *Descriptive statistics for the measures of cumulative dopaminergic genetic risk, parental behaviour, executive function, and covariates*

Construct	*N*	*M*	*SD*	Range
Cumulative dopaminergic genetic risk (composite score)	135	2.04	1.04	0.00 – 4.00
Problematic discipline (composite score)	135	0.92	1.32	0.00 – 7.00
Responsiveness (composite score)	135	13.01	2.79	6.00 – 18.00
Goody Shelf (rule-breaking)	129	3.22	6.94	0.00 – 33.00
Snack Delay (movement score)	125	52.38	32.20	4.00 – 117.00
Snack Delay (ate treat)	125	0.31	0.47	0.00 – 1.00
Delayed Alternation (accuracy)	130	0.50	0.18	0.00 – 0.94
Nebraska Barnyard (composite score)	124	3.31	1.75	0.75 – 8.06
Big-Little Stroop (conflict trial accuracy)	124	0.25	0.25	0.00 – 1.00
Go/No-go (d')	132	0.57	1.02	-1.37 – 3.12
Shape School inhibit (accuracy)	93	0.35	0.27	0.00 – 1.00
Self-control factor score	135	0.05	0.95	-1.50 – 1.91
WMIC factor score	135	0.03	0.80	-1.47 – 2.53
Child ethnicity	135			
European American		56%		
African American		24%		
Latinx American		20%		
Prenatal tobacco exposure status (% exposed)	135	55%		
Psychological distress (composite score)	135	0.50	0.53	0.00 – 3.64
Parental education (years)	135	14.03	1.57	11.00 – 18.00
Child sex (% male)	135	53%		

Table 3-3. *Correlations between cumulative dopaminergic genetic risk, parental behaviour, executive function factor scores, and covariates*

Measures	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. Cumulative dopaminergic genetic risk	.08	.00	-.20*	-.09	.11	.09	-.07	.06	.04	-.12
2. Problematic discipline	--	-.34**	-.10	-.17*	-.16^{+}	.23**	.23**	.03	-.18*	.13
3. Responsiveness		--	.12	.10	-.05	-.13	-.07	-.27**	.27**	-.03
4. Self-control factor score			--	.54**	-.21*	.13	-.26**	-.25**	.02	-.13
5. WMIC factor score				--	-.21*	.03	-.13	-.26**	.21*	.04
6. African American					--	-.28**	-.05	.07	-.14^{+}	-.07
7. Latinx American						--	.04	.06	-.07	-.13
8. Prenatal tobacco exposure status							--	.11	-.17*	-.04
9. Psychological distress								--	-.17*	-.07
10. Parental education									--	.15^{+}
11. Sex										--

Note. WMIC = working memory/inhibitory control. $^{+}p < .10$; $^{*}p < .05$; $^{**}p < .01$.

Figure 3-1. Path diagram illustrating the main and interaction effects of cumulative dopaminergic risk and parental behaviour on self-control and working memory/inhibitory control (WMIC). Both unstandardized and standardized (in parentheses) parameters are presented; error variances and covariates (child ethnicity, prenatal tobacco exposure status, psychological distress, parental education, and child sex) are not shown. $^{+}p < .10$; $^{*}p < .05$; $^{**}p < .01$.

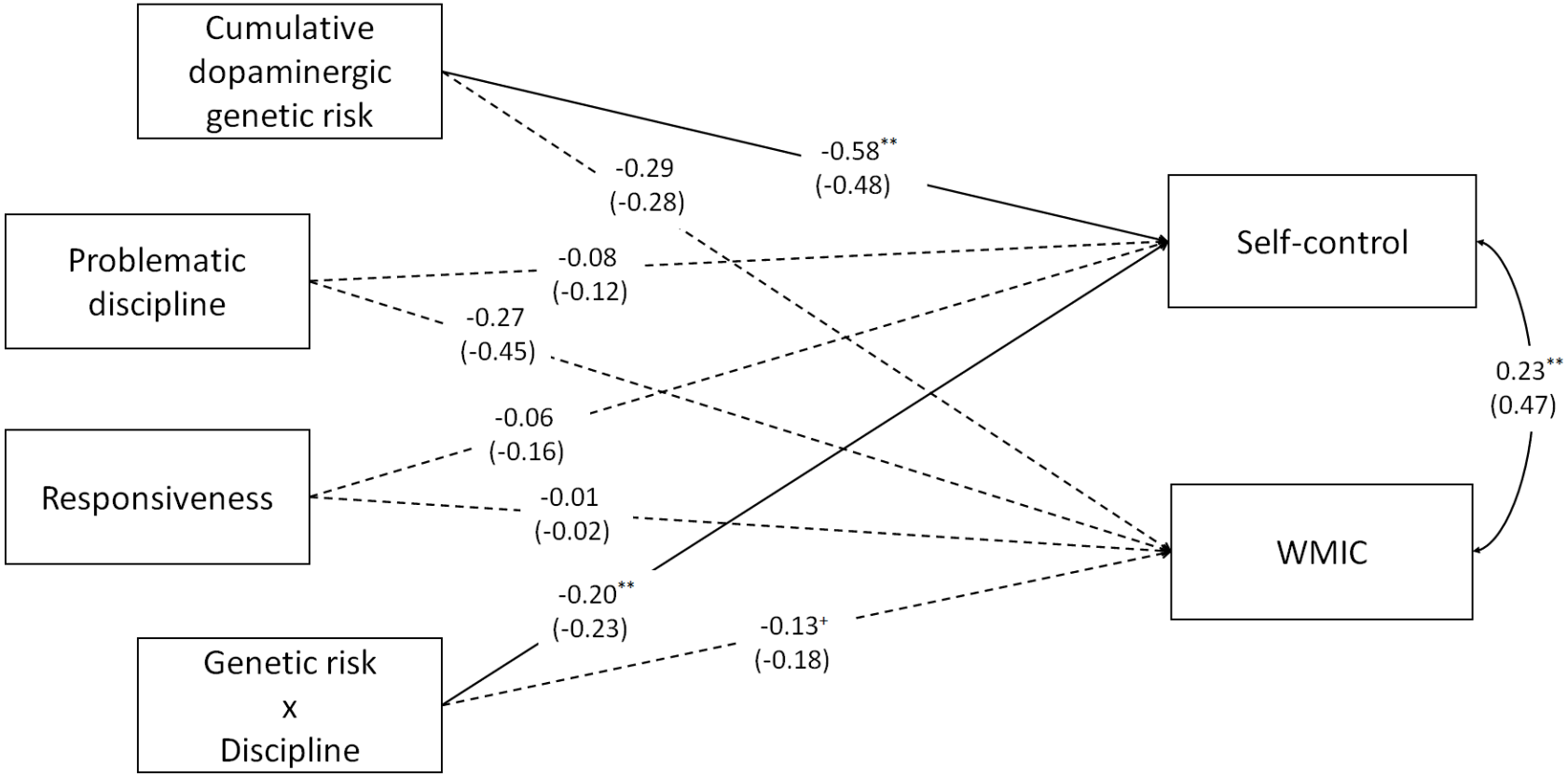
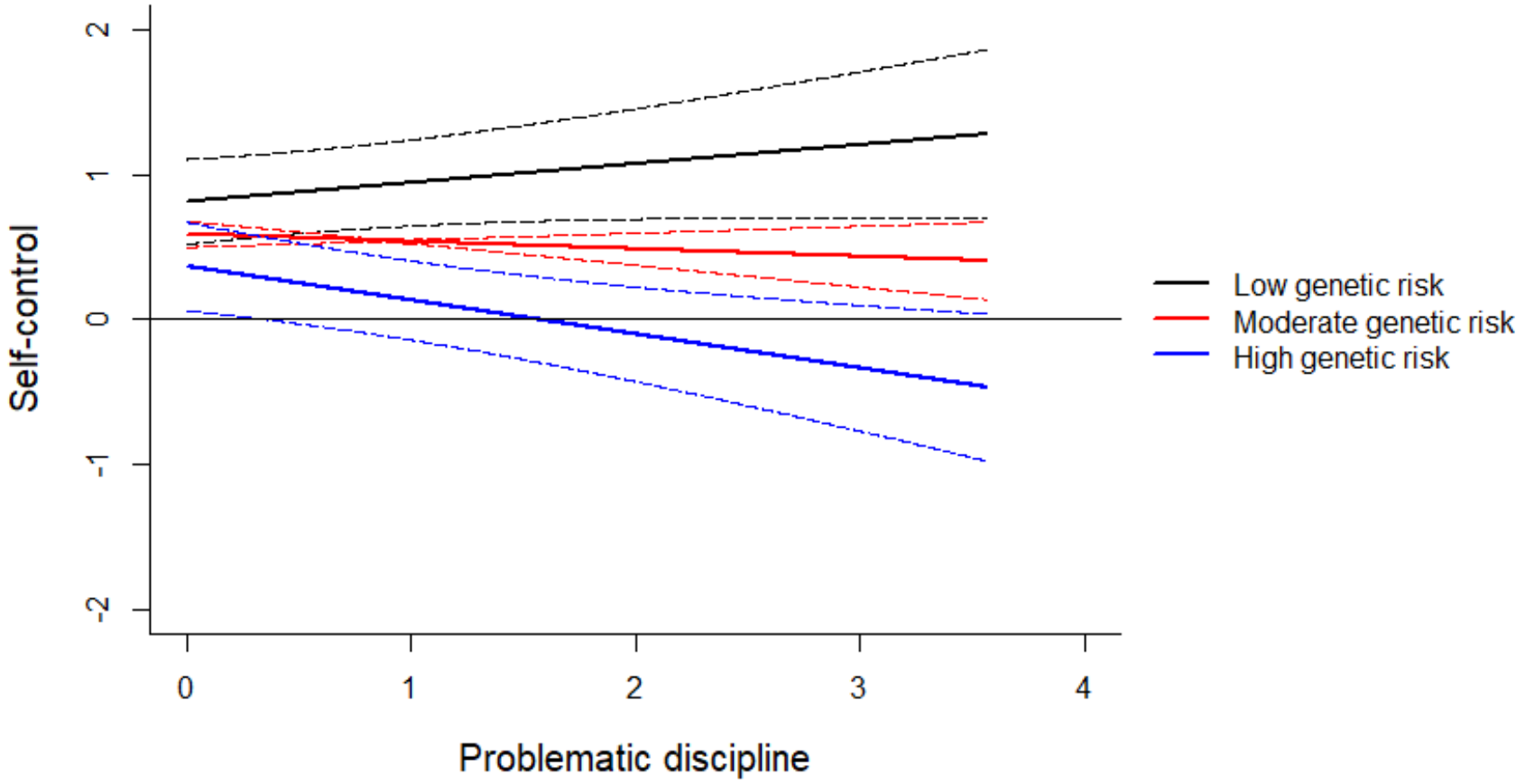


Figure 3-2. The relation between problematic discipline and self-control by cumulative dopaminergic genetic risk, including upper and lower bounds.



Chapter 4

Examining Bidirectional Relations Between Parental Discipline and Children's Inhibitory Control

Examining Bidirectional Relations Between Parental Discipline and Children's Inhibitory Control

Inhibitory control (IC) is one component of executive function (EF), the set of higher-order cognitive processes necessary for carrying out goal-directed behaviour (Garon et al., 2008). IC is the ability to control one's attention, behaviours, thoughts, or emotions to carry out a subdominant response in favor of a prepotent or automatic response (Diamond, 2013). IC during the transition to middle childhood (ages 4 to 7) is associated with the development of emotion regulation (Hudson & Jacques, 2014) and social competence (Nigg, Quamma, Greenberg, & Kusche, 1999). Deficits in IC are also a risk factor for the development of externalizing behaviour problems, including ADHD, conduct disorder, and aggression (Matthys et al., 2013).

Parents' discipline techniques predict children's IC between the ages of 3 and 6 (for a meta-analysis see Valcan et al. 2017). Yet, children's characteristics are associated with the frequency and duration of the parental behaviours to which they are exposed (Bell, 1968). Transactional models of the development of EF argue that parents and children mutually influence each other to drive the development of EF (J. Belsky et al., 2007; Kiff et al., 2011). Examining bidirectional relations between parental behaviour and children's IC is an emerging research area (Klein et al., 2018; Merz, Landry, Montroy, & Williams, 2017). The aim of this study was to examine bidirectional relations between parent's harsh and inductive discipline and children's IC in 4- to 7-year-olds.

Parental Discipline and Children's IC

Discipline refers to the ways parents' manage their children's behaviour, including issuing directions, monitoring, and responding to children's compliance (Hill et al., 2008). Discipline can be harsh or inductive. Harsh discipline is marked by coercive or inconsistent

parental behaviour, or the use of harsh punishment to manage children's behaviour. This includes the use of physical discipline, power assertion, or overly controlling or restrictive behaviour. Inductive discipline techniques are those that help children develop appropriate EF skills. Inductive discipline includes behaviour management strategies like reasoning, explaining the impact of the child's behaviour on others, and reminding the child of the rules (Kerr, Lopez, Olson, & Sameroff, 2004). Harsh discipline is associated with poorer IC in 3- to 6-year-olds (Valcan et al., 2017) and inductive discipline is associated with better IC in 2- to 8-year-olds (Klein et al., 2018; Roskam et al., 2014).

Transactional Relations Between Discipline and IC

The continuous, dynamic interplay between parent-child dyads, where children are systematically exposed to different parental behaviours as a function of their innate characteristics, is thought to be important for the development of EF (Choe et al., 2013; Eisenberg et al., 2015; Kiff et al., 2011). Children with poorer EF are argued to elicit more harsh discipline from their parents as parents try to manage their children's difficult behaviour. In turn, more harsh discipline is associated with poorer EF because it increases children's distress and physiological arousal (C. Blair, 2016). For example, compared to infants with controlling, rejecting, or sensitive parents, infants exposed to harsh discipline exhibit signs of physiological dysregulation as indexed by significantly higher cortisol levels that do not decrease following completion of the Strange Situation (Bugental, Martorell, & Barraza, 2003; Spangler & Grossmann, 1993).

Bidirectional relations between harsh discipline and children's IC have yet to be examined. Nonetheless, there is support for bidirectional relations between overcontrolling, intrusive parental behaviour and children's effortful control, a construct that overlaps with IC,

between 30- and 54-months of age (Eisenberg et al., 2015). More overcontrolling behaviour when children were 30 months old predicted poorer effortful control at 54 months, and poorer effortful control at 30 months predicted more overcontrolling behaviour at 54 months. There is also robust support for bidirectional relations between parent's use of harsh discipline and children's behaviour problems (Patterson, Debaryshe, & Ramsey, 1990; Patterson, Dishion, & Bank, 1984). Children with behaviour problems tend to elicit more harsh discipline from their parents, which, in turn, predicts more problematic behaviour (Choe et al., 2013). Given that deficits in IC are associated with the development of behaviour problems (Matthys et al., 2013), these bidirectional relations may hold for the development of IC as well.

Children with better EF are theorized to elicit more appropriate discipline techniques, such as inductive discipline, which are associated with better EF (J. Belsky et al., 2007; Kiff et al., 2011). Thus, the use of inductive discipline is thought to be beneficial for the development of children's EF. Only one study has examined bidirectional relations between parents' inductive discipline and children's IC and it did not find evidence for bidirectional relations (Klein et al., 2018). More inductive discipline when children were 3 years old predicted better child IC at age 5. However, child IC at age 3 did not predict parents' inductive discipline at age 5. Therefore, it remains to be seen whether parents' inductive discipline and children's IC mutually influence each other.

Changes in Transactional Relations Across Developmental Stages

The strength of bidirectional relations between parental discipline and children's IC may differ depending on children's developmental stage. Expectations for appropriate parental behaviour and the frequency of specific behaviours (e.g., physical discipline) change in relation to children's developmental stage (Collins, Madsen, & Susman-Stillman, 2002; Dallaire &

Weinraub, 2005). For example, parents view physical discipline as most appropriate for preschoolers and least appropriate for infants and children over the age of 5 (Gershoff, 2002). Paralleling these views, parents' use of physical discipline increases through infancy, peaks when children are 3- and 4-years-old, and declines thereafter (Straus & Stewart, 1999). This suggests that children's characteristics may elicit different behavioural responses from their parents at different stages in their development. In support of this suggestion, a study of bidirectional relations between children's conduct problems and parental monitoring from ages 6 to 16 found that conduct problems did not predict parental monitoring at age 7, but did predict it after age 11 (Burke, Pardini, & Loeber, 2008). This change in the relation between children's conduct problems and parental monitoring was thought to reflect an increased need for parental monitoring as children grew older.

