Executive Functioning in Context: Measurement, Etiology, and Biological Embedding

by

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A dissertation submitted in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy
(Psychology)
in The University of Michigan
2023

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DEDICATION

This dissertation is dedicated to Claire.

ACKNOWLEDGMENTS

This dissertation would not have been possible without a whole host of people who taught me about science. First and foremost, I have to thank my graduate mentor, Dr. Luke Hyde. Luke took a chance on a student with interests only tangential to his lab's previous work, in a year when he was already admitting a superstar. In doing so, he gave me not only a place in a fabulous graduate program, but a lifelong best friend in Hailey Dotterer (the aforementioned superstar) and an incredible clinical cohort. I can't say enough about the roles Luke has played in shaping both my trajectories as a clinical scientist and as a person. Luke taught me my most foundational science skills while also giving me the space to test out and develop my own ideas. He taught me how to match a person's pace and volume in a clinical setting, and how to consider context at every step of the diagnostic and treatment process. He taught me how to teach undergraduates, and encouraged me to guest lecture and develop my own class materials. When my life got really, really hard, Luke didn't give up on me.

I also want to thank my dissertation committee, who I have repeatedly described as "my dream committee", for taking the time to support me during this process. I have been lucky to work with Dr. S. Alexandra Burt for several years, and she always took the time to walk through complex twin models and discuss project ideas when I needed help. Dr. John Jonides provided thoughtful and challenging input when I wanted to dig deeper into cognitive outcomes. Dr. Vonnie McLoyd and Dr. Chandra Sripada graciously joined my committee and provided invaluable input as true experts in their areas. Additionally, though not on my committee, Dr.

Alexander Weigard has also served as a mentor and a collaborator on these projects. He wrote the coolest paper I had ever read in 2020, inspiring me to re-think how I analyzed my data, and enthusiastically collaborated with me when he was under no obligation.

I also want to thank my first "lab family", Dr. Elizabeth Brannon's lab. My science journey began with an interview with Dr. Joonkoo Park in which I expressed major enthusiasm about working with monkeys and/or children, and he informed me that the job would involve neither. Joon, Liz, and the rest of the Brannon Lab welcomed me as a part of the group and taught me how much I didn't know about the brain.

Of course, I have to thank my own little family. My husband Laken was traded across the country after my first year of graduate school, and he never once questioned my decision to stay in Michigan and continue the program. An absurd amount of my graduate coursework was completed on planes, but we made it work. My daughter Taylor has spent her whole life watching me work on this dissertation, and I hope one day she understands why it was so important not to bang on the laptop keyboard. My identity as "mom" has shaped the direction of my work (two out of three of these studies involve parenting!) and I am so grateful to her for inspiring and challenging me.

I would also like to acknowledge the funding sources that made my education and this dissertation possible. My parents decided to shell out for a Duke education, and I am forever grateful. I received graduate funding from the NSF Graduate Research Fellowship Program as well as Rackham Graduate School. The incredible MTwiNS study is funded by numerous sources including several grants from the National Institutes of Health and from the Brain and Behavior Foundation. I would also like to thank the families who participated in MTwiNS for sharing their lives with us. None of this would have been possible without their time and effort.

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ABSTRACT

Executive functioning abilities measured early in life are known to predict important outcomes later in life, including academic outcomes, physical health, and symptoms of psychopathology. Unfortunately, a growing literature identifies a consistent relationship between socioeconomic disadvantage in childhood and worse executive functioning performance, and the mechanisms underlying this troubling relationship are not well understood. This dissertation is comprised of three studies that address several complications muddying our understanding of executive functioning and its relationship with disadvantage, including (1) how we measure executive functioning, (2) how we can disentangle non-genetic from genetic effects of parenting on executive functioning, and (3) how multiple levels of context may become biologically embedded to affect executive functioning. In Study 1, we find encouraging evidence for a novel computational measure of task-general executive functioning, Efficiency of Evidence Accumulation (EEA), as a transdiagnostic risk factor associated with externalizing psychopathology in adolescents. In Study 2, we employ a genetically informed design, find little evidence of passive or evocative genotype-environment correlation, and instead finding that the relationship between harsh parenting and child executive functioning is due in part to non-shared environmental influences. In Study 3, we find that neighborhood poverty is associated with executive functioning performance via inferior frontal gyrus activation during a go/no-go task, over and above other contextual effects. However, we do not find evidence for nurturing, supportive parenting as a buffer, nor for harsh parenting as an activator of neighborhood risk.

The general discussion chapter of this dissertation highlights the implications of this research and important future directions.

CHAPTER I

Introduction to the Dissertation

Executive functioning describes a set of top-down processes that involve regulation of goal-directed behavior, including processes like inhibitory control, working memory, and cognitive flexibility (Diamond, 2013; Miyake et al., 2000). Executive functioning abilities measured early in life consistently predict important outcomes later in life, including academic outcomes like literacy, vocabulary, and math skills (Blair & Razza, 2007; McClelland et al., 2007), learning (Marcovitch et al., 2008), and even physical health (Kubzansky et al., 2009; Mischel et al., 2011; Seeyave et al., 2009). In fact, a gradient of childhood self-control has been shown to predict physical health, substance dependence, finances, and criminal outcomes at age 32 (Moffitt et al., 2011). Additionally, deficits in executive functioning have been linked to various forms of psychopathology (Bloemen et al., 2018; Wright et al., 2014), including attention-deficit/hyperactivity disorder (ADHD; Castellanos et al., 2006; Cherkasova et al., 2013), antisocial behavior (Moffitt, 2018), substance abuse (Smith et al., 2014; Verdejo-Garcia et al., 2008), and depression (Gotlib & Joormann, 2010). Based on this constellation of evidence, understanding the origins of children's executive functioning abilities and using this knowledge to support executive functioning development appears to be a clear target for clinical psychology.

Unfortunately, childhood socioeconomic disadvantage has emerged as a major risk factor for reduced executive functioning performance. A growing literature identifies a consistent

relationship between socioeconomic disadvantage in childhood and lower executive functioning scores, both during childhood and adolescence (Hackman et al., 2014; Lawson et al., 2018; Raver et al., 2013; Rochette & Bernier, 2014; Shameem & Hamid, 2014), and continuing into adulthood (Evans et al., 2021; Last et al., 2018). Meta-analytic evidence indicates that the effect of socioeconomic disadvantage on executive functioning is small-to-medium in size, with the biggest effect sizes coming from studies with meaningful socioeconomic variability and multiple measures of executive functioning (Lawson et al., 2018). However, there is substantial heterogeneity in effect sizes between studies (Lawson et al., 2018), and there is not yet consensus regarding the mechanisms underlying the relationship between socioeconomic disadvantage and executive functioning. The present dissertation will take a deep dive into some of the complications muddying our understanding of executive functioning and how it is affected by socioeconomic disadvantage, including (1) how we measure executive functioning, (2) how we can disentangle non-genetic from genetic effects on executive functioning, and (3) how multiple levels of context may become biologically embedded to affect executive functioning.

Measuring Executive Functioning: Improving Reliability and Predictive Power with Computational Modeling

The executive functioning literature is complicated by a lack of consensus around the underlying structure of executive functioning (Karr et al., 2018). Studies of individual differences in executive functioning tend to assume that executive functioning is made up of several intercorrelated, but distinct, subcomponents (Sripada & Weigard, 2021). The most prominent theory of executive functioning, the *unity-yet-diversity* theory (Miyake et al., 2000), subdivides executive functioning into three separable functions: shifting between tasks or sets ("shifting"), updating and monitoring of representations in working memory ("updating"), and

deliberate inhibition of prepotent responses ("inhibition"). The general *unity-yet-diversity* framework is supported by years of factor analytic work (Friedman & Miyake, 2017; Miyake et al., 2000). Though the focus of clinical neuroscience research is often on the "*diversity*" of executive functioning, the "*unity*" of the theory reflects the fact that these three subcomponents, though separable, are correlated, addressing the paradox of a hypothesized domain-general executive functioning coupled with troublingly low intercorrelations between executive functioning tasks (Friedman & Miyake, 2017; Miyake et al., 2000). However, factor analyses often identify variations on the original three subcomponents, and no single subcomponent has been shown to consistently relate to any single form of psychopathology (Karr et al., 2018). In fact, a recent re-analysis of 46 eligible confirmatory factor analyses found that no one model consistently converged and met acceptability criteria in all samples, suggesting that many previously published models based upon the "*unity-yet-diversity*" framework executive functioning may have been underpowered and overfit (Karr et al., 2018). Rather than focusing on the *diversity* of executive functioning, it may be helpful to focus on *unity* via a measure of task-general executive functioning.

One way to index task-general executive functioning is to model task performance in a way that can be applied to any subcomponent executive functioning task, generating a common metric that measures a domain-general construct (Weigard & Sripada, 2021). Model-based approaches, like the drift diffusion model, can be applied to a variety of behavioral tasks and provide richer information about cognitive performance than traditional metrics by accounting for both accuracy and reaction time (Weigard, Brislin, et al., 2021; Wiecki et al., 2015). One model-based construct in particular, the "efficiency of evidence accumulation" (EEA), indexed by drift rate in the drift diffusion model, shows promise as a task-general individual difference

that contributes to cognitive impairments across various forms of psychopathology in adults (Weigard & Sripada, 2021). The drift diffusion model assumes that evidence driving a decision (i.e. a choice on a two-choice task) is accumulated continuously over time (Ratcliff & Rouder, 1998). EEA describes the rate at which a person gathers relevant evidence among background noise in order to make an accurate choice (Weigard & Sripada, 2021). In other words, EEA indexes how efficiently an individual tends to "drift" toward a correct decision across various tasks and conditions. EEA exhibits good test-retest reliability (Lerche & Voss, 2017), and outperforms traditional metrics both in temporal stability and in prediction of psychopathology (Weigard, Clark, et al., 2021). In adults, reduced EEA is associated with schizophrenia, bipolar disorder, and ADHD, as well as a global severity of psychopathology (Sripada & Weigard, 2021), and predicts substance use behaviors (Weigard, Brislin, et al., 2021). Given its improved reliability and relation to real-world measures of psychopathology, using a model-based measure of executive functioning, like EEA, may improve our ability to pinpoint the mechanisms underlying disadvantage-related executive functioning differences. However, though it shows great promise in adult samples, the reliability and validity of this measure have not yet been investigated in samples of children and adolescents.

Intergenerational Transmission of Executive Functioning

Beyond measurement, a key question is the etiology of executive functioning — where do individual differences in executive functioning come from? The executive functioning literature is complicated by the issue of disentangling non-genetic (i.e. parenting, neighborhood poverty) contributors from genetic contributors to individual differences in executive functioning. Executive functioning runs in families, such that parents with better executive functioning skills tend to have children with better executive functioning skills (Deater-Deckard,

2014). Non-genetic, environmental influences such as parenting have been implicated as potential mediators of the relationship between parent and child executive functioning (e.g. Distefano et al., 2018; Kao et al., 2018; Korucu et al., 2020). However, the heritability of a general executive functioning factor is thought to be quite high (Friedman & Miyake, 2017), with estimates as high as 99% (Friedman et al., 2008) or 100% (Engelhardt et al., 2015). Because parents pass 50% of their genes to their children, and parenting behaviors are known to be influenced by genotype (Elkins et al., 1997; McGuire, 2003; Narusyte et al., 2008), it could be argued that any apparent effect of context on executive functioning is driven by *genotype-environment correlation (rGE),* or the correlation between the parents' (and thus children's) genetic tendencies and the environment they provide for their children (Deater-Deckard, 2014; Knopik et al., 2017; Manuck & McCaffery, 2014). Disentangling the role of genotype-environment correlation in classical twin studies of executive functioning has proven challenging (Li & Roberts, 2017), and the majority of studies linking context to child executive functioning are not genetically informed, with a few notable exceptions (Bridgett et al., 2018; Cioffi et al., 2020). Thus, it is necessary to extend beyond classical twin studies in order to better illuminate the role, or lack thereof, of non-genetic contributors to the family transmission of executive functioning.

The Embedding of Context: The Role of the Neighborhood

A final consideration is how contexts may be biologically embedded to contribute to individual differences in executive functioning. A growing literature points to neighborhood disadvantage as a particularly notable risk factor for executive functioning differences, over and above family-level socioeconomic variables. Neighborhood contexts confer additional risk beyond family-level factors because they increase exposure to additional adverse experiences

(e.g., neighborhood danger, under-resourced schools, toxicant exposure; Evans, 2004; Leventhal & Brooks-Gunn, 2000). These neighborhood-adversity-related stressors may generate "wear" on stress regulatory systems, with implications for the brain circuitry underlying executive functioning (Finegood et al., 2017; McEwen, 2004). As many of the earlier studies reporting disadvantage-related executive functioning differences only measured family-level variables like income or education (Lawson et al., 2018), the neighborhood context may represent an unmeasured but crucial third variable.

Indeed, there is a growing literature that finds effects of neighborhood context on brain structure and function. Recent work from our group demonstrated that children raised in neighborhoods with higher levels of poverty show reduced behavioral and brain measures of executive functioning (Tomlinson et al., 2020). Notably, this relationship held even when controlling for family-level socioeconomic variables (i.e., family income, maternal education), indicating that *where children live* is particularly important for their executive functioning development. There is also evidence of effects of neighborhood disadvantage on other corticolimbic structures and processes: for example, neighborhood disadvantage is associated with greater amygdala reactivity to faces (Gard et al., 2021), reduced prefrontal activation to working memory load (Murtha et al., 2021), changes in amygdala-prefrontal connectivity (Ramphal et al., 2020), reduced cortical thickness (Hackman et al., 2021; Hunt et al., 2020; Taylor et al., 2020), altered trajectories of brain age (Rakesh, Cropley, et al., 2021), and worse performance on neurocognitive tasks (Hackman et al., 2021; Taylor et al., 2020; Webb et al., 2021). Thus, considering the role of the neighborhood in children's executive functioning development is of critical importance.

The Embedding of Context: The Role of Parenting

A more proximal context likely to contribute to individual differences in children's executive functioning skills is the parenting they receive. Indeed, there is substantial evidence that parenting matters for executive functioning development, such that nurturing, supportive behaviors promote executive functioning skills while harsh, controlling behaviors impede executive functioning skill development (Fay-Stammbach et al., 2014; Hughes & Devine, 2019; Li et al., 2019). Rather than falling along a continuum, positive and negative parenting behaviors appear to have separable influences on executive functioning, such that even when included in the same model supportive parenting behaviors have a specific positive effect while harsh parenting behaviors have a specific negative effect (Hughes & Devine, 2019). Thus, interventions either to promote positive parenting behaviors or to reduce negative parenting behaviors could be helpful to support executive functioning skills.

However, parenting does not occur in a vacuum. It is unlikely that the neighborhood context and the parenting context operate independently from each other. Instead, the reality is likely closer to what was outlined in Bronfenbrenner's ecological systems theory, in which individuals develop within multiple nested environmental contexts (Bronfenbrenner, 1977). The family environment, including the parenting children receive, is necessarily embedded within the neighborhood context (Bronfenbrenner, 1977; Hyde et al., 2020). Additionally, as outlined in Sameroff's transactional model of development, these contexts necessarily influence each other and are simultaneously influencing and influenced by the developing child (Sameroff, 2009). The more proximal parenting context may act as a protective buffer against the more distal stressors associated with neighborhood disadvantage. Indeed, positive parenting behaviors were recently shown to moderate the relationship between neighborhood disadvantage and resting state functional connectivity in several networks (Rakesh, Seguin, et al., 2021). Thus, work is

needed that considers the neighborhood and parenting contexts through an *ecological neuroscience* lens, in which development is thought to occur within nested, transactional contexts (Hyde et al., 2020).

Additional Considerations in the Literature

Many studies of socioeconomic disadvantage effects on executive functioning have focused on childhood or early adolescence (Blair, 2016; Evans et al., 2021; Lawson et al., 2018). Similarly, many studies of parenting effects on executive functioning focus on infants and young children (i.e. age 6 months to 4 years, e.g. Bernier et al., 2012; Bernier et al., 2010; Blair et al., 2014; Bridgett et al., 2018; Broomell et al., 2020; Cioffi et al., 2020; Distefano et al., 2018; Hammond et al., 2012; Helm et al., 2020; Hughes & Devine, 2019; Kao et al., 2018; Korucu et al., 2020; Zeytinoglu et al., 2017). This is indeed an important time period to study, as the prefrontal cortex and associated executive functioning skills are rapidly developing during this time (Kolb et al., 2012; Zelazo & Carlson, 2012). However, the prefrontal cortex has a prolonged developmental course, including an overproduction of neurons and their connections during early childhood, followed by pruning and myelination in later childhood and adolescence (Kolb et al., 2012). Executive functioning development follows this trajectory, with rapid improvement in executive functioning skills during early childhood, followed by slow and steady improvement through adolescence (Diamond, 2013). Such a prolonged developmental course makes the prefrontal cortex susceptible to environmental input even into adolescence (Blakemore & Choudhury, 2006; Kolb et al., 2012). Additionally, externalizing disorders associated with executive functioning difficulties, such as ADHD and antisocial behavior, often emerge or become more impairing during the adolescent period (Cherkasova et al., 2013; Moffitt, 2018).

Thus, work that considers parenting influences on executive functioning in later childhood and adolescence may have particular clinical relevance.

Specific Aims of this Dissertation

The purpose of this dissertation was to address these three major complications to our understanding of executive functioning and its relation to socioeconomic disadvantage by (1) testing the viability of efficiency of evidence accumulation (EEA) as a task-general executive functioning metric in adolescents; (2) disentangling non-genetic from genetic influences in the intergenerational transmission of executive functioning; and (3) studying the interacting effects of neighborhood poverty and parenting on brain and behavioral measures of executive functioning. The three studies described in this dissertation all utilized data from a large sample of adolescents representative of families living in impoverished neighborhoods. Such a sampling approach is uncommon within the neuroscience (Falk et al., 2013) and behavioral genetics (Burt et al., 2021) literatures, but does appear to be necessary to adequately measure the effects of socioeconomic disadvantage (Lawson et al., 2018).

The first study examined the utility of a model-based measurement of executive functioning in an adolescent sample. This study tested three hypotheses examining the reliability and validity of this model-based measure: first, that model-based executive functioning would be more reliable than traditional metrics via test-retest reliability; and second, that model-based executive functioning would predict concurrent and prospective parent- and child-reported symptoms of several forms of psychopathology, including ADHD and a global psychopathology score. Finally, we tested the hypothesis that EEA would correlate with inhibition-related brain

activation within inhibitory (i.e., inferior frontal gyrus) and error-monitoring (i.e., anterior cingulate cortex) brain regions.

The second study examined the intergenerational transmission of executive functioning by disentangling non-genetic influences from genetic influences. This study first utilized twin modeling methods to decompose the variance in child executive functioning into genetic and non-genetic components and then quantify passive rGE (i.e., correlation between additive genetic influences and the family environment). We hypothesized that twins who received more positive parenting behaviors (e.g., nurturance, warmth, scaffolding) and less negative parenting behaviors (e.g., harshness, negative control) would demonstrate higher executive functioning scores, and that the association between parenting and executive functioning would not be driven entirely by genetic or family-level confounds.

The third study examined the associations between neighborhood poverty, parenting, and EF-related brain activation. First, we replicated the results of Tomlinson et al. (2020) in a larger sample, asking whether neighborhood poverty predicted executive functioning performance via executive-functioning-related (EF-related) inferior frontal gyrus activation during a go/no-go task. Second, we investigated whether positive parenting, thoroughly assessed via child-report and parent-report, correlated with EF-related brain activation. Third, we examined whether positive parenting buffered the effects of neighborhood poverty on neural and behavioral measures of executive functioning. In line with emerging evidence in the field (Rakesh, Seguin, et al., 2021), we hypothesized that positive parenting would buffer the effect of neighborhood poverty on EF-related brain activation.

Study 1: Measuring Task-General executive functioning in Children Using Efficiency of Evidence Accumulation

Aim 1. Examine whether EEA is more reliable than traditional executive functioning metrics via test-retest reliability.

Aim 2. Examine the construct validity of EEA measurement via examining associations with concurrent and prospective parent- and child-reported symptoms of several forms of psychopathology.

Aim 3. Examine the inhibition-related neural correlates of EEA within inhibitory (i.e. inferior frontal gyrus, IFG) and error-monitoring (i.e. anterior cingulate cortex, ACC) regions.

Study 2: A Genetically Informed Approach to the Role of Parenting in the Intergenerational Transmission of Executive Functioning

Aim 1. Test whether parent executive functioning has an indirect effect on child executive functioning via harsh or nurturing parenting using structural equation modeling.

Aim 2. Use the nuclear twin family model to decompose the variance in child executive functioning into genetic and environmental components, quantifying passive rGE.

Aim 3. Decompose the covariance between harsh parenting and child executive functioning.

Study 3: Parenting as a Buffer Against the Effects of Neighborhood Poverty on Executive Functioning

Aim 1. Examine whether neighborhood poverty predicts executive functioning performance via EF-related inferior frontal gyrus (IFG) activation during a go/no-go task.

Aim 2. Investigate whether nurturing or harsh parenting, assessed via child-report and parent-report, correlates with EF-related brain activation.

Aim 3. Investigate whether nurturing parenting buffers, or harsh parenting activates, the effects of neighborhood poverty on neural and behavioral measures of executive functioning.

REFERENCES

Bernier, A., Carlson, S. M., Deschenes, M., & Matte-Gagne, C. (2012). Social Factors in the Development of Early Executive Functioning: A Closer Look at the Caregiving Environment. *Dev Sci, 15*(1), 12-24. https://doi.org/10.1111/j.1467-7687.2011.01093.x

Bernier, A., Carlson, S. M., & Whipple, N. (2010). From External Regulation to Self-Regulation: Early Parenting Precursors of Young Children's Executive Functioning. *Child Dev, 81*(1), 326-339. https://doi.org/10.1111/j.1467-8624.2009.01397.x

Blair, C. (2016). Developmental Science and Executive Function. *Curr Dir Psychol Sci, 25*(1), 3-7. https://doi.org/10.1177/0963721415622634

Blair, C., Raver, C. C., Berry, D. J., & Family Life Project, I. (2014). Two Approaches to Estimating the Effect of Parenting on the Development of Executive Function in Early Childhood. *Dev Psychol, 50*(2), 554-565. https://doi.org/10.1037/a0033647

Blair, C., & Razza, R. P. (2007). Relating Effortful Control, Executive Function, and False Belief Understanding to Emerging Math and Literacy Ability in Kindergarten. *Child Dev, 78*(2), 647-663. https://doi.org/10.1111/j.1467-8624.2007.01019.x

Blakemore, S.-J., & Choudhury, S. (2006). Development of the Adolescent Brain: Implications for Executive Function and Social Cognition. *Journal of Child Psychology and Psychiatry, 47*(3-4), 296-312. https://doi.org/10.1111/j.1469-7610.2006.01611.x

Bloemen, A. J. P., Oldehinkel, A. J., Laceulle, O. M., Ormel, J., Rommelse, N. N. J., & Hartman, C. A. (2018). The Association between Executive Functioning and Psychopathology: General or Specific? *Psychol Med, 48*(11), 1787-1794. https://doi.org/10.1017/S0033291717003269

Bridgett, D. J., Ganiban, J. M., Neiderhiser, J. M., Natsuaki, M. N., Shaw, D. S., Reiss, D., & Leve, L. D. (2018). Contributions of Mothers' and Fathers' Parenting to Children's Self-Regulation: Evidence from an Adoption Study. *Dev Sci, 21*(6), e12692. https://doi.org/10.1111/desc.12692

Bronfenbrenner, U. (1977). Toward an Experimental Ecology of Human Development. *American Psychologist, 32*(7), 513-531. https://doi.org/10.1037/0003-066x.32.7.513

Broomell, A. P. R., Smith, C. L., Calkins, S. D., & Bell, M. A. (2020). Context of Maternal Intrusiveness During Infancy and Associations with Preschool Executive Function. *Infant Child Dev, 29*(1). https://doi.org/10.1002/icd.2162

Burt, S. A., Klump, K. L., Vazquez, A. Y., Shewark, E. A., & Hyde, L. W. (2021). Identifying Patterns of Youth Resilience to Neighborhood Disadvantage. *Research in Human Development*, 1-16. https://doi.org/10.1080/15427609.2021.1935607

Castellanos, F. X., Sonuga-Barke, E. J., Milham, M. P., & Tannock, R. (2006). Characterizing Cognition in Adhd: Beyond Executive Dysfunction. *Trends Cogn Sci, 10*(3), 117-123. https://doi.org/10.1016/j.tics.2006.01.011

Cherkasova, M., Sulla, E. M., Dalena, K. L., Ponde, M. P., & Hechtman, L. (2013). Developmental Course of Attention Deficit Hyperactivity Disorder and Its Predictors. *J Can Acad Child Adolesc Psychiatry, 22*(1), 47-54. https://www.ncbi.nlm.nih.gov/pubmed/23390433

Cioffi, C. C., Leve, L. D., Natsuaki, M. N., Shaw, D. S., Reiss, D., & Neiderhiser, J. M. (2020). Does Maternal Warmth Moderate Longitudinal Associations between Infant Attention Control and Children's Inhibitory Control? *Infant Child Dev, 29*(1). https://doi.org/10.1002/icd.2147