Previous work on bidirectional relations between parental behaviour and child IC has focused on 3- to 5-year-olds (Klein et al., 2018; Merz et al., 2017). It remains to be seen whether the strength of bidirectional relations between parental behaviour and IC differ across the transition to middle childhood. Early childhood (birth to age 5) is thought to be a sensitive period for environmental effects on IC because there is greater plasticity in the prefrontal cortex, one of the main brain regions underlying IC, and because IC matures fastest during this period (Fay-Stammbach, Hawes, & Meredith, 2014; Valcan et al., 2017). Further, there is greater variability in parental behaviour in early childhood than during the transition to middle childhood, with parent's behaviour becoming increasingly stable between 6 months and 72 months (Dallaire & Weinraub, 2005). Greater plasticity in child IC and parental behaviour in early childhood (i.e., birth to age 5) than middle childhood (i.e., ages 6 to 12) suggests that bidirectional relations may be stronger in early childhood.

The Present Study

The aims of this study were twofold. First, we were interested in examining bidirectional relations between parents' harsh and inductive discipline and children's IC in a year long study of 4- to 7-year-olds and their primary caregivers. Second, we were interested in examining whether child age moderated these relations (see Figure 4-1 for the conceptual model). We hypothesized that higher harsh discipline at the start of the year would predict slower growth in IC over the year, and that higher inductive discipline would predict faster growth in IC. We also hypothesized that better child IC at the beginning of the year would predict a decrease in parents' harsh discipline and an increase in parents' inductive discipline over the year. Finally, we expected bidirectional relations to be stronger for younger children.

Methods

Study Design and Participants

Participants were 136 children and their primary caregiver (4-year-olds: n = 46; 5-year-olds: n = 36; 6-year-olds: n = 54; 70 girls, 66 boys; M_{age} = 5 years, 170 days; SD = 312 days) drawn from the Longitudinal Executive Function Study, a cohort-sequential study of the development of EF during the transition to middle childhood. The cohort had an original enrollment of 192 children. Seven-year-olds (n = 54) were excluded from the present analyses because they only had data available at one timepoint. Two participants were excluded because they were missing IC data due to participant noncompliance. Parents were recruited through flyers placed in childcare facilities, schools, websites, and newspaper advertisements in Edmonton, Alberta. Parents provided written, informed consent prior to their participation in the study. Children born preterm (> 3 weeks or < 2500 g) or diagnosed with neurological or developmental disorders known to affect cognitive outcomes were excluded from the study.

Children included in the final sample were from predominantly high socioeconomic status households. Family income ranged from less than $10,000 to over $90,000 (*Mdn* ≥ $90,000). Mothers completed an average of 16 years of education, equivalent to the completion of an undergraduate degree. Most parents (91%) were married or cohabitating. Parent's reported children's ethnic backgrounds as European Canadian (n = 83), Asian Canadian (n = 10), African Canadian (n = 10), Indigenous (n = 4), Middle Eastern or North African Canadian (n = 4), Latinx Canadian (n = 3), or multi-ethnic (n = 22). Approximately 56% of children were exposed to more than one language. French (34%), Arabic (8%), and Spanish (4%) were the most common second languages.

Procedure

Parent-child dyads visited a developmental laboratory at the University of Alberta to complete a laboratory session (time 1; T1). Twelve months later (time 2; T2), dyads returned to complete a second session. At each timepoint, a trained research assistant tested children individually. Children completed a battery of tasks meant to assess inhibitory control, set shifting, working memory, and general cognitive skills. Tasks were administered in a fixed order to ensure that potential carry-over effects across tasks were consistent for all participants. Parents completed background and parental behaviour questionnaires. At each timepoint, children received a small toy and parents received a gift card as compensation.

Measures

Parental behaviour. At both timepoints, a parent (T1: 85% mothers; T2: 80% mothers) completed the *Parenting Styles and Dimensions Questionnaire* (*PSDQ*; Robinson, Mandleco, Olsen, & Hart, 1995), a 62 item self-report assessment examining parental warmth, positive parenting strategies, and disciplinary practices. Items were rated on a 5-point scale ranging from

"1 = Never" to "5 = Always." The harsh discipline measure included 16 items, such as "I explode in anger towards our child." A harsh discipline score was created for each timepoint by dividing the summed score of the verbal hostility, corporal punishment, and non-reasoning/punitive strategies subscales by the number of questions answered. Three questions, "disagrees with child," "slaps child when the child misbehaves," and "appears to be more concerned with own feelings than with child's feelings," were dropped because of poor loadings on internal consistency analyses (corrected item-total *r*s < .30). Internal consistencies for the harsh discipline measures were adequate (αs = .77 – .82). The inductive discipline measure consisted of 7 items, such as "explains the consequences of the child's behaviour." An inductive discipline score was created for each timepoint by dividing the summed score of the reasoning/induction subscale by the number of questions answered. Internal consistencies for the measures of inductive discipline were adequate (αs = .76 – .82). The harsh (*r*s = -.33 – .47, *p*s < .01) and inductive discipline (*r*s = -.29 – .31, *p*s < .01) measures are correlated with self-report measures of parental behaviour, such as *Coping with Children's Negative Emotions* (Topham et al., 2011).

Confirmatory factor analysis (CFA) was used to determine whether the measures of harsh and inductive discipline corresponded to one or two constructs at T1. Because the discipline measures consisted of scale scores, parcelling was used to create three indicators for each measure (Matsunaga, 2008). For harsh discipline, parcels were created by taking the means of the verbal hostility, corporal punishment, and non-reasoning/punitive strategies subscales of harsh discipline. For inductive discipline, parcels were created by calculating the means of two or three items, chosen at random, from the reasoning/inductive discipline subscale used to measure inductive discipline. A two-factor model corresponding to harsh discipline and

inductive discipline fit the data well, $\chi^2(8) = 12.91$, $p = .12$, *RMSEA* = .07, *CFI* = .98, *SRMR* = .05. This model fit significantly better than a one-factor model, $\Delta\chi^2(1) = 72.47$, $p < .01$. Details of the measurement model are presented in Appendix D. The three harsh discipline parcels loaded on a harsh discipline factor and the three inductive discipline parcels loaded on an inductive discipline factor. All factor loadings were significant. Longitudinal measurement invariance is an assumption of latent difference score models (Kievit et al., 2018). It was tested for using a series of nested CFA models (Widaman, Ferrer, & Conger, 2010). There was configural, metric, and scalar invariance for both scales (see Appendix E for model fit statistics).

Inhibitory control. Children completed four tasks assessing IC: *Go/No-Go*, *Simon Task*, *Flanker Task*, and *Global-Local*. In *Go/No-Go* (Wiebe et al., 2012; adapted from Simpson & Riggs, 2007), participants were shown pictures of coloured fish and told to catch the fish by pressing a button on a button box (75% of trials). On no-go trials (25% of trials), an image of a shark appeared, and children were told to withhold pressing the button on the button box. The dependent measure for this task was d prime (d'), the difference between the z-scores for the hit rate (the proportion of correct responses on go trials) and false alarm rate (the proportion of incorrect responses on no-go trials) (Stanislaw & Todorov, 1999). Performance on the *Go/No-Go* task is correlated with performance on the *Day Night* task ($r = .68$; Simpson & Riggs, 2006).

In the *Simon Task* (adapted from Shing, Lindenberger, Diamond, Li, & Davidson, 2010), children were asked to sort images of beach balls and seashells by pressing the right button on a button box when they saw a seashell and the left button when they saw a beach ball. Images appeared on the right or left side of a computer screen. On congruent trials, the image appeared on the same side of the screen as the button that needed to be pressed. On incongruent trials, the image appeared on the side of the screen opposite the button that needed to be pressed. The

dependent measure was accuracy on incongruent trials. The *Simon Task* demonstrates convergent validity with the *Antisaccade Task*, *Attention Network Task*, and *Colour-Shape Switching Task* (*r*s < .66; Paap & Oliver, 2016).

The *Flanker Task* (adapted from Rueda et al., 2004) required children to press a button corresponding to the direction of a central target fish while ignoring two distractors to the left of the target and two to the right. The distracters could be (1) fish swimming in the same direction as the target fish (congruent trials), (2) fish swimming in the opposite direction of the target fish (incongruent trials), or (3) starfish (neutral trials). The dependent measure was accuracy on incongruent trials. The *Flanker Task* demonstrates convergent validity with the *Delis-Kaplan Executive Function Scales Colour-Word Test* (r = .52; Zelazo et al., 2014).

In the *Global-Local Task* (adapted from Bialystok, 2010), children were shown a big shape made up of smaller shapes (e.g., a large star made up of small hearts) and asked to identify the big or small shape depending on the cue. The big and small shape stimuli could be the same (congruent trials), different (incongruent trials), or a neutral stimuli that was never a response option (neutral trials). The dependent measure was accuracy on incongruent trials. *Global-Local* is a set shifting measure, but because there was a significant congruency effect, analogous to the congruency effect in the *Big-Little Stroop,* an IC task (Kochanska, Murray, & Harlan, 2000), we calculated an IC measure. Accuracy on incongruent trials is orthogonal to switching cost, the measure used as an index of set shifting (not reported in this study). There is convergent validity for accuracy on incongruent trials as an IC measure because it loaded on a latent factor with the three other IC measures used in this study,

CFA was used to establish the latent construct of IC at T1. A one-factor measurement model of IC fit the data well, $\chi^2(2) = 3.81$, $p = .15$, *RMSEA* = .08, *CFI* = .98, *SRMR* = .03.

Details of the measurement model are presented in Appendix D. All four tasks loaded on an IC factor. All factor loadings were significant. Model fit statistics for the tests of measurement invariance are reported in Appendix E. Tests of measurement invariance supported invariance by age group (i.e., for the 4-, 5-, and 6-year-olds) at the configural, metric, and scalar levels. Tests of longitudinal measurement invariance supported configural invariance, partial metric invariance for the *Go/No-Go* and *Global-Local* task indicators, and partial scalar invariance for the *Flanker* task indicator.

A structural equation model with latent IC factors was too complex to estimate. Therefore, we opted to use factor scores for the IC measures. Factor scores for the latent IC factors at each timepoint were generated based on the final partial scalar invariance model and exported from MPlus 8.1 (Muthén & Muthén, 2017) for use in a path model.

Covariates. Child sex (Carlson & Wang, 2007), ethnicity (Lansford, Wager, Bates, Dodge, & Pettit, 2012), and household socioeconomic status (Vrantsidis et al., 2020) were included as covariates because they are sometimes associated with parental behaviour or IC. Child sex was dummy coded (girls = 0, boys = 1), as was child ethnicity (European Canadian = 0, non-European Canadian = 1). Parental education and financial stress at T1 were used as the measures of socioeconomic status. Mothers reported on each parent or caregiver's highest degree. For single-parent households, mother's highest degree was used as the measure of parental education. For two-parent households, the highest degree in the household was used as the measure. Parents were asked how hard it was to pay their monthly bills on their income. Responses to this question were used as the measure of financial stress. To minimize the number of participants lost due to missingness on exogenous variables, missing scores on the financial stress variable (n = 1) were replaced with financial stress scores from T2.

Analytic Strategy

Univariate distributions for all variables were examined for non-normality and outliers. In total, 3% of the data were missing, ranging from < 1% (financial stress) to 24% (*Simon Task* T2); see Table 4-1 for more details. Missing data on endogenous variables were dealt with using full information maximum likelihood estimation (FIML) using an expectation maximization algorithm. FIML assumes that data are missing at random. Therefore, a series of logistic regression models were computed to test whether missingness on outcome measures was related to demographic characteristics. Younger children were more likely to be missing data on the *Flanker, Go/No-Go,* and *Simon* tasks at T1 ($\chi^2(1) = 7.50$, 4.65, and 3.53, ps < .05, respectively). Non-European Canadian children were more likely to be missing data on the *Flanker* task at T2 ($\chi^2(1) = 4.37$, $p < .05$). Less parental education predicted missing data on the harsh and inductive discipline measures, and *Flanker*, *Global-Local*, *Go/No-Go*, and *Simon* tasks at T2 ($\chi^2(1) =$ 10.54, 10.54, 9.21, 11.36, 8.54, and 8.00, ps < .05, respectively). Missing data on all other measures were unrelated to child age, sex, or ethnicity (ps > .05).

Structural equation modeling (SEM) was conducted using MPlus 8.1. Model fit was assessed using the chi-square (χ^2) statistic, root mean square error of approximation (*RMSEA*), comparative fit index (*CFI*), and standardized root mean square residual (*SRMR*). Values indicating good fit were .06 for the RMSEA, .95 – 1.00 for the CFI, and less than .08 for the SRMR (Hu & Bentler, 1999). Values indicating adequate model fit were .90 – .94 for the CFI, .06 – .08 for RMSEA, and less than .10 for the SRMR (Hu & Bentler, 1999; Kline, 2015).

A latent difference score model (McArdle & Hamagami, 2001) was used to examine whether parents' harsh and inductive discipline predicted change in children's IC, and whether children's IC predicted changes in parents' harsh and inductive discipline. The latent difference

score model that best fit the data was established using a backward trimming approach (Kline, 2015). First, parental discipline at T1, child age, and financial stress were mean centered, and parental education was centered at 16 years, equivalent to the completion of an undergraduate degree. Second, product terms representing the two-way interactions between child age and T1 harsh discipline, inductive discipline, and IC, respectively, were calculated. Third, we tested whether there were significant mean changes and variability in the harsh discipline, inductive discipline, and IC latent change scores. To do this, we created latent difference scores for each variable by estimating a single-indicator factor model consisting of the T2 harsh discipline, inductive discipline, and IC measures, respectively, with the factor loading constrained to one. A significant mean for the latent difference scores ($p < .05$) was evidence for mean change over the year. A significant variance ($p < .05$) was evidence for individual differences in change over the year. Fourth, a latent difference score model was estimated. T2 harsh discipline was regressed on T1 harsh discipline, and the path was constrained to zero. This was also done for inductive discipline and IC. The intercept and variance of each T2 measure was constrained to zero. Correlations between the three latent change scores were estimated, as were correlations between the three T1 measures and child age. Autoregressive and cross-lagged regression paths from each change score to T1 measure were estimated. The three latent change scores were also regressed on the interaction terms and covariates. Parental education and financial stress were correlated because they both capture aspects of socioeconomic status (Evans & English, 2002). Finally, non-significant regression paths ($p > .10$) were trimmed sequentially, beginning with the path that had the highest p-value (Kline, 2015).

Moderation was tested using a series of path models. Paths from the harsh discipline × age, inductive discipline × age, and IC × age interaction terms were sequentially constrained to

zero. A chi-square difference ($\Delta\chi^2$) test was used to compare the constrained model to the unconstrained model (Kline, 2015). Moderation was supported if constraining the path resulted in a significant chi-square difference test ($p < .05$). For significant tests, the constrained model was retained; otherwise, the more parsimonious model was favored. Significant interactions were probed using simple slopes (Aiken & West, 1991) to determine whether the strength of the associations differed by age. Because a continuous measure of child age was used in all models, simple slopes were testing using the mean of each age group ($M_{4\text{-}year\text{-}olds} = 4.41$; $M_{5\text{-}year\text{-}olds} = 5.43$; $M_{6\text{-}year\text{-}olds} = 6.39$).

Results

Descriptive Statistics

Descriptive statistics are presented in Table 4-1, and correlations among these variables are presented in Table 4-2. Four-, five-, and six-year-olds did not differ in their exposure to harsh discipline (T1: $F(2, 133) = .10$, $p = .81$; T2: $\chi^2(2) = 4.28$, $p = .12$) or inductive discipline (T1: $F(2, 133) = 2.73$, $p = .07$; T2: $F(2, 105) = .47$, $p = .63$). Mean latent IC differed between age groups at T1 ($\Delta\chi^2(2) = 49.55$, $p < .01$). Four-year-olds ($M = .00$, $SD = 1.00$) had lower mean latent IC than five-year-olds ($M = .57$, $SD = .24$; $\Delta\chi^2(1) = 33.11$, $p < .01$) and six-year-olds ($M = .65$; $SD = .28$; $\Delta\chi^2(1) = 48.04$, $p < .01$). Mean latent IC did not differ for the five- and six-year-olds ($\Delta\chi^2(1) = 1.25$, $p = .26$).

Latent Difference Score Model

A model consisting of the latent difference scores for harsh discipline, inductive discipline, and IC was just identified. Children tended to show an approximate .44 standard deviation increase in latent IC between T1 and T2 ($\Delta IC_\alpha = .38$, $p < .01$). There were negligible changes in parents' harsh discipline ($\Delta HD_\alpha = -.04$, $p = .13$) or inductive discipline ($\Delta ID_\alpha = .02$, p

=.67). There was between-person variability in the extent to which children's IC (ψ = .23, p < .01) and parent's harsh discipline (ψ = .07, p < .01) and inductive discipline (ψ = .20, p < .01) changed over the year, suggesting it was appropriate to continue with a latent difference score model (Kievit et al., 2018).

Change in IC, harsh discipline, and inductive discipline were regressed on the T1 predictors, interaction terms, and covariates. Non-significant paths were trimmed sequentially, beginning with the path that had the highest p-value. For change in IC, only the paths from T1 IC, age, IC × age, and parental education were retained in the final model. For change in harsh discipline, the paths from harsh and inductive discipline at T1, age, inductive discipline × age, and financial stress were retained. For change in inductive discipline, the paths from T1 inductive discipline, T1 harsh discipline, financial stress, and ethnicity were retained.