Deater-Deckard, K. (2014). Family Matters: Intergenerational and Interpersonal Processes of Executive Function and Attentive Behavior. *Curr Dir Psychol Sci, 23*(3), 230-236. https://doi.org/10.1177/0963721414531597

Diamond, A. (2013). Executive Functions. *Annu Rev Psychol, 64*, 135-168. https://doi.org/10.1146/annurev-psych-113011-143750

Distefano, R., Galinsky, E., McClelland, M. M., Zelazo, P. D., & Carlson, S. M. (2018). Autonomy-Supportive Parenting and Associations with Child and Parent Executive Function. *Journal of Applied Developmental Psychology, 58*, 77-85. https://doi.org/10.1016/j.appdev.2018.04.007

Elkins, I. J., McGue, M., & Iacono, W. G. (1997). Genetic and Environmental Influences on Parent-Son Relationships: Evidence for Increasing Genetic Influence During Adolescence. *Dev Psychol, 33*(2), 351-363. https://doi.org/10.1037//0012-1649.33.2.351

Engelhardt, L. E., Briley, D. A., Mann, F. D., Harden, K. P., & Tucker-Drob, E. M. (2015). Genes Unite Executive Functions in Childhood. *Psychol Sci, 26*(8), 1151-1163. https://doi.org/10.1177/0956797615577209

Evans, G. W. (2004). The Environment of Childhood Poverty. *Am Psychol, 59*(2), 77-92. https://doi.org/10.1037/0003-066X.59.2.77

Evans, G. W., Farah, M. J., & Hackman, D. A. (2021). Early Childhood Poverty and Adult Executive Functioning: Distinct, Mediating Pathways for Different Domains of Executive Functioning. *Dev Sci*, e13084. https://doi.org/10.1111/desc.13084

Falk, E. B., Hyde, L. W., Mitchell, C., Faul, J., Gonzalez, R., Heitzeg, M. M., Keating, D. P., Langa, K. M., Martz, M. E., Maslowsky, J., Morrison, F. J., Noll, D. C., Patrick, M. E., Pfeffer, F. T., Reuter-Lorenz, P. A., Thomason, M. E., Davis-Kean, P., Monk, C. S., & Schulenberg, J. (2013). What Is a Representative Brain? Neuroscience Meets Population Science. *Proc Natl Acad Sci U S A, 110*(44), 17615-17622. https://doi.org/10.1073/pnas.1310134110

Fay-Stammbach, T., Hawes, D. J., & Meredith, P. (2014). Parenting Influences on Executive Function in Early Childhood: A Review. *Child Development Perspectives, 8*(4), 258-264. https://doi.org/10.1111/cdep.12095

Finegood, E. D., Rarick, J. R. D., Blair, C., & Family Life Project, I. (2017). Exploring Longitudinal Associations between Neighborhood Disadvantage and Cortisol Levels in Early Childhood. *Dev Psychopathol, 29*(5), 1649-1662. https://doi.org/10.1017/S0954579417001304

Friedman, N. P., & Miyake, A. (2017). Unity and Diversity of Executive Functions: Individual Differences as a Window on Cognitive Structure. *Cortex, 86*, 186-204. https://doi.org/10.1016/j.cortex.2016.04.023

Friedman, N. P., Miyake, A., Young, S. E., DeFries, J. C., Corley, R. P., & Hewitt, J. K. (2008). Individual Differences in Executive Functions Are Almost Entirely Genetic in Origin. *J Exp Psychol Gen, 137*(2), 201-225. https://doi.org/10.1037/0096-3445.137.2.201

Gard, A. M., Maxwell, A. M., Shaw, D. S., Mitchell, C., Brooks-Gunn, J., McLanahan, S. S., Forbes, E. E., Monk, C. S., & Hyde, L. W. (2021). Beyond Family-Level Adversities: Exploring the Developmental Timing of Neighborhood Disadvantage Effects on the Brain. *Dev Sci, 24*(1), e12985. https://doi.org/10.1111/desc.12985

Gotlib, I. H., & Joormann, J. (2010). Cognition and Depression: Current Status and Future Directions. *Annu Rev Clin Psychol, 6*, 285-312. https://doi.org/10.1146/annurev.clinpsy.121208.131305

Hackman, D. A., Betancourt, L. M., Gallop, R., Romer, D., Brodsky, N. L., Hurt, H., & Farah, M. J. (2014). Mapping the Trajectory of Socioeconomic Disparity in Working Memory: Parental and Neighborhood Factors. *Child Dev, 85*(4), 1433-1445. https://doi.org/10.1111/cdev.12242

Hackman, D. A., Cserbik, D., Chen, J. C., Berhane, K., Minaravesh, B., McConnell, R., & Herting, M. M. (2021). Association of Local Variation in Neighborhood Disadvantage in Metropolitan Areas with Youth Neurocognition and Brain Structure. *JAMA Pediatr, 175*(8), e210426. https://doi.org/10.1001/jamapediatrics.2021.0426

Hammond, S. I., Muller, U., Carpendale, J. I., Bibok, M. B., & Liebermann-Finestone, D. P. (2012). The Effects of Parental Scaffolding on Preschoolers' Executive Function. *Dev Psychol, 48*(1), 271-281. https://doi.org/10.1037/a0025519

Helm, A. F., McCormick, S. A., Deater-Deckard, K., Smith, C. L., Calkins, S. D., & Bell, M. A. (2020). Parenting and Children's Executive Function Stability across the Transition to School. *Infant Child Dev, 29*(1). https://doi.org/10.1002/icd.2171

Hughes, C., & Devine, R. T. (2019). For Better or for Worse? Positive and Negative Parental Influences on Young Children's Executive Function. *Child Dev, 90*(2), 593-609. https://doi.org/10.1111/cdev.12915

Hunt, J. F. V., Vogt, N. M., Jonaitis, E. M., Buckingham, W. R., Koscik, R. L., Zuelsdorff, M., Okonkwo, O. C., Clark, L. R., Johnson, S. C., Asthana, S., Bendlin, B. B., & Kind, A. J. (2020). Neighborhood Disadvantage Is Associated with Accelerated Cortical Thinning and Cognitive Decline in Cognitively Unimpaired Adults. *Alzheimer's & Dementia, 16*(S10). https://doi.org/10.1002/alz.043170

Hyde, L. W., Gard, A. M., Tomlinson, R. C., Burt, S. A., Mitchell, C., & Monk, C. S. (2020). An Ecological Approach to Understanding the Developing Brain: Examples Linking Poverty, Parenting, Neighborhoods, and the Brain. *Am Psychol, 75*(9), 1245-1259. https://doi.org/10.1037/amp0000741

Kao, K., Nayak, S., Doan, S. N., & Tarullo, A. R. (2018). Relations between Parent Ef and Child Ef: The Role of Socioeconomic Status and Parenting on Executive Functioning in Early Childhood. *Translational Issues in Psychological Science, 4*(2), 122-137. https://doi.org/10.1037/tps0000154

Karr, J. E., Areshenkoff, C. N., Rast, P., Hofer, S. M., Iverson, G. L., & Garcia-Barrera, M. A. (2018). The Unity and Diversity of Executive Functions: A Systematic Review and Re-Analysis of Latent Variable Studies. *Psychol Bull, 144*(11), 1147-1185. https://doi.org/10.1037/bul0000160

Knopik, V. S., Neiderhiser, J. M., DeFries, J. C., & Plomin, R. (2017). *Behavioral Genetics* (7th ed.). Worth Publishers.

Kolb, B., Mychasiuk, R., Muhammad, A., Li, Y., Frost, D. O., & Gibb, R. (2012). Experience and the Developing Prefrontal Cortex. *Proc Natl Acad Sci U S A, 109 Suppl 2*, 17186-17193. https://doi.org/10.1073/pnas.1121251109

Korucu, I., Litkowski, E., Purpura, D. J., & Schmitt, S. A. (2020). Parental Executive Function as a Predictor of Parenting Practices and Children's Executive Function. *Infant and Child Development, 29*(1). https://doi.org/10.1002/icd.2152

Kubzansky, L. D., Martin, L. T., & Buka, S. L. (2009). Early Manifestations of Personality and Adult Health: A Life Course Perspective. *Health Psychol, 28*(3), 364-372. https://doi.org/10.1037/a0014428

Last, B. S., Lawson, G. M., Breiner, K., Steinberg, L., & Farah, M. J. (2018). Childhood Socioeconomic Status and Executive Function in Childhood and Beyond. *PLoS One, 13*(8), e0202964. https://doi.org/10.1371/journal.pone.0202964

Lawson, G. M., Hook, C. J., & Farah, M. J. (2018). A Meta-Analysis of the Relationship between Socioeconomic Status and Executive Function Performance among Children. *Dev Sci, 21*(2). https://doi.org/10.1111/desc.12529

Lerche, V., & Voss, A. (2017). Retest Reliability of the Parameters of the Ratcliff Diffusion Model. *Psychol Res, 81*(3), 629-652. https://doi.org/10.1007/s00426-016-0770-5

Leventhal, T., & Brooks-Gunn, J. (2000). The Neighborhoods They Live In: The Effects of Neighborhood Residence on Child and Adolescent Outcomes. *Psychological Bulletin, 126*(2), 309-337. https://doi.org/10.1037/0033-2909.126.2.309

Li, J. B., Willems, Y. E., Stok, F. M., Dekovic, M., Bartels, M., & Finkenauer, C. (2019). Parenting and Self-Control across Early to Late Adolescence: A Three-Level Meta-Analysis. *Perspect Psychol Sci, 14*(6), 967-1005. https://doi.org/10.1177/1745691619863046

Li, J. J., & Roberts, D. K. (2017). Genetic Influences on Executive Functions across the Life Span. In S. A. Wiebe & J. Karbach (Eds.), *Executive Function*. Routledge. https://doi.org/10.4324_9781315160719-8

Manuck, S. B., & McCaffery, J. M. (2014). Gene-Environment Interaction. *Annu Rev Psychol, 65*, 41-70. https://doi.org/10.1146/annurev-psych-010213-115100

Marcovitch, S., Jacques, S., Boseovski, J. J., & Zelazo, P. D. (2008). Self-Reflection and the Cognitive Control of Behavior: Implications for Learning. *Mind, Brain, and Education, 2*(3), 136-141. https://doi.org/10.1111/j.1751-228X.2008.00044.x

McClelland, M. M., Cameron, C. E., Connor, C. M., Farris, C. L., Jewkes, A. M., & Morrison, F. J. (2007). Links between Behavioral Regulation and Preschoolers' Literacy, Vocabulary, and Math Skills. *Dev Psychol, 43*(4), 947-959. https://doi.org/10.1037/0012-1649.43.4.947

McEwen, B. S. (2004). Protection and Damage from Acute and Chronic Stress: Allostasis and Allostatic Overload and Relevance to the Pathophysiology of Psychiatric Disorders. *Ann N Y Acad Sci, 1032*, 1-7. https://doi.org/10.1196/annals.1314.001

McGuire, S. (2003). The Heritability of Parenting. *Parenting-Science and Practice, 3*(1), 73-94. https://doi.org/Pii 785828830
10.1207/S15327922par0301_04

Mischel, W., Ayduk, O., Berman, M. G., Casey, B. J., Gotlib, I. H., Jonides, J., Kross, E., Teslovich, T., Wilson, N. L., Zayas, V., & Shoda, Y. (2011). 'Willpower' over the Life Span: Decomposing Self-Regulation. *Soc Cogn Affect Neurosci, 6*(2), 252-256. https://doi.org/10.1093/scan/nsq081

Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The Unity and Diversity of Executive Functions and Their Contributions to Complex "Frontal Lobe" Tasks: A Latent Variable Analysis. *Cogn Psychol, 41*(1), 49-100. https://doi.org/10.1006/cogp.1999.0734

Moffitt, T. E. (2018). Male Antisocial Behaviour in Adolescence and Beyond. *Nature Human Behaviour, 2*(3), 177-186. https://doi.org/10.1038/s41562-018-0309-4

Moffitt, T. E., Arseneault, L., Belsky, D., Dickson, N., Hancox, R. J., Harrington, H., Houts, R., Poulton, R., Roberts, B. W., Ross, S., Sears, M. R., Thomson, W. M., & Caspi, A.

(2011). A Gradient of Childhood Self-Control Predicts Health, Wealth, and Public Safety. *Proc Natl Acad Sci U S A, 108*(7), 2693-2698. https://doi.org/10.1073/pnas.1010076108

Murtha, K., Larsen, B., Pines, A., Parkes, L., Moore, T. M., Adebimpe, A., Alexander-Bloch, A., Calkins, M. E., Davila, D. G., Lindquist, M. A., Mackey, A. P., Roalf, D. R., Scott, J. C., Wolf, D. H., Gur, R. C., Gur, R. E., Barzilay, R., & Satterthwaite, T. D. (2021). https://doi.org/10.1101/2021.08.12.454903

Narusyte, J., Neiderhiser, J. M., D'Onofrio, B. M., Reiss, D., Spotts, E. L., Ganiban, J., & Lichtenstein, P. (2008). Testing Different Types of Genotype-Environment Correlation: An Extended Children-of-Twins Model. *Dev Psychol, 44*(6), 1591-1603. https://doi.org/10.1037/a0013911

Rakesh, D., Cropley, V., Zalesky, A., Vijayakumar, N., Allen, N. B., & Whittle, S. (2021). Neighborhood Disadvantage and Longitudinal Brain-Predicted-Age Trajectory During Adolescence. *Dev Cogn Neurosci, 51*, 101002. https://doi.org/10.1016/j.dcn.2021.101002

Rakesh, D., Seguin, C., Zalesky, A., Cropley, V., & Whittle, S. (2021). Associations between Neighborhood Disadvantage, Resting-State Functional Connectivity, and Behavior in the Adolescent Brain Cognitive Development Study: The Moderating Role of Positive Family and School Environments. *Biol Psychiatry Cogn Neurosci Neuroimaging*. https://doi.org/10.1016/j.bpsc.2021.03.008

Ramphal, B., DeSerisy, M., Pagliaccio, D., Raffanello, E., Rauh, V., Tau, G., Posner, J., Marsh, R., & Margolis, A. E. (2020). Associations between Amygdala-Prefrontal Functional Connectivity and Age Depend on Neighborhood Socioeconomic Status. *Cereb Cortex Commun, 1*(1), tgaa033. https://doi.org/10.1093/texcom/tgaa033

Ratcliff, R., & Rouder, J. N. (1998). Modeling Response Times for Two-Choice Decisions. *Psychological Science, 9*(5), 347-356. https://doi.org/Doi 10.1111/1467-9280.00067

Raver, C. C., Blair, C., & Willoughby, M. (2013). Poverty as a Predictor of 4-Year-Olds' Executive Function: New Perspectives on Models of Differential Susceptibility. *Dev Psychol, 49*(2), 292-304. https://doi.org/10.1037/a0028343

Rochette, É., & Bernier, A. (2014). Parenting, Family Socioeconomic Status, and Child Executive Functioning: A Longitudinal Study. *Merrill-Palmer Quarterly, 60*(4). https://doi.org/10.13110/merrpalmquar1982.60.4.0431

Sameroff, A. J. (2009). The Transactional Model. In A. J. Sameroff (Ed.), *The Transactional Model of Development: How Children and Contexts Shape Each Other.* (pp. 3-21). American Psychological Association. https://doi.org/10.1037/11877-001

Seeyave, D. M., Coleman, S., Appugliese, D., Corwyn, R. F., Bradley, R. H., Davidson, N. S., Kaciroti, N., & Lumeng, J. C. (2009). Ability to Delay Gratification at Age 4 Years and Risk of Overweight at Age 11 Years. *Arch Pediatr Adolesc Med, 163*(4), 303-308. https://doi.org/10.1001/archpediatrics.2009.12

Shameem, F., & Hamid, S. (2014). Socioeconomic Status and Adolescent Aggression: The Role of Executive Functioning as a Mediator. *Am J Psychol, 127*(4), 419-430. https://doi.org/10.5406/amerjpsyc.127.4.0419

Smith, J. L., Mattick, R. P., Jamadar, S. D., & Iredale, J. M. (2014). Deficits in Behavioural Inhibition in Substance Abuse and Addiction: A Meta-Analysis. *Drug Alcohol Depend, 145*, 1-33. https://doi.org/10.1016/j.drugalcdep.2014.08.009

Sripada, C., & Weigard, A. (2021). Impaired Evidence Accumulation as a Transdiagnostic Vulnerability Factor in Psychopathology. *Front Psychiatry, 12*, 627179. https://doi.org/10.3389/fpsyt.2021.627179

Taylor, R. L., Cooper, S. R., Jackson, J. J., & Barch, D. M. (2020). Assessment of Neighborhood Poverty, Cognitive Function, and Prefrontal and Hippocampal Volumes in Children. *JAMA Netw Open, 3*(11), e2023774. https://doi.org/10.1001/jamanetworkopen.2020.23774

Tomlinson, R. C., Burt, S. A., Waller, R., Jonides, J., Miller, A. L., Gearhardt, A. N., Peltier, S. J., Klump, K. L., Lumeng, J. C., & Hyde, L. W. (2020). Neighborhood Poverty Predicts Altered Neural and Behavioral Response Inhibition. *Neuroimage, 209*, 116536. https://doi.org/10.1016/j.neuroimage.2020.116536

Verdejo-Garcia, A., Lawrence, A. J., & Clark, L. (2008). Impulsivity as a Vulnerability Marker for Substance-Use Disorders: Review of Findings from High-Risk Research, Problem Gamblers and Genetic Association Studies. *Neurosci Biobehav Rev, 32*(4), 777-810. https://doi.org/10.1016/j.neubiorev.2007.11.003

Webb, E. K., Weis, C. N., Huggins, A. A., Parisi, E. A., Bennett, K. P., Miskovich, T., Krukowski, J., deRoon-Cassini, T. A., & Larson, C. L. (2021). Neighborhood Disadvantage Is Associated with Stable Deficits in Neurocognitive Functioning in Traumatically-Injured Adults. *Health Place, 67*, 102493. https://doi.org/10.1016/j.healthplace.2020.102493

Weigard, A., Clark, D. A., & Sripada, C. (2021). Cognitive Efficiency Beats Top-Down Control as a Reliable Individual Difference Dimension Relevant to Self-Control. *Cognition, 215*, 104818. https://doi.org/10.1016/j.cognition.2021.104818

Weigard, A., & Sripada, C. (2021). Task-General Efficiency of Evidence Accumulation as a Computationally Defined Neurocognitive Trait: Implications for Clinical Neuroscience. *Biological Psychiatry Global Open Science, 1*(1), 5-15. https://doi.org/10.1016/j.bpsgos.2021.02.001

Weigard, A. S., Brislin, S. J., Cope, L. M., Hardee, J. E., Martz, M. E., Ly, A., Zucker, R. A., Sripada, C., & Heitzeg, M. M. (2021). Evidence Accumulation and Associated Error-Related Brain Activity as Computationally-Informed Prospective Predictors of Substance Use in Emerging Adulthood. *Psychopharmacology (Berl)*. https://doi.org/10.1007/s00213-021-05885-w

Wiecki, T. V., Poland, J., & Frank, M. J. (2015). Model-Based Cognitive Neuroscience Approaches to Computational Psychiatry. *Clinical Psychological Science, 3*(3), 378-399. https://doi.org/10.1177/2167702614565359

Wright, L., Lipszyc, J., Dupuis, A., Thayapararajah, S. W., & Schachar, R. (2014). Response Inhibition and Psychopathology: A Meta-Analysis of Go/No-Go Task Performance. *J Abnorm Psychol, 123*(2), 429-439. https://doi.org/10.1037/a0036295

Zelazo, P. D., & Carlson, S. M. (2012). Hot and Cool Executive Function in Childhood and Adolescence: Development and Plasticity. *Child Development Perspectives*, n/a-n/a. https://doi.org/10.1111/j.1750-8606.2012.00246.x

Zeytinoglu, S., Calkins, S. D., Swingler, M. M., & Leerkes, E. M. (2017). Pathways from Maternal Effortful Control to Child Self-Regulation: The Role of Maternal Emotional Support. *J Fam Psychol, 31*(2), 170-180. https://doi.org/10.1037/fam0000271

CHAPTER II

Measuring Task-General Executive Functioning in Children Using Efficiency of Evidence Accumulation

Executive functioning in childhood is important for both child and adult outcomes, including literacy, vocabulary, math skills, and mental and physical health (Blair & Razza, 2007; Marcovitch et al., 2008; McClelland et al., 2007; Mischel et al., 2011; Moffitt et al., 2011). Executive functioning is thought to be a set of top-down processes that involve regulation of behavior when autopilot is not sufficient (Diamond, 2013), though there are many other terms often used to capture similar constructs (e.g. self-regulation, effortful control; Nigg, 2017). Deficits in executive functioning have long been linked to various forms of psychopathology (Bloemen et al., 2018; Gotlib & Joormann, 2010; Moffitt, 2018; Smith et al., 2014; Verdejo-Garcia et al., 2008; Wright et al., 2014). Thus, understanding the factors that lead to individual differences in executive functioning is of interest to clinical psychology.

Studies of individual differences in executive functioning tend to assume that executive functioning is made up of several intercorrelated, but distinct, subcomponents (Sripada & Weigard, 2021). Perhaps the most prominent theory of executive functioning, the "unity-yet-diversity" theory (Miyake et al., 2000), subdivides executive functioning into "shifting", "updating", and "inhibition" functions. This framework is based on and supported by years of factor analytic work (Friedman & Miyake, 2017; Miyake et al., 2000). A proliferation of tasks have thus been designed to capture individual performance within subcomponents of executive functioning (e.g. the go/no-go task measures "inhibition"; Packwood et al., 2011). Such tasks,

designed to assess specific subcomponents of executive functioning, have been used extensively in clinical neuropsychology (Packwood et al., 2011) as well as in clinical neuroscience research (e.g. Hull et al., 2008; Lee et al., 2013; Metin et al., 2012; Smith et al., 2014; Wright et al., 2014). However, this focus on subdivision, though pervasive, has not led to consensus regarding specific links between executive functioning and psychopathology.

Indeed, there is a striking level of disagreement regarding the exact structure of the subcomponents of executive functioning (Lee et al., 2013; McKenna et al., 2017; Packwood et al., 2011) and there has been little success consistently relating any one subcomponent of executive functioning to one specific form of psychopathology (see Karr et al., 2018 for a systematic review). Additionally, metrics traditionally extracted from these subcomponent executive functioning tasks suffer from test-retest reliability issues and do not consistently relate to real-world outcomes such as mental health, physical health, or income (Eisenberg et al., 2019). These issues with validity and reliability raise questions regarding the clinical utility of task performance on these subdivided executive functioning tasks: for example, though attention deficit/hyperactivity disorder (ADHD) is thought to be a disorder of executive dysfunction, there is substantial heterogeneity in profiles of executive functioning performance for children with ADHD when using traditional metrics on experimental and neuropsychological tasks (Boonstra et al., 2005; Lambek et al., 2010; Metin et al., 2012; Packwood et al., 2011). At the same time, the ongoing NIMH Research Domain Criteria (RDoC) project has emphasized the importance of identifying reliable cross-cutting metrics with clear biological bases (Cuthbert & Insel, 2013). To complement ongoing work into the underlying structure of executive functioning, it may be helpful to consider whether task-general, rather than subdivided, executive functioning metrics have potential as transdiagnostic correlates of psychopathology (Weigard & Sripada, 2021).

Model-based measurement of EF

Though less frequently used in clinical neuroscience, model-based approaches popular in computational psychology may be able to improve prediction of clinical outcomes by providing richer information about cognitive performance than traditional metrics (Weigard, Brislin, et al., 2021; Wiecki et al., 2015). Indeed, traditional executive functioning metrics may oversimplify the complex neural processes underlying task performance. Any single dependent variable provides an incomplete picture of task performance, as these metrics (e.g., accuracy, reaction time) are being measured simultaneously (Huang-Pollock et al., 2017; Ratcliff, 2002). Instead, a model-based approach, like the drift diffusion model, can quantify task performance as a broader decision process, accounting for both speed and reaction time (Huang-Pollock et al., 2017; Ratcliff, 2002; Ratcliff et al., 2018). The drift diffusion model assumes that evidence driving a decision (i.e. a choice on a two-choice task) is accumulated continuously over time (Ratcliff & Rouder, 1998). This model has been successfully applied to the go/no-go (Ratcliff et al., 2018) and stop signal (Nigg et al., 2018) tasks commonly used in the clinical literature. Crucially, this model has biological plausibility: diffusion model parameters have been shown to relate to brain activity (Turner et al., 2017; Weigard et al., 2020) and the diffusion model reflects what is known about the spiking behavior of neurons (Killeen et al., 2013; Ratcliff & McKoon, 2008). Thus, though model-based approaches have not yet been widely adopted in clinical neuroscience, they show promise as a method for generating task-general executive functioning metrics.

There is recent evidence that one model-based metric in particular, the "efficiency of evidence accumulation" (EEA), calculated from drift rate in the drift diffusion model, may represent a task-general individual difference that contributes to cognitive impairments across various forms of psychopathology in adults (Weigard & Sripada, 2021). EEA describes the rate

at which a person gathers relevant evidence among background noise in order to make an accurate choice (Weigard & Sripada, 2021). EEA exhibits good test-retest reliability (Lerche & Voss, 2017), and has recently been shown to outperform traditional metrics both in temporal stability and in prediction of psychopathology in adults (Weigard, Clark, et al., 2021). Additionally, recent work suggests clear clinical relevance for EEA, with evidence that reduced EEA in adults may be a transdiagnostic risk factor associated with schizophrenia, bipolar disorder, and ADHD, as well as a global severity of psychopathology (Sripada & Weigard, 2021). EEA and related brain activation have also been shown to predict substance use behaviors in adults (Weigard, Brislin, et al., 2021). Thus, a growing constellation of evidence points to EEA as a promising task-general transdiagnostic correlate of psychopathology in adults.

Less is known about the utility of the EEA paradigm in children. However, there is promising evidence that drift rate, an overlapping construct, is impaired in clinical samples of children with ADHD (Huang-Pollock et al., 2020; Karalunas et al., 2014; Weigard et al., 2018). Validation of the EEA paradigm in community samples of children is a crucial step toward understanding its role as a transdiagnostic process. In order to be considered a transdiagnostic process for psychopathology in children, EEA must meet some basic criteria: it should be reliable, and it should show concurrent and predictive validity (Cronbach & Meehl, 1955; Cuthbert & Insel, 2013; Smith, 2005). First, because EEA theoretically indexes a relatively stable individual difference, EEA should show stability over testing occasions, or good test-retest reliability (Cronbach & Meehl, 1955). Second, EEA should show construct validity, indexed via its relation to other constructs in predictable ways, both concurrently and prospectively (Cronbach & Meehl, 1955; Smith, 2005). Like in adults, this validity can be assessed via the relation between individual differences in EEA and concurrent and/or prospective symptoms of

psychopathology (Sripada & Weigard, 2021; Weigard, Brislin, et al., 2021). Finally, in the spirit of the integrative RDoC framework, the behavioral EEA metric should have a substantive link to underlying neural circuitry (Cuthbert & Insel, 2013), which could be demonstrated via a correlation with EF-related brain activation (Weigard et al., 2020).

Present Study

The present study examined the potential utility of EEA as a transdiagnostic risk factor for psychopathology in a large community sample of twins. We utilized go/no-go and stop signal data from the Michigan Twin Neurogenetics Study (MTwiNS), a unique longitudinal twin study (N=354 pairs) with oversampling for twins living in low-income neighborhoods. We calculated several traditional metrics from both tasks for comparison to a task-general EEA metric. First, we tested the hypothesis that EEA would demonstrate better test-retest reliability than traditional metrics. Second, we tested the hypothesis that EEA would show acceptable construct validity. We tested construct validity via concurrent and prospective correlations with parent- and child-reported symptoms of several forms of psychopathology, including attention problems and a global psychopathology score. Finally, we tested the hypothesis that EEA would relate to brain circuitry, via its correlation with inhibition-related brain activation within inhibitory (i.e., inferior frontal gyrus, IFG) and error-monitoring (i.e., anterior cingulate cortex, ACC) regions.

Methods

Participants

The present study included data from the Michigan Twins Neurogenetics Study (MTwiNS), a longitudinal neuroimaging project within the large-scale Michigan State University Twin Registry (see Burt & Klump, 2019). The 354 families participating in MTwiNS were

originally identified through birth records. Families were recruited into two cohorts — a population-based cohort that represented families living within 120 miles of Michigan State University and an at-risk cohort recruited from the same area, but only including families living in U.S. Census tracts where at least 10.5% of families lived below the poverty line (the mean for the state of Michigan at the time; Burt & Klump, 2019). MTwiNS participants were re-recruited from the original participant pool based upon criteria for the second, "at-risk" cohort. Thus, MTwiNS includes families from the "at-risk" cohort as well as those from the population-based cohort that would have qualified for the "at-risk" cohort. This re-recruitment strategy yielded a sample representative of families living in neighborhoods with above average levels of poverty, a unique sampling frame within both the behavioral genetics and neuroimaging literatures (Burt et al., 2021).

The 637 twins (from 337 pairs, 300 complete, 128 monozygotic) included in the present study successfully completed at least one of two behavioral executive functioning tasks during their first MTwiNS visit. Of these 637 twins, 325 (from 174 pairs, 151 complete, 71 monozygotic) participated in a second visit at least one year after their first visit. The twins were 7 to 19 years old upon first visit (Mean=15.7, SD=2.2; 54.5% male; less than 2% of the present sample was 10 or younger) and 10 to 19 years old upon second visit (Mean=14.7, SD=2.1; 54.5% male). The breakdown of twins' parent-reported ethnicity reflected the surrounding area (80% White, 12% Black, 5% Other, 1% Latino/Latina, 1% Pacific Islander, 1% Native American, 1% Asian). Median reported family annual income for this sample was $70,000 to $79,999 and ranged from less than $4,999 to greater than $90,000. 11% of included children were from families reporting an annual income below the 2020 federal poverty line of $26,246 per year and 53% below the living wage for a family of 4 in Michigan (with one parent working;

http://livingwage.mit.edu/states/26). Zygosity was established using physical similarity questionnaires (administered to the twins and/or their parents) that show accuracies of 95% or better (Bouchard et al., 1990; Iacono et al., 1999; Peeters et al., 1998). Discrepancies were resolved through review of zygosity items or by DNA markers. Parents provided informed consent and children provided assent in compliance with the policies of the Institutional Review Board of the University of Michigan.

Procedure

Twins and their primary caregivers took part in a day-long visit to the University of Michigan which included a mock scan as well as a one-hour blood-oxygen-level-dependent (BOLD) fMRI scan for each twin. Twins completed several tasks in the scanner, including a go/no-go task. Outside of the scanner, twins completed several additional computer tasks, including a stop signal task (SST), and a battery of child-report questionnaires. Primary caregivers completed a demographic interview with an examiner and a battery of self- and parent-report questionnaires. Return visits, which take place roughly a year after the first visit (M = 1.33 years, SD = 0.44 years, Range = 0.67 – 3.00), are ongoing and follow a nearly identical protocol.

Measures

Go/No-Go Task

The go/no-go task used in this study was adapted from Casey et al. (1997), in which neural reactivity during inhibition is elicited via a "whack-a-mole" game (stimuli courtesy of Sarah Getz and the Sackler Institute for Developmental Psychobiology; task downloaded from http://fablab.yale.edu/page/assays-tools). In the present task, participants were instructed to press a button as quickly as possible in response to one stimulus ("go", a mole) and avoid responding

to a less frequent non-target ("No-Go", a vegetable). The target stimuli (moles) were modified with various "disguises" to make the task more interesting and difficult given the relatively slow speed of stimuli due to MRI scanning requirements. The task consisted of four runs, each with approximately 55 trials, for a total of 255 trials of which 55 were no-go (21.6% no-go). Each no-go trial was preceded by 1-5 go trials. Each trial lasted 2300ms, including a maximum of 1800ms stimulus presentation, 400ms feedback, and 100 – 1000ms fixation to account for individual differences in reaction time. Participants practiced the task briefly in an MRI simulator before the MRI scan.

In order to thoroughly compare EEA to traditional metrics, we extracted several frequently used metrics from the go/no-task. **No-go Accuracy** was calculated as the percent of "no-go" trials for which a participant correctly avoided responding. **Go Accuracy** was calculated as the percent of "go" trials for which a participant correctly responded. **Mean Reaction Time** was calculated as the average reaction time for "go" trials, excluding failed "go" trials (no response). **Standard Deviation of Reaction Time** was calculated as the standard deviation of reaction time for "go" trials, excluding failed "go" trials. **Inhibitory efficiency** (Tomlinson et al., 2020) was calculated by dividing accuracy by mean reaction time. This measure was designed to account for the potential of two individuals to obtain the same accuracy score while one individual trades reaction time for accuracy. Participants with overall below-chance performance (<55%), below-chance performance on "go" trials (<55%), or with more than 5% of trials implausibly "fast guesses" (<150ms; Voss et al., 2013) were not considered to be meaningfully participating in the go/no-go task, and were therefore excluded from go/no-go analyses (N=13 first visit, N=5 second visit), leaving N=589 individuals with viable go/no-go data at the first visit, and N=307 individuals with viable go/no-go data at the second visit.

Stop-Signal Task

The child-friendly stop-signal task used in this study was a 10 minute, 150 trial task adapted from Bissett & Logan (2012) as described previously (Begolli et al., 2018). Participants were presented with a fish for 850ms and told to push the "a" or "l" keys as quickly as possible based on the orientation of the fish, unless a visual stop-signal stimulus (Martin the Manta Ray) appeared on the screen, which occurred on 50 of the task trials (33%; "stop" trials). This stop-signal was presented following a stop signal delay (SSD) that was determined through a standard "staircase tracking" algorithm (Logan, 1994) designed to lead to a roughly 50% probability of inhibition on "stop" trials for each participant. This algorithm began with a 250ms SSD and was thereafter increased or decreased by 50ms on each subsequent "stop" trial based on whether the participant was able to successfully inhibit. Participants with overall below-chance performance (<55%), below-chance performance on "go" trials (<55%), a high non-response rate on "go" trials (>25%), or with more than 5% of trials implausibly "fast guesses" (<150ms; Voss et al., 2013) were not considered to be meaningfully participating in the task and were therefore excluded from stop-signal analyses (N=59 first visit, N=63 second visit), leaving N=335 individuals with viable stop-signal data at the first visit, and N=295 individuals at the second visit. A number of participants who did complete go/no-go did not have the opportunity to complete stop-signal upon first visit (N=254) as the stop-signal task was added to the protocol partway through data collection. These participants were still included in all possible analyses given viable go/no-go data (N=253). The same metrics that were extracted from the go/no-go task were extracted from the stop-signal task, to the extent possible due to differing task designs (i.e. No-Go Accuracy and Efficiency are not meaningful for this task due to the stair stepping procedure). Due to a programming error, two slightly different task versions were used; to

account for this difference, task version was regressed out of all metrics extracted from the stop-signal task before continuing with analyses.

Psychopathology

Symptoms of psychopathology were assessed via parent- (primary caregiver) and child-report on the clinically-oriented subscales of the Child Behavior Checklist and Youth Self Report (CBCL and YSR; Achenbach & Rescorla, 2001). Subscales of interest included the total problems, externalizing problems, internalizing problems, attention problems, anxious/depressed problems, withdrawn/depressed problems, rule breaking, and aggressive behavior subscales. We used a combined informant approach, creating a composite of all available family informants (i.e. mother, father, child; Burt et al., 2006). Such an approach allows for a more complete assessment of symptoms than would the use of any single informant (Achenbach et al., 1987). Z-scored raw symptom counts for each subscale were averaged across the available informants to create each composite. When only one informant report was available, that report was used for analyses.

Functional neuroimaging

Functional imaging data were acquired using one of two GE Discovery MR750 3T scanners located at the University of Michigan Functional MRI Laboratory. To leverage improvements in MRI data acquisition and to be consistent with the Adolescent Brain Cognitive Development (ABCD) Study (Casey et al., 2018), we altered our acquisition protocol after the first 140 MtwiNS families. For the first 140 MtwiNS families, one run of 284 volumes was collected for each participant using an 8-channel head coil. BOLD functional images were acquired using a gradient-echo reverse spiral sequence (repetition time = 2000ms, echo time = 30ms, flip angle = 90°, FOV = 22cm). Images included 43 interleaved oblique slices of 3mm

thickness with 3.44x3.44mm^2 in-plane resolution. High-resolution T1-weighted SPGR images (156 slices, slice thickness = 1mm, in plane resolution of 1x1mm^2) were aligned with the AC-PC plane and used during normalization of the functional images. For the remaining MtwiNS participants (families 141-354), one run of 685 volumes was collected for each participant. BOLD functional images were acquired using a gradient-echo multiband sequence (repetition time = 800ms, echo time = 30ms, flip angle = 52°, FOV = 21.6 cm) with a 32-channel head coil, which covered 697 interleaved axial slices of 2.4 mm thickness. High-resolution T1-weighted SPGR images (208 slices, slice thickness = 1 mm) were aligned with the AC-PC plane and used during normalization of the functional images. Scan protocol was included as a nuisance regressor in fMRI analyses.

Preprocessing for both acquisition sequences were identical unless otherwise specified. Functional data were preprocessed and analyzed using Statistical Parametric Mapping software version 12 (SPM12; Wellcome Centre for Human Neuroimaging). The first four volumes of each run were discarded to allow for stabilization of the MR signal. Raw k-space data were de-spiked before reconstruction to image space in reverse-spiral sequence acquisition. For gradient-echo sequence data with multiband acquisition, task-specific field maps were constructed from volumes of both anterior-to-posterior and posterior-to-anterior phase encoding; field maps were applied after image construction to reduce spatial distortions and minimize movement artifacts. Slice timing correction was performed using the median slice as the reference slice. Functional data were then spatially realigned to the 10th slice of the volume. These spatially realigned data were coregistered to a high-resolution T1-weighted image, segmented, and spatially normalized into standard stereotactic space to the Montreal Neurological Institute (MNI) template. For participants with unusable high-resolution T1-weighted structural images (N=4), we spatially

realigned using the T1-weighted overlay image (first 140 families: 43 slices, slice thickness=3mm, in plane resolution of 0.85x0.85mm^2; families 141-354: 60 slices, slice thickness = 2.4mm, in plane resolution of 0.9x0.9mm^2). Finally, functional data were smoothed using a 6mm Gaussian kernel.

After preprocessing, the Artifact detection Tools toolbox (ART; https://www.nitrc.org/projects/artifact_detect/) was used to detect translation or rotational motion outlier volumes that remained after earlier QA (>2mm movement or 3.5 rotation) and to scrub them from the dataset. Preprocessed images were also visually inspected for major artifacts. Coverage of the frontal lobe was checked using the WFU PickAtlas "frontal lobe" structural mask (Maldjian et al., 2003). A participant's fMRI data were considered unusable if they contained obvious prefrontal artifacts, had less than 90% coverage of the frontal mask, or had more than 5% of scans identified as motion outliers. Preprocessing was conducted in containerized versions of SPM12, and the standard pipeline is accessible via Github (https://github.com/UMich-Mind-Lab/pipeline-task-standard). After quality checks, 549 participants with behavioral data had usable fMRI data (Table II.1).

Functional data were modeled using the general linear model in SPM12. Three conditions were modeled: correct No-Go trials, in which a participant correctly withheld a response to a No-Go stimulus; incorrect No-Go trials, in which a participant incorrectly responded to a No-Go stimulus; and Go trials, in which a participant saw a Go stimulus. Incorrect Go trials were not modeled due to the expected high hit rates for Go trials (median 100%). For each participant, the main contrast of interest was all No-Go (including both correct and incorrect) > Go.

Analytic Plan

Efficiency of Evidence Accumulation (EEA)

Drift Diffusion Model Analysis. Drift rate parameters from a drift diffusion model were calculated using Bayesian methods for each condition and task (i.e. no-go, go, stop-signal) using the Dynamic Models of Choice functions (Heathcote, 2019), an adaptation of the differential evolution Markov chain Monte Carlo model (Turner et al., 2013), in R version 4.1.0 (R Core Team, 2021). Trials with implausibly fast response rates (<150ms; Voss et al., 2013) were excluded from the drift diffusion model analysis. Drift rate was calculated separately for "go" and "no-go" trial types within the go/no-go task due to previous evidence that it may systematically differ across the two conditions (Huang-Pollock et al., 2017; Ratcliff et al., 2018).

EEA Score. Individual participants' EEA scores were calculated as latent factor scores from the three drift rate parameters (go, no-go, stop-signal) in Mplus version 8.6 (Muthén & Muthén, 1998-2017) via R (R Core Team, 2021), tidyverse (Wickham et al., 2019), and the *MplusAutomation* package (Hallquist & Wiley, 2018).

Question 1: Is EEA more reliable than traditional metrics?

To assess the reliability of EEA, we assessed test-retest reliabilities of EEA and traditional metrics within our sample for those individuals who returned for a second visit and had usable data at that timepoint (N=309). We also assessed across-task reliability for those metrics available from both the go/no-go and stop signal tasks at the first visit. For both sets of analyses, we ran zero-order Pearson correlations to assess rank-order stability. Finally, in order to provide the best possible comparison to the EEA latent factor score, which combined performance from the go/no-go and stop-signal tasks, we repeated these analyses with "average" scores for each metric including data from both tasks when available. These "average" scores were created by averaging z-scored performance on a given metric from both tasks (i.e.,

averaging z-scored go/no-go SDRT and z-scored stop-signal SDRT). When data from only one task was available, that z-score was used without averaging.

Question 2: Is EEA concurrently or prospectively related to psychopathology?

To assess whether EEA and traditional metrics related concurrently to psychopathology, we first ran zero-order Pearson correlations for all metrics with all psychopathology subscales at the first visit. To reduce the number of comparisons, we used the "average" task metrics described above when metrics were available for both tasks. We then used linear regression with maximum likelihood estimation within Mplus to repeat these analyses, controlling for age and sex, and accounting for relatedness within families using the "cluster" command.

To assess whether EEA and traditional metrics related prospectively to psychopathology, we ran zero-order correlations for all metrics at the first visit with all psychopathology subscales at the second visit. We then used linear regression with maximum likelihood estimation within Mplus to repeat these analyses, controlling for age (at the second visit), sex, and time between visits, and accounting for relatedness within families using the "cluster" command. Finally, we re-ran these models with controls for previous levels of psychopathology to assess whether performance predicted *change* in symptoms of psychopathology between visits.

Question 3: Is EEA related to brain activation?

To assess the biological relevance of executive functioning task performance as measured by EEA and more traditional metrics, we used linear regression in SPM12 to investigate whether executive functioning performance as measured by these variables might covary with brain activation during the contrast of interest (all no-go > go). We used a stringent, whole-brain approach, with cluster thresholding determined for each model by 3dttest++ within AFNI (Cox, 1996; Cox et al., 2017a, 2017b) based on an overall error rate (alpha) of 0.05 with a voxel-wise

threshold of $p<0.001$. To reduce the number of comparisons, we ran these regressions using the "average" task metrics including data from both tasks when possible.

Results

The drift diffusion model fit the data well for all conditions (Figure II.1) and parameter recovery for the EEA parameter (drift rate, *v*) was excellent for all tasks and conditions (Figure II.2). The EEA factor score converged, and loadings for all three conditions (go, no-go, stop-signal) were significant and within the acceptable range at both visits, though loadings for stop-signal were lower than those for the go and no-go conditions of the go/no-go task (Figure II.3). Descriptive statistics for and correlations between all of the extracted metrics from each task are available in Tables II.2 and II.3. Correlations between all extracted metrics from both tasks are available in Table II.4, and correlations between EEA and "average" metrics are available in Table II.5.

Question 1: Is EEA more reliable than traditional metrics?

EEA demonstrated comparable test-retest reliability to the traditional metrics (r=.52, 95% CI [.43,.59], $p<0.001$; Tables II.4 & II.5; Figure II.4). The metric with the highest test-retest reliability was Go/No-Go Efficiency (Accuracy / Reaction Time; r=.64, 95% CI [.57,.71], $p<0.001$) and with the lowest was Go Accuracy for either task (e.g., for Go/No-Go, r=.37, 95% CI [.26,.47], $p<0.001$; Table II.4). Findings were very similar when considering "average" metrics which included performance from both tasks when available (Table II.5).

EEA demonstrated better cross-task reliability than the other extracted metrics that were available for both tasks at the first visit (r=.28 and .30 for Go and No-Go with Stop-Signal, respectively; 95% CI [.17,.38] and [.19,.40]; $p<0.001$; Table II.6; Figure II.4). No other available metric demonstrated cross-task reliability significantly different from 0 at the first visit, meaning

that a participant who performed better than their peers per a given metric (i.e., reaction time) on one task did not reliably perform better than others per that same metric on the second task.

Question 2: Is EEA concurrently or prospectively related to psychopathology?

Zero-order correlations for EEA and traditional metrics with concurrent psychopathology are reported in Table II.7. When accounting for age, sex, and relatedness within families, as expected worse EEA scores related to more concurrent externalizing problems (β=-.11; 95% CI [-.20,-.01]; p=0.03; Table II.8), more concurrent attention problems (β=-.21; 95% CI [-.30,-.11]; p<0.01; Table II.8), and more concurrent rule-breaking problems (β=-.17; 95% CI [-.27,-.07]; p<0.01; Table II.8), but no longer related to concurrent anxious/depressed problems (β=.06; 95% CI [-.04,.15]; p=0.22; Table II.8).

Zero-order correlations for EEA and traditional metrics with prospective outcome variables are reported in Table II.9. When accounting for age, sex, time between visits, and relatedness within families, worse EEA scores no longer related to prospective attention problems (β=-.12; 95% CI [-.26,.01]; p=0.07; Table II.10), but did relate to more prospective externalizing problems (β=-.12; 95% CI [-.22,.00]; p=0.04; Table II.10), less prospective internalizing problems (β=.12; 95% CI [.00,.23]; p=0.04; Table II.10), less prospective anxious/depressed problems (β=.17; 95% CI [.05,.29]; p=0.01; Table II.10), and more prospective rule breaking (β=-.16; 95% CI [-.26,-.04]; p=0.01; Table II.10). When adding an additional control for previous levels of psychopathology, no significant relationships remained, meaning that EEA did not predict change in psychopathology over time when accounting for the stability of psychopathology.

Question 3: Is EEA related to brain activation?

When accounting for age, sex, and scan protocol, better performance as measured by four of the extracted metrics (EEA, Reaction Time, Standard Deviation of Reaction Time, and Efficiency) related to increased inhibition-related (no-go > go) brain activation in the anterior cingulate cortex and the right superior temporal gyrus (Table II.11; significant activation for these metrics with overlap displayed in Figure II.6). Better go/no-go Efficiency was additionally related to increased inhibition-related activation in the bilateral inferior frontal gyrus and the left precuneus (Table II.8; Figure II.7). Though all four superior temporal gyrus clusters had similar peak voxels, their shapes varied: for example, the superior temporal gyrus cluster associated with faster Reaction Time notably extended to include the right angular gyrus, while the superior temporal gyrus cluster associated with EEA notably extended along the temporal lobe to include the medial temporal gyrus (Figure II.8). Details regarding all significant clusters are available in Table II.11.

Discussion

In the present study, we examined the utility of a novel computational executive functioning performance metric, Efficiency of Evidence Accumulation (EEA), as a transdiagnostic risk factor for psychopathology in a large community sample of twins. We calculated EEA scores for each participant from go/no-go and stop-signal task data via drift diffusion and latent factor modeling, and extracted additional, more traditional performance metrics from these tasks as well (No-Go Accuracy, Go Accuracy, Reaction Time, Efficiency (No-Go Accuracy/Reaction Time), Standard Deviation of Reaction Time). We found that EEA demonstrated similar test-retest reliability, and much better cross-task reliability, when compared to the more traditional metrics. Results from zero-order correlations and multilevel models accounting for relatedness within families revealed similar patterns of associations with

concurrent and prospective psychopathology for many of the executive functioning metrics. In particular, better scores tended to relate to more anxious/depressed problems, less attention problems, and less rule-breaking behaviors. EEA was the only metric to relate prospectively to externalizing psychopathology when accounting for age, sex, and relatedness within families. Notably, better EEA scores did not relate to fewer total problems as hypothesized, and no metrics predicted any future psychopathology when accounting for the stability of psychopathology over time. Finally, fMRI analyses revealed that executive functioning performance as measured by EEA and several traditional metrics (Efficiency, Reaction Time, Standard Deviation of Reaction Time) did relate to inhibition-related brain activation within a major error-monitoring hub, the anterior cingulate cortex, as well as within the superior temporal gyrus. Interestingly, the Efficiency metric extracted from the go/no-go task correlated with activity in these regions as well as a broader group of salience network structures, including the inferior frontal gyrus. In sum, these analyses suggest that EEA provides some advantages over traditional metrics, particularly in terms of cross-task reliability, and that EEA does demonstrate robust relationships to concurrent and prospective externalizing behaviors and related concurrent brain activation in an adolescent sample.