The final model is presented in Figure 4-2. This model fit the data well, $\chi^2(32) = 45.91$, p = .05, *RMSEA* = .06, *CFI* = .98, *SRMR* = .07. The predictors in the final model accounted for 97% of the variability in change in children's IC. IC at T1 predicted change in IC, but this main effect was qualified by an interaction with age that was marginal ($\Delta\chi^2(1) = 3.75$, p = .053). Results of the simple slopes tests are presented in Figure 4-3. Poorer IC at T1 predicted faster growth in IC and this trend got stronger with age (4-year-olds: b = -.52, p < .01; 5-year-olds: b = -.55, p < .01; 6-year-olds: b = -.57, p < .01). Age did not predict change in IC. Of the covariates, higher parental education was a marginal predictor of slower growth in IC (b = -.01, p = .07).

The final model accounted for 25% of the variability in change in parents' harsh discipline. Inductive discipline at T1 predicted change in harsh discipline and this main effect was qualified by an interaction with child age ($\Delta\chi^2(1) = 6.20$, p = .01). Results of the simple slopes tests are presented in Figure 4-4. For parents of 5- (b = -.14, p < .01) and 6-year-olds (b =

-.26, $p < .01$), but not parents of 4-year-olds ($b = -.01$, $p = .95$), higher inductive discipline at T1 predicted a decrease in harsh discipline over the year. A one standard deviation increase in harsh discipline at T1 predicted a .41 standard deviation decrease in harsh discipline over the year. Child age did not predict change in harsh discipline. Of the covariates, higher financial stress predicted a decrease in harsh discipline over the year ($b = -.05$, $p = .03$).

The final model accounted for 31% of the variability in change in parents' inductive discipline. A one standard deviation increase in inductive discipline at T1 predicted a .45 standard deviation decrease in inductive discipline over the year. Harsh discipline at T1 predicted a decrease in inductive discipline over the year, although this was marginal. Child age did not predict change in inductive discipline. Of the covariates, non-European Canadian parents ($b = .14$, $p = .09$) and those with higher financial stress ($b = .08$, $p = .07$) had marginal increases in inductive discipline over the year.

Discussion

The aims of this study were to (1) examine bidirectional relations between parent's harsh and inductive discipline and children's IC over the transition to middle childhood, and (2) to examine child age as a moderator of these relations. We hypothesized that higher harsh discipline and inductive discipline at the beginning of the year would predict slower growth, and faster growth, respectively, in children's IC over the year. We also expected better child IC to predict a decrease in harsh discipline and an increase in inductive discipline over the year. Further, we expected bidirectional relations to be stronger for younger children. Results did not support these hypotheses. Parental discipline at the beginning of the year did not predict change in children's IC and children's IC did not predict change in parental discipline over the year. Instead, higher inductive discipline at T1 predicted a decrease in harsh discipline over the year

for the parents of 5- and 6-year-olds, but not parents of 4-year-olds. In addition, poorer IC at T1 predicted faster growth in IC and this trend got stronger with age. These results do not support bidirectional relations between parental discipline and children's IC over the transition to middle childhood or child age as a moderator of these bidirectional relations.

Parental discipline did not predict change in children's IC over the year and children's IC did not predict change in parental discipline. While these findings were unexpected based on transactional models of the development of EF (e.g., J. Belsky et al., 2007; Kiff et al., 2011), support for bidirectional relations between parental discipline, and parental behaviour, more generally, and children's IC is mixed (Eisenberg et al., 2015; Klein et al., 2018; Merz et al., 2017). It is possible that aspects of parental discipline and IC, unmeasured in the present study, are involved in bidirectional relations. For example, intrusion and overcontrol predict poorer effortful control in children 30- to 54-months-old (Eisenberg et al., 2015). Likewise, inductive discipline techniques, such as monitoring children's behaviour and limit setting, predict better IC in 2- to 8-year-olds (Klein et al., 2018; Roskam et al., 2014). Finally, research on child temperament as a predictor of parental behaviour has found strong and consistent evidence for bidirectional relations between parental discipline and children's negative affect and impulsivity (for a review see Kiff et al., 2011). This may suggest that aspects of IC that reflect regulation of behaviour in emotionally or motivationally arousing situations, rather than aspects of IC used in situations of cognitive load, are implicated in bidirectional relations with parental discipline.

It is also possible that bidirectional relations between parental discipline and children's IC were not significant because of the high degree of stability in IC and parental discipline over the year. In particular, the two IC measures were highly correlated (r = .97) and T1 IC accounted for the majority of variability in change in IC. By comparison, in previous studies the

correlations between IC measures ranged from .53 to .59 (Klein et al., 2018; Merz et al., 2017). The high degree of stability in the present study may be due to the demographics of the sample. Compared to previous studies, children in this study were older (i.e., 4- to 7-years-old versus 3- to 5-years-olds) and families were from primarily middle and high socioeconomic status households, rather than low socioeconomic households (e.g., Klein et al., 2018; Merz et al., 2017). There tends to be less variability in IC and parental behaviour in dyads with older children and more socially advantaged backgrounds (Clark et al., 2013; Dallaire & Weinraub, 2005; Lawson, Hook, & Farah, 2018; Razza, Martin, & Brooks-Gunn, 2010; Wiebe et al., 2012). These demographic differences may have contributed to the divergent findings across studies.

For parents of 5- and 6-year-olds, higher inductive discipline at T1 predicted a decrease in harsh discipline over the year. For parents of 4-year-olds, inductive discipline did not predict change in harsh discipline. This pattern of results is consistent with previous research (Choe et al., 2013; Kerr et al., 2004), and with expectations for normative change in parents' use of discipline techniques as children change developmental stages. During middle childhood, ages 5 to 12, parents' use of physical discipline decreases, and their use of inductive discipline increases (Collins et al., 2002; Lansford, Staples, Bates, Pettit, & Dodge, 2013). This shift in discipline techniques is thought to occur in response to children's growing self-regulatory and cognitive abilities (e.g., understanding more complex reasoning and perspective taking) (Collins et al., 2002). In middle childhood, children are better able to understand inductive discipline techniques and appropriately modify their behaviour. The results of this study may have captured parents at different stages in this transition. It is possible that parents of 4-year-olds had yet to shift to using discipline techniques more commonly seen in middle childhood, while the parents of 5- and 6-

year-olds were increasing their use of inductive discipline and decreasing their use of harsh discipline as appropriate for their child's developmental stage.

Poorer child IC at T1 predicted faster growth in IC, and this trend was stronger for older children. Faster growth in IC for children with poorer IC at T1 may reflect a catch-up effect. It may also reflect a ceiling effect (e.g., 53% of 6-year-olds were at ceiling on the *Flanker Task* at T2 compared to 20% of 4-year-olds) as there was greater variability in low IC scores compared to high IC scores. This finding is consistent with a study on the development of EF, including IC, that found children with poorer EF at age 3 had faster growth in EF between the ages of 3 and 5 (C. Blair et al., 2014). The results of the present study extend this finding to 4- to 7-year-olds.

Findings from the current study should be interpreted in light of the study's strengths and limitations. Key strengths of the present study were the use of a latent variable approach to the measurement of child IC, the establishment of longitudinal measurement invariance for the measures of IC and parental discipline, and the use of a model that explicitly models change over time. The use of factor scores based on a latent variable approach to measuring IC likely improved construct reliability. Individual IC tasks are often unreliable measures of IC because performance reflects variations in both IC ability and the basic abilities required to complete the tasks; for example, motor abilities (Miyake et al., 2000). The use of a latent variable approach resulted in a model with good fit to the data, separating IC contributors to task performance from extraneous task-specific contributors. Second, we were able to establish longitudinal measurement invariance for the IC and discipline measures. By establishing longitudinal measurement invariance, it was possible to rule out differences in the measurement of the constructs at T1 and T2 as a possible explanation for the results. Third, by using a latent change score model, compared to an autoregressive cross-lagged panel model, we were able to explicitly

model change in IC and parental discipline (Usami, Hayes, & McArdle, 2016). This meant it was possible to test whether there were individual differences in change over the year (Hertzog & Nesselroade, 2003).

This study had several limitations. First, we used a self-report questionnaire to assess parental behaviour. Questionnaires may capture parenting styles (e.g., parents' attitudes, behaviours, beliefs, and values about parenting) which tend to be stable over time; whereas, observational measures of parental behaviour may capture context-specific behaviours that have the potential to alter child behaviour, and fluctuate over time (Bornstein, Cote, & Venuti, 2001; Dallaire & Weinraub, 2005). It is possible that the discipline measures in the present study captured parenting styles, rather than specific behaviours that occur in the context of parent-child interactions. Observational measures of parental behaviour may be more sensitive to parental discipline behaviours that are related to children's IC. Second, the study had a relatively small sample size for structural equation modelling. A larger sample size would have allowed us to estimate a more complex model that included latent IC factors. It also would have increased the precision of estimates. Third, because data were only available at two timepoints, we were unable to model within-person change overtime. Transactional models of child development are theories of within-person change (e.g., Patterson et al., 1984). Cross-lagged panel models of within-person change, such as the random intercept-cross-lagged panel model, require three timepoints to be identified (Hamaker, Kuiper, & Grasman, 2015).

Most research on the relation between parental behaviour and child IC has not considered child-driven effects (see Valcan et al., 2017). Children tend to elicit behaviours from their caregivers on the basis of their characteristics (Bell, 1968). The behaviours children elicit also tend to change with their developmental stage (Collins et al., 2002). This study examined

bidirectional relations between parent's harsh and inductive discipline and children's IC over the course of a year and child age as a moderator of these relations. There was no support for bidirectional relations between parental discipline and children's IC in 4- to 7-year-olds. This finding conflicts with research on 3- to 5-year-olds (Eisenberg et al., 2015; Klein et al., 2018; Merz et al., 2017). These differential findings for 4- to 7-year-olds and 3- to 5-year-olds is broadly consistent with the suggestion that early childhood is a sensitive period for the effects of parental behaviour on IC (Fay-Stammbach et al., 2014; Valcan et al., 2017). Additional longitudinal research examining bidirectional relations between parental behaviour and children's IC across early and middle childhood is necessary to be able to ascertain whether early childhood is a sensitive period for the effects of parental behaviour on IC.

Table 4-1. *Descriptive statistics for the measures of parental discipline, inhibitory control, and covariates*

Construct	N	M	SD	Range
Harsh discipline T1 (composite score)	136	1.67	.40	1 – 3.69
Harsh discipline T2 (composite score)	108	1.63	.37	1 – 3.15
Inductive discipline T1 (composite score)	136	4.23	.51	2.43 – 5.00
Inductive discipline T2 (composite score)	108	4.23	.51	2.57 – 5.00
Go/No-Go T1 (d')	125	3.08	.67	1.38 – 3.77
Simon T1 (accuracy)	126	.85	.13	.40 – 1.00
Flanker T1 (accuracy)	128	.76	.23	.17 – 1.00
Global-Local T1 (accuracy)	132	.68	.19	.31 – 1.00
Go/No-Go T2 (d')	106	3.25	.51	1.83 – 3.77
Simon T2 (accuracy)	104	.87	.11	.45 – 1.00
Flanker T2 (accuracy)	107	.90	.14	.33 – 1.00
Global-Local T2 (accuracy)	109	.78	.17	.25 – 1.00
IC factor score T1	136	-.01	.87	-2.17 – 1.52
IC factor score T2	136	.38	.40	-.61 – 1.07
Child age (years)	136	5.47	.85	4.27 – 6.65
Child sex (% male)	136	49%		
Child ethnicity (% European Canadian)	136	62%		
Parental education (years)	136	16.85	2.38	10 – 20
Financial stress	136	1.67	.96	1 – 5

Note. IC = Inhibitory control. T1 = time 1. T2 = time 2.

Table 4-2. *Correlations between measures of parental discipline, inhibitory control, and covariates*

Measures	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.
1. Harsh discipline T1	.74**	-.14	-.22*	.02	-.16+	-.05	-.16+	-.04	-.16+	.01	.04	-.11	-.11	.05	.18*	.17*	-.05	.13
2. Harsh discipline T2	--	-.37**	-.33**	.01	-.17+	.00	-.11	-.05	-.11	.01	.01	-.09	-.10	-.09	.07	.14	-.05	.02
3. Inductive discipline T1		--	.63**	.16+	.09	.09	-.06	.11	.22*	.05	.12	.15+	.17*	.04	-.15+	.01	.17*	.00
4. Inductive discipline T2			--	.11	.11	.04	-.02	-.04	.20*	.05	.06	.11	.13	.03	-.04	.05	.09	.11
5. *Flanker* T1				--	.39**	.45**	.22*	.44**	.41**	.19*	.05	.71**	.65**	.58**	.04	.04	.14	-.12
6. *Global-Local* T1					--	.35**	.35**	.41**	.40**	.11	.02	.70**	.64**	.41**	-.04	-.04	.23**	-.18*
7. *Go/No-Go* T1						--	.36**	.39**	.47**	.36**	.07	.78**	.72**	.33**	.00	.01	.15+	-.09
8. *Simon* T1							--	.29**	.30**	.16	.35**	.59**	.53**	.28**	-.05	.11	.08	-.04
9. *Flanker* T2								--	.41**	.22*	.08	.63**	.68**	.44**	.03	.14	.13	-.11
10. *Global-Local* T2									--	.32**	.20*	.76**	.86**	.47**	-.13	-.06	.02	.00
11. *Go/No-Go* T2										--	.09	.38**	.45**	.09	-.02	-.04	.00	-.06
12. *Simon* T2											--	.17+	.24*	.11	-.08	.04	-.06	.13
13. IC factor score T1												--	.97**	.59**	-.04	.01	.19*	-.12
14. IC factor score T2													--	.58**	-.06	-.01	.15+	-.10
15. Age														--	.07	.09	.03	-.11
16. Sex															--	-.07	-.03	.15+
17. Ethnicity																--	.15+	-.04
18. Parental education																	--	-.14
19. Financial stress																		--

Note. IC = inhibitory control. T1 = time 1. T2 = time 2. $^{+}p < .10$; $^{*}p < .05$; $^{**}p < .01$.

Figure 4-1. Hypothesized model of the bidirectional relations between parental discipline and children's IC, and child age as a moderator of these relations. Dashed lines indicate paths hypothesized to be non-significant and solid lines indicate paths hypothesized to be significant. IC = inhibitory control. ΔIC = change in inhibitory control. ΔHD = change in harsh discipline. ΔID = change in inductive discipline. T1 = time 1. T2 = time 2.

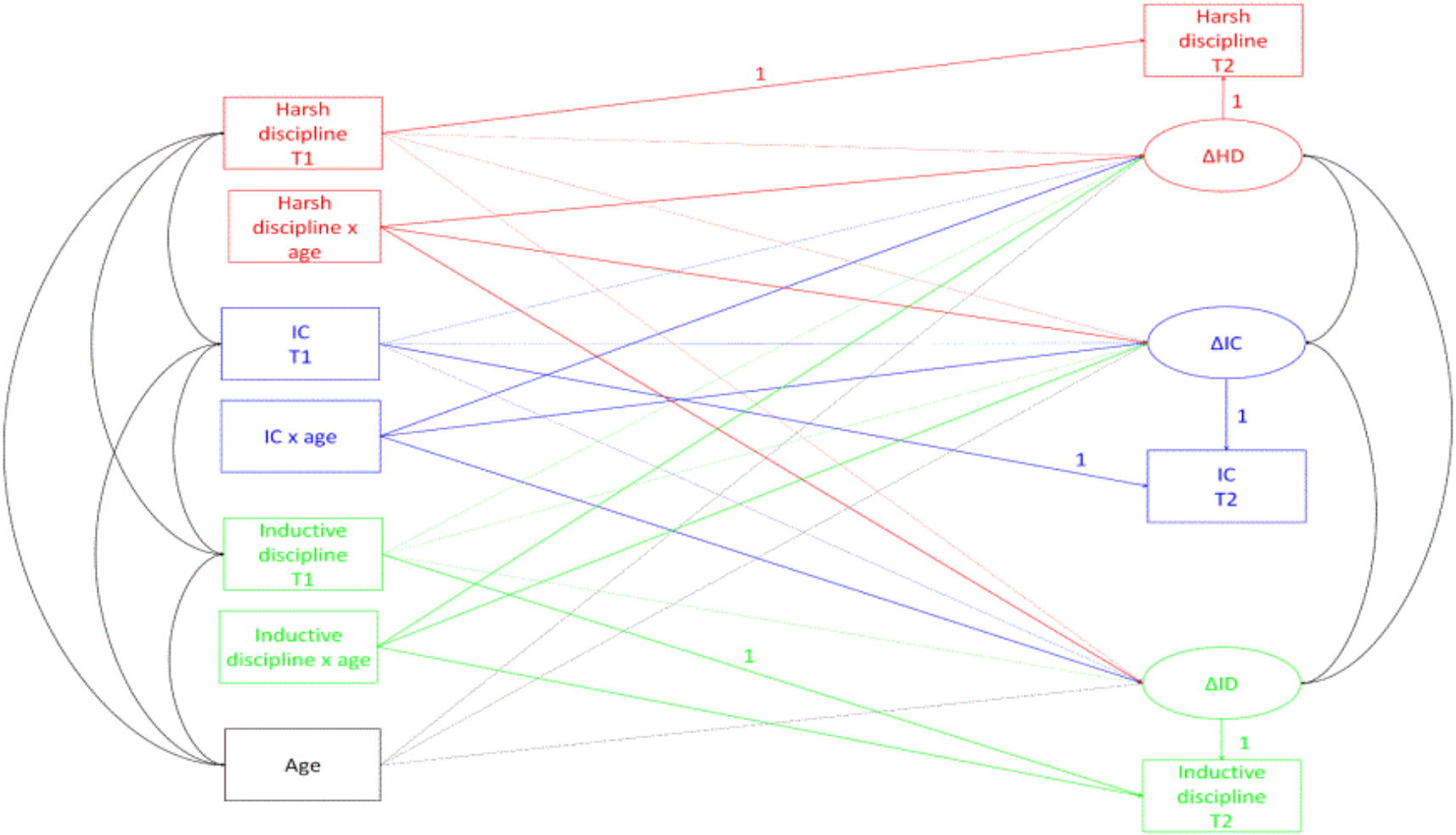


Figure 4-2. Path diagram illustrating child age as a moderator of the bidirectional relations between parents' harsh and inductive discipline and children's IC, controlling for the effects of ethnicity, parental education, and financial stress. Both unstandardized and standardized (in parentheses) parameters are presented; error variances and covariates are not shown. IC = inhibitory control. ΔIC = change in inhibitory control. ΔHD = change in harsh discipline. ΔID = change in inductive discipline. T1 = time 1. T2 = time 2. $^{+}p <$ .10; $^{*}p < .05$; $^{**}p < .01$.