We found that our EEA metric demonstrated reasonable test-retest reliability (r=.52) in an adolescent sample, comparable to other, more traditional metrics (ranging from r=0.37 for Go/No-Go Go Accuracy to r=0.64 for Go/No-Go Efficiency). This finding is in line with previous work which has found EEA and related drift diffusion model parameters to be quite reliable (Lerche & Voss, 2017; Schubert et al., 2016; Weigard, Clark, et al., 2021). The EEA test-retest reliability estimate for our sample may seem much lower than previous work (e.g. Lerche et al. 2017 found rs>0.70 for drift diffusion model parameters); however, this stability is

remarkable given the timespan between visits (>1 year) during adolescence, a developmental period when executive functioning has not yet stabilized (Kolb et al., 2012). The finding of similar test-retest reliability for EEA and traditional metrics, however, does not necessarily align with recent work demonstrating poor test-retest reliability for traditional behavioral executive functioning measures and suggesting computational methods as a solution (Eisenberg et al., 2019; Karr et al., 2018; Weigard, Clark, et al., 2021). However, we did find that our EEA metric outperformed traditional metrics when comparing across tasks, demonstrating significant cross-task reliability at the first visit (r=.=.28 and .30 for Go and No-Go with Stop-Signal, respectively), while the more traditional metrics that were available for both tasks (Go Accuracy, Reaction Time, SD Reaction Time) did not. Notably, the metric demonstrating the best test-retest reliability, Go/No-Go Efficiency, could not be calculated for the Stop Signal task used in the present study and thus its cross-task reliability could not be assessed. The significant cross-task reliability of the EEA metric aligns with its proposal as a task-general individual difference dimension (Lerche et al., 2020; Weigard, Clark, et al., 2021), extending this work to include adolescence. Future work could use a wider variety of tasks, especially tasks not traditionally designed to measure "inhibition", to further establish task-generality of EEA in adolescence. However, if these findings hold, it would mean that scores from an EEA analysis of one EF task would generalize to other tasks – implying that using different tasks across studies or only using a single task in clinical practice could suffice to provide the same information on individual differences as a more extensive multi-tasks battery.

Using multiple modeling approaches, we found relatively consistent relationships between executive functioning performance, as measured by both EEA as well as several traditional metrics, and concurrent and prospective symptoms of psychopathology. Though EEA

did not relate to total problems as hypothesized, better performance as indexed by EEA did relate to fewer concurrent and prospective externalizing problems and rule-breaking problems, and to fewer concurrent attention problems, even when accounting for age, sex, and relatedness within families. While all other metrics related to at least one of concurrent externalizing, attention problems, or rule-breaking problems in similar models (i.e., No-Go Accuracy, Efficiency, Standard Deviation of Reaction Time), EEA was the only metric to relate to broad externalizing problems over a year after task completion. These findings in adolescents reflect similar results in adults: for example, EEA and related brain activation have recently been shown to relate prospectively to substance use, another externalizing behavior, in young adults (Weigard, Brislin, et al., 2021), and reductions in EEA have been associated with Attention-Deficit/Hyperactivity Disorder, among other diagnoses, in adults (Sripada & Weigard, 2021). Thus, as hypothesized elsewhere (Weigard & Sripada, 2021), computational modeling of task performance may provide a more complete picture of the underlying cognitive mechanisms than simpler summary metrics, allowing better prediction of real-world behaviors. It is important to note, however, that neither EEA nor traditional metrics predicted future psychopathology when controlling for the stability of psychopathology. In other words, performance as measured by any metric did not predict change in psychopathology over time. This result could be driven by a relatively high stability of symptoms of psychopathology over the course of one year. On the other hand, this could reflect limited predictive value of these metrics in general. Longitudinal work with more timepoints and a longer timespan could clarify this issue.

Interestingly, better performance as measured by EEA and several other metrics (No-Go Accuracy, Go Accuracy, Reaction Time, Efficiency, and Standard Deviation of Reaction Time) related to more concurrent and prospective anxious/depressed problems at the zero-order level,

and with more prospective anxious/depressed problems even after accounting for age, sex, time between visits, and relatedness within families. This result ran counter to the hypothesized relationship between better EEA and fewer overall problems, but it is not without precedent in the literature. Another drift diffusion modeling study recently found associations between increased internalizing symptoms and greater boundary separation, a different diffusion model parameter indexing response caution (Fosco et al., 2022). It is possible that the EEA metric may tap into a neurocognitive mechanism that is problematic when underactive, leading to externalizing behaviors (i.e., rule-breaking), but also when overactive, leading to internalizing behaviors (i.e., anxiety, behavioral inhibition). On the other hand, these findings may simply reflect the long-held idea (e.g. Yerkes & Dodson, 1908) that anxiety, at normative levels, promotes better task performance. Future work could more closely inspect the relationship between EEA and anxiety across the spectrum of anxiety symptoms, ranging from normative to clinically impairing.

Functional neuroimaging analyses revealed strikingly consistent clusters of activation in which better executive functioning performance as measured by EEA and several other metrics (Efficiency, Reaction Time, Standard Deviation of Reaction Time) correlated with increased inhibition-related brain activation. Performance as measured by all four metrics was associated with activation in the anterior cingulate cortex, as hypothesized, as well as the superior temporal gyrus. The anterior cingulate cortex is a major salience network hub (Seeley et al., 2007), important for error processing (Bush et al., 2000) and conflict monitoring (Botvinick et al., 2004). This region has previously been implicated when employing computational models of the go/no-go task (Weigard et al., 2020). The superior temporal gyrus is associated with change detection (Opitz et al., 2002) and is frequently implicated in fMRI studies of response inhibition,

particularly regarding action cancellation (Zhang et al., 2017). Inhibition-related activation in the superior temporal gyrus has also been reported to correlate with informant-report trait impulsivity (Horn et al., 2003). Overall, it is clear that, within this adolescent sample, executive functioning task performance as indexed by EEA and several other metrics have robust and reasonable brain correlates.

Notably, the Efficiency metric was associated with activation in several additional clusters, including the bilateral inferior frontal gyrus and the precuneus. This finding extends a previous, very similar, finding within a smaller version of this sample (Tomlinson et al., 2020). However, it is not clear why a simpler executive functioning metric would be associated with more widespread brain activation than the computational EEA measure. It is possible that the Efficiency metric indexes a broader "effort" or "salience" process, reflected by activation within several important salience network hubs (Seeley et al., 2007), while the EEA metric indexes a more specific, underlying neurocognitive process. On the other hand, more parsimonious computational modeling approaches have previously been found to generate more stable results even if some of the complexities of the underlying neural processes are lost (Lerche & Voss, 2016, 2017). Though forgoing computational modeling to use the Efficiency metric, which simply combines two traditional summary metrics (No-Go Accuracy / Reaction Time), might be an extreme example of a parsimonious approach, the same principle may apply. Finally, while the EEA latent factor score reflected performance on two tasks, the Efficiency metric reflected performance specific to the Go/No-Go task, which is also the task performed in the scanner. Future work could investigate whether this more widespread Efficiency-related activation persists when Efficiency is calculated to include tasks performed outside of the scanner.

Given the ease with which Efficiency can be calculated (Accuracy / RT), it is worth considering whether Efficiency might be a reasonable stand-in for the more computationally intensive EEA score in some cases. In the present study, Efficiency and EEA were highly correlated (r=.82). Efficiency had the highest test-retest correlation of any metric extracted for the study (r=.64). Efficiency also had robust brain correlates within relevant brain regions in the salience network. On the other hand, EEA was a slightly better predictor of psychopathology: while both metrics correlated with attention problems, EEA showed stronger associations with overall externalizing problems and with rule-breaking problems. Additionally, EEA can be calculated from a stop-signal task, which is widely used in the executive functioning literature, while Efficiency as defined in the present study cannot. In studies considering only go/no-go data, it may be reasonable to use the simple Efficiency metric rather than calculating EEA.

This study does have several limitations. For one, the associations between EEA and symptoms of psychopathology were not corrected for multiple comparisons, and would not survive such corrections. Additionally, the large, ongoing MTwiNS study was not designed specifically for testing the reliability and validity of various executive functioning metrics, and visits were scheduled a year apart in order to observe developmental change. Thus, future work could extend these results by assessing test-retest reliability of executive functioning metrics in adolescents with a much shorter time (~1 week) between visits. Along those lines, the go/no-go and stop signal tasks were each only about 10 minutes long in the present study due to time constraints, limiting our ability to assess split-task reliability. Future work could implement much longer tasks to allow for computational model fitting to portions of the task for within-task comparison. Additionally, the present study only incorporated one parameter from one type of computational model, the drift diffusion model. Though not within the scope of this study,

consideration of other parameters within the drift diffusion model, or of parameters from other models entirely, would be of interest to the field. For example, the drift diffusion model is one example of a "horse-race" computational model, in which there is a "race" between two processes (i.e. "go" and "stop"), the outcome of which is dependent on the relative time it takes to complete each process (Logan & Cowan, 1984; Verbruggen & Logan, 2009). Comparison to parameters from other models would be an interesting next step. Finally, the inclusion of additional tasks, especially tasks not designed to test response inhibition, could greatly broaden the scope of this work and potentially further differentiate computational methods like EEA from more traditional summary metrics.

In summary, we found encouraging evidence for a novel computational measure of task-general executive functioning, EEA, as a transdiagnostic risk factor associated with externalizing psychopathology. EEA demonstrated reasonable test-retest reliability, and it was the only performance metric in the present study that related to both concurrent and prospective externalizing psychopathology when accounting for age, sex, and relatedness within families. Functional neuroimaging analyses also revealed that EEA had robust brain correlates, relating to inhibition-related brain activation in two relevant regions, the anterior cingulate cortex and the right superior temporal gyrus. Notably, however, several other traditional summary metrics correlated with brain activation in strikingly similar locations and had similar test-retest reliablity. None of the metrics predicted change in psychopathology over time, highlighting an important limitation in this area germane to translation efforts for neuropsychology. Overall, these findings highlight that computational methods may provide some advantages over more traditional summary metrics of executive functioning performance in adolescents, while also

revealing that some traditional metrics (i.e. Efficiency, Standard Deviation of Reaction Time) may provide a reasonable alternative when computational modeling is not possible.

References

Achenbach, T. M., McConaughy, S. H., & Howell, C. T. (1987). Child/Adolescent Behavioral and Emotional Problems: Implications of Cross-Informant Correlations for Situational Specificity. *Psychol Bull, 101*(2), 213-232. https://www.ncbi.nlm.nih.gov/pubmed/3562706

Achenbach, T. M., & Rescorla, L. A. (2001). *Manual for the Aseba School-Age Forms & Profiles*. University of Vermont, Research Center for Children, Youth, & Families.

Begolli, K. N., Richland, L. E., Jaeggi, S. M., Lyons, E. M., Klostermann, E. C., & Matlen, B. J. (2018). Executive Function in Learning Mathematics by Comparison: Incorporating Everyday Classrooms into the Science of Learning. *Thinking & Reasoning, 24*(2), 280-313. https://doi.org/10.1080/13546783.2018.1429306

Bissett, P. G., & Logan, G. D. (2012). Post-Stop-Signal Slowing: Strategies Dominate Reflexes and Implicit Learning. *J Exp Psychol Hum Percept Perform, 38*(3), 746-757. https://doi.org/10.1037/a0025429

Blair, C., & Razza, R. P. (2007). Relating Effortful Control, Executive Function, and False Belief Understanding to Emerging Math and Literacy Ability in Kindergarten. *Child Dev, 78*(2), 647-663. https://doi.org/10.1111/j.1467-8624.2007.01019.x

Bloemen, A. J. P., Oldehinkel, A. J., Laceulle, O. M., Ormel, J., Rommelse, N. N. J., & Hartman, C. A. (2018). The Association between Executive Functioning and Psychopathology: General or Specific? *Psychol Med, 48*(11), 1787-1794. https://doi.org/10.1017/S0033291717003269

Boonstra, A. M., Oosterlaan, J., Sergeant, J. A., & Buitelaar, J. K. (2005). Executive Functioning in Adult Adhd: A Meta-Analytic Review. *Psychol Med, 35*(8), 1097-1108. https://doi.org/10.1017/s003329170500499x

Botvinick, M. M., Cohen, J. D., & Carter, C. S. (2004). Conflict Monitoring and Anterior Cingulate Cortex: An Update. *Trends Cogn Sci, 8*(12), 539-546. https://doi.org/10.1016/j.tics.2004.10.003

Bouchard, T. J., Jr., Lykken, D. T., McGue, M., Segal, N. L., & Tellegen, A. (1990). Sources of Human Psychological Differences: The Minnesota Study of Twins Reared Apart. *Science, 250*(4978), 223-228. https://doi.org/10.1126/science.2218526

Burt, S. A., & Klump, K. L. (2019). The Michigan State University Twin Registry (Msutr): 15 Years of Twin and Family Research. *Twin Res Hum Genet, 22*(6), 741-745. https://doi.org/10.1017/thg.2019.57

Burt, S. A., Klump, K. L., Vazquez, A. Y., Shewark, E. A., & Hyde, L. W. (2021). Identifying Patterns of Youth Resilience to Neighborhood Disadvantage. *Research in Human Development*, 1-16. https://doi.org/10.1080/15427609.2021.1935607

Burt, S. A., McGue, M., Iacono, W. G., & Krueger, R. F. (2006). Differential Parent-Child Relationships and Adolescent Externalizing Symptoms: Cross-Lagged Analyses within a Monozygotic Twin Differences Design. *Dev Psychol, 42*(6), 1289-1298. https://doi.org/10.1037/0012-1649.42.6.1289

Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and Emotional Influences in Anterior Cingulate Cortex. *Trends in Cognitive Sciences, 4*(6), 215-222. https://doi.org/10.1016/s1364-6613(00)01483-2

Casey, B. J., Cannonier, T., Conley, M. I., Cohen, A. O., Barch, D. M., Heitzeg, M. M., Soules, M. E., Teslovich, T., Dellarco, D. V., Garavan, H., Orr, C. A., Wager, T. D., Banich, M. T., Speer, N. K., Sutherland, M. T., Riedel, M. C., Dick, A. S., Bjork, J. M., Thomas, K. M., Chaarani, B., Mejia, M. H., Hagler, D. J., Jr., Daniela Cornejo, M., Sicat, C. S., Harms, M. P., Dosenbach, N. U. F., Rosenberg, M., Earl, E., Bartsch, H., Watts, R., Polimeni, J. R., Kuperman, J. M., Fair, D. A., Dale, A. M., & Workgroup, A. I. A. (2018). The Adolescent Brain Cognitive Development (Abcd) Study: Imaging Acquisition across 21 Sites. *Dev Cogn Neurosci, 32*, 43-54. https://doi.org/10.1016/j.dcn.2018.03.001

Casey, B. J., Trainor, R. J., Orendi, J. L., Schubert, A. B., Nystrom, L. E., Giedd, J. N., Castellanos, F. X., Haxby, J. V., Noll, D. C., Cohen, J. D., Forman, S. D., Dahl, R. E., & Rapoport, J. L. (1997). A Developmental Functional Mri Study of Prefrontal Activation During Performance of a Go-No-Go Task. *Journal of Cognitive Neuroscience, 9*(6), 835-847. https://doi.org/10.1162/jocn.1997.9.6.835

Cox, R. W. (1996). Afni: Software for Analysis and Visualization of Functional Magnetic Resonance Neuroimages. *Comput Biomed Res, 29*(3), 162-173. https://doi.org/10.1006/cbmr.1996.0014

Cox, R. W., Chen, G., Glen, D. R., Reynolds, R. C., & Taylor, P. A. (2017a). Fmri Clustering and False-Positive Rates. *Proc Natl Acad Sci U S A, 114*(17), E3370-E3371. https://doi.org/10.1073/pnas.1614961114

Cox, R. W., Chen, G., Glen, D. R., Reynolds, R. C., & Taylor, P. A. (2017b). Fmri Clustering in Afni: False-Positive Rates Redux. *Brain Connect, 7*(3), 152-171. https://doi.org/10.1089/brain.2016.0475

Cronbach, L. J., & Meehl, P. E. (1955). Construct Validity in Psychological Tests. *Psychol Bull, 52*(4), 281-302. https://doi.org/10.1037/h0040957

Cumming, G. (2014). The New Statistics: Why and How. *Psychol Sci, 25*(1), 7-29. https://doi.org/10.1177/0956797613504966

Cuthbert, B. N., & Insel, T. R. (2013). Toward the Future of Psychiatric Diagnosis: The Seven Pillars of Rdoc. *BMC Med, 11*, 126. https://doi.org/10.1186/1741-7015-11-126

Diamond, A. (2013). Executive Functions. *Annu Rev Psychol, 64*, 135-168. https://doi.org/10.1146/annurev-psych-113011-143750

Eisenberg, I. W., Bissett, P. G., Zeynep Enkavi, A., Li, J., MacKinnon, D. P., Marsch, L. A., & Poldrack, R. A. (2019). Uncovering the Structure of Self-Regulation through Data-Driven Ontology Discovery. *Nat Commun, 10*(1), 2319. https://doi.org/10.1038/s41467-019-10301-1

Fosco, W. D., Meisel, S. N., Weigard, A., White, C. N., & Colder, C. R. (2022). Computational Modeling Reveals Strategic and Developmental Differences in the Behavioral Impact of Reward across Adolescence. *Dev Sci, 25*(2), e13159. https://doi.org/10.1111/desc.13159

Friedman, N. P., & Miyake, A. (2017). Unity and Diversity of Executive Functions: Individual Differences as a Window on Cognitive Structure. *Cortex, 86*, 186-204. https://doi.org/10.1016/j.cortex.2016.04.023

Gotlib, I. H., & Joormann, J. (2010). Cognition and Depression: Current Status and Future Directions. *Annu Rev Clin Psychol, 6*, 285-312. https://doi.org/10.1146/annurev.clinpsy.121208.131305

Hallquist, M. N., & Wiley, J. F. (2018). Mplusautomation: An R Package for Facilitating Large-Scale Latent Variable Analyses in Mplus. *Struct Equ Modeling, 25*(4), 621-638. https://doi.org/10.1080/10705511.2017.1402334

Heathcote, A. (2019). *Dynamic Models of Choice.* In https://osf.io/pbwx8/

Horn, N. R., Dolan, M., Elliott, R., Deakin, J. F. W., & Woodruff, P. W. R. (2003). Response Inhibition and Impulsivity: An Fmri Study. *Neuropsychologia, 41*(14), 1959-1966. https://doi.org/10.1016/s0028-3932(03)00077-0

Huang-Pollock, C., Ratcliff, R., McKoon, G., Roule, A., Warner, T., Feldman, J., & Wise, S. (2020). A Diffusion Model Analysis of Sustained Attention in Children with Attention Deficit Hyperactivity Disorder. *Neuropsychology, 34*(6), 641-653. https://doi.org/10.1037/neu0000636

Huang-Pollock, C., Ratcliff, R., McKoon, G., Shapiro, Z., Weigard, A., & Galloway-Long, H. (2017). Using the Diffusion Model to Explain Cognitive Deficits in Attention Deficit Hyperactivity Disorder. *J Abnorm Child Psychol, 45*(1), 57-68. https://doi.org/10.1007/s10802-016-0151-y

Hull, R., Martin, R. C., Beier, M. E., Lane, D., & Hamilton, A. C. (2008). Executive Function in Older Adults: A Structural Equation Modeling Approach. *Neuropsychology, 22*(4), 508-522. https://doi.org/10.1037/0894-4105.22.4.508

Iacono, W. G., Carlson, S. R., Taylor, J., Elkins, I. J., & McGue, M. (1999). Behavioral Disinhibition and the Development of Substance-Use Disorders: Findings from the Minnesota Twin Family Study. *Dev Psychopathol, 11*(4), 869-900. https://doi.org/10.1017/s0954579499002369

Karalunas, S. L., Geurts, H. M., Konrad, K., Bender, S., & Nigg, J. T. (2014). Annual Research Review: Reaction Time Variability in Adhd and Autism Spectrum Disorders:

Measurement and Mechanisms of a Proposed Trans-Diagnostic Phenotype. *J Child Psychol Psychiatry, 55*(6), 685-710. https://doi.org/10.1111/jcpp.12217

Karr, J. E., Areshenkoff, C. N., Rast, P., Hofer, S. M., Iverson, G. L., & Garcia-Barrera, M. A. (2018). The Unity and Diversity of Executive Functions: A Systematic Review and Re-Analysis of Latent Variable Studies. *Psychol Bull, 144*(11), 1147-1185. https://doi.org/10.1037/bul0000160

Killeen, P. R., Russell, V. A., & Sergeant, J. A. (2013). A Behavioral Neuroenergetics Theory of Adhd. *Neurosci Biobehav Rev, 37*(4), 625-657. https://doi.org/10.1016/j.neubiorev.2013.02.011

Kolb, B., Mychasiuk, R., Muhammad, A., Li, Y., Frost, D. O., & Gibb, R. (2012). Experience and the Developing Prefrontal Cortex. *Proc Natl Acad Sci U S A, 109 Suppl 2*, 17186-17193. https://doi.org/10.1073/pnas.1121251109

Lambek, R., Tannock, R., Dalsgaard, S., Trillingsgaard, A., Damm, D., & Thomsen, P. H. (2010). Validating Neuropsychological Subtypes of Adhd: How Do Children with and without an Executive Function Deficit Differ? *J Child Psychol Psychiatry, 51*(8), 895-904. https://doi.org/10.1111/j.1469-7610.2010.02248.x

Lee, K., Bull, R., & Ho, R. M. (2013). Developmental Changes in Executive Functioning. *Child Dev, 84*(6), 1933-1953. https://doi.org/10.1111/cdev.12096

Lerche, V., von Krause, M., Voss, A., Frischkorn, G. T., Schubert, A. L., & Hagemann, D. (2020). Diffusion Modeling and Intelligence: Drift Rates Show Both Domain-General and Domain-Specific Relations with Intelligence. *J Exp Psychol Gen, 149*(12), 2207-2249. https://doi.org/10.1037/xge0000774

Lerche, V., & Voss, A. (2016). Model Complexity in Diffusion Modeling: Benefits of Making the Model More Parsimonious. *Front Psychol, 7*, 1324. https://doi.org/10.3389/fpsyg.2016.01324

Lerche, V., & Voss, A. (2017). Retest Reliability of the Parameters of the Ratcliff Diffusion Model. *Psychol Res, 81*(3), 629-652. https://doi.org/10.1007/s00426-016-0770-5

Logan, G. D., & Cowan, W. B. (1984). On the Ability to Inhibit Thought and Action: A Theory of an Act of Control. *Psychological Review, 91*(3), 295-327. https://doi.org/10.1037/0033-295x.91.3.295

Maldjian, J. A., Laurienti, P. J., Kraft, R. A., & Burdette, J. H. (2003). An Automated Method for Neuroanatomic and Cytoarchitectonic Atlas-Based Interrogation of Fmri Data Sets. *Neuroimage, 19*(3), 1233-1239. https://www.ncbi.nlm.nih.gov/pubmed/12880848

Marcovitch, S., Jacques, S., Boseovski, J. J., & Zelazo, P. D. (2008). Self-Reflection and the Cognitive Control of Behavior: Implications for Learning. *Mind, Brain, and Education, 2*(3), 136-141. https://doi.org/10.1111/j.1751-228X.2008.00044.x

McClelland, M. M., Cameron, C. E., Connor, C. M., Farris, C. L., Jewkes, A. M., & Morrison, F. J. (2007). Links between Behavioral Regulation and Preschoolers' Literacy, Vocabulary, and Math Skills. *Dev Psychol, 43*(4), 947-959. https://doi.org/10.1037/0012-1649.43.4.947

McKenna, R., Rushe, T., & Woodcock, K. A. (2017). Informing the Structure of Executive Function in Children: A Meta-Analysis of Functional Neuroimaging Data. *Front Hum Neurosci, 11*, 154. https://doi.org/10.3389/fnhum.2017.00154

Metin, B., Roeyers, H., Wiersema, J. R., van der Meere, J., & Sonuga-Barke, E. (2012). A Meta-Analytic Study of Event Rate Effects on Go/No-Go Performance in Attention-Deficit/Hyperactivity Disorder. *Biol Psychiatry, 72*(12), 990-996. https://doi.org/10.1016/j.biopsych.2012.08.023

Mischel, W., Ayduk, O., Berman, M. G., Casey, B. J., Gotlib, I. H., Jonides, J., Kross, E., Teslovich, T., Wilson, N. L., Zayas, V., & Shoda, Y. (2011). 'Willpower' over the Life Span: Decomposing Self-Regulation. *Soc Cogn Affect Neurosci, 6*(2), 252-256. https://doi.org/10.1093/scan/nsq081

Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The Unity and Diversity of Executive Functions and Their Contributions to Complex "Frontal Lobe" Tasks: A Latent Variable Analysis. *Cogn Psychol, 41*(1), 49-100. https://doi.org/10.1006/cogp.1999.0734

Moffitt, T. E. (2018). Male Antisocial Behaviour in Adolescence and Beyond. *Nature Human Behaviour, 2*(3), 177-186. https://doi.org/10.1038/s41562-018-0309-4

Moffitt, T. E., Arseneault, L., Belsky, D., Dickson, N., Hancox, R. J., Harrington, H., Houts, R., Poulton, R., Roberts, B. W., Ross, S., Sears, M. R., Thomson, W. M., & Caspi, A. (2011). A Gradient of Childhood Self-Control Predicts Health, Wealth, and Public Safety. *Proc Natl Acad Sci U S A, 108*(7), 2693-2698. https://doi.org/10.1073/pnas.1010076108

Muthén, L. K., & Muthén, B. O. (1998-2017). *Mplus User's Guide* (Eighth Edition ed.). Muthén & Muthén.

Nigg, J. T. (2017). Annual Research Review: On the Relations among Self-Regulation, Self-Control, Executive Functioning, Effortful Control, Cognitive Control, Impulsivity, Risk-Taking, and Inhibition for Developmental Psychopathology. *J Child Psychol Psychiatry, 58*(4), 361-383. https://doi.org/10.1111/jcpp.12675

Nigg, J. T., Gustafsson, H. C., Karalunas, S. L., Ryabinin, P., McWeeney, S. K., Faraone, S. V., Mooney, M. A., Fair, D. A., & Wilmot, B. (2018). Working Memory and Vigilance as Multivariate Endophenotypes Related to Common Genetic Risk for Attention-Deficit/Hyperactivity Disorder. *J Am Acad Child Adolesc Psychiatry, 57*(3), 175-182. https://doi.org/10.1016/j.jaac.2017.12.013

Opitz, B., Rinne, T., Mecklinger, A., von Cramon, D. Y., & Schroger, E. (2002). Differential Contribution of Frontal and Temporal Cortices to Auditory Change Detection: Fmri and Erp Results. *Neuroimage, 15*(1), 167-174. https://doi.org/10.1006/nimg.2001.0970

Packwood, S., Hodgetts, H. M., & Tremblay, S. (2011). A Multiperspective Approach to the Conceptualization of Executive Functions. *J Clin Exp Neuropsychol, 33*(4), 456-470. https://doi.org/10.1080/13803395.2010.533157

Peeters, H., Van Gestel, S., Vlietinck, R., Derom, C., & Derom, R. (1998). Validation of a Telephone Zygosity Questionnaire in Twins of Known Zygosity. *Behav Genet, 28*(3), 159-163. https://doi.org/10.1023/a:1021416112215

R Core Team. (2021). *R: A Language and Environment for Statistical Computing.* In R Foundation for Statistical Computing. https://www.R-project.org/

Ratcliff, R. (2002). A Diffusion Model Account of Response Time and Accuracy in a Brightness Discrimination Task: Fitting Real Data and Failing to Fit Fake but Plausible Data. *Psychon Bull Rev, 9*(2), 278-291. https://doi.org/10.3758/bf03196283

Ratcliff, R., Huang-Pollock, C., & McKoon, G. (2018). Modeling Individual Differences in the Go/No-Go Task with a Diffusion Model. *Decision (Wash D C), 5*(1), 42-62. https://doi.org/10.1037/dec0000065

Ratcliff, R., & McKoon, G. (2008). The Diffusion Decision Model: Theory and Data for Two-Choice Decision Tasks. *Neural Comput, 20*(4), 873-922. https://doi.org/10.1162/neco.2008.12-06-420

Ratcliff, R., & Rouder, J. N. (1998). Modeling Response Times for Two-Choice Decisions. *Psychological Science, 9*(5), 347-356. https://doi.org/Doi 10.1111/1467-9280.00067

Rorden, C., & Brett, M. (2000). Stereotaxic Display of Brain Lesions. *Behav Neurol, 12*(4), 191-200. https://doi.org/10.1155/2000/421719

Schubert, A.-L., Frischkorn, G., Hagemann, D., & Voss, A. (2016). Trait Characteristics of Diffusion Model Parameters. *Journal of Intelligence, 4*(3). https://doi.org/10.3390/jintelligence4030007

Seeley, W. W., Menon, V., Schatzberg, A. F., Keller, J., Glover, G. H., Kenna, H., Reiss, A. L., & Greicius, M. D. (2007). Dissociable Intrinsic Connectivity Networks for Salience Processing and Executive Control. *J Neurosci, 27*(9), 2349-2356. https://doi.org/10.1523/JNEUROSCI.5587-06.2007

Smith, G. T. (2005). On Construct Validity: Issues of Method and Measurement. *Psychol Assess, 17*(4), 396-408. https://doi.org/10.1037/1040-3590.17.4.396

Smith, J. L., Mattick, R. P., Jamadar, S. D., & Iredale, J. M. (2014). Deficits in Behavioural Inhibition in Substance Abuse and Addiction: A Meta-Analysis. *Drug Alcohol Depend, 145*, 1-33. https://doi.org/10.1016/j.drugalcdep.2014.08.009

Sripada, C., & Weigard, A. (2021). Impaired Evidence Accumulation as a Transdiagnostic Vulnerability Factor in Psychopathology. *Front Psychiatry, 12*, 627179. https://doi.org/10.3389/fpsyt.2021.627179

Stanley, D. J., & Spence, J. R. (2018). Reproducible Tables in Psychology Using the Apatables Package. *Advances in Methods and Practices in Psychological Science, 1*(3), 415-431. https://doi.org/10.1177/2515245918773743

Tomlinson, R. C., Burt, S. A., Waller, R., Jonides, J., Miller, A. L., Gearhardt, A. N., Peltier, S. J., Klump, K. L., Lumeng, J. C., & Hyde, L. W. (2020). Neighborhood Poverty Predicts Altered Neural and Behavioral Response Inhibition. *Neuroimage, 209*, 116536. https://doi.org/10.1016/j.neuroimage.2020.116536

Turner, B. M., Forstmann, B. U., Love, B. C., Palmeri, T. J., & Van Maanen, L. (2017). Approaches to Analysis in Model-Based Cognitive Neuroscience. *J Math Psychol, 76*(B), 65-79. https://doi.org/10.1016/j.jmp.2016.01.001

Turner, B. M., Sederberg, P. B., Brown, S. D., & Steyvers, M. (2013). A Method for Efficiently Sampling from Distributions with Correlated Dimensions. *Psychol Methods, 18*(3), 368-384. https://doi.org/10.1037/a0032222

Verbruggen, F., & Logan, G. D. (2009). Models of Response Inhibition in the Stop-Signal and Stop-Change Paradigms. *Neurosci Biobehav Rev, 33*(5), 647-661. https://doi.org/10.1016/j.neubiorev.2008.08.014

Verdejo-Garcia, A., Lawrence, A. J., & Clark, L. (2008). Impulsivity as a Vulnerability Marker for Substance-Use Disorders: Review of Findings from High-Risk Research, Problem Gamblers and Genetic Association Studies. *Neurosci Biobehav Rev, 32*(4), 777-810. https://doi.org/10.1016/j.neubiorev.2007.11.003

Voss, A., Nagler, M., & Lerche, V. (2013). Diffusion Models in Experimental Psychology: A Practical Introduction. *Exp Psychol, 60*(6), 385-402. https://doi.org/10.1027/1618-3169/a000218

Weigard, A., Clark, D. A., & Sripada, C. (2021). Cognitive Efficiency Beats Top-Down Control as a Reliable Individual Difference Dimension Relevant to Self-Control. *Cognition, 215*, 104818. https://doi.org/10.1016/j.cognition.2021.104818

Weigard, A., Huang-Pollock, C., Brown, S., & Heathcote, A. (2018). Testing Formal Predictions of Neuroscientific Theories of Adhd with a Cognitive Model-Based Approach. *J Abnorm Psychol, 127*(5), 529-539. https://doi.org/10.1037/abn0000357

Weigard, A., Soules, M., Ferris, B., Zucker, R. A., Sripada, C., & Heitzeg, M. (2020). Cognitive Modeling Informs Interpretation of Go/No-Go Task-Related Neural Activations and Their Links to Externalizing Psychopathology. *Biol Psychiatry Cogn Neurosci Neuroimaging, 5*(5), 530-541. https://doi.org/10.1016/j.bpsc.2019.11.013

Weigard, A., & Sripada, C. (2021). Task-General Efficiency of Evidence Accumulation as a Computationally Defined Neurocognitive Trait: Implications for Clinical Neuroscience. *Biological Psychiatry Global Open Science, 1*(1), 5-15. https://doi.org/10.1016/j.bpsgos.2021.02.001

Weigard, A. S., Brislin, S. J., Cope, L. M., Hardee, J. E., Martz, M. E., Ly, A., Zucker, R. A., Sripada, C., & Heitzeg, M. M. (2021). Evidence Accumulation and Associated Error-Related Brain Activity as Computationally-Informed Prospective Predictors of Substance Use in Emerging Adulthood. *Psychopharmacology (Berl)*. https://doi.org/10.1007/s00213-021-05885-w

Wellcome Centre for Human Neuroimaging. *Spm12*. https://www.fil.ion.ucl.ac.uk/spm/

Wickham, H., Averick, M., Bryan, J., Chang, W., McGowan, L., François, R., Grolemund, G., Hayes, A., Henry, L., Hester, J., Kuhn, M., Pedersen, T., Miller, E., Bache, S., Müller, K., Ooms, J., Robinson, D., Seidel, D., Spinu, V., Takahashi, K., Vaughan, D., Wilke, C., Woo, K., & Yutani, H. (2019). Welcome to the Tidyverse. *Journal of Open Source Software, 4*(43). https://doi.org/10.21105/joss.01686

Wiecki, T. V., Poland, J., & Frank, M. J. (2015). Model-Based Cognitive Neuroscience Approaches to Computational Psychiatry. *Clinical Psychological Science, 3*(3), 378-399. https://doi.org/10.1177/2167702614565359

Wright, L., Lipszyc, J., Dupuis, A., Thayapararajah, S. W., & Schachar, R. (2014). Response Inhibition and Psychopathology: A Meta-Analysis of Go/No-Go Task Performance. *J Abnorm Psychol, 123*(2), 429-439. https://doi.org/10.1037/a0036295

Yarkoni, T., Poldrack, R. A., Nichols, T. E., Van Essen, D. C., & Wager, T. D. (2011). Large-Scale Automated Synthesis of Human Functional Neuroimaging Data. *Nat Methods, 8*(8), 665-670. https://doi.org/10.1038/nmeth.1635

Yerkes, R. M., & Dodson, J. D. (1908). The Relation of Strength of Stimulus to Rapidity of Habit-Formation. *Journal of Comparative Neurology and Psychology, 18*(5), 459-482. https://doi.org/10.1002/cne.920180503

Zhang, R., Geng, X., & Lee, T. M. C. (2017). Large-Scale Functional Neural Network Correlates of Response Inhibition: An Fmri Meta-Analysis. *Brain Struct Funct, 222*(9), 3973-3990. https://doi.org/10.1007/s00429-017-1443-x

Table II.1

Summary of data included in fMRI analyses

	Number Lost	Participants With Usable Data
Participated in First Visit		**708** (354 complete pairs)
Sample with Any Go/No-Go fMRI Data		**600**
• Incomplete MRI scan	(16)	
• Software malfunction	(3)	
• Declined MRI scan	(27)	
• Uncomfortable with scan	(18)	
• Dental (e.g. braces, retainer)	(17)	
• Metal in/on the body	(13)	
• Exceeded scanner size restrictions	(5)	
• Major medical/neurological disorder	(9)	
Total Lost	*108*	
Final Sample with Usable fMRI Data		**549**
• Frontal lobe coverage <90%	(40)	
• ART Outliers >5%	(7)	
• Task performance unacceptable for EEA score generation	(4)	
Total Lost	*51*	

Note. Breakdown of available fMRI data based on lab standard fMRI quality checks.

Table II.2

Means, standard deviations, and correlations with confidence intervals of all metrics extracted from the Go/No-Go task

Variable	*M*	*SD*	1	2	3	4	5	6	7	8	9	10	11
1. Total Accuracy (1)	0.00	1.00											
2. Total Accuracy (2)	-0.00	1.00	**.41**** [.31, .50]										
3. No-Go Accuracy (1)	0.00	1.00	.69** [.65, .73]	.23** [.11, .33]									
4. No-Go Accuracy (2)	0.00	1.00	.39** [.28, .48]	.53** [.44, .61]	**.48**** [.38, .56]								
5. Go Accuracy (1)	0.00	1.00	.82** [.79, .84]	.37** [.27, .47]	.15** [.07, .23]	.15* [.03, .26]							
6. Go Accuracy (2)	0.00	1.00	.30** [.19, .40]	.92** [.90, .94]	.04 [-.07, .16]	.16** [.04, .27]	**.37**** [.26, .47]						
7. Reaction Time (1)	0.00	1.00	-.18** [-.26, -.10]	-.19** [-.30, -.07]	.12** [.04, .20]	-.02 [-.14, .09]	-.34** [-.41, -.27]	-.21** [-.32, -.09]					
8. Reaction Time (2)	-0.00	1.00	-.22** [-.33, -.10]	-.28** [-.39, -.17]	-.08 [-.20, .03]	.04 [-.07, .16]	-.23** [-.33, -.11]	-.35** [-.45, -.24]	**.60**** [.52, .67]				
9. Efficiency (1)	0.00	1.00	.55** [.50, .61]	.28** [.17, .38]	.52** [.45, .57]	.30** [.19, .40]	.35** [.28, .42]	.19** [.07, .30]	-.77** [-.80, -.73]	-.56** [-.64, -.47]			
10. Efficiency (2)	-0.00	1.00	.37** [.26, .46]	.49** [.39, .57]	.30** [.19, .41]	.47** [.37, .55]	.26** [.14, .36]	.35** [.24, .45]	-.53** [-.61, -.44]	-.84** [-.87, -.81]	**.64**** [.57, .71]		
11. SD Reaction Time (1)	0.00	1.00	-.55** [-.61, -.49]	-.41** [-.50, -.31]	-.30** [-.37, -.22]	-.31** [-.41, -.20]	-.52** [-.58, -.46]	-.34** [-.44, -.23]	.72** [.68, .76]	.49** [.39, .57]	-.79** [-.82, -.76]	-.57** [-.64, -.48]	
12. SD Reaction Time (2)	-0.00	1.00	-.39**	-.62**	-.24**	-.34**	-.33**	-.56**	.41**	.72**	-.49**	-.80**	**.52****

	[-.48, -.28]	[-.68, -.54]	[-.35, -.13]	[-.44, -.23]	[-.43, -.22]	[-.64, -.48]	[.31, .50]	[.66, .77]	[-.57, -.39]	[-.84, -.75]	[.42, .60]

Note. (1) and (2) indicate scores at Time 1 and Time 2, respectively. *M* and *SD* are used to represent mean and standard deviation, respectively. Values representing test-retest Pearson correlations are indicated in bold. Values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014). * indicates $p < .05$. ** indicates $p < .01$. Table generated with the *apaTables* package (Stanley & Spence, 2018).

Table II.3

Means, standard deviations, and correlations with confidence intervals for all metrics extracted from Stop-Signal task

Variable	*M*	*SD*	1	2	3	4	5
1. Go Accuracy (1)	-0.00	1.00					
2. Go Accuracy (2)	-0.00	1.00	.37** [.18, .53]				
3. Reaction Time (1)	-0.00	1.00	.41** [.31, .49]	.23* [.04, .41]			
4. Reaction Time (2)	0.00	1.00	.15 [-.05, .33]	.31** [.20, .42]	.56** [.41, .68]		
5. SD Reaction Time (1)	0.00	1.00	.03 [-.08, .13]	-.04 [-.24, .15]	.40** [.30, .48]	.18 [-.01, .37]	
6. SD Reaction Time (2)	0.00	1.00	-.08 [-.27, .12]	-.06 [-.19, .06]	.09 [-.10, .29]	.32** [.21, .43]	.38** [.20, .54]

Note. (1) and (2) indicate scores from Time 1 and Time 2, respectively. *M* and *SD* are used to represent mean and standard deviation, respectively. Values representing test-retest Pearson correlations are indicated in bold. Values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014). * indicates $p < .05$. ** indicates $p < .01$. Table generated with the *apaTables* package (Stanley & Spence, 2018).

Table II.4

Correlations between EEA and all extracted metrics for both the Go/No-Go and Stop-Signal tasks at both time points, with confidence intervals

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
1. EEA (1)																			
2. EEA (2)	**.52**** [.43, .59]																		
3. GNG Accuracy (1)	.75** [.71, .78]	.43** [.33, .52]																	
4. GNG Accuracy (2)	.36** [.25, .45]	.73** [.67, .78]	**.41**** [.31, .50]																
5. GNG Accuracy, No-Go (1)	.67** [.63, .72]	.35** [.24, .44]	.69** [.65, .73]	.23** [.11, .33]															
6. GNG Accuracy, No-Go (2)	.36** [.25, .46]	.65** [.57, .71]	.39** [.28, .48]	.53** [.44, .61]	**.48**** [.38, .56]														
7. GNG Accuracy, Go (1)	.49** [.43, .55]	.32** [.21, .42]	.82** [.79, .84]	.37** [.27, .47]	.15** [.07, .23]	.15* [.03, .26]													
8. GNG Accuracy, Go (2)	.25**	.55**	.30**	.92**	.04	.16**	**.37****												

	[.14, .36]	[.46, .63]	[.19, .40]	[.90, .94]	[-.07, .16]	[.04, .27]	[.26, .47]									
9. SST Accuracy, Go (1)	.40**	.19	.17**	.13	.16**	-.12	.10	.22*								
	[.30, .48]	[-.00, .37]	[.06, .28]	[-.08, .33]	[.04, .27]	[-.32, .09]	[-.02, .21]	[.01, .41]								
10. SST Accuracy, Go (2)	.35**	.18**	.32**	.19**	.28**	.14*	.20**	.16*	**.37****							
	[.24, .46]	[.05, .29]	[.20, .42]	[.06, .31]	[.17, .39]	[.01, .26]	[.08, .32]	[.03, .28]	[.18, .53]							
11. GNG RT (1)	-.46**	-.32**	-.18**	-.19**	.12**	-.02	-.34**	-.21**	-.09	-.14*						
	[-.52, -.40]	[-.42, -.22]	[-.26, -.10]	[-.30, -.07]	[.04, .20]	[-.14, .09]	[-.41, -.27]	[-.32, -.09]	[-.20, .03]	[-.26, -.02]						
12. GNG RT (2)	-.41**	-.54**	-.22**	-.28**	-.08	.04	-.23**	-.35**	-.27*	-.07	**.60****					
	[-.50, -.31]	[-.62, -.46]	[-.33, -.10]	[-.39, -.17]	[-.20, .03]	[-.07, .16]	[-.33, -.11]	[-.45, -.24]	[-.46, -.07]	[-.20, .06]	[.52, .67]					
13. SST RT (1)	.08	.00	.12*	-.12	.12*	-.00	.06	-.13	.41**	.23*	.04	.05				
	[-.03, .18]	[-.19, .19]	[.00, .23]	[-.32, .10]	[.00, .23]	[-.21, .21]	[-.06, .17]	[-.33, .08]	[.31, .49]	[.04, .41]	[-.07, .16]	[-.17, .25]				
14. SST RT (2)	.04	-.02	.12	.10	.10	.10	.09	.07	.15	.31**	.08	.16*	**.56****			
	[-.08, .17]	[-.14, .10]	[-.00, .24]	[-.03, .23]	[-.03, .22]	[-.04, .22]	[-.03, .21]	[-.06, .20]	[-.05, .33]	[.20, .42]	[-.05, .20]	[.03, .28]	[.41, .68]			
15. GNG Efficiency (1)	.82**	.48**	.55**	.28**	.52**	.30**	.35**	.19**	.18**	.29**	-.77**	-.56**	.04	-.03		
	[.79, .84]	[.39, .57]	[.50, .61]	[.17, .38]	[.45, .57]	[.19, .40]	[.28, .42]	[.07, .30]	[.07, .29]	[.18, .40]	[-.80, -.73]	[-.64, -.47]	[-.08, .15]	[-.15, .10]		
16. GNG Efficiency (2)	.54**	.82**	.37**	.49**	.30**	.47**	.26**	.35**	.16	.12	-.53**	-.84**	-.04	-.10	**.64****	
	[.45, .62]	[.77, .85]	[.26, .46]	[.39, .57]	[.19, .41]	[.37, .55]	[.14, .36]	[.24, .45]	[-.06, .35]	[-.01, .25]	[-.61, -.44]	[-.87, -.81]	[-.25, .17]	[-.23, .03]	[.57, .71]	
17. GNG SDRT (1)	-.81**	-.52**	-.55**	-.41**	-.30**	-.31**	-.52**	-.34**	-.25**	-.27**	.72**	.49**	-.10	-.02	-.79**	-.57**
	[-.84, -.78]	[-.60, -.44]	[-.61, -.49]	[-.50, -.31]	[-.37, -.22]	[-.41, -.20]	[-.58, -.46]	[-.44, -.23]	[-.36, -.14]	[-.38, -.15]	[.68, .76]	[.39, .57]	[-.21, .02]	[-.14, .11]	[-.82, -.76]	[-.64, -.48]

18. GNG SDRT (2)	-.48** [-.56, -.38]	-.88** [-.90, -.85]	-.39** [-.48, -.28]	-.62** [-.68, -.54]	-.24** [-.35, -.13]	-.34** [-.44, -.23]	-.33** [-.43, -.22]	-.56** [-.64, -.48]	-.28** [-.46, -.07]	-.08 [-.21, .05]	.41** [.31, .50]	.72** [.66, .77]	-.03 [-.24, .18]	.06 [-.07, .19]	-.49** [-.57, -.39]	-.80** [-.84, -.75]	**.52**** [.42, .60]		
19. SST SDRT (1)	-.19** [-.29, -.08]	-.10 [-.29, .09]	-.06 [-.18, .06]	-.06 [-.27, .15]	-.03 [-.15, .08]	.06 [-.15, .27]	-.06 [-.17, .06]	-.11 [-.31, .10]	.03 [-.08, .13]	-.04 [-.24, .15]	.06 [-.06, .18]	.22* [.01, .41]	.40** [.30, .48]	.18 [-.01, .37]	-.08 [-.20, .03]	-.13 [-.33, .08]	.08 [-.04, .19]	.15 [-.06, .35]	
20. SST SDRT (2)	-.17** [-.28, -.04]	-.18** [-.30, -.06]	-.00 [-.12, .12]	-.07 [-.20, .06]	-.01 [-.14, .11]	.03 [-.10, .16]	.01 [-.11, .14]	-.10 [-.23, .03]	-.08 [-.27, .12]	-.06 [-.19, .06]	.15* [.03, .27]	.17* [.04, .29]	.09 [-.10, .29]	.32** [.21, .43]	-.16* [-.28, -.03]	-.14* [-.26, -.01]	.14* [.02, .26]	.14* [.01, .27]	**.38**** [.20, .54]

Note. This table depicts correlations between all task metrics for both the go/no-go and stop-signal tasks at both timepoints. Time 1 is depicted with (1) and Time 2 is depicted with (2). Values which represent test-retest Pearson correlations are indicated in bold. Values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014). * indicates $p < .05$. ** indicates $p < .01$. Table generated with the *apaTables* package (Stanley & Spence, 2018).

Table II.5

Means, standard deviations, and correlations with confidence intervals for EEA and "average" metrics

Variable	*M*	*SD*	1	2	3	4	5	6	7
1. EEA (1)	0.00	0.67							
2. EEA (2)	0.00	0.61	**.52**** [.43, .59]						
3. Go Accuracy (1)	-0.01	0.96	.52** [.46, .57]	.32** [.22, .42]					
4. Go Accuracy (2)	-0.01	0.86	.36** [.26, .45]	.48** [.39, .56]	**.39**** [.29, .48]				
5. Reaction Time (1)	0.04	0.91	-.38** [-.45, -.31]	-.30** [-.40, -.20]	-.19** [-.27, -.12]	-.21** [-.31, -.10]			
6. Reaction Time (2)	0.02	0.83	-.26** [-.36, -.15]	-.39** [-.48, -.29]	-.11* [-.22, -.00]	-.10 [-.21, .01]	**.54**** [.46, .61]		
7. SD Reaction Time (1)	0.04	0.95	-.73** [-.76, -.69]	-.50** [-.58, -.41]	-.42** [-.49, -.36]	-.36** [-.45, -.26]	.65** [.60, .69]	.38** [.28, .47]	
8. SD Reaction Time (2)	0.01	0.83	-.41** [-.50, -.31]	-.71** [-.76, -.65]	-.27** [-.37, -.16]	-.38** [-.47, -.28]	.37** [.27, .46]	.60** [.52, .66]	**.46**** [.37, .54]

Note. This table depicts summary statistics for and correlations between the "average" task metrics, calculated as the mean of a participant's performance on the metric from the go/no-go and stop-signal tasks when both were available. *M* and *SD* are used to represent mean and standard deviation, respectively. Values representing test-retest Pearson correlations are indicated in bold. Values

in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014). * indicates $p < .05$. ** indicates $p < .01$. Table generated with the *apaTables* package (Stanley & Spence, 2018).

Table II.6

Correlations with confidence intervals for metrics available for both tasks at first visit

Variable	1	2	3	4	5	6	7	8
1. EEA (Go)								
2. EEA (No-Go)	.61** [.56, .66]							
3. EEA (SST)	**.28**** [.17, .38]	**.30**** [.19, .40]						
4. Go Accuracy (GNG)	.54** [.48, .59]	.36** [.29, .43]	.16** [.05, .28]					
5. Go Accuracy (SST)	.21** [.10, .32]	.29** [.18, .39]	.71** [.65, .76]	**.10** [-.02, .21]				
6. Reaction Time (GNG)	-.50** [-.56, -.44]	-.33** [-.40, -.26]	-.17** [-.28, -.05]	-.34** [-.41, -.27]	-.09 [-.20, .03]			
7. Reaction Time (SST)	.12* [.01, .24]	.06 [-.06, .17]	-.02 [-.13, .09]	.06 [-.06, .17]	.41** [.31, .49]	**.04** [-.07, .16]		
8. SD Reaction Time (GNG)	-.88** [-.90, -.86]	-.59** [-.64, -.54]	-.32** [-.42, -.21]	-.52** [-.58, -.46]	-.25** [-.36, -.14]	.72** [.68, .76]	-.10 [-.21, .02]	
9. SD Reaction Time (SST)	-.07 [-.18, .05]	-.07 [-.18, .05]	-.57** [-.64, -.49]	-.06 [-.17, .06]	.03 [-.08, .13]	.06 [-.06, .18]	.40** [.30, .48]	**.08** [-.04, .19]

Note. *M* and *SD* are used to represent mean and standard deviation, respectively. Values representing cross-task Pearson correlations are indicated in bold. Values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014). * indicates $p < .05$. ** indicates $p < .01$. Table generated with the *apaTables* package (Stanley & Spence, 2018).