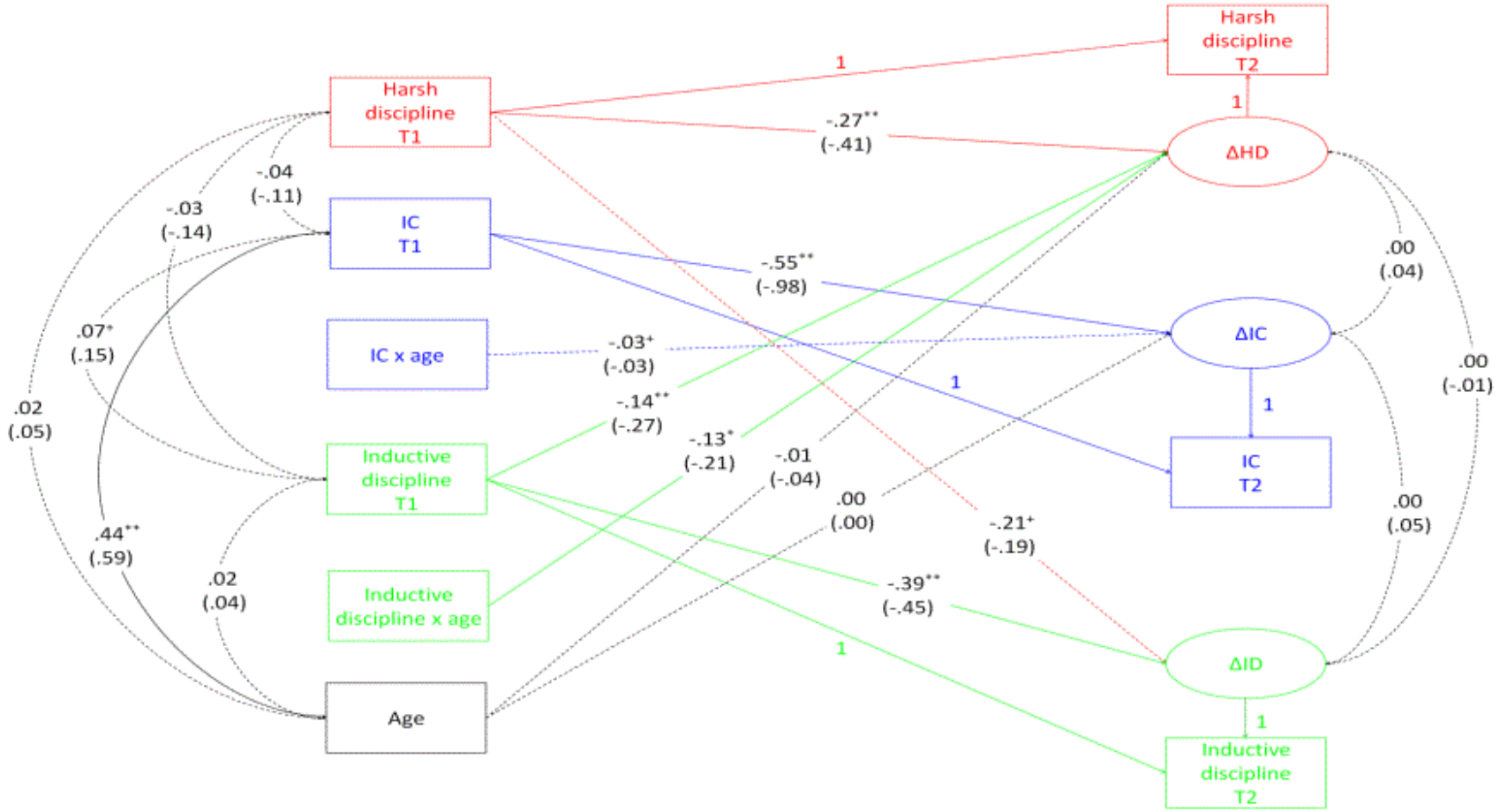


Figure 4-3. Effect of children's inhibitory control (IC) at time 1 (T1) on the predicted developmental trajectory of IC for 4-, 5-, and 6-year-olds.

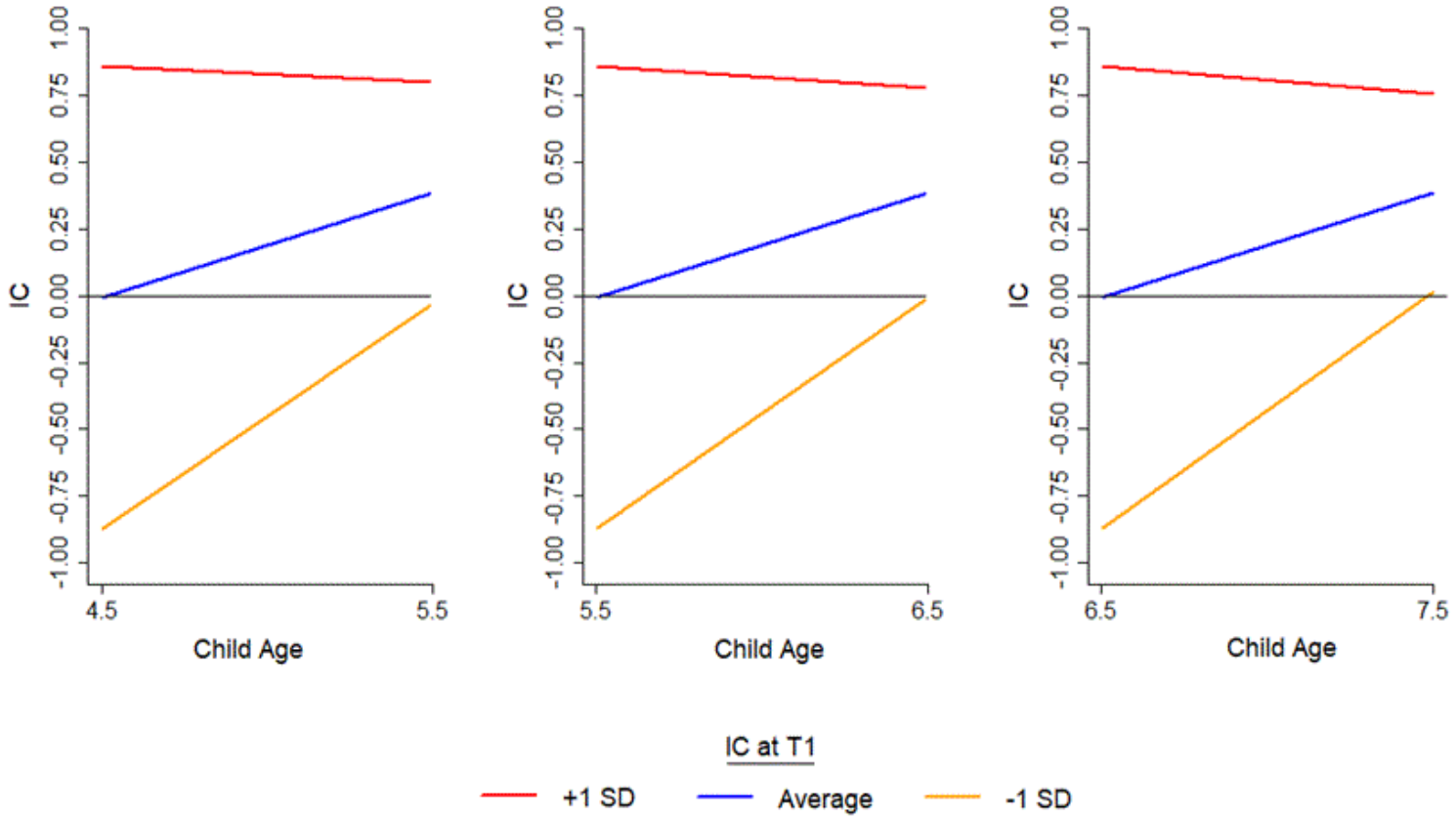
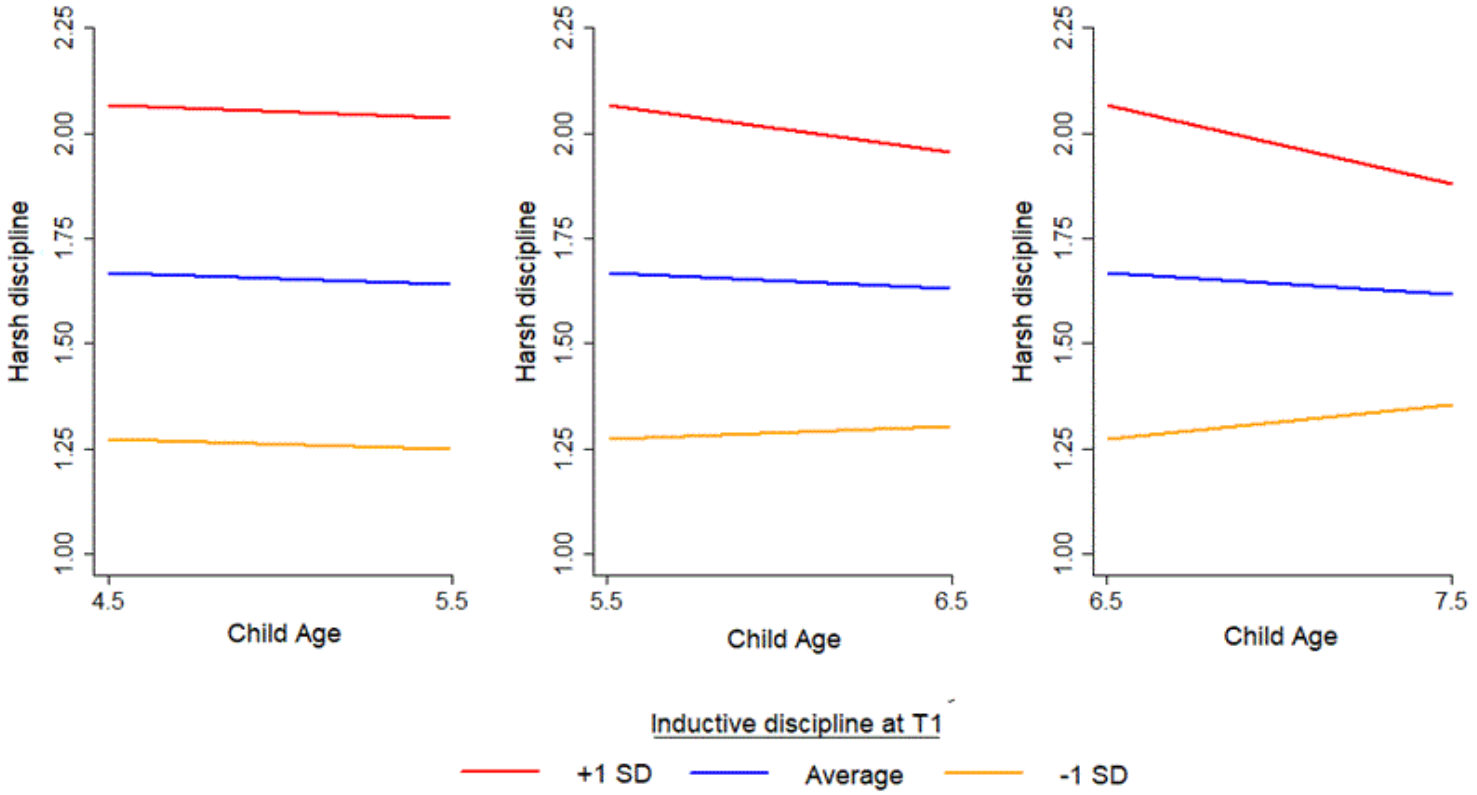


Figure 4-4. Effect of parents' inductive discipline at time 1 (T1) on the predicted trajectory of harsh discipline for parents of 4-, 5-, and 6-year-olds.



Chapter 5

General Discussion and Conclusion

General Discussion and Conclusion

Contemporary theories of child development emphasize the role of children's innate characteristics in shaping how they are impacted by their environment (Sameroff, 1975; Scarr & McCartney, 1983). While findings from empirical studies leave little doubt about the importance of parental behaviours for the development of children's executive function (EF) (Valcan et al., 2017), most research to date has not considered the role of children's characteristics in this relation. The development of a more comprehensive understanding of how parental behaviour impacts children's EF requires understanding (a) how children's characteristics interact with parental behaviour, and (b) how the parent-child dyad mutually influence each other to drive the development of EF. The objectives of this dissertation were to examine interactions and bidirectional relations between parental behaviour and children's characteristics (genotype and EF) on children's EF in early and middle childhood.

To better understand how parental behaviour and children's characteristics interact to predict children's EF, I systematically reviewed the literature on gene × parental behaviour interactions for children's EF and examined whether cumulative dopaminergic genetic risk × parental behaviour interactions were associated with EF in preschool children. Both studies found support for interactions between dopaminergic genes and parental behaviour on EF in early childhood. To better understand bidirectional relations between parental behaviour and children's characteristics, I examined whether parent's harsh and inductive discipline predicted change in children's inhibitory control, one component of EF, and if children's inhibitory control predicted changes in parental discipline during the transition to middle childhood. I did not find support for bidirectional relations between parental discipline and children's inhibitory control. The findings from this dissertation add to the literature on the role of children's characteristics in shaping the relation between parental behaviour and children's EF.

This chapter will focus on integrating findings across the three studies comprising this dissertation and situating them within the broader literature on parental behaviour and children's EF. First, I will provide a brief overview of each study, including main findings. Next, I will discuss the results of the studies in the context of existing research on gene × environment interactions, evocative effects of children's EF on parental behaviour, and parental behaviour. Finally, I will conclude by highlighting directions for future research and addressing limitations common to all three studies.

Study 1: The Effects of Candidate Gene × Parental Behaviour Interactions on Executive Function in Early Childhood: A Systematic Review

Findings from twin studies suggest that the development of EF is driven by both genetic and environmental factors (J. Li & Roberts, 2017). However, results of candidate gene studies are conflicting and many findings have failed to replicate (Klaus et al., 2019). Gene × environment interactions may help explain the variability in the results of candidate gene studies. Parents are a particularly important aspect of children's home environment in early childhood (Hughes & Devine, 2017). Candidate genes associated with EF (e.g., *DRD2* and *DRD4*) are associated with individual differences in sensitivity to parental behaviour (J. Belsky & van IJzendoorn, 2017). Therefore, genes and parental behaviour are theorized to interact to predict children's EF. Yet, the heterogeneity of findings from gene × parental behaviour interaction studies make it difficult to draw firm conclusions about the role of gene × parental behaviour interactions in the development of children's EF. The first study in this dissertation systematically reviewed the literature on gene × parental behaviour interactions on children's EF in early childhood (birth to age 5).