Table II.7

Correlations with confidence intervals for concurrent symptoms of psychopathology and extracted metrics

Variable	EEA	No-Go Accuracy[a]	Go Accuracy	Reaction Time	Efficiency[a]	SDRT
Total Problems	-.00 [-.08, .07]	-.04 [-.12, .04]	-.01 [-.09, .07]	-.08* [-.16, -.01]	.02 [-.06, .10]	-.02 [-.10, .06]
Externalizing	-.04 [-.12, .04]	-.07 [-.15, .01]	-.04 [-.12, .04]	-.07 [-.14, .01]	.01 [-.07, .09]	.04 [-.04, .12]
Internalizing	.11** [.04, .19]	.09* [.01, .17]	.05 [-.03, .13]	-.09* [-.17, -.01]	.11** [.03, .19]	-.11** [-.19, -.04]
Attention Problems	-.14** [-.21, -.06]	-.16** [-.24, -.08]	-.10* [-.17, -.02]	-.03 [-.10, .05]	-.11** [-.19, -.03]	.05 [-.02, .13]
Anxious/Depressed Problems	.15** [.08, .23]	.09* [.01, .17]	.08* [.00, .16]	-.13** [-.21, -.06]	.15** [.07, .23]	-.14** [-.22, -.06]
Withdrawn/Depressed Problems	.07 [-.00, .15]	.05 [-.03, .13]	.04 [-.04, .11]	-.05 [-.13, .02]	.07 [-.01, .15]	-.08* [-.16, -.00]
Rule Breaking	-.06 [-.14, .02]	-.08 [-.16, .00]	-.08* [-.16, -.00]	-.06 [-.14, .02]	.03 [-.05, .11]	.05 [-.02, .13]
Aggression	-.02 [-.10, .06]	-.05 [-.13, .03]	-.01 [-.08, .07]	-.06 [-.14, .02]	-.00 [-.08, .08]	.02 [-.06, .10]

Note. Values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014). * indicates $p < .05$. ** indicates $p < .01$. [a] indicates calculated only for Go/No-Go task.

Table II.8

Standardized coefficients for multilevel models of extracted metrics predicting concurrent symptoms of psychopathology

Variable	EEA	No-Go Accuracy[a]	Go Accuracy	Reaction Time	Efficiency[a]	SDRT
Total Problems	-.09 [-.18, .01]	-.11* [-.20, .00]	-.03 [-.10, .03]	-.05 [-.12, .04]	-.06 [-.16, .03]	.04 [-.03, .12]
Externalizing	-.11* [-.20, -.01]	-.11* [-.21, .00]	-.06 [-.13, .00]	-.02 [-.14, .03]	-.07 [-.17, .04]	.10** [.03, .16]
Internalizing	.02 [-.08, .11]	-.01 [-.09, .08]	.02 [-.05, .09]	-.06 [-.10, .05]	.05 [-.04, .14]	-.04 [-.12, .03]
Attention Problems	-.21** [-.30, -.11]	-.19** [-.29, -.08]	-.11* [-.20, -.03]	.02 [-.06, .10]	-.19** [-.30, -.08]	.10** [.02, .18]
Anxious/Depressed Problems	.06 [-.04, .15]	-.02 [-.12, .08]	.05 [-.02, .12]	-.10* [-.17, -.02]	.08 [-.02, .17]	-.07 [-.14, .00]
Withdrawn/Depressed Problems	-.02 [-.10, .07]	-.02 [-.11, .07]	.01 [-.06, .08]	.00 [-.08, .09]	-.01 [-.11, .08]	-.01 [-.09, .06]
Rule Breaking	-.17** [-.27, -.07]	-.13* [-.23, -.03]	-.11** [-.19, -.04]	.03 [-.03, .10]	-.10 [-.21, .01]	.15** [.08, .21]
Aggression	-.05 [-.14, .04]	-.08 [-.17, .02]	-.01 [-.09, .05]	-.06 [-.12, .02]	-.03 [-.13, .07]	.05 [-.02, .12]

Note. Values indicate the standardized estimate (β) for each metric predicting symptoms of psychopathology. Models controlled for age and sex, and clustered by family to account for relatedness. Values in square brackets indicate the 95% confidence interval for each standardized estimate. * indicates $p < .05$. ** indicates $p < .01$. [a] indicates calculated only for Go/No-Go task.

Table II.9

Correlations with confidence intervals for prospective symptoms of psychopathology and extracted metrics

Variable	EEA	No-Go Accuracy[a]	Go Accuracy	Reaction Time	Efficiency[a]	SDRT
Total Problems	.01 [-.10, .11]	-.00 [-.11, .11]	-.02 [-.12, .09]	-.05 [-.15, .06]	.02 [-.09, .13]	.00 [-.11, .11]
Externalizing	-.10 [-.21, .00]	-.10 [-.20, .01]	-.07 [-.18, .04]	-.02 [-.12, .09]	-.06 [-.16, .05]	.09 [-.02, .20]
Internalizing	.16** [.05, .26]	.14* [.03, .24]	.06 [-.05, .17]	-.09 [-.20, .02]	.15** [.04, .26]	-.11* [-.22, -.00]
Attention Problems	-.12* [-.22, -.01]	-.12* [-.23, -.01]	-.08 [-.19, .03]	.01 [-.10, .12]	-.10 [-.21, .01]	.09 [-.02, .20]
Anxious/Depressed Problems	.22** [.12, .32]	.17** [.06, .27]	.12* [.02, .23]	-.13* [-.23, -.02]	.21** [.10, .31]	-.17** [-.27, -.06]
Withdrawn/Depressed Problems	.07 [-.04, .18]	.06 [-.05, .17]	.01 [-.10, .12]	-.05 [-.15, .06]	.09 [-.02, .20]	-.02 [-.13, .08]
Rule Breaking	-.07 [-.18, .04]	-.08 [-.19, .03]	-.07 [-.18, .04]	-.06 [-.17, .05]	.03 [-.08, .14]	.06 [-.05, .17]
Aggression	-.11 [-.21, .00]	-.09 [-.20, .02]	-.06 [-.17, .05]	.02 [-.09, .12]	-.10 [-.21, .01]	.10 [-.01, .20]

Note. Values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014). * indicates $p < .05$. ** indicates $p < .01$. [a] indicates calculated only for Go/No-Go task.

Table II.10

Standardized coefficients for multilevel models of extracted metrics predicting prospective symptoms of psychopathology

Variable	EEA	No-Go Accuracy[a]	Go Accuracy	Reaction Time	Efficiency[a]	SDRT
Total Problems	-.01 [-.12, .12]	-.04 [-.20, .00]	.00 [-.10, .09]	-.07 [-.18, .04]	.03 [-.10, .17]	.01 [-.10, .11]
Externalizing	-.12* [-.22, .00]	-.11 [-.21, .00]	-.07 [-.18, .05]	-.03 [-.15, .08]	-.06 [-.18, .07]	.10 [-.02, .21]
Internalizing	.12* [.00, .23]	.06 [-.09, .08]	.07 [-.01, .14]	-.10* [-.20, -.01]	.14* [.02, .27]	-.10 [-.19, .00]
Attention Problems	-.12 [-.26, .01]	-.13 [-.29, -.08]	-.07 [-.17, .02]	-.02 [-.15, .11]	-.09 [-.23, .06]	.09 [-.04, .21]
Anxious/Depressed Problems	.17* [.05, .29]	.08 [-.12, .08]	.13* [.05, .19]	-.12* [-.24, -.02]	.18** [.06, .31]	-.13* [-.24, -.03]
Withdrawn/Depressed Problems	.06 [-.07, .18]	.03 [-.11, .16]	.01 [-.09, .11]	-.06 [-.16, .06]	.08 [-.05, .22]	-.01 [-.12, .10]
Rule Breaking	-.16* [-.26, -.04]	-.13* [-.23, -.03]	-.09 [-.22, .03]	.00 [-.12, .10]	-.06 [-.18, .09]	.14** [.03, .24]
Aggression	-.07 [-.18, .05]	-.08 [-.20, .05]	-.04 [-.15, .07]	-.05 [-.15, .06]	-.05 [-.17, .07]	.06 [-.06, .17]

Note. Values indicate the standardized estimate (β) for each metric predicting symptoms of psychopathology. Models controlled for age and sex, and clustered by family to account for relatedness. Values in square brackets indicate the 95% confidence interval for each standardized estimate. * indicates $p < .05$. ** indicates $p < .01$. [a] indicates calculated only for Go/No-Go task.

Table II.11

Clusters in which better executive functioning performance correlates with increased inhibition-related activation

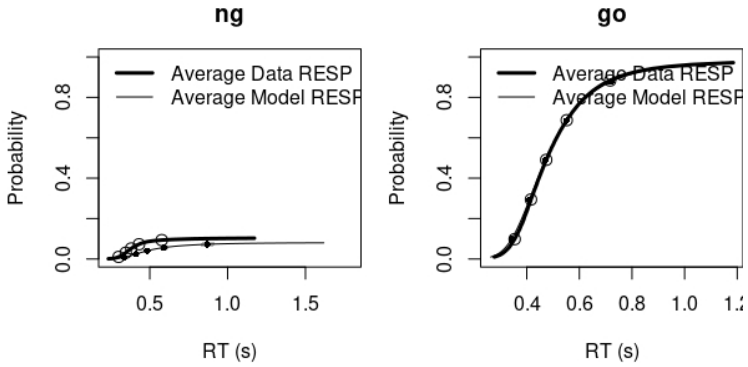
Variable	Structure	Peak (X, Y, Z)	T	k (# voxels)
EEA	Anterior Cingulate Cortex	(-2, 48, 2)	4.40	787
	Superior Temporal Gyrus	(54, -44, 14)	5.13	814
No-Go Accuracy[a]	-			
Go Accuracy	-			
Reaction Time	Anterior Cingulate Cortex	(-8, 40, 16)	4.23	494
	Superior Temporal Gyrus *extending to Angular Gyrus	(54, -44, 12)	3.96	691
Efficiency[a]	Anterior Cingulate Cortex	(-4, 28, 22)	4.81	1498
	Superior Temporal Gyrus	(54, -44, 12)	5.37	616
	Inferior Frontal Gyrus (R)	(38, 24, -14)	4.56	164
	Inferior Frontal Gyrus (L)	(-40, 18, -12)	4.39	157
	Precuneus	(-18, -62, 20)	4.24	348
SD Reaction Time	Anterior Cingulate Cortex	(-6, 42, 14)	4.85	712
	Superior Temporal Gyrus	(56, -56, 10)	4.21	185

Note. Brain regions in which better executive functioning task performance correlated with increased inhibition-related (no-go>go) activation in a sample of 549 twins. False positive rate is controlled across the whole brain using 3dttest++ for cluster-level correction ($p < 0.001$, alpha < 0.05, $k > 164$) [a] indicates metric was calculated only for Go/No-Go task, $N = 540$, $k > 152$. Structure labels for brain regions not originally hypothesized (e.g. STG) were determined via Neurosynth.org (Yarkoni et al., 2011) and SPM Neuromorphometrics.

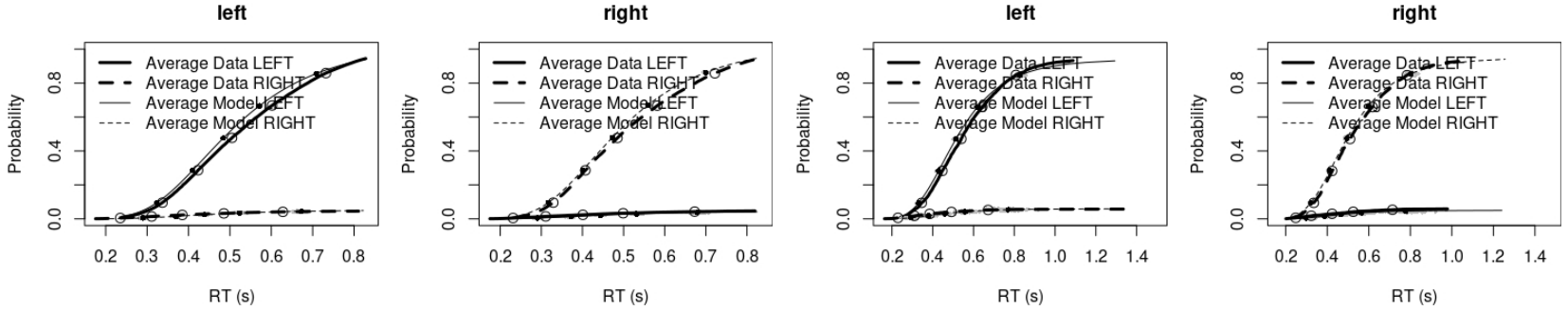
Figure II.1

Drift diffusion model fit for all task versions and conditions

a) Go/No-Go



b) Stop Signal (both task versions)

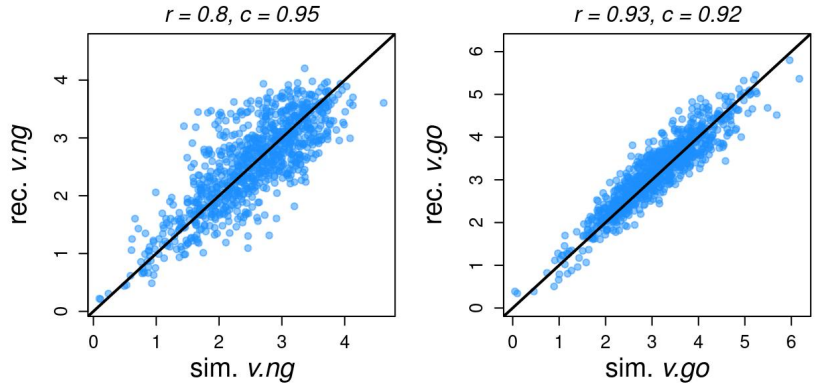


Note. These graphs depict the model predicted data and the actual data for the go/no-go (a) and stop-signal (b) tasks. Model fit to the data was good for most conditions and tasks. The model had some difficulty predicting commission errors on no-go trials, but this was within expectations as "no-go" was the most rare trial type and commission errors were low overall (average no-go accuracy was 89%).

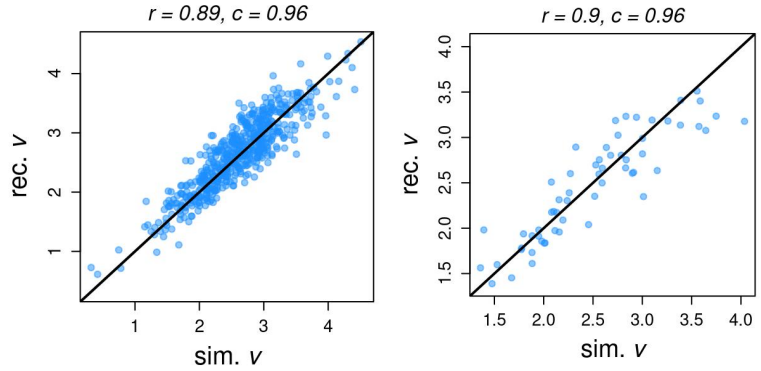
Figure II.2

Drift diffusion model drift rate (v) parameter recovery for all task versions and conditions

a) Go/No-Go



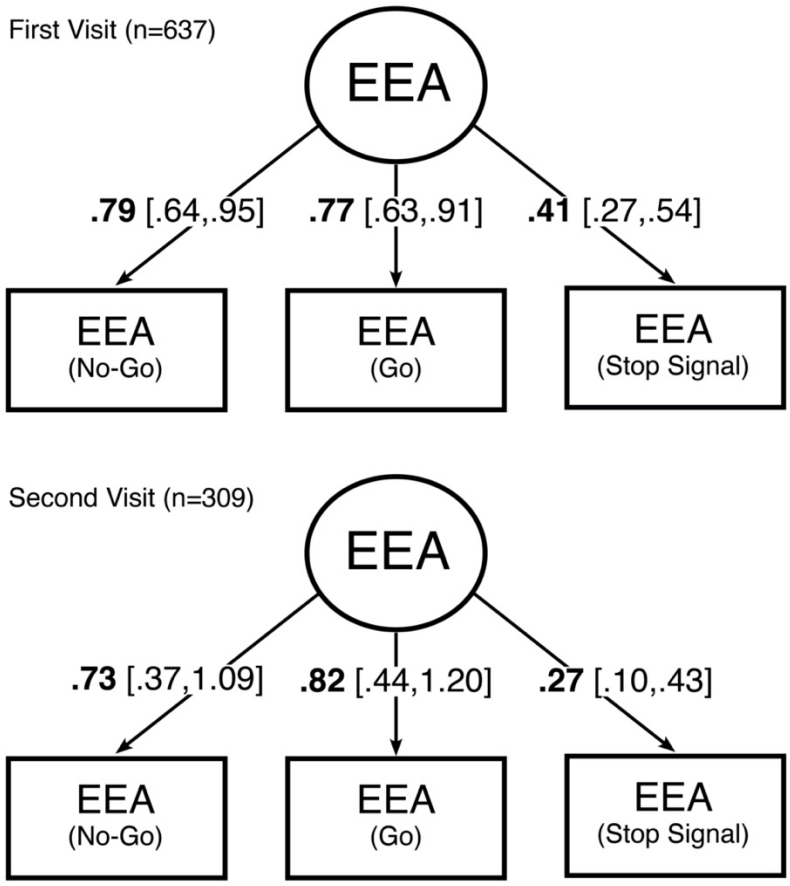
b) Stop-Signal (both task versions)



Note. These graphs depict the drift rate (*v*) parameters recovered from simulated data (sim, x axis) and the actual data (rec) for the go/no-go (a) and stop-signal (b) tasks. Parameter recovery was excellent.

Figure II.3

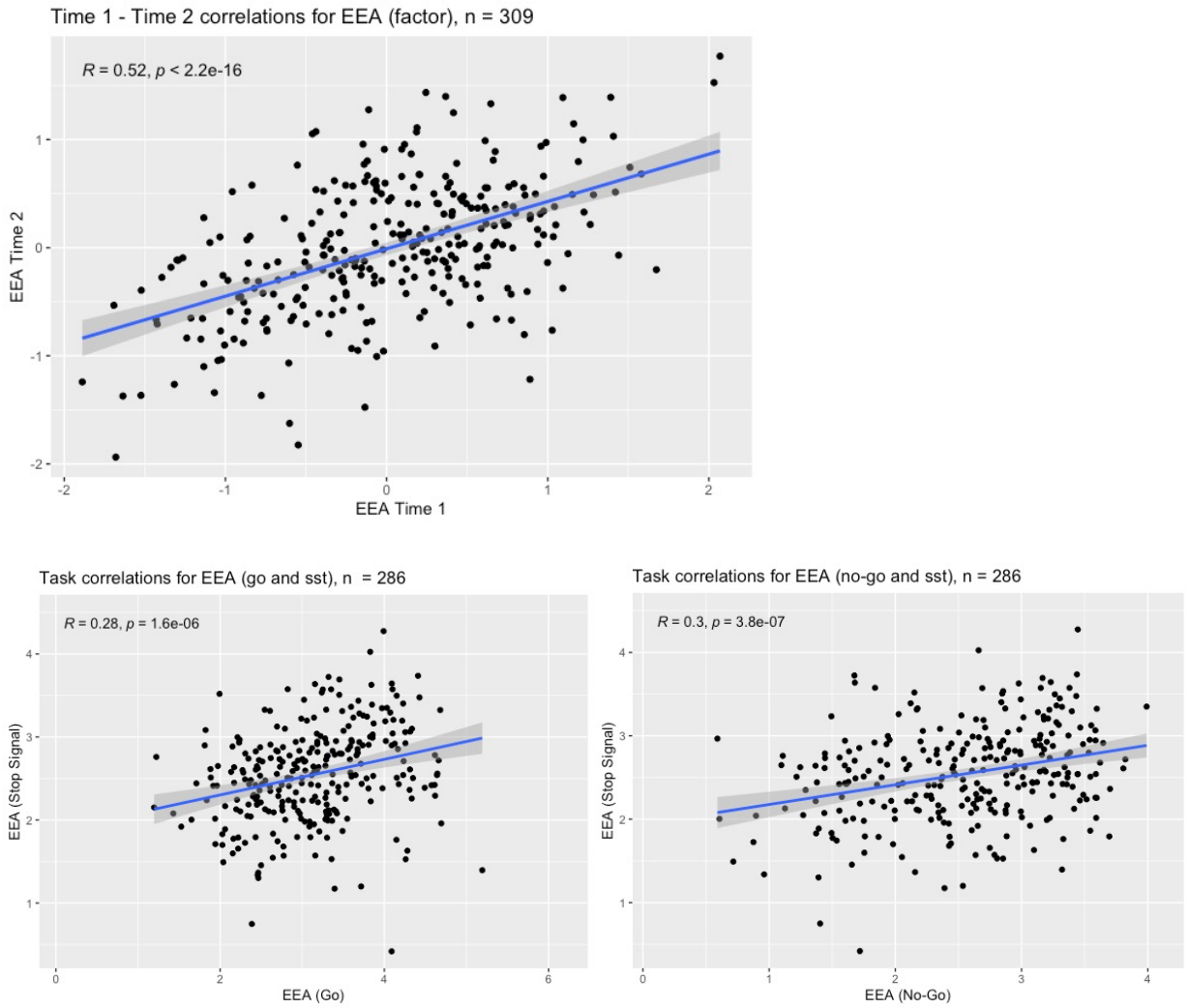
EEA Latent Factor Models at First and Second Visits, With Standardized Loadings



Note. This figure depicts the two latent EEA models generated in Mplus (Muthén & Muthén, 1999-2017) via R (R Core Team, 2021) and the MplusAutomation package (Hallquist & Wiley, 2018). Models utilized maximum likelihood estimation with bootstrapping (1000), and factor scores were extracted for further analysis.

Figure II.4

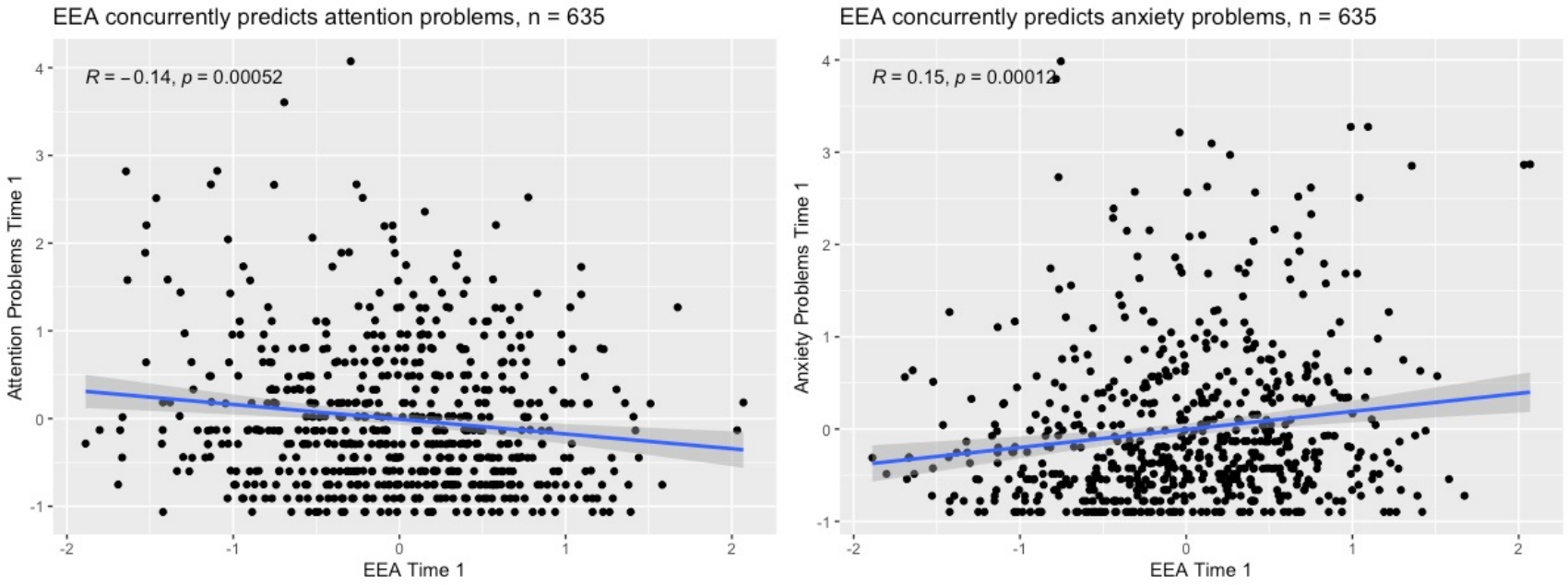
Test-retest and cross-task reliability for the EEA factor score and EEA metrics



Note. This figure depicts test-retest (top) and across-task (bottom) Pearson correlations for EEA. Task correlations used the individual EEA scores for each condition (go, no-go, stop signal) rather than the latent factor score.