Of the 17 studies identified in the review, 13 (76%) reported at least one significant interaction. Overall, 24 of 51 (47%) candidate gene × parental behaviour interactions were significant. In addition, at least one significant interaction was reported in 9 of 12 (75%) studies examining catecholaminergic genes (14 of 31 (45%) interactions were significant), 4 of 6 (67%) studies examining serotonergic genes (9 of 17 (53%) interactions were significant), 0 of 1 (0%) study examining cholinergic genes (0 of 2 (0%) interactions were significant), and 1 of 1 (100%) study examining oxytocinergic genes (1 of 1 (100%) interactions were significant). Results suggest that genes and parental behaviour interact to predict EF in early childhood and that catecholaminergic and serotoninergic genotypes may be promising biological-based individual differences that confer differential sensitivity to the effects of parental behaviour on EF.

Importantly, I was not able to evaluate the strength of the evidence for gene × parental behaviour interactions on children's EF or determine whether particular genes or parental behaviours were implicated in these interactions. This was because heterogeneity in the samples; measurement of genes, parental behaviour, and EF; statistical methods; and reporting of results (e.g., effect sizes); a lack of direct replications; and limited research on some genes (e.g., *OXTR*) and parental behaviours (e.g., negative parental behaviours) meant I could not make comparisons across studies. In sum, this review pointed to several shortcomings and gaps in the literature that should be addressed in future research to strengthen the evidence for gene × parental behaviour interactions on EF.

Study 2: Interplay of Cumulative Dopaminergic Genetic Risk and Parental Behaviour is Associated with Executive Function in Early Childhood: Support for the Diathesis-Stress Model

The second study in this dissertation examined whether the interactions between cumulative dopaminergic genetic risk, operationalized as the number of risk alleles for the *DAT1*, *DRD2*, *DRD4*, and *COMT* genes, and parental behaviour (problematic discipline and responsiveness) were associated with EF (self-control and WMIC) in 3-year-olds. This study also examined whether interactions were consistent with the diathesis-stress or differential susceptibility model of gene × environment interaction. The diathesis stress model (Monroe & Simons, 1991) proposes that particular genotypes confer vulnerability to the negative effects of negative environmental factors. The differential susceptibility model (J. Belsky et al., 2009; J. Belsky & Pluess, 2009) proposes that certain genotypes confer increased sensitivity to both positive and negative environmental factors.

Consistent with the diathesis-stress model, higher problematic discipline was associated with poorer self-control for children at higher genetic risk relative to children at lower genetic risk. Models of the development of behaviour problems (e.g., Beauchaine & Gatzke-Kopp, 2012) posit that problematic discipline increases the risk for developing behaviour problems in children at-risk for reduced dopaminergic functioning. The results of this study suggest that problematic discipline may increase the risk for developing behaviour problems in children at higher genetic risk via its association with poorer self-control.

Tests for an interaction between genetic risk × problematic discipline on WMIC were inconclusive as the chi-square difference test was significant but follow-up probes of the interaction were not significant. As demonstrated in study 1, previous research has not examined

gene × parental behaviour interactions on WMIC. Additional research examining associations between gene × parental behaviour interactions and WMIC is needed.

The interaction between genetic risk and responsiveness was not associated with EF nor was there a main effect of responsiveness on EF. This result conflicts with the findings of study 1. Differences in the measurement of responsiveness across studies may have contributed to the conflicting findings. Parents' supportive presence and engagement, which were unmeasured in the present study, may drive the association between responsiveness and EF in children at higher genetic risk (Kok et al., 2013; Smith et al., 2013). It is also possible that the measure of responsive parental behaviour used in the present study was not sufficiently sensitive to variations in responsiveness that distinguish between "good enough" parents.

Study 3: Examining Bidirectional Relations Between Parental Discipline and Children's Inhibitory Control

Children with poorer EF are thought to elicit more problematic discipline techniques (e.g., harsh discipline), which are associated with poorer EF (Choe et al., 2013; Kiff et al., 2011). Children with better EF are theorized to elicit more adaptive parental discipline techniques (e.g., inductive discipline), which are associated with better EF. Further, the frequency of parental behaviours changes depending on children's developmental stage (Dallaire & Weinraub, 2005). This suggests that the behaviours important for EF development, and that are evoked by children's EF, may vary depending on children's developmental stage. Study 3 used a latent difference score model to examine bidirectional relations between parents' harsh and inductive discipline and children's inhibitory control, one component of EF, and child age as a moderator of these relations, in a year-long study covering the period from 4 to 7 years of age.

Results did not support bidirectional relations between parental discipline and children's inhibitory control or child age as a moderator of these relations. Neither harsh nor inductive discipline at the beginning of the study predicted change in children's inhibitory control. Children's inhibitory control at the beginning of the study did not predict change in parents' harsh or inductive discipline. Child age did not moderate the relations between parental discipline and children's inhibitory control.

Evidence for bidirectional relations between parental discipline and children's inhibitory control is mixed (Eisenberg et al., 2015; Klein et al., 2018). The null findings in the present study may suggest that aspects of parental discipline and inhibitory control, not assessed in the present study, are implicated in bidirectional relations between these two constructs. For example, bidirectional relations involving parental intrusion and children's self-control have been reported (Eisenberg et al., 2015; Merz et al., 2017). It is also possible that the high degree of stability in inhibitory control and parental discipline, the use of self-report parental behaviour questionnaires, or characteristics of the sample (child age and household socioeconomic status) limited my ability to detect significant relations between parental discipline and children's inhibitory control. Additional research examining bidirectional relations between parental discipline and inhibitory control during the transition to middle childhood is needed to distinguish between these possibilities.

For the parents of 5- and 6-year-olds, but not 4-year-olds, higher inductive discipline at the beginning of the study was associated with a decrease in harsh discipline over the year. This result is consistent with expectations for normative change in parents' use of discipline techniques during middle childhood, ages 6 to 12 (Collins et al., 2002). As children transition to middle childhood, physical discipline is increasingly viewed as inappropriate and children are

increasingly able to benefit from inductive discipline techniques. Therefore, parents typically transition from using harsh discipline to using inductive discipline to manage children's behaviour. The results of this study suggest that parents of 4-year-olds and 5- to 6-year-olds were at different stages in this transition.

Poorer child inhibitory control at the beginning of the study predicted faster growth in inhibitory control, and this trend was stronger for older children. This finding extends the results of a longitudinal study on the development of EF between the ages of 3 and 5, which found more positive gains in EF for children with poorer EF at age 3 (C. Blair et al., 2014). Faster growth in inhibitory control may reflect a catch-up effect among children with poorer inhibitory control. It may also suggest the presence of a ceiling effect as there was greater variability in low inhibitory control scores than high inhibitory control scores.

Gene × Parental Behaviour Interactions in the Development of EF

Studies 1 and 2 examined the associations between gene × parental behaviour interactions and children's EF in early childhood using a range of candidate genes, parental behaviours, and EF components. Results of both studies suggest gene × parental behaviour interactions are associated with the development of children's EF. Study 1 found support for interactions across diverse candidate genes, parental behaviours, and EF components. Study 2 examined the specificity of gene × parental behaviour interactions and found that the interaction between cumulative dopaminergic genetic risk and problematic discipline, but not responsiveness, was uniquely associated with self-control, the motivational component of EF. The results of these studies are consistent with the tenants of transactional models of child development which argue that children's innate characteristics modify the impact of the environment on developmental outcomes (Sameroff, 1975). Nonetheless, questions remain regarding which genes, parental

behaviours, and EF components are implicated in gene × parental behaviour interactions. The results of studies 1 and 2 may be able to shed some light on the genes and components of EF implicated in gene × parental behaviour interactions. Questions regarding the role of specific parental behaviours in the development of EF remain unanswered and will be discussed in the limitations section of this chapter.

Findings from studies 1 and 2 suggest that dopamine genotype may confer differential sensitivity to environmental factors. This is consistent with meta-analyses of genes involved in differential susceptibility to environmental influences (Bakermans-Kranenburg & van IJzendoorn, 2011; van IJzendoorn & Bakermans-Kranenburg, 2014), and with the role of dopamine in processing and influencing sensitivity to rewards and punishers in the environment (Matthys et al., 2013). Study 1 also identified genes involved in the serotonergic and cholinergic systems as possible moderators of the relation between parental behaviour and EF. It is likely that the cumulative effect of multiple genes involved in neurotransmitter systems that impact sensitivity to rewards and punishers, such as serotonin (Pearson, Mcgeary, & Beevers, 2014), also moderate the relation between parental behaviour and EF, particularly self-control. Future research should examine this possibility.

Importantly, while there was broad support for genes involved in dopaminergic functioning conferring increased sensitivity to the effects of parental behaviour, the risk alleles associated with increased sensitivity differed across studies. In study 2 children at higher dopaminergic genetic risk (as determined by the presence of the 10-repeat allele of *DAT1*, A1 allele of *DRD2*, 7-repeat allele of *DRD4*, and Val allele of *COMT*) had poorer self-control when problematic discipline was higher relative to children at lower genetic risk. Conversely, in study 1, Kok et al. (2013) found that parental behaviour was only associated with EF for children with

the Met allele of *COMT.* Similarly, Sulik et al. (2015) found that, for boys, Met was associated with increased sensitivity to the effects of parental behaviour on EF. For girls, Val was associated with increased sensitivity to parental behaviour. These differences may suggest that there are developmental or sex differences in how dopaminergic genes confer differential sensitivity to the environment.

The effects of candidate genes may be emergent because of the protracted development of neurotransmitter systems (Hariri & Holmes, 2006; Tunbridge et al., 2006). For example, the Val allele of *COMT* is thought to increase sensitivity to the environment because it is associated with reduced dopamine availability (J. Belsky & Pluess, 2009). It is also associated with poorer EF in adults for the same reason (Tunbridge et al., 2006). Dopamine levels increase through childhood, peak at puberty, and decline into adulthood (Dumontheil et al., 2011). A cohort sequential study of 6- to 20-year-olds found that Val-Val homozygotes had better working memory than Met-Met homozygotes before the age of 10 (Dumontheil et al., 2011), suggesting that the relation between *COMT* alleles and EF changes with the development of the dopaminergic system. Alleles of *COMT* that reduce dopamine availability may reduce sensitivity to the environment and be associated with better EF at stages in the development of the dopaminergic system where dopamine levels are higher than in adulthood. The results of Kok et al. (2013) and a study of *COMT* as a moderator of the relation between socioeconomic status and EF in early childhood (C. Blair et al., 2015) are consistent with developmental differences in dopamine genotype as a moderator of relations between environmental factors and children's EF.

Second, Sulik et al. (2015) found that sex moderated the interaction between *COMT* and parenting quality on EF. The sample size in study 2 was not large enough to be able to examine sex differences in the interaction between dopamine genotype and parental behaviour on EF, and

the majorities of studies reviewed in study 1 did not test this possibility. There are sex differences in the dopaminergic system. Males have lower basal dopamine levels than females (Laakso, Vilkman, Haaparanta, & Solin, 2002), but increased dopamine response to agonists like alcohol (Urban et al., 2010). Further, estrogen modulates dopaminergic functioning (Harrison & Tunbridge, 2008; Petersen & London, 2018). For example, it inhibits COMT enzyme activity (Harrison & Tunbridge, 2008). These sex differences in the dopaminergic system have lead researchers to suggest that dopaminergic candidate genes may differentially affect males and females (Petersen & London, 2018) in a manner consistent with the results of Sulik et al. (2015).

The results of studies 1 and 2 also shed some light on the question of which EF components are most strongly associated with gene × parental behaviour interactions. Study 2 found that the interaction between dopamine genotype and problematic discipline was associated with self-control. Whether this interaction held for WMIC was inconclusive. Results of study 1 were broadly consistent with this pattern of results: 6 of 8 studies examining interactions between dopamine genes and parental behaviour on a motivational component of EF (e.g., compliance) found a significant interaction, compared to 1 of 3 studies examining a cognitive component of EF (e.g., executive attention). These results are surprising given that dopamine is involved in both self-control and WMIC (Berridge, 2007; Robbins & Arnsten, 2009). As mentioned in chapter 3, this pattern of results may be due to children's tendency to respond impulsively in early childhood (Carlson, 2005). These results may also suggest that parental behaviour or the social context are better predictors of performance on motivational EF tasks than cognitive EF tasks. For example, the behaviour of the experimenter predicts children's performance on the *Marshmallow Test* (Kidd, Palmeri, & Aslin, 2013; Michaelson & Munakata, 2016). If the experimenter has a track record of not returning with items as promised, children

are four times faster to eat the marshmallow (mean wait time of 3 minutes) than when the experimenter is reliable in returning with items as promised (mean wait time of 12 minutes) (Kidd et al., 2013). There is some suggestion that children's expectations regarding their parent's behaviour may also impact performance on self-control tasks in a similar fashion (Jacobsen, Huss, Fendrich, Kruesi, & Ziegenhain, 1997; Mittal, Russell, Britner, & Peake, 2013). Conversely, successful completion of EF tasks in situations of cognitive load is not dependent on the experimenter's behaviour. These tasks may be better indices of children's ability to initiate and sustain goal-directed behaviours rather than regulate performance in the presence of external pressure (e.g., an experimenter) (Landry, Smith, Swank, & Miller-Loncar, 2000).

Evocative Effects of Children's EF on Parental Behaviour

Transactional theories of child development also propose that children's characteristics evoke particular behaviours from the environment (e.g., Scarr & McCartney, 1983). Study 3 examined bidirectional relations between parental discipline and children's inhibitory control. Children's inhibitory control did not predict change in parental discipline over the year. Support for an evocative effect of children's inhibitory control, and EF more broadly, on parental behaviour is mixed (C. Blair et al., 2014; Klein et al., 2018; Merz et al., 2017). This may suggest that the relation between children's EF and parental behaviour is moderated by characteristics of the child or parent.

Child sex may moderate the relation between children's EF and parental behaviour. Boys have poorer EF and more behaviour problems than girls (for meta-analyses see Else-Quest, Hyde, Goldsmith, & van Hulle, 2006; Gershon, 2002). Boys may elicit more negative and less responsive behaviours from their caregivers because of their poorer EF (Mileva-Seitz et al., 2015). In support of this assertion, Pener-Tessler et al. (2013) found that child sex moderated the

relation between children's self-control and repsonsive parenting. For boys, but not girls, poorer self-control predicted less responsive parenting.

Parents' sensitivity to the environment may also moderate the relation between children's EF and parental behaviour. Parents are more likely to use harsh discipline when they are in a negative mood or experiencing stress (Critchley & Sanson, 2006). Emerging research suggests parents' physiological reactivity, a measure of stress regulation, moderates the relation between children's behaviour (e.g., compliance and crying) and parents use of physical discipline, such that only more physiological reactive parents respond to children's behaviour with harsh discipline (Norman Wells, Skowron, Scholtes, & Degarmo, 2019; Skowron et al., 2011). This is consistent with the finding that parents who are better able to regulate their emotions are less likely to use harsh discipline and more likely to be warm and responsive (Crandall, Deater-Deckard, & Riley, 2015). This may suggest that the behaviours children evoke from their caregivers are partially dependent on the caregiver's characteristics, such as the ability to regulate negative affect.

Parental Behaviour and EF in Early and Middle Childhood

In study 3, neither parents' harsh nor inductive discipline predicted change in children's inhibitory control. Previous work with the MIDS cohort, used in study 2, also found inconsistent and contradictory main effects of harsh parenting, problematic discipline, and responsiveness on children's WMIC and self-control (Vrantsidis, 2016; Vrantsidis et al., 2020). Consistent with the results of studies 1 and 2, this may suggest that the relation between parental discipline and children's EF is moderated by children's characteristics. In addition to child genotype, functioning of the HPA axis (Xing, Yin, & Wang, 2019), physiological reactivity (Obradović, Portilla, & Ballard, 2016), temperament (Rochette & Bernier, 2016), and child sex (Mileva-Seitz

et al., 2015; Vrantsidis, Volk, Wakschlag, Espy, & Wiebe, under review) moderate the relation between parental behaviour and children's EF. The differential susceptibility theory (J. Belsky & Pluess, 2009) and biological sensitivity to context theory (Boyce & Ellis, 2005) argue that some children are more sensitive to environmental factors based on their individual characteristics. In particular, children with heightened stress reactivity are argued to be more sensitive to environmental factors (Boyce & Ellis, 2005). Functioning of the HPA axis and physiological reactivity directly index individual's stress reactivity. Temperament (Calkins, Dedmon, Gill, Lomax, & Johnson, 2002), sex (Elsmén, Steen, & Hellström-Westas, 2004), and dopamine genotype (Propper et al., 2008) are associated with differences in physiological and stress reactivity. These moderators provide support for the assertion that heightened stress reactivity increases sensitivity to the environment, which, in turn, is associated with children's EF (C. Blair & Raver, 2012). This has important implications for our understanding of the development of EF.

Boyce and Ellis (2005) argue that developmental plasticity in the stress response system allows early experience to alter the functioning of these systems to adaptively match children's environment, with poorer regulation of stress physiology associated with poorer EF (C. Blair & Raver, 2012). For example, infants exposed to harsh discipline have higher cortisol levels and poorer regulation of cortisol in novel situations than infants who are not exposed to harsh discipline (Bugental et al., 2003; Spangler & Grossmann, 1993). This suggests that children's stress physiology may moderate the relation between parental behaviour and children's EF in a dynamic, rather than static, fashion. Early differences in caregiver behaviours (e.g., responsiveness versus harsh discipline) may alter children's stress reactivity, in turn impacting how children respond to the environment and their EF. Thus, parental behaviour in early and

middle childhood may have long-term effects on children's EF via the canalization of the neurobiological systems underlying EF (C. Blair & Raver, 2012).

Likewise, parenting interventions to improve EF may be most impactful and have longer-lasting effects if carried out in early childhood because this is when plasticity in the neurobiological systems underlying EF is greatest (Boyce & Ellis, 2005). For example, an intervention to improve responsive behaviours in foster parents found that maltreated infants whose foster parents completed the intervention had cortisol levels during the Strange Situation comparable to those of non-maltreated infants (Dozier, Peloso, Lewis, Laurenceau, & Levine, 2008). This suggests that early childhood interventions can ameliorate maladaptive changes to HPA axis functioning. It remains to be seen if these changes in stress physiology are related to gains in EF.

Future Directions for Research

The findings of this dissertation suggest several directions for future research. First, all three studies in this dissertation primarily focused on the relation between maternal behaviour and children's EF. This was not intentional. The search strategy in study 1 was designed to capture studies on maternal and paternal behaviour. However, no studies on gene × paternal behaviour interactions were identified, and studies of primary caregivers typically studied mothers (e.g., study 3). Maternal and paternal behaviour may independently and differentially relate to children's EF (Baptista et al., 2017; Lucassen et al., 2015). For example, fathers' and mothers' use of harsh and inductive discipline have been differentially linked to inhibitory control in 3-year-olds (Karreman et al., 2008). Fathers' use of harsh discipline, but not inductive discipline, was associated with poorer inhibitory control, but the opposite pattern of results was found for mothers' use of harsh and inductive discipline. Furthermore, paternal sensitivity may

protect against the negative effect of low maternal sensitivity on children's EF (Feldman, 2012). A full understanding of the impact of parental behaviour on children's EF involves understanding the contribution of paternal behaviour to this relation.

Second, more research with ethnically diverse samples will add to our understanding of the universality of the relation between parental behaviour and children's EF. Study 1 found some evidence for ethnic differences in gene × environment interactions on EF. Further, ethnicity and parental behaviour were correlated in study 2, and the relation between inductive discipline and harsh discipline reported in study 3 may differ depending on parents' ethnicity (Gu & Kwok, 2020). It was not possible to examine ethnic differences in studies 2 or 3 because of the limited number of non-European American or Canadian families in the studies. Nonetheless, ethnicity moderates the relation between physical discipline and externalizing behaviour problems, such that there is some evidence that physical discipline is associated with behaviour problems for European American children but not African American children (Deater-Deckard & Dodge, 1997). Among African American families living in low socioeconomic status households, parental behaviour that is high in physical discipline and warmth is a protective factor for the development of self-regulation, a construct that includes EF (Brody & Flor, 1998). Similar to African American parents, Latinx American, and Chinese American parents also tend to be higher in control than European American parents and have higher expectations for child compliance (Chao, 1994; Dearing, 2004; Rodriguez, Donovick, & Crowley, 2009). This may suggest that parental behaviours, particularly discipline, differentially relate to children's EF depending on the family's ethnicity.

Third, more work is needed examining how parental behaviour is related to children's EF at different developmental stages. Is early childhood a critical period for the effects of parental

behaviour on EF (Valcan et al., 2017)? Do different parental behaviours support the development of EF at different stages of children's development (C. Blair et al., 2014)? For example, children are supposed to be better able to benefit from inductive discipline between the ages of 6 and 12 (Collins et al., 2002). In study 3, parents' inductive discipline did not predict children's inhibitory control. However, inductive discipline has been linked to better inhibitory control in 2- to 5-year-olds (Karreman et al., 2008; Klein et al., 2018). Therefore, it remains to be seen whether inductive discipline is particularly beneficial for children in middle childhood versus early childhood. Longitudinal studies examining parental behaviour at multiple timepoints, starting in early childhood, as predictors of children's EF are necessary to answer questions about the timing of parental effects. Likewise, more longitudinal studies examining the relation between parental behaviour and EF at multiple timepoints, particularly in middle childhood, are necessary to be able to answer questions about how parental behaviour is associated with EF at different stages in the development of EF.

Fourth, future research should examine the role of passive gene × environment correlations in the relation between parental behaviour and children's EF. A passive gene × environment correlation occurs when parents that are genetically related to the child provide an environment that is correlated with the genotype of the child (Scarr & McCartney, 1983). Passive gene × environment correlations were not examined in the studies reviewed in study 1, and it was not possible to test for these in studies 2 or 3 as we did not collect information on parents' EF or genotype. Emerging research suggests parent's behaviour partially mediates the relation between parents' EF and children's EF (Deater-Deckard & Bell, 2017; Distefano, Galinsky, McClelland, Zelazo, & Carlson, 2018). Further, *DAT1* and *DRD2* are linked to individual differences in both parent's behaviour and children's EF (Mileva-Seitz, Bakermans-Kranenburg,

& van IJzendoorn, 2016). The identification of passive gene × environment correlations in the relation between parental behaviour and children's EF may help researchers identify which parental behaviours associated with children's EF are most impacted by the environment and, therefore, most amenable to interventions.

Limitations

Findings from this dissertation should be interpreted in light of two limitations. First, this dissertation hoped to answer questions about which parental behaviours are important for the development of children's EF. All three studies used measures of parental behaviour that were heterogenous, making it difficult to ascertain which parental behaviours are important for the development of EF. The majority of studies reviewed in study 1 included both positive (e.g., sensitivity) and negative (e.g., intrusiveness) behaviours in their measure of parental behaviour (e.g., parenting quality; Sulik et al., 2015). Therefore, it was not possible to determine whether positive or negative parental behaviours interacted with candidate genes. Studies 2 and 3 benefited from the use of separate measures of positive and negative parental behaviours. However, except for the measure of inductive discipline, the measures in these studies were still heterogenous. For example, the measure of problematic discipline used in study 2 included physical discipline, negative affect, and overcontrolling behaviour. Some parents high in problematic discipline are high in negative affect, others are over controlling, and some are warm but use physical discipline as their primary discipline technique (Gershoff, 2002; McFadden & Tamis-Lemonda, 2013). This implies that the construct of problematic discipline can be broken down further. It was not possible to do this in study 2 because each aspect of problematic discipline was only assessed using one or two items. Future research examining specific parental

behaviours will add to researchers' understanding of which behaviours are associated with children's EF and the mechanisms involved in these relations.

Second, study 2 followed Roisman et al.'s (2012) guidelines for distinguishing between the diathesis-stress and differential susceptibility models of gene × environment interaction. Likewise, in study 1, 7 of 9 studies that probed an interaction at least partially followed Roisman et al.'s (2012) guidelines. It is important to note that there is a debate in the literature regarding how to best test competing models of gene × environmental interaction (Del Giudice, 2017; Jolicoeur-Martineau, Belsky, et al., 2019). Roisman et al.'s (2012) guidelines are considered exploratory as the guidelines propose methods for probing statistically significant interactions. An alternative method to testing competing models of gene × environment interaction is the competitive-confirmatory approach (J. Belsky et al., 2013; J. Belsky & Widaman, 2018; Widaman et al., 2012). The competitive-confirmatory approach involves re-parameterizing the multiple regression equation to estimate the value of the environmental variable at which the slopes of the genetic risk groups cross-over. If the cross-over point falls within the range of observed data, models consistent with the diathesis-stress and differential susceptibility are estimated and compared to determine which model best fits the data. Re-parameterizing the equation for the interaction between cumulative dopaminergic genetic risk and problematic discipline in study 2 yields a cross-over point of -1.25. This value is consistent with the diathesis-stress model as the cross-over point falls outside the observed range of problematic discipline (*range* = 0 – 18). Roisman et al.'s (2012) guidelines are the most widely used approach and a more conservative test of competing models of gene × environment interaction (Del Giudice, 2017). However, compared to the competitive-confirmatory approach, they have less power to accurately quantify the form of an interaction (Jolicoeur-Martineau, Belsky, et al., 2019). The

major draw back of the competitive-confirmatory approach is that it does not formally test the significance of the interaction term (J. Belsky & Widaman, 2018).

Conclusion

This dissertation examined whether parent's behaviour and children's characteristics were mutually associated with the development of children's EF in early childhood and the transition to middle childhood. Child dopaminergic genotype × parental behaviour interactions were associated with children's EF between 6 and 60 months of age. There were no significant bidirectional relations between parental discipline and children's inhibitory control between the ages of 4 and 7. These results suggest that the relation between parental behaviour and children's EF is not unidirectional. Children's characteristics, namely genotype, moderated the association between parent's behaviour and EF, such that the association was stronger for children at genetic risk for greater sensitivity to environmental factors. Understanding how children's individual characteristics shape how they are impacted by parental behaviour will allow for a more nuanced understanding of the association between parental behaviour and children's developing EF. This will hopefully allow for a more personalized approach towards parenting interventions aimed at improving children's EF. For example, researchers and practitioners may be able to determine which interventions are most beneficial for children based on their characteristics, or they may be able to tailor interventions to children's characteristics to be maximally beneficial.

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Appendix A

Search Strategies for Chapter 2

Legend

Symbol	Definition
?, $	Symbol for any character or no character. E.g., behavio?r returns behaviour or behaviour
*(at termination of word root)	Truncation symbol. Indicates 0 to many characters
*(before MeSH term)	Indicates that the subject term has been focused, returning only records with a higher relevance weighting.
.ab.	In Ovid databases searches in the abstract field
.kw., .kf., id.	In Ovid databases searches in the keyword field
.ti.	In Ovid databases limits search to title field
/ (at end of term)	In Ovid databases indicates a Subject heading
/ge [Genetics]	In Ovid Medline indicates a MeSH subheading added to the search term; in this case restricting to records with the Genetics subheading attached
adj2, NEAR/2, W/2	Adjacency operators. Defines the number of words that can occur between two search terms. E.g., adj2 allows two terms to occur within 2 words of each other
exp	In Ovid databases the explode function searches for the term being exploded and all narrower terms indexed under the term being exploded
TITLE-ABS	In SCOPUS database limits search to title and abstract fields
TS	In Web of Science databases limits search to topic field
GN	In Web of Science databases limits search to gene name data field

OVID PsycINFO 1806 to August Week 1 2019 (*N* = 504)

1. exp *Genotypes/ or exp *Polymorphism/ or exp *Genes/ or exp *Alleles/ or (gene or genes or genotype* or polymorphism* or allele* or haplotype*).ti,ab,id.
2. adoptive parents/ or attachment behaviour/ or authoritarian parenting/ or authoritative parenting/ or exp child discipline/ or exp childrearing practices/ or coparenting/ or family relations/ or exp fathers/ or foster parents/ or homosexual parents/ or exp mothers/ or exp parent child communication/ or exp parent child relations/ or parental characteristics/ or parental expectations/ or parental investment/ or parental involvement/ or parenting/ or parenting skills/ or exp parenting style/ or parents/ or permissive parenting/ or exp single parents/ or exp family conflict/ or child care/
3. (parent* or maternal* or paternal* or mother* or father* or caregiver* or attachment or childrearing or child-rearing or child care).ti,ab,id.
4. 2 or 3
5. exp approach behaviour/ or approach avoidance/ or decision making/ or choice behaviour/ or exp self-control/ or exp *Executive Function/ or problem solving/ or prefrontal cortex/ or frontal lobe/ or *response inhibition/ or frustration/ or Emotional Regulation/ or Emotional Control/ or *attention/ or selective attention/ or self-regulation/ or Cognitive Flexibility/ or Impulsiveness/ or risk taking/ or gambling/ or reinforcement delay/ or monetary rewards/ or monetary incentives/ or compliance/
6. (cognitive control or cognitive flexibility or executive control or executive function* or inhibitory control or proactive control or problem solving or reactive control or response inhibition or set shifting or task switching or (memory adj4 updating) or working memory).ti,ab,id.
7. (affect regulat* or affect control* or affective regulat* or affective control* or approach behavio?r or approach avoidance or decision making or delay aversion or (delay* adj2 discount*) or (delay* adj2 gratification) or inhibitory control or emotion* regulat* or emotion* control* or frustration or gambling or impulse control or impulsiv* or reversal learning or reward discounting or reward processing or reward sensitivity or risk taking or time discounting or inter-temporal choice or intertemporal choice or delayed reward or monetary incentive delay or monetary choice task or compliance).ti,ab,id.
8. (frontal cortex or frontal lobe or prefrontal cortex or self control or self regulat* or (attention* adj2 shift*) or executive attention or selective attention or attention or temperament).ti,ab,id.
9. or/5-8
10. 1 and 4 and 9
11. limit 10 to animal
12. limit 11 to human
13. 11 not 12
14. 10 not 13
15. limit 14 to (100 childhood or 120 neonatal or 140 infancy <2 to 23 mo> or 160 preschool age)
16. (preschool* or infant* or toddler* or baby or babies or newborn* or kindergarten* or kindergarden* or early child* or young child*).ti,ab,id.
17. (("1* mo*" or "2* mo*" or "3* mo*" or "4* mo*" or "5* mo*" or "6* mo*") adj old*).ti,ab,id.
18. (("1 y*" or "2 y*" or "3 y*" or "4 y*" or "5 y*") adj old*).ti,ab,id.

19. or/16-18
20. 14 and 19
21. 15 or 20
22. child*.ti,ab,id.
23. 14 and 22
24. limit 23 to online first publication
25. 21 or 24
26. limit 25 to all journals

Ovid MEDLINE(R) ALL 1946 to August 07, 2019 (*N* = 1369)

1. exp *Receptors, Adrenergic/ge [Genetics]
2. exp *Apolipoproteins E/ge [Genetics]
3. exp *Brain-Derived Neurotrophic Factor/ge [Genetics]
4. exp *Receptors, Cholinergic/ge [Genetics]
5. *Catechol O-Methyltransferase/ge [Genetics]
6. exp *Cell Adhesion Molecules/ge [Genetics]
7. exp *Dopa Decarboxylase/ge [Genetics]
8. exp *Receptors, Dopamine/ge [Genetics]
9. exp *Dopamine Plasma Membrane Transport Proteins/ge [Genetics]
10. *Monoamine Oxidase/ge [Genetics]
11. *Receptors, Nicotinic/ge [Genetics]
12. *Norepinephrine Plasma Membrane Transport Proteins/ge [Genetics]
13. *Receptors, Oxytocin/ge [Genetics]
14. exp *Receptors, Serotonin/ge [Genetics]
15. *Serotonin Plasma Membrane Transport Proteins/ge [Genetics]
16. exp *Synaptosomal-Associated Protein 25/ge [Genetics]
17. *Solute Carrier Proteins/ge [Genetics]
18. exp *Tryptophan Hydroxylase/ge [Genetics]
19. exp *Dopamine/ge [Genetics]
20. exp *Norepinephrine/ge [Genetics]
21. exp *Tryptophan/ge [Genetics]
22. exp *Serotonin/ge [Genetics]
23. exp *Choline/ge [Genetics]
24. exp *Acetylcholine/ge [Genetics]
25. exp *Oxytocin/ge [Genetics]
26. exp *Epinephrine/ge [Genetics]
27. exp *Vasopressins/ge [Genetics]
28. exp *Receptors, Vasopressin/ge [Genetics]
29. or/1-28
30. exp *Genotype/ or exp *Polymorphism, Genetic/ or exp *Genes/ or exp *Alleles/ or (gene or genes or genotype* or polymorphism* or allele* or haplotype*).ti,ab,kf.
31. 29 or 30
32. exp child rearing/ or exp family characteristics/ or exp family relations/ or exp nuclear family/ or single-parent family/ or child care/
33. (parent* or maternal* or paternal* or mother* or father* or caregiver* or attachment or childrearing or child-rearing or child care).ti,ab,kf.
34. 32 or 33
35. exp "Reinforcement (Psychology)"/ or decision making/ or choice behaviour/ or delay discounting/ or problem solving/ or frustration/ or frontal lobe/ or prefrontal cortex/ or *"Executive Function"/ or Reversal Learning/ or exp *"Inhibition (Psychology)"/ or *"Attention"/ or exp Temperament/ or exp Self-Control/ or Impulsive Behaviour/ or exp Risk Taking/
36. (cognitive control or cognitive flexibility or executive control or executive function* or inhibitory control or proactive control or problem solving or reactive control or response

inhibition or set shifting or task switching or (memory adj4 updating) or working memory).ti,ab,kf.
37. (affect regulat* or affect control* or affective regulat* or affective control* or approach behavio?r or approach avoidance or decision making or delay aversion or (delay* adj2 discount*) or (delay* adj2 gratification) or inhibitory control or emotion* regulat* or emotion* control* or frustration or gambling or impulse control or impulsiv* or reversal learning or reward discounting or reward processing or reward sensitivity or risk taking or time discounting or inter-temporal choice or intertemporal choice or delayed reward or monetary incentive delay or monetary choice task or compliance).ti,ab,kf.
38. (frontal cortex or frontal lobe or prefrontal cortex or self control or self regulat* or (attention* adj2 shift*) or executive attention or selective attention or attention or temperament).ti,ab,kf.
39. or/35-38
40. 31 and 34 and 39
41. limit 40 to animals
42. limit 41 to humans
43. 41 not 42
44. 40 not 43
45. (("1* mo*" or "2* mo*" or "3* mo*" or "4* mo*" or "5* mo*" or "6* mo*") adj old*).ti,ab,kf.
46. (("1 y*" or "2 y*" or "3 y*" or "4 y*" or "5 y*") adj old*).ti,ab,kf.
47. (preschool* or infant* or toddler* or baby or babies or newborn* or kindergarten* or kindergarden* or early child* or young child*).ti,ab,kf.
48. or/45-47
49. 44 and 48
50. limit 40 to ("all child (0 to 18 years)" or "newborn infant (birth to 1 month)" or "infant (1 to 23 months)" or "preschool child (2 to 5 years)")
51. 49 or 50
52. child*.ti,ab,kf.
53. 44 and 52
54. limit 53 to ("in data review" or in process or publisher or "pubmed not medline")
55. 51 or 54