Figure II.5

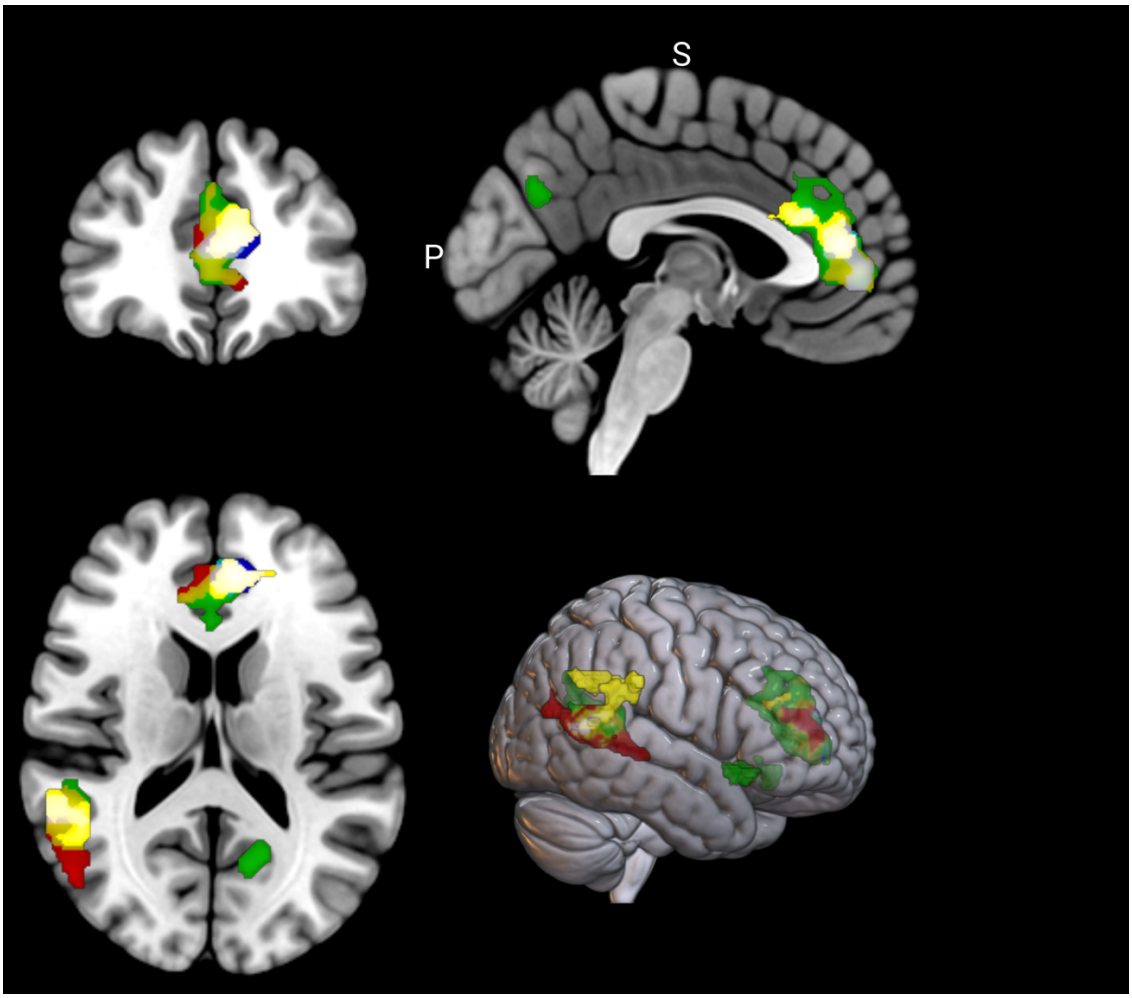
Zero-order correlations between EEA and symptoms of psychopathology for select subscales



Note. This figure depicts zero-order correlations between task performance as measured by EEA and concurrent attention problems (left) and concurrent anxious/depressed problems (right) as reported by children and their parents.

Figure II.6

Unique and overlapping regions in which better EEA (red), Efficiency (green), SDRT (blue), and RT (yellow) correlated with increased inhibition-related brain activation

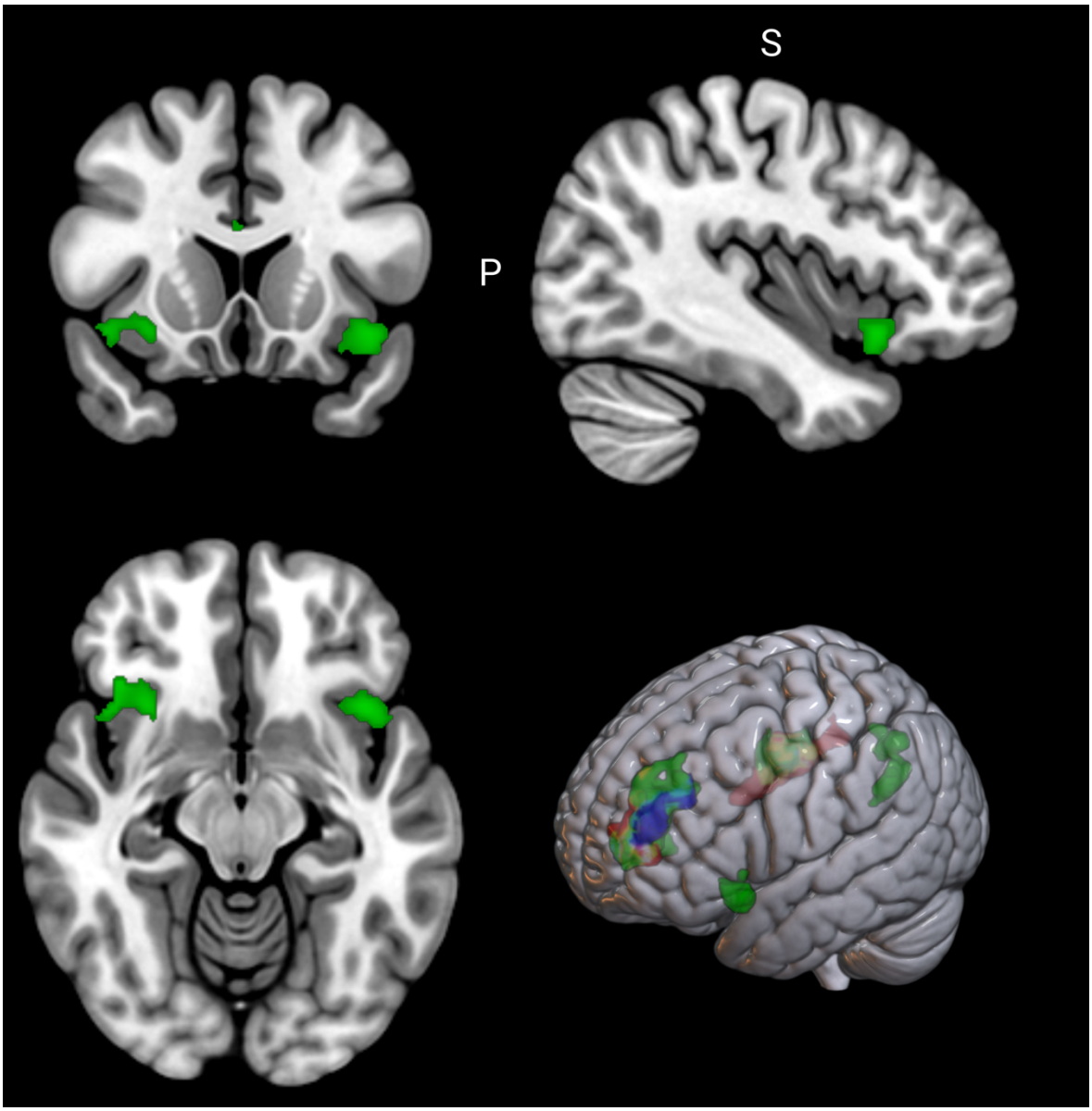


Note. Slice location of this image is (-33, 40, 16). This image depicts substantial overlap in regions correlating with executive functioning performance as measured by EEA (red), Efficiency (green), Standard Deviation of reaction time (blue), and Reaction Time (bright yellow) particularly within the anterior cingulate cortex and superior temporal gyrus. Additive overlap between images is indicated with color blending; therefore, locations indicated in white

would represent areas with the most overlap. Neither no-go nor go accuracy scores correlated with inhibition-related activation. Image generated from activation maps cluster-thresholded with 3dttest++ (Cox, 1996; Cox et al., 2017a, 2017b) in SPM (Wellcome Centre for Human Neuroimaging) and rendered via MRIcroGL (Rorden & Brett, 2000).

Figure II.7

Additional clusters in which better Go/No-Go Efficiency correlated with increased inhibition-related activation

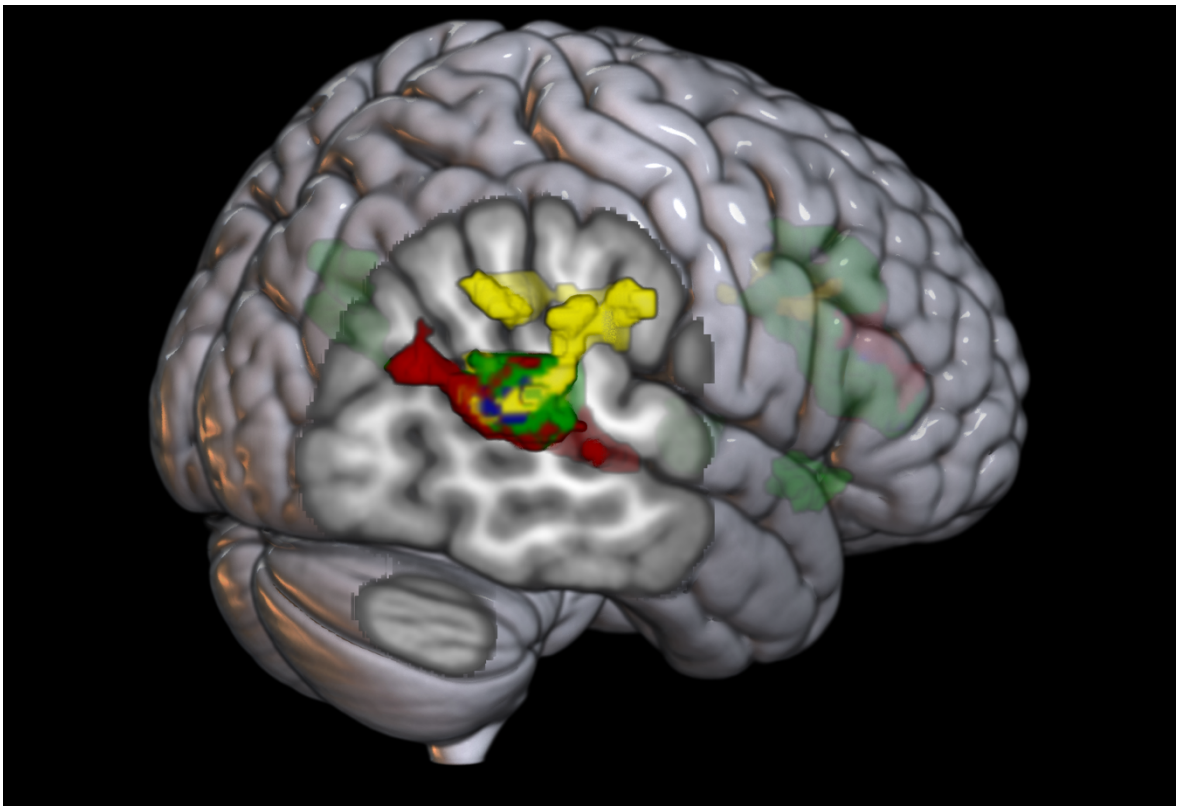


Note. Slice location of this image is (-40, 16, -12). This image depicts bilateral inferior frontal gyrus clusters that correlated with executive functioning performance as measured by Go/No-Go Efficiency (green). No other executive functioning metric correlated with activation within this region after correcting for multiple comparisons across the whole brain. Image generated from

activation maps cluster-thresholded with 3dttest++ (Cox, 1996; Cox et al., 2017a, 2017b) in SPM (Wellcome Centre for Human Neuroimaging) and rendered via MRIcroGL (Rorden & Brett, 2000).

Figure II.8

Variance in shape of the superior temporal gyrus clusters associated with better EEA, Efficiency, Reaction Time, and Standard Deviation of Reaction Time



Note. This image depicts variance in superior temporal gyrus cluster extent despite similar peak voxels for the four executive functioning performance metrics (EEA, red; Efficiency, green; SDRT, blue; Reaction Time, yellow). Render location (56, -37, -2) with clip location (0.09, 67, 0) in MRIcroGL (Rorden & Brett, 2000).

CHAPTER III

The Role of Parenting in the Intergenerational Transmission of Executive Functioning: A Genetically Informed Approach

Deficits in executive functioning, a set of top-down processes that involve regulation of goal-directed behavior, including processes like inhibitory control, working memory, and cognitive flexibility (Diamond, 2013), have long been linked to various forms of psychopathology (Bloemen et al., 2018; Wright et al., 2014), including attention-deficit/hyperactivity disorder (Castellanos et al., 2006; Cherkasova et al., 2013), antisocial behavior (Moffitt, 2018), substance abuse (Smith et al., 2014; Verdejo-Garcia et al., 2008), and depression (Gotlib & Joormann, 2010). Executive functioning runs in families, such that parents with better executive functioning skills tend to have children with better executive functioning skills (Deater-Deckard, 2014; Jester et al., 2009). However, much observational work does not disentangle the genetic and non-genetic influences on individual differences in executive functioning (Cuevas, Deater-Deckard, Kim-Spoon, Wang, et al., 2014), and genetically informed work is needed to clarify the mechanisms that contribute to its intergenerational transmission.

Parenting behaviors are one proposed mechanism underlying the intergenerational transmission of executive functioning. There is substantial evidence that parenting behaviors like nurturance and scaffolding promote better executive functioning development, whereas behaviors like harshness and negative control impede executive functioning development (Fay-Stammbach et al., 2014; Hughes & Devine, 2019; Li et al., 2019). Parent executive functioning is thought to impact parenting (and the broader home environment), which in turn, impacts child

development (e.g. Distefano et al., 2018; Kao et al., 2018; Korucu et al., 2020). For example, a parent with better executive functioning skills may have a better ability to utilize more nurturing and less harsh parenting behaviors, promoting better executive functioning development in their child. However, with some notable exceptions (Bridgett et al., 2018; Cioffi et al., 2020), the majority of developmental studies linking parent executive functioning to child executive functioning via parenting behaviors are not genetically informed.

A separate line of research implicates genetic influences as a primary contributor to the intergenerational transmission of executive functioning. Performance on executive functioning tasks is moderately heritable, though with evidence of moderate non-shared environmental influences (Gagne & Saudino, 2010; Li & Roberts, 2017; Vasilopoulos et al., 2012). The heritability of task-general executive functioning, as indexed via latent modeling, is thought to be much higher (Friedman & Miyake, 2017), with estimates as high as 99% (Friedman et al., 2008) or 100% (Engelhardt et al., 2015). The high heritability of latent executive functioning also appears to be consistent across development, from childhood into adulthood (Friedman & Miyake, 2017).

Given these heritability estimates, it is entirely possible that observational studies linking parenting to executive functioning are actually capturing unmeasured genetic processes. That is, because parents and children share genes, the extent to which parenting mediates the relationship between parent and child executive functioning could be driven by *genotype-environment correlation,* or the correlation between the parents' (and thus children's) genetic tendencies and the environments the children encounter (Deater-Deckard, 2014; Knopik et al., 2017; Manuck & McCaffery, 2014). Two types of genotype-environment correlation are particularly important in this case: *passive* and *evocative genotype-environment correlation* (see Manuck & McCaffery,

2014 for a discussion). First, passive genotype-environment correlation emerges because the environment parents provide to their biological children is influenced by the parents' own genotype, the latter which is passed onto their children genetically. As such, the child's genes are necessarily correlated with their environmental experiences. For example, parents with worse executive functioning may pass on trait-relevant genes of risk for executive dysfunction and react more harshly to misbehavior on the part of their child as a consequence of their compromised executive functioning (Bridgett et al., 2017; Deater-Deckard et al., 2010). Put another way what appears to be an effect of parental reactivity on child executive dysfunction may in fact reflect genes common to parents and their children. Second, evocative genotype-environment correlation captures the extent to which children elicit reactions from their environment that are consistent with their genetically-influenced tendencies. In this case, children with a genetic predisposition for lower executive functioning could exhibit behaviors that evoke harsher parenting. Put another way, parenting would be a consequence of child executive functioning, such that the genetic influences on executive functioning would correlate with those on parenting. Consistent with this idea, previous adoption work has found evidence for an evocative genotype-environment correlation in which higher levels of child anger evokes harsher parenting from adoptive mothers (Bridgett et al., 2018). In sum, some non-genetically informed studies may implicate parenting as a causal mechanism when these associations are actually a function of passive or evocative genotype-environment correlation.

Fortunately, there are an array of twin modeling approaches that can leverage the genetic relatedness of twins to elucidate the role(s) of genotype-environment correlation. First, the nuclear twin family model (Burt & Klump, 2012; Keller et al., 2010) leverages similarities across twins and their biological parents to determine the etiology of a given phenotype. This

model provides several advantages over the classical twin design, including the ability to divide shared environmental influences (C in the classical twin model) into those shared by siblings (S; “sibling-level shared environmental influences”) and those shared by twins and their parents (F; "family-level shared environmental influences"; Burt & Klump, 2012; Keller et al., 2010). Sibling-level shared environmental influences (S) include anything that makes siblings more similar to each other, but not to their parents, such as shared school environments, shared parenting experiences, and shared peers (Burt & Klump, 2012; Keller et al., 2010). Family-level shared environmental influences (F), on the other hand, include influences that make twins and their parents more similar to each other, such as shared cultural influences, family socioeconomic status, and broader societal norms (Burt & Klump, 2012; Keller et al., 2010). Additionally, this model can directly quantify passive genotype-environment correlation, calculated as the covariance between additive genetic (A) and family shared environmental effects (F; Burt & Klump, 2012; Keller et al., 2010).

To examine evocative genotype-environment correlation, we can make use of a bivariate ACE model. This model decomposes the association between parenting and child executive functioning into genetic (A), shared environmental (C), and non-shared environmental (E) components, providing insight into the genetic and environmental origins of their association. Moreover, because genetic influences (A) on parenting in a child twin design necessarily index the extent to which twins’ genetic tendencies shape the parenting they receive (Klahr & Burt, 2014), the presence of a genetic correlation would largely capture the role of evocative genotype-environment correlation. Moreover, the bivariate modeling approach can test whether there are shared or non-shared environmental influences that overlap between parenting and child executive functioning, which would support the notion that some of the association between

parenting and child executive functioning is environmental in origin. Thus, between these two modeling approaches, both passive and evocative genotype-environment correlation can be directly measured. We are also able to clarify whether there are environmental mechanisms through which parenting impacts child executive function.

In sum, these complementary modeling approaches can clarify the role that various forms of genotype-environment correlation play in the complex relationship between parent executive functioning, parenting, and child executive functioning. In doing so, however, it would be important to attend to a few key issues: First, it is important to consider the reliability and validity of frequently used executive functioning measures (Eisenberg et al., 2019; Karr et al., 2018). The metrics traditionally extracted from behavioral executive functioning tasks suffer from test-retest reliability issues, do not consistently relate to real-world outcomes such as mental health, physical health, or income, and do not load into a consistent factor structure (Eisenberg et al., 2019; Enkavi et al., 2019; Hedge et al., 2018; Karr et al., 2018; Rouder & Haaf, 2019; Weigard et al., 2021). One emerging and promising alternative to traditional behavioral measures is to use computational cognitive models to capture the processes underlying performance across a variety of tasks that engage executive functioning skills (Weigard & Sripada, 2021). Evidence accumulation models (Brown & Heathcote, 2008; Evans & Wagenmakers, 2020; Ratcliff et al., 2016), which are widely used to model choice response time tasks (including many executive functioning measures) suggest that a single higher-order individual difference dimension drives performance across many tasks: the efficiency with which individuals can gather evidence for correct choices in the context of background noise (Lerche et al., 2020; Schmiedek et al., 2007; Weigard et al., 2021). “Efficiency of evidence accumulation”, as indexed by these computational models, exhibits good test-retest reliability (Lerche & Voss,

2017; Schubert et al., 2016) and outperforms traditional executive functioning metrics both in temporal stability and in prediction of individuals' self-regulation (Weigard et al., 2021). In addition to using computational models of behavioral performance, behavioral measures can be complemented via the inclusion of survey measures. Though often weakly correlated with these behavioral measures of executive functioning (Dang et al., 2020), self-report, survey measures of executive functioning show stronger associations with real-world outcomes than do behavioral measures (Eisenberg et al., 2019). Thus, to maximize relevance to real-world outcomes, the present study combines computational and self-report measures of child executive functioning.

Second, much of the extant work focuses on infants and young children (e.g. Bernier et al., 2012; Bernier et al., 2010; Blair et al., 2014; Bridgett et al., 2018; Broomell et al., 2020; Cioffi et al., 2020; Distefano et al., 2018; Hammond et al., 2012; Helm et al., 2020; Hughes & Devine, 2019; Kao et al., 2018; Korucu et al., 2020; Zeytinoglu et al., 2017). Early childhood is indeed an important time period to study, as parenting influences may be especially important for young children (Gee, 2016; Shaw & Bell, 1993) and the prefrontal cortex and associated executive functioning skills are rapidly developing during this time (Kolb et al., 2012; Zelazo & Carlson, 2012). However, prefrontal cortex development continues through later childhood and adolescence (Kolb et al., 2012) and adolescence is a peak period for the onset of disorders associated with executive functioning difficulties (Cherkasova et al., 2013; Moffitt, 2018). Given evidence that parenting is related to executive functioning and related constructs during adolescence (Li et al., 2019), work is needed that considers the role of parenting on the intergenerational transmission of executive functioning in later childhood and adolescence as these skills are refined and begin to approach adult levels.

Thus, the present study examined the roles of shared genes and parenting in the intergenerational transmission of executive functioning in a large, socioeconomically diverse sample of children and adolescents. We utilized behavioral and informant-report measures of executive functioning for twins and their parents from the Michigan Twin Neurogenetics Study (MTwiNS), a unique longitudinal study of twins (N=708 in 354 pairs) that were oversampled for residence in low-income neighborhoods. For our first aim, we assessed whether harsh or nurturing parenting were phenotypically related to a computationally derived, task-general measure of executive functioning. We then tested whether parent executive functioning was associated with child executive functioning via parenting behaviors. We hypothesized that more nurturing, less harsh parenting would be associated with better child executive functioning.

For our second aim, we assessed the etiology of executive functioning via the nuclear twin family model. Using the nuclear twin family model, we assessed the role of sibling-level and family-level influences on executive functioning and quantified the role of passive genotype-environment correlation. We hypothesized that executive functioning would be moderately heritable, and that there would be evidence of passive genotype-environment correlation, reflecting that the genes underlying parent executive functioning relate to the general family environment, including the overall parenting environment.

For our third aim, we employed a bivariate twin model to examine the etiology of the phenotypic association between parenting and executive functioning. Our hypotheses for this aim were informed by results of Bridgett et al. (2018), an adoption study which, by design, eliminated the impact of passive genotype-environment correlation. This study found evidence of evocative genotype-environment correlation (i.e., child anger evoked harsher parenting) and an environmental effect of parenting on child self-regulation after controlling for evocative

genotype-environment correlation. Thus, we hypothesized that there would be some environmental influence of parenting on child executive functioning, as well as evidence of evocative genotype-environment correlation.

Methods

Participants

The present study included data from families in the Michigan Twins Neurogenetics Study (MTwiNS), a project within the Michigan State University Twin Registry (MSUTR; Burt & Klump, 2019). The 354 families participating in MTwiNS were originally identified through birth records as part of the Twin Study of Behavioral and Emotional Development in Children (TBED-C; for details see Burt & Klump, 2019) and recruited into one of two cohorts — a population-based cohort that represented families living within 120 miles of Michigan State University and an at-risk cohort recruited from the same area, but that was restricted to families living in U.S. Census tracts where at least 10.5% of families lived below the poverty line (the mean for the state of Michigan at the time; Burt & Klump, 2019). MTwiNS participants were re-recruited from the original participant pool based upon criteria for the second, "at-risk" cohort. Thus, MTwiNS includes families from the "at-risk" cohort as well as those from the population-based cohort that would have qualified for the "at-risk" cohort. This re-recruitment strategy yielded a sample representative of families living in neighborhoods with above average levels of poverty, a unique sampling frame within both the behavioral genetics and neuroimaging literatures (Burt et al., 2021).

The 708 twins (354 pairs, 167 monozygotic) included in the present study were 7 to 19 years old (Mean=14.6, SD=2.2; 54.5% male; only 3.3% of the present sample was 10 or

younger). The breakdown of twins' parent-reported ethnicity reflected the surrounding area (78% White/Caucasian, 13% African-American, 5% Other, 1% Latino/Latina, 1% Pacific Islander, 1% Native American, <1% Asian). Median reported family annual income for this sample was $70,000 to $79,999 and ranged from less than $4,999 to greater than $90,000. 9% of included families reported an annual income below the 2020 federal poverty line of $26,246 per year and 33% reported annual income below the living wage for a family of 4 in Michigan (http://livingwage.mit.edu/states/26). Zygosity was established using physical similarity questionnaires administered to the twins and/or their parents (Bouchard et al., 1990; Iacono et al., 1999; Peeters et al., 1998). Discrepancies were resolved through review of zygosity items or by DNA markers. Parents provided informed consent and children provided assent.

Procedure

Primary caregivers completed executive functioning self-report measures during their preliminary visit to Michigan State University as part of their participation in the TBED-C (twin ages ranged from 6-11 at the time; Mean= 8.05; SD=1.43). All other measures were collected in a day-long return visit to the University of Michigan (twin ages at follow-up ranged from 7-19 as described above). Each twin completed a variety of behavioral tasks, some of which occurred during an MRI scan, including a go/no-go task (during imaging) and a stop-signal task (in the lab). Twins also completed a battery of child-report questionnaires. Primary caregivers completed a demographic interview with an examiner and a battery of self- and parent-report questionnaires.

Measures

Child Executive Functioning

Go/No-go Task. The child friendly go/no-go task used in this study was adapted from Casey et al. (1997) and assesses inhibition via a "whack-a-mole" game (stimuli courtesy of Sarah Getz and the Sackler Institute for Developmental Psychobiology; task downloaded from http://fablab.yale.edu/page/assays-tools). In the present task, participants were instructed to press a button as quickly as possible in response to one stimulus ("go", a mole) and avoid responding to a less frequent non-target ("No-Go", a vegetable). The target stimuli (moles) were modified with various "disguises" to make the task more interesting and difficult given the relatively slow speed of stimuli used to accommodate fMRI data acquisition. The task consisted of four runs, each with approximately 55 trials, for a total of 255 trials of which 55 were no-go (21.6% no-go). Each no-go trial was preceded by 1-5 go trials. Each trial lasted 2300ms, including a maximum of 1800ms stimulus presentation, 400ms feedback, and 100 – 1000ms fixation to account for individual differences in reaction time. Participants practiced the task briefly in an MRI simulator before the MRI scan. Participants with below-chance "go accuracy" (correct responses on <55% of "go" trials) were not considered to be meaningfully participating in the task (N=2), leaving N=589 individuals with viable go/no-go data.

Stop-Signal Task. The child friendly stop-signal task used in this study was a 10 minute, 150 trial task adapted from Bissett & Logan (2012) as described previously (Begolli et al., 2018). Participants were presented with a fish for 850ms and told to push the "a" or "l" keys as quickly as possible based on the orientation of the fish, unless a visual stop-signal stimulus (Martin the Manta Ray) appeared on the screen, which occurred on 50 of the task trials (33%; "stop" trials). This stop-signal was presented following a stop signal delay (SSD) that was determined through a standard "staircase tracking" algorithm (Logan, 1994) designed to lead to a roughly 50% probability of inhibition on "stop" trials for each participant. This algorithm began with a 250ms

SSD and was thereafter increased or decreased by 50ms on each subsequent ”stop” trial based on whether the participant was able to successfully inhibit. The stop-signal task was added to the protocol partway through data collection and was therefore only available for a subset of participants (N=332 individuals).

Evidence Accumulation Model Analyses. Parameters of the linear ballistic accumulator (LBA) model were estimated in an individual-level Bayesian framework for the go/no-go task and “go” choice trials from the stop-signal task using the Dynamic Models of Choice functions (Heathcote, 2019), an adaptation of the differential evolution Markov chain Monte Carlo model (Turner et al., 2013), in R version 4.1.0 (R Core Team, 2021). Plots of model fit (Gelman et al., 1996) and parameter recovery studies (Heathcote, Brown, et al., 2015) indicated good model fit to the data in each task and acceptable parameter recovery given the task designs. Following previous work (Heathcote, Suraev, et al., 2015; Weigard et al., 2020), efficiency of evidence accumulation (hereafter, “efficiency”) was estimated as the difference in accumulation rates between the evidence accumulator for the correct choice (*vc*) and that for the incorrect choice (*ve*) scaled by the between-trial variability in accumulation rates pooled across both choices (*sv*): (*vc*-*ve*)/*sv*. It was estimated separately for “go” and “no-go” trial types within the go/no-go task due to previous evidence that it may systematically differ across the two conditions (Huang-Pollock et al., 2017; Ratcliff et al., 2018).

Self-Report of Executive Functioning. Twins reported on their EF abilities via the attention, activation control, and inhibitory control subscales of the Early Adolescent Temperament Questionnaire (EATQ; Capaldi & Rothbart, 1992). These scales consist of 6, 5, and 5 items, respectively, and can be combined to make up an “effortful control” superscale, though for the purposes of this study each scale was used as a separate indicator (in a latent

factor) of executive functioning (Snyder et al., 2015). The attention scale measures the ability to focus and shift attention, the activation control scale measures the ability to begin and complete tasks, and the inhibitory control scale measures the ability to suppress unwanted behaviors (Snyder et al., 2015). Within our sample, internal consistencies for the attention and activation subscales were borderline to acceptable (α = 0.674 and 0.748) while internal consistency of the inhibitory control subscale was poor (α = 0.355).

Executive Functioning Factor Score. Individual twins' executive functioning scores were calculated as latent factor scores combining the three efficiency parameters (go, no-go, stop-signal) with the three self-report executive functioning scores (attention, activation control, inhibitory control) using maximum likelihood estimation with bootstrapping in Mplus version 8.6 (Muthén & Muthén, 1998-2017) via R, tidyverse (Wickham et al., 2019), and the *MplusAutomation* package (Hallquist & Wiley, 2018). This modeling approach with full information estimation is robust to missing data, and executive functioning scores were therefore available for all participants (N=708 individuals). Correlations between all executive functioning measures are available in Table III.1.

Parent Executive Functioning

Multidimensional Personality Questionnaire. Parents reported on their own self-control abilities via the self-control scale of the Multidimensional Personality Questionnaire, a 24-item scale which measures planfulness versus impulsivity (MPQ; Tellegen & Waller, 2008). A higher score on this scale indicates better self-control. Within our sample, internal consistency for this scale was acceptable (α = 0.743 and 0.752 for mothers and fathers, respectively).

Adult Self Report. Parents reported on their own attention problems via the attention problems scale of the Adult Self Report (Achenbach & Rescorla, 2001). This scale measures

attention problems (e.g., “I have trouble concentrating or paying attention for long”) and was reverse scored such that a higher score on this scale indicated less attention problems. Within our sample, internal consistency for this scale was acceptable (α = 0.818 and 0.809 for mothers and fathers, respectively).

Parent Executive Functioning Composite. Control and attention scales were z-scored and then averaged for each parent to create an “average” reported executive functioning score. If parents had one measure but not the other (N=24 mothers and 15 fathers), the one remaining report was used. With this approach, almost all families (N=339 families) had parent executive functioning data. Correlations between all parent and child executive functioning measures are available in Table III.1.

Parenting

Parental Environment Questionnaire. The Parental Environment Questionnaire (PEQ; Elkins et al., 1997) was administered to assess nurturance and conflict in each parent-child dyad. The involvement or nurturance subscale (12 items) assesses communication, closeness, and support in the parent-child relationship (e.g., “I praise my child when he/she does something well”). The conflict subscale (12 items) assesses disagreement, tension, and anger in the parent-child relationship (e.g., “I often criticize my child”). Mothers and fathers reported on their relationships with each twin, and each twin reported on their relationship with their parent(s). Within our sample, internal consistencies for the PEQ subscales were acceptable (for conflict, α = 0.901, 0.883, and 0.873 for twin, mother, and father report, respectively; for involvement, α = 0.859, 0.794, and 0.858 for twin, mother, and father report, respectively).

Alabama Parenting Questionnaire. The Alabama Parenting Questionnaire (APQ; Shelton et al., 1996) was administered to assess parenting in each parent-child dyad. The parental

involvement subscale (10 items) assesses closeness and communication in the parent-child relationship (e.g., "You have a friendly talk with your child"). The positive parenting subscale (6 items) assesses positive reinforcement behaviors (e.g., "You praise your child if he/she behaves well"). The inconsistent discipline subscale (6 items) assesses unpredictability in parental discipline (e.g., "The punishment you give your child depends on your mood"). The corporal punishment subscale (3 items) assesses physical discipline (e.g., "You spank your child with your hand when he/she has done something wrong). Some participants received a shortened version of the corporal punishment subscale; these participants' scores were prorated accordingly. Mothers and fathers reported on their relationships with each twin, and each twin reported on their relationship with their mother. Within our sample, internal consistencies for the APQ subscales were acceptable (for inconsistent discipline, $\alpha = 0.749$, and 0.701 for mother and father report, respectively; for positive parenting, $\alpha = 0.785$ and 0.801 for mother, and father report, respectively). Because some families received only one corporal punishment item, internal consistency was not computed for that scale.

Parenting Composite. Consistent with prior work (e.g. Burt et al., 2003), we created a composite of all reporters for each subscale of the PEQ and APQ to assess the overall parenting environment for each child, averaging z-scored twin reports, mother reports, and father reports, and then averaging these three reporters together. Using this method, we created a "harsh parenting" composite including data from all available reporters on the PEQ conflict, APQ inconsistent discipline, and APQ corporal punishment subscales (Table III.2); we created a "nurturing parenting" composite from all available reporters on the PEQ involvement, APQ parental involvement, and APQ positive parenting subscales (Table III.3). When any of these reporters were missing (N=292 (harsh) and N=325 (nurturing) individuals were missing at least 1

informant-report, primarily father), we calculated the composite from the available reports. Using this method, all twins had composite parenting data (N=708 individuals).

Analytic Plan

Question 1: Is parenting one pathway through which executive functioning is transmitted intergenerationally?

First, we employed a regression approach to test whether harsh or nurturing parenting were associated with child executive functioning. We then tested whether parent executive functioning might have an indirect effect on child executive functioning via harsh or nurturing parenting using structural equation modeling in Mplus version 8.6 (Muthén & Muthén, 1998-2017). We used a maximum likelihood estimator with bootstrapping (bootstrap = 1000). We tested for specific indirect effects of parent executive functioning on child executive functioning via harsh or nurturing parenting, clustering by family to account for relatedness of twins. In all models, we included child age and sex as controls. We also included twin race/ethnicity (white vs. nonwhite), a socially constructed category, to control for differences in exposure to systemic racism and related systematic differences by race and ethnicity in exposure to stress, trauma, and opportunity.

Question 2: What is the etiology of executive functioning?

Twin analyses leverage the difference in the proportion of genes shared between monozygotic twins (who share 100% of their segregating genes) and dizygotic twins (who share roughly 50% of their segregating genes) to make inferences about additive genetic (A), shared environmental (i.e. environmental factors that make twins similar to each other; C) and non-shared environmental (i.e. factors that make twins different from each other, including measurement error; E) contributions to a given phenotype (Figure III.2b; see Plomin et al.,

2012). In the classical twin model, these three estimates are calculated based on just two pieces of information: the covariance between monozygotic twins and the covariance between dizygotic twins (along with multiple assumptions; Burt & Klump, 2012; Plomin et al., 2012). To address this second question, we employed the nuclear twin family model, an extension of the classical twin model. The nuclear twin family model incorporates two additional pieces of information, the covariance between parents and the covariance between parents and children, allowing researchers to account for assortative mating and differentiate shared environmental influences from passive genotype-environment correlation (Figure III.2a; Burt & Klump, 2012). More specifically, the inclusion of parent data on the phenotype of interest allows for the differentiation of shared environmental influences (C) into those shared between siblings (S; "sibling-level shared environmental influences") and those passed between parents and offspring (F; "family-level shared environmental influences). Within this model, passive genotype-environment correlation (w; Figure III.2a) can be represented by the covariance between additive genetic factors (A) and familial environmental variance (F). Given that a major focus of our question is on family-level factors and passive genotype-environment correlation, we first ran a full nuclear twin family model including S and F (i.e. ASFE; Figure III.2a). All twin models were run in Mplus version 8.6 (Muthén & Muthén, 1998-2017) via R version 4.1.0 (R Core Team, 2021) and the mplusAutomation package (Hallquist & Wiley, 2018).

Twin analyses included all twins with any relevant data (N=708 individuals, N=354 pairs, N=167 monozygotic pairs). Data were prepared for twin analysis using tidyverse (Wickham et al., 2019) within R version 4.1.0 (R Core Team, 2021). To eliminate mean age or sex differences, we regressed out age and sex effects from all phenotypes of interest (i.e.,

extracted the residuals from a regression with age and sex predicting the phenotype of interest); We used the standardized residual from this regression as our phenotype score (Mean=0, SD=1).

Question 3: Does parenting have a unique environmental effect on executive functioning? Is there evidence of evocative genotype-environment correlation?

To further examine the relation between harsh parenting and child executive functioning, we fit classical bivariate ACE models to decompose the covariance between harsh parenting and child executive functioning. Like the nuclear twin family model, the classical twin model estimates additive genetic (A) and non-shared environmental (E) contributions to a given phenotype; however, as described above, this model estimates broad shared environmental influences (C) rather than differentiating sibling-level (S) or family-level (F) influences. The bivariate classical ACE model incorporates two phenotypes of interest into the same model, parsing the phenotypic covariance between them into that which is due to genetic (A), shared environmental (C), and non-shared environmental (E) factors (Figure III.2c); these covariances can then be standardized on their respective variances to produce genetic and environmental correlations. These correlations can then be used to make inferences about the source of the overlap between parenting and child executive functioning. For example, because twin modeling of parenting indexes the extent to which twins' genetic tendencies influence the parenting they receive, genetic (A) overlap would indicate genetic confounds, and more specifically evocative genotype-environment correlation (Klahr & Burt, 2014). On the other hand, shared environmental overlap (C) would indicate either passive genotype-environment correlation or environmental mediation, while non-shared environmental overlap (E) would indicate environmental mediation of their relationship.

Results

Descriptive statistics are reported in Tables III.4 and III.5. Harsh and nurturing parenting were moderately negatively correlated (r=-.33) and both harsh and nurturing parenting correlated with child executive functioning (r=-.29 and .26, respectively; Table III.4). ICCs for executive functioning were .35 for MZ twins and .23 for DZ twins, while ICCs for harsh parenting were rMZ=.75 and rDZ=.64, and for nurturing parenting were rMZ=.81 and rDZ=.70 (see Table III.5 for cross-trait cross-twin correlations). The executive functioning factor score fit was below traditionally accepted metrics for acceptable fit (Table III.6). Loadings were substantially higher for self-report measures than for efficiency measures.

Harsh parenting is a phenotypic pathway linking parenting executive functioning to child executive functioning

In a path model that linked parent executive functioning to child executive functioning via both harsh and nurturing parenting (see Figure III.1), links between parent executive functioning and harsh parenting, and between harsh parenting and child executive functioning were both significant (Beta=-0.22, 95% CI [-0.32 -0.12]; Beta=-0.21, 95% CI [-0.30 -0.12]). Similarly, links between parent executive functioning and nurturing parenting, and between nurturing parenting and child executive functioning were both significant (Beta=0.10, 95% CI [0.02 0.18]; Beta=-0.16, 95% CI [0.08 0.24]). Moreover, there was an indirect effect of parent executive functioning on child executive functioning via parenting (total indirect Beta=0.062, 95% CI [0.03, 0.10]), and significant specific indirect effects of both harsh (Beta=0.05, 95% CI [0.02, 0.07]) and nurturing (Beta=0.02, 95% CI [0.003 0.04]) parenting. There was also a small direct effect, such that a portion of the relationship between parent and child executive function was not mediated by parenting. This model clustered by family to account for relatedness of

twins, and included controls for child age, sex, and race. Simple linear regressions with each set of variables yielded consistent results (Tables III.7- III.9).

Executive functioning shows substantial non-shared environmental effects with some evidence for sibling-level shared environmental effects

We first ran a full ASFE nuclear twin family model, which differentiated shared environmental influences (C) into those shared among the family unit (F) and those shared by siblings (S; Figure III.2a). The full ASFE model estimated large non-shared environmental influences on child executive functioning (E=0.68, 95% CI [0.54, 0.81]; Table III.10). Sibling-level shared environmental influences, by contrast, were small and were not significant (S=0.07, 95% CI [0, 0.23]; Table III.10), as were the family-level (F) influences (F=0.00, 95% CI [0, 0.05]; Table III.10). The model also estimated passive genotype-environment correlation, or the correlation between genetic (A) and family (F) influences, to be 0. This correlation of 0 indicates that any family-level influences are unlikely to be a function of the genetically influenced tendencies of the parents. A reduced ASE model, which set F to 0, fit the data better and also indicated low-to-moderate heritability (A=0.22, 95% CI [0.12, 0.33]; Table III.10) as well as a trend toward sibling-level shared environmental influences (S=0.09, 95% CI [0, 0.19]; Table III.10). The very low F estimate would suggest that any shared environmental influences on executive functioning are more likely to be those that make siblings more similar, rather than those that make children and their parents more similar. This absence of environmentally driven parent-child similarity precludes passive genotype-environment correlation effects on child executive functioning (as also seen in the above point estimate of zero for passive genotype-environment correlation).

Because child and parent executive functioning were measured in different ways (factor score including both behavioral performance and self-report for youth vs. self-report alone for parents), we tested two supplemental models to confirm that the very low F estimate was not caused by measurement issues. In the first supplemental model, we ran the full ASFE model using twin self-report executive functioning alone as measured by the effortful control superscale of the EATQ, which combines the attention, activation control, inhibitory control scales used in the factor score for the primary model. In the second supplemental model, we ran the full ASFE model using twin behavioral executive functioning alone, as measured by an average of the three computational metrics available (go, no-go, stop signal). Both supplemental models also estimated F at 0 (Table III.11).

In sum, this series of nuclear twin family models suggested substantial non-shared environmental influences on executive functioning (E=0.69), which include both measurement error and experiences that make family members more different from each other. There was also evidence for low-to-moderate genetic influences on executive functioning (A=0.22), and trend-level evidence for small shared environmental influences, particularly those that are shared among siblings, but not the whole family unit (e.g., exposure to similar peers, similar parenting style, similar school environment).

The effect of harsh parenting on child executive functioning is due, at least in part, to non-shared environmental influences

We employed bivariate ACE models (Figure III.2c) to examine the etiology of the association between both harsh and nurturing parenting and child executive functioning, finding some overlap between harsh parenting and child executive functioning on the non-shared environmental component (rE=-0.21, p=0.014; 29% of observed association explained by E

correlation; Table III.10). This finding indicates that at least part of the effect of harsh parenting on executive functioning was not due to genetic or family-level confounds, but to unique environmental influences. On the other hand, neither genetic nor non-shared environmental influences overlapped between nurturing parenting and executive functioning, though the proportion of overlap explained by the genetic component, while not significant, was estimated to be moderate (prA=0.31; Table III.10). Both harsh parenting and nurturing parenting had significant shared environmental overlap with executive functioning (rC; Table III.10). Though univariate models estimated very little C for child executive functioning, the bivariate model benefits from the MZ and DZ cross-trait cross-twin covariances for parenting and executive functioning (correlations in Table III.5), rather than univariate intraclass correlations. For reference, univariate classical ACE estimates for harsh parenting, nurturing parenting, and child executive functioning are also included in Table III.10. Interestingly, harsh parenting did not have a significant genetic component (A=0.17, 95% CI [0, 0.41]; C=0.57, 95% CI [0.37, 0.72]; E=0.26, 95% CI [0.19, 0.33]; Table III.10), while nurturing parenting did have evidence of moderate genetic influences (A=0.26, 95% CI [0.07, 0.47]; C=0.56, 95% CI [0.37, 0.72]; E=0.18, 95% CI [0.13, 0.24]; Table III.10). On the other hand, both harsh and nurturing parenting had evidence of large shared environmental influences (C), which is often thought to reflect either family environmental influences or passive genotype-environment correlation in a child twin design (Neiderhiser et al., 2004). In the case of a relationship variable, as presented here, C might also index effects of parent personality, family socioeconomic status, or culture, on the parenting parents provide, as these effects would not vary between twins (Klahr & Burt, 2014).

Discussion

In the present study, we leveraged a genetically informed design to better understand the role of parenting in the intergenerational transmission of executive functioning. Using a latent factor capturing both self-report measures of child executive functioning and computational measures of cognitive processes underlying executive functioning, we found that parent executive functioning was related to child executive functioning via both harsh and nurturing parenting. Results from nuclear twin family models then revealed large non-shared environmental influences on executive functioning, with some evidence for genetic influences and no evidence of passive genotype-environment correlation. Finally, bivariate twin models indicated overlap in shared environmental influences between both nurturing and harsh parenting and child executive functioning, likely indicating environmental mediation given the aforementioned absence of passive genotype-environment correlation. These bivariate twin models also revealed overlap between non-shared environmental influences on harsh (but not nurturing) parenting and child executive functioning, but little evidence for evocative genotype-environment correlation, suggesting that the effect of harsh parenting on executive functioning is not solely due to genetic confounds. In sum, these analyses suggest that executive functioning in parents is related to executive functioning in children via their parenting behavior, and that this association reflects at least in part an environmental effect of harsh parenting.

We found that harsher parenting was associated with worse child executive functioning at the phenotypic level. This finding is in line with substantial evidence within the developmental literature that parenting matters for children's executive functioning development, such that harsh, controlling behaviors are associated with worse child executive functioning (Cuevas, Deater-Deckard, Kim-Spoon, Watson, et al., 2014; Valcan et al., 2017). Additionally, we found

that more nurturance was associated with better child executive functioning, consistent with a growing body of literature that indexes nurturing, supportive parenting as a key promoter of executive functioning development (Cioffi et al., 2020; Deater-Deckard, 2014; Distefano et al., 2018; Helm et al., 2020; Hughes & Devine, 2019; Towe-Goodman et al., 2014). We also found evidence that these parenting behaviors play a role in the intergenerational transmission of more nurturing parenting, and there was an indirect effect of parent executive functioning on child executive functioning via both harsh and nurturing parenting behaviors. This finding is consistent with other studies suggesting that parenting may be one mechanism linking parent executive functioning and child executive functioning (e.g. Korucu et al., 2020). Much of the extant work regarding parenting and executive functioning focuses on early childhood (see Valcan et al., 2017 for a meta-analysis). Thus, our study extends these findings to adolescence (96.7% of the sample was age 11 – 17). Additionally, while previous work measured specific and highly relevant parenting behaviors like scaffolding or intrusiveness (Broomell et al., 2020; Distefano et al., 2018; Fay-Stammbach et al., 2014; Hammond et al., 2012; Hughes & Devine, 2019; Valcan et al., 2017), our parenting composites indexed a much broader picture of the parent-child relationship, incorporating multiple reporters and multiple questionnaire measures, thus extending previous research to broad indexes of positive and negative parenting. This robust association between broad parenting and child executive functioning could imply that clinical strategies to generally decrease harshness and increase nurturance may support child executive functioning even without teaching more targeted parenting behaviors like scaffolding.

Using twin modeling, we found evidence for large non-shared environmental influences on executive functioning. In contrast to other twin studies of executive functioning (Engelhardt et al., 2015; Friedman & Miyake, 2017; Friedman et al., 2008), we found the heritability of

executive functioning to be modest (A=0.29). A notable difference between the present study and previous work is the measure of executive functioning. While previous work used latent factors of performance across various executive functioning tasks to assess task-general executive functioning (Friedman & Miyake, 2017), the present study instead used computational modeling to index efficiency of evidence accumulation, a task-general cognitive mechanism thought to underlie performance on executive functioning tasks (Weigard & Sripada, 2021) and then combined these computational measures with self-report executive functioning data. The task-general efficiency measure is reliable (Lerche et al., 2020; Schubert et al., 2016; Weigard et al., 2021) and has recently shown consistent links with self-regulatory processes and promise as a transdiagnostic risk factor for psychopathology (Sripada & Weigard, 2021; Weigard & Sripada, 2021). Thus, our inclusion of this computational measure of executive functioning may have contributed to different heritability estimates. On the other hand, supplemental models did find that there was evidence of large non-shared environmental (E) influences even when measuring executive functioning with behavioral or self-report measures alone. Future work could make use of latent modeling of the computational measures across more than two tasks.

Additionally, the lower estimates of heritability in this study could be due to the sample itself. Because residence in low-income neighborhoods is a robust risk factor for externalizing behaviors, this sample was oversampled for families living in lower-income neighborhoods, a novel approach which captures greater levels of risk and subsequent externalizing than most other twin studies, which typically contain fewer families facing substantial adversity. Indeed