OVID Embase 1974 to 2019 August 07 (*N* = 1088)

1. exp *genotype/ or exp *genetic polymorphism/ or exp *gene/ or exp *allele/ or (gene or genes or genotype* or polymorphism* or allele* or haplotype*).ti,ab,kw.
2. exp child parent relation/ or exp domestic violence/ or exp dysfunctional family/ or exp family attitude/ or exp family conflict/ or exp family life/ or family relation/ or exp maternal deprivation/ or nuclear family/ or exp parent/ or exp parental behaviour/ or exp parenthood/ or exp single-parent family/ or child care/ or child rearing/ or infant care/
3. (parent* or maternal* or paternal* or mother* or father* or caregiver* or attachment or childrearing or child-rearing or child care).ti,ab,kw.
4. 2 or 3
5. decision making/ or delay discounting/ or problem solving/ or exp reinforcement/ or high risk behaviour/ or gambling/ or frontal lobe/ or frontal cortex/ or exp prefrontal cortex/ or *"executive function"/ or reversal learning/ or exp *"Inhibition (Psychology)"/ or frustration/ or working memory/ or self control/ or *"attention"/ or selective attention/ or exp temperament/
6. (cognitive control or cognitive flexibility or executive control or executive function* or inhibitory control or proactive control or problem solving or reactive control or response inhibition or set shifting or task switching or (memory adj4 updating) or working memory).ti,ab,kw.
7. (affect regulat* or affect control* or affective regulat* or affective control* approach behavio?r or approach avoidance or decision making or delay aversion or (delay* adj2 discount*) or (delay* adj2 gratification) or inhibitory control or emotion* regulat* or emotion* control* or frustration or gambling or impulse control or impulsiv* or reversal learning or reward discounting or reward processing or reward sensitivity or risk taking or time discounting or inter-temporal choice or intertemporal choice or delayed reward or monetary incentive delay or monetary choice task or compliance).ti,ab,kw.
8. (frontal cortex or frontal lobe or prefrontal cortex or self control or self regulat* or (attention* adj2 shift*) or executive attention or selective attention or attention or temperament).ti,ab,kw.
9. or/5-8
10. 1 and 4 and 9
11. limit 10 to animals
12. limit 11 to humans
13. 11 not 12
14. 10 not 13
15. limit 14 to (infant <to one year> or preschool child <1 to 6 years> or child <unspecified age>)
16. (preschool* or infant* or toddler* or baby or babies or newborn* or kindergarten* or kindergarden* or early child* or young child*).ti,ab,kw.
17. (("1* mo*" or "2* mo*" or "3* mo*" or "4* mo*" or "5* mo*" or "6* mo*") adj old*).ti,ab,kw.
18. (("1 y*" or "2 y*" or "3 y*" or "4 y*" or "5 y*") adj old*).ti,ab,kw.
19. or/16-18
20. 14 and 19
21. 15 or 20
22. child*.ti,ab,kw.
23. 14 and 22

24. limit 23 to in-process status
25. limit 23 to article-in-press status
26. 21 or 24 or 25
27. limit 26 to (conference abstract or "conference review")
28. 26 not 27

Web of Science BIOSIS Citation Index All Years (1926-2019) (*N* = 433)

#1 GN=(5-HT* or 5HT* or ADRA1* or ADRA2* or ANKK1 or APOE or BDNF or CADM2 or CHRNA or cht or COMT or DAT1 or DbH or DDC or DRD* or HTR1* or HTR2* or MAO* or NAT1 or NET1 or OXTR or SERT or SERT1 or SLC5A* or SLC6A* or SNAP 25 or TPH* or dopamine* or tryptophan or seroton* or choline* or acetylcholine* or oxytocin or norepinephrine or epinephrine or adrenalin or noradrenaline or "Apolipoprotein E*" or "Brain-derived neurotrophic factor" or "catechol o-Methyltransferase" or "cell adhesion molecule" or "Choline* NEAR/4 transport*" or "acetylcholine* NEAR/4 transport*" or "Choline* NEAR/4 receptor*" or "acetylcholine* NEAR/4 receptor*" or "DOPA decarboxylase" or "dopamine* NEAR/4 receptor*" or "dopamine* NEAR/4 transport*" or monoamine* or "monoamine oxidase*" or "monoamine NEAR/4 receptor*" or "monoamine NEAR/4 transport*" or "Nicotinic NEAR/4 receptor*" or "norepinephrine NEAR/4 transport*" or "epinephrine NEAR/4 transport*" or "adrenal* NEAR/4 transport*" or "noradrenal* NEAR/4 transport*" or "norepinephrine NEAR/4 receptor*" or "epinephrine NEAR/4 receptor*" or "adrenal* NEAR/4 receptor*" or "noradrenal* NEAR/4 receptor*" or "adrenergic NEAR/4 receptor*" or "noradrenergic NEAR/4 receptor*" or "oxytocin NEAR/4 receptor*" or "serotonin* NEAR/4 receptor*" or "serotonin* NEAR/4 transport*" or "solute carrier*" or "synaptosomal associated protein" or "Tryptophan hydroxylase*" or "vasopressin*" or "vasopressin* NEAR/4 receptor*")
#2 TS=(gene or genes or genotype* or polymorphism* or allele* or haplotype*)
#3 #1 OR #2
#4 TS=(parent* or maternal* or paternal* or mother* or father* or caregiver* or attachment or childrearing or "child-rearing" or "child care")
#5 TS=(preschool* or infant* or toddler* or baby or babies or newborn* or kindergarten* or kindergarden* or child* or p$ediatric* or "1* mo* old*" or "2* mo* old*" or "3* mo* old*" or "4* mo* old*" or "5* mo* old*" or "6* mo* old*" or "1 y* old*" or "2 y* old*" or "3 y* old*" or "4 y* old*" or "5 y* old*")
#6 TS=("cognitive control" or "cognitive flexibility" or "executive control" or "executive function*" or "inhibitory control" or "proactive control" or "problem solving" or "reactive control" or "response inhibition" or "set shifting" or "task switching" or "memory updating" or "working memory" or "affect regulat*" or "affect control*" or "affective regulat*" or "affective control*" or "approach behavio$r" or "approach avoidance" or "decision making" or "delay aversion" or delay* NEAR/2 discount* or delay* NEAR/2 gratification or "inhibitory control" or "emotion* regulat*" or "emotion* control*" or frustration or gambling or "impulse control" or impulsiv* or "reversal learning" or "reward discounting" or "reward processing" or "reward sensitivity" or "risk taking" or "time discounting" or "inter-temporal choice" or "intertemporal choice" or "delayed reward" or "monetary incentive delay" or "monetary choice task" or "frontal cortex" or "frontal lobe" or "prefrontal cortex" or "self control" or "self regulat*" or "attention* shift*" or "executive attention" or "selective attention" or temperament or "emotional intelligence" or "choice behavio$r" or "reinforcement delay" or compliance)
#7 #3 AND #4 AND #5 AND #6

Web of Science Core Collection All Years (1900 – 2019) (*N* = 934)

#1 TS=(gene or genes or genotype* or polymorphism* or allele* or haplotype*)
#2 TS=(parent* or maternal* or paternal* or mother* or father* or caregiver* or attachment or childrearing or “child-rearing” or “child care”)
#3 TS=("cognitive control" or "cognitive flexibility" or "executive control" or "executive function*" or "inhibitory control" or "proactive control" or "problem solving" or "reactive control" or "response inhibition" or "set shifting" or "task switching" or "memory updating" or "working memory" or "affect regulat*" or "affect control*" or "affective regulat*" or "affective control*" or "approach behavio$r" or "approach avoidance" or "decision making" or "delay aversion" or delay* NEAR/2 discount* or delay* NEAR/2 gratification or "inhibitory control" or "emotion* regulat*" or "emotion* control*" or frustration or gambling or "impulse control" or impulsiv* or "reversal learning" or "reward discounting" or "reward processing" or "reward sensitivity" or "risk taking" or "time discounting" or "inter-temporal choice" or "intertemporal choice" or "delayed reward" or "monetary incentive delay" or "monetary choice task" or "frontal cortex" or "frontal lobe" or "prefrontal cortex" or "self control" or "self regulat*" or "attention* shift*" or "executive attention" or "selective attention" or temperament or "emotional intelligence" or "choice behavio$r" or "reinforcement delay" or compliance)
#4 TS=(preschool* or infant* or toddler* or baby or babies or newborn* or kindergarten* or kindergarden* or child* or p$ediatric* or "1* mo* old*" or "2* mo* old*" or "3* mo* old*" or "4* mo* old*" or "5* mo* old*" or "6* mo* old*" or "1 y* old*" or "2 y* old*" or "3 y* old*" or "4 y* old*" or "5 y* old*")
#5 #1 AND #2 AND #3 AND #4
Refined by: [excluding] **DOCUMENT TYPES:** (MEETING ABSTRACT)

Elsevier Scopus (*N* = 527)

(TITLE-ABS (gene OR genes OR genotype* OR polymorphism* OR allele* OR haplotype*)) AND (TITLE-ABS (parent* OR maternal* OR paternal* OR mother* OR father* OR caregiver* OR attachment OR childrearing OR "child-rearing" OR "child care")) AND (TITLE-ABS ("cognitive control" OR "cognitive flexibility" OR "executive control" OR "executive function*" OR "inhibitory control" OR "proactive control" OR "problem solving" OR "reactive control" OR "response inhibition" OR "set shifting" OR "task switching" OR "memory updating" OR "working memory" OR "affect regulat*" OR "affect control*" OR "affective regulat*" OR "affective control*" OR "approach behaviour" OR "approach behaviour" OR "approach avoidance" OR "decision making" OR "delay aversion" OR (delay* W/2 discount*) OR (delay* W/2 gratification) OR "inhibitory control" OR "emotion* regulat*" OR "emotion* control*" OR frustration OR gambling OR "impulse control" OR impulsiv* OR "reversal learning" OR "reward discounting" OR "reward processing" OR "reward sensitivity" OR "risk taking" OR "time discounting" OR "inter-temporal choice" OR "intertemporal choice" OR "delayed reward" OR "monetary incentive delay" OR "monetary choice task" OR "frontal cortex" OR "frontal lobe" OR "prefrontal cortex" OR "self control" OR "self regulat*" OR "attention* shift*" OR "executive attention" OR "selective attention" OR temperament OR "emotional intelligence" OR "choice behaviour" OR "choice behaviour" OR "reinforcement delay" OR compliance)) AND (TITLE-ABS (preschool* OR child* OR infant* OR toddler* OR baby OR babies OR pediatric* OR paediatric* OR newborn* OR kindergarten* OR kindergarden*))

Appendix B

Results of the Sensitivity Analyses in Chapter 3

1) European American participants (n = 76)

For WMIC, the paths from genetic risk × problematic discipline ($\Delta\chi^2(1) = .49$, $p = .48$) and genetic risk × responsiveness ($\Delta\chi^2(1) = 2.79$, $p = .10$) were dropped. For self-control, the paths from genetic risk × problematic discipline ($\Delta\chi^2(1) = 3.29$, $p = .07$) and genetic risk × responsiveness ($\Delta\chi^2(1) = .02$, $p = .89$) were dropped. Results for the final model are presented below.

Predictor	*b*	*SE(b)*	*β*	*p*	95% CI	R^2
Self-control						
Cumulative dopaminergic genetic risk	-.32	.16	-.37	.05^{+}	-.736, .101	
Problematic discipline	-.42	.24	-.64	.08^{+}	-1.029, .195	
Responsiveness	-.06	.06	-.18	.35	-.219, .103	
Prenatal tobacco exposure status	.09	.21	.05	.69	-.461, .632	
Psychological distress	-.23	.18	-.13	.21	-.689, .238	
Parental education	.02	.06	.03	.81	-.148, .179	
Child sex	-.15	.23	-.07	.51	-.737, .438	
Cumulative dopaminergic genetic risk × prenatal tobacco exposure status	.37	.18	.28	**.04***	-.084, .828	
Cumulative dopaminergic genetic risk × psychological distress	.07	.19	.06	.72	-.429, .568	
Cumulative dopaminergic genetic risk × parental education	.02	.06	.03	.77	-.138, .173	
Cumulative dopaminergic genetic risk × child sex	.44	.30	.18	.14	-.331, 1.212	
Problematic discipline × prenatal tobacco exposure status	.30	.21	.43	.15	-.237, .843	
Problematic discipline × psychological distress	.20	.18	.26	.26	-.253, .647	
Problematic discipline × parental education	.14	.06	.29	**.01***	-.002, .287	
Problematic discipline × child sex	.34	.23	.18	.14	-.258, .933	
Responsiveness × prenatal tobacco exposure status	.04	.08	.09	.66	-.179, .253	
Responsiveness × psychological distress	.19	.09	.40	**.03***	-.031, .409	
Responsiveness × parental education	.04	.02	.17	.13	-.026, .098	
Responsiveness × child sex	.14	.09	.18	.12	-.089, .360	
						.43
WMIC						
Cumulative dopaminergic genetic risk	-.13	.17	-.17	.45	-.573, .311	

Problematic discipline	-.20	.25	-.33	.42	-.847, .446
Responsiveness	.07	.07	.23	.30	-.102, .237
Prenatal tobacco exposure status	.10	.22	.06	.65	-.475, .679
Psychological distress	-.05	.19	-.03	.81	-.536, .444
Parental education	.08	.07	.14	.23	-.093, .252
Child sex	-.35	.24	-.17	.15	-.968, .273
Cumulative dopaminergic genetic risk × prenatal tobacco exposure status	-.03	.19	-.03	.86	-.516, .448
Cumulative dopaminergic genetic risk × psychological distress	.29	.20	.29	.16	-.239, .814
Cumulative dopaminergic genetic risk × parental education	.10	.06	.18	.13	-.068, .261
Cumulative dopaminergic genetic risk × child sex	.55	.32	.25	.08^{+}	-.260, 1.369
Problematic discipline × prenatal tobacco exposure status	.09	.22	.14	.69	-.481, .659
Problematic discipline × psychological distress	-.04	.19	-.06	.83	-.514, .436
Problematic discipline × parental education	-.05	.06	-.11	.42	-.201, .104
Problematic discipline × child sex	-.11	.24	-.06	.65	-.741, .517
Responsiveness × prenatal tobacco exposure status	-.09	.09	-.24	.31	-.319, .138
Responsiveness × psychological distress	.04	.09	.10	.63	-.189, .276
Responsiveness × parental education	.03	.03	.14	.28	-.038, .093
Responsiveness × child sex	-.10	.09	-.14	.29	-.335, .139
					.25

Note. WMIC = working memory/inhibitory control. $^{+}p < .10$; $^{*}p < .05$; $^{**}p < .01$.

2) European American and African American participants (n = 108)

For WMIC, the paths from genetic risk × problematic discipline ($\Delta\chi^2(1) = .32$, $p = .57$) and genetic risk × responsiveness ($\Delta\chi^2(1) = 1.39$, $p = .24$) were dropped. For self-control, the path from genetic risk × problematic discipline ($\Delta\chi^2(1) = 5.13$, $p = .02$) was retained; while the path genetic risk × responsiveness ($\Delta\chi^2(1) = .01$, $p = .95$) was dropped. Results for the final model are presented below. This model fit the data well ($\chi^2(1) = .08$, $p = .78$, RMSEA = .00, CFI = 1.00, SRMR = .00).

Predictor	*b*	*SE(b)*	*β*	*p*	95% CI	R^2
Self-control						
Cumulative dopaminergic genetic risk	-.31	.15	-.34	**.03***	-.681, .066	
Problematic discipline	-.29	.21	-.38	.17	-.823, .249	
Responsiveness	-.07	.05	-.21	.18	-.199, .062	
Cumulative dopaminergic genetic risk × problematic discipline	-.17	.07	-.22	**.01***	-.348, .003	
African American	-.27	.19	-.13	.17	-.761, .230	
Prenatal tobacco exposure status	-.53	.15	-.29	**.001****	-.917, -.137	
Psychological distress	-.24	.19	-.14	.20	-.732, .244	
Parental education	.02	.05	.04	.65	-.109, .155	
Child sex	-.15	.16	-.08	.34	-.566, .260	
Cumulative dopaminergic genetic risk × African American	.02	.18	.01	.90	-.431, .473	
Cumulative dopaminergic genetic risk × prenatal tobacco exposure status	.09	.15	.07	.57	-.305, .476	
Cumulative dopaminergic genetic risk × psychological distress	.44	.18	.24	**.02***	-.024, .898	
Cumulative dopaminergic genetic risk × parental education	.03	.05	.05	.58	-.097, .151	
Cumulative dopaminergic genetic risk × child sex	.09	.14	.07	.51	-.270, .458	
Problematic discipline × African American	.16	.15	.17	.30	-.232, .542	
Problematic discipline × prenatal tobacco exposure status	-.13	.18	-.07	.46	-.579, .322	
Problematic discipline × psychological distress	-.01	.05	-.02	.83	-.132, .112	
Problematic discipline × parental education	.21	.17	.25	.22	-.233, .657	
Problematic discipline × child sex	-.10	.07	-.17	.14	-.267, .072	
Responsiveness × African American	.12	.07	.25	.06^{+}	-.046, .288	
Responsiveness × prenatal tobacco exposure status	.11	.05	.33	**.02***	-.011, .228	
Responsiveness × psychological distress	.04	.02	.18	.06	-.013, .087	
Responsiveness × parental education	.10	.06	.23	.10	-.059, .268	

Responsiveness × child sex	.28	.20	.14	.16	-.239, .803	.46
WMIC						
Cumulative dopaminergic genetic risk	-.12	.15	-.15	.41	-.497, .255	
Problematic discipline	-.18	.21	-.26	.41	-.724, .373	
Responsiveness	-.002	.05	-.01	.96	-.137, .133	
African American	-.45	.19	-.25	.02	-.940, .050	
Prenatal tobacco exposure status	-.13	.16	-.08	.42	-.529, .275	
Psychological distress	-.45	.20	-.30	.02	-.949, .057	
Parental education	.10	.05	.19	.06	-.036, .234	
Child sex	.04	.17	.02	.82	-.388, .461	
Cumulative dopaminergic genetic risk × African American	.08	.18	.06	.65	-.374, .536	
Cumulative dopaminergic genetic risk × prenatal tobacco exposure status	.17	.15	.16	.26	-.220, .563	
Cumulative dopaminergic genetic risk × psychological distress	.11	.18	.07	.56	-.368, .582	
Cumulative dopaminergic genetic risk × parental education	.05	.05	.10	.32	-.078, .178	
Cumulative dopaminergic genetic risk × child sex	.004	.15	.004	.98	-.370, .378	
Problematic discipline × African American	.08	.20	.04	.71	-.450, .602	
Problematic discipline × prenatal tobacco exposure status	.02	.15	.03	.89	-.374, .416	
Problematic discipline × psychological distress	-.004	.18	-.003	.98	-.455, .447	
Problematic discipline × parental education	-.03	.05	-.06	.56	-.151, .095	
Problematic discipline × child sex	.04	.18	.05	.84	-.421, .491	
Responsiveness × African American	-.11	.07	-.21	.10	-.287, .062	
Responsiveness × prenatal tobacco exposure status	.03	.07	.06	.71	-.147, .197	
Responsiveness × psychological distress	-.001	.05	-.004	.98	-.124, .122	
Responsiveness × parental education	.02	.02	.11	.34	-.032, .070	
Responsiveness × child sex	.02	.07	.04	.79	-.151, .186	.27

Note. WMIC = working memory/inhibitory control. $^{+}p < .10$; $^{*}p < .05$; $^{**}p < .01$.

Appendix C

Complete Results for the Final Model in Chapter 3

Predictor	*b*	*SE(b)*	*β*	*p*	95% CI	R^2
Self-control						
Cumulative dopaminergic genetic risk	-.39	.14	-.26	**.01***	-.761, -.018	
Problematic discipline	-.15	.20	-.45	.47	-.671, .375	
Responsiveness	-.06	.05	-.004	.25	-.189, .073	
Cumulative dopaminergic genetic risk × problematic discipline	-.18	.07	-.18	**.01***	-.348, -.016	
African American	-.30	.20	-.18	.13	-.799, .207	
Latinx American	.16	.20	.06	.42	-.357, .684	
Prenatal tobacco exposure status	-.48	.14	-.02	**.001****	-.845, -.110	
Psychological distress	-.38	.17	-.33	**.03***	-.817, .061	
Parental education	-.08	.05	.08	.13	-.204, .053	
Child sex	-.30	.15	.05	**.05***	-.683, .088	
Cumulative dopaminergic genetic risk × African American	.06	.18	.02	.74	-.410, .533	
Cumulative dopaminergic genetic risk × Latinx American	.09	.18	-.03	.63	-.383, .558	
Cumulative dopaminergic genetic risk × prenatal tobacco exposure status	.20	.14	.26	.16	--.167, .574	
Cumulative dopaminergic genetic risk × psychological distress	.19	.17	.03	.25	-.236, .616	
Cumulative dopaminergic genetic risk × parental education	-.03	.05	.08	.48	-.150, .085	
Cumulative dopaminergic genetic risk × child sex	.10	.14	.01	.47	-.259, .462	
Problematic discipline × African American	.24	.21	.09	.25	-.304, .787	
Problematic discipline × Latinx American	.05	.14	.10	.71	-.306, .410	
Problematic discipline × prenatal tobacco exposure status	.12	.13	.13	.36	-.221, .468	
Problematic discipline × psychological distress	-.06	.16	-.06	.71	-.460, .343	
Problematic discipline × parental education	.01	.05	-.05	.89	-.111, .123	
Problematic discipline × child sex	.03	.14	.09	.86	-.342, .394	
Responsiveness × African American	-.08	.07	-.18	.24	-.261, .097	
Responsiveness × Latinx American	-.08	.08	-.09	.30	-.274, .117	
Responsiveness × prenatal tobacco exposure status	.10	.06	-.06	.09^{+}	-.049, .248	
Responsiveness × psychological distress	.06	.04	-.06	.16	-.051, .175	
Responsiveness × parental education	.02	.02	.10	.19	-.023, .070	
Responsiveness × child sex	.12	.06	.02	**.05***	-.034, .264	

.40

WMIC					
Cumulative dopaminergic genetic risk	-.20	.13	-.26	.13	-.548, .144
Problematic discipline	-.28	.19	-.45	.15	-.763, .212
Responsiveness	-.001	.05	-.004	.98	-.123, .121
Cumulative dopaminergic genetic risk × problematic discipline	-.12	.06	-.18	.05^{+}	-.272, .038
African American	-.34	.18	-.13	.06^{+}	-.812, .125
Latinx American	.11	.19	.06	.56	-.375, .594
Prenatal tobacco exposure status	-.03	.13	-.02	.85	-.368, .317
Psychological distress	-.50	.16	-.33	**.002****	-.907, -.089
Parental education	.04	.05	.08	.37	-.078, .162
Child sex	.08	.14	.05	.57	-.280, .438
Cumulative dopaminergic genetic risk × African American	.04	.17	.02	.82	-.400, .479
Cumulative dopaminergic genetic risk × Latinx American	-.04	.17	-.03	.80	-.482, .394
Cumulative dopaminergic genetic risk × prenatal tobacco exposure status	.26	.13	.26	**.05***	-.081, .609
Cumulative dopaminergic genetic risk × psychological distress	.05	.15	.03	.76	-.350, .444
Cumulative dopaminergic genetic risk × parental education	.04	.04	.08	.37	-.071, .147
Cumulative dopaminergic genetic risk × child sex	.01	.13	.01	.91	-.321, .350
Problematic discipline × African American	.17	.20	.09	.38	-.336, .680
Problematic discipline × Latinx American	.11	.13	.10	.40	-.225, .443
Problematic discipline × prenatal tobacco exposure status	.10	.13	.13	.45	-.226, .415
Problematic discipline × psychological distress	-.09	.15	-.06	.56	-.460, .288
Problematic discipline × parental education	-.02	.04	-.05	.59	-.132, .086
Problematic discipline × child sex	-.07	.13	.09	.61	-.274, .412
Responsiveness × African American	-.10	.07	-.18	.12	-.268, .065
Responsiveness × Latinx American	-.07	.0	-.09	.36	-.247, .117
Responsiveness × prenatal tobacco exposure status	.03	.05	.06	.63	-.112, .164
Responsiveness × psychological distress	-.02	.04	-.06	.65	-.124, .087
Responsiveness × parental education	.02	.02	.10	.28	-.025, .061
Responsiveness × child sex	.01	.05	.02	.86	-.129, .148

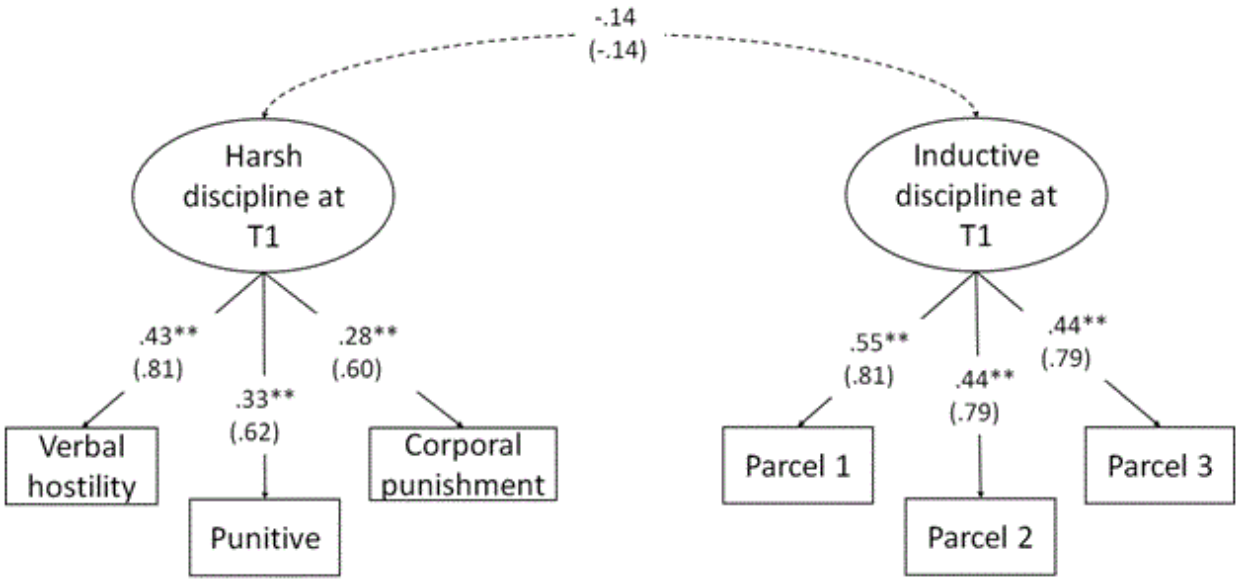
.27

Note. WMIC = working memory/inhibitory control. $^{+}p < .10$; $^{*}p < .05$; $^{**}p < .01$.

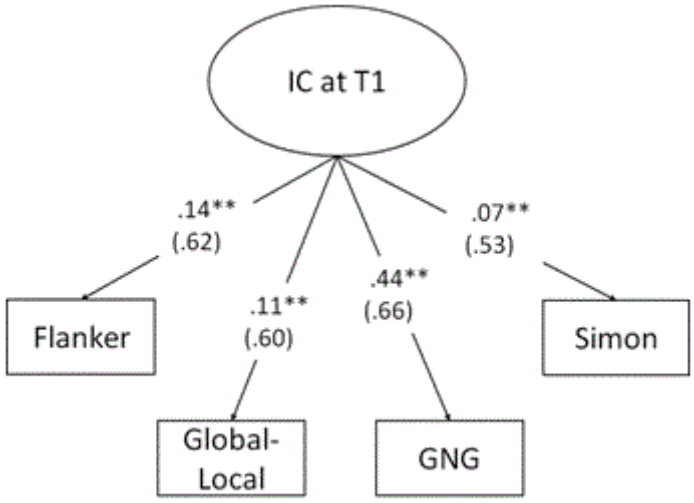
Appendix D

Measurement Models for Chapter 4

1) Two-factor measurement model of harsh and inductive discipline. For inductive discipline, parcel 1 included questionnaire items 25 and 58, parcel two included item 29 and 62, and parcel three included item 16, 42, and 53. T1 = time 1. $^{+}p < .10$; $^{*}p < .05$; $^{**}p < .01$.



2) One-factor measurement model of inhibitory control. IC = inhibitory control. GNG = *Go/No-Go*. T1 = time 1. $^{+}p < .10$; $^{*}p < .05$; $^{**}p < .01$.



Appendix E

Results of Measurement Invariance Testing for Chapter 4

Model fit indices for measurement invariance models of inhibitory control at time 1 for 4-, 5-, and 6-year-olds

Model	χ^2	*df*	*p*	*RMSEA*	*CFI*	*SRMR*	Model comparison	$\Delta\chi^2$	*df*	*p*
1. Configural invariance	8.39	6	.21	.09	.92	.07				
2. Metric invariance	9.42	12	.67	.00	1.00	.07	1 vs. 2	1.03	6	.98
3. Scalar invariance	21.07	18	.28	.06	.90	.16	2 vs. 3	11.65	6	.07

Note. For model comparisons, the preferred model is underlined. Where two nested models showed equivalent fit to the data, the more parsimonious model was preferred.

Model fit indices for longitudinal measurement invariance models for harsh discipline, inductive discipline, and inhibitory control

Model	χ^2	*df*	*p*	*RMSEA*	*CFI*	*SRMR*	Model comparison	$\Delta\chi^2$	*df*	*p*
Harsh discipline										
1. Configural invariance	3.21	5	.67	.00	1.00	.02				
2. Metric invariance	6.96	7	.43	.00	1.00	.05	1 vs. 2	3.75	2	.15
3. Scalar invariance	10.46	9	.32	.03	1.00	.06	2 vs. 3	3.50	2	.17
Inductive discipline										
1. Configural invariance	11.03	5	.05	.09	.98	.03				
2. Metric invariance	16.72	7	.02	.10	.97	.11	1 vs. 2	5.70	2	.06
3. Scalar invariance	17.02	9	.05	.08	.98	.11	2 vs. 3	0.30	2	.86
Inhibitory control										
1. Configural invariance	13.71	15	.55	.00	1.00	.04				
2. Metric invariance	28.11	18	.06	.06	.94	.13	1 vs. 2	14.40	3	.002

3. Partial metric invariance: *Flanker Task* indicators unconstrained	28.09	17	.04	.07	.94	.13	1 vs. 3	14.39	2	.001
4. Partial metric invariance: *Global-Local Task* indicators unconstrained	16.43	17	.49	.00	1.00	.07	1 vs. 4	2.73	2	.26
5. Partial metric invariance: *Simon Task* indicators unconstrained	23.15	17	.15	.05	.96	.10	1 vs. 5	9.44	2	.01
6. Final partial metric invariance model	16.43	17	.49	.00	1.00	.07	1 vs. 6	2.73	2	.26
7. Scalar invariance	27.77	20	.12	.05	.95	.08	6 vs. 7	11.33	3	.01
8. Partial scalar invariance: *Go/No-Go* intercepts unconstrained	26.08	19	.12	.05	.96	.09	6 vs. 8	9.64	2	.01
9. Partial scalar invariance: *Flanker Task* intercepts unconstrained	16.51	19	.62	.00	1.00	.07	6 vs. 9	.07	2	.96
10. Partial scalar invariance: *Global-Local Task* intercepts unconstrained	24.09	19	.19	.04	.97	.06	6 vs. 10	7.66	2	.02
11. Partial scalar invariance: *Simon Task* intercepts unconstrained	27.26	19	.10	.06	.95	.09	6 vs. 11	10.83	2	.004
12. Final partial scalar invariance model	16.51	19	.62	.00	1.00	.07	6 vs. 12	.07	2	.97

Note. For model comparisons, the preferred model is underlined. Where two nested models showed equivalent fit to the data, the more parsimonious model was preferred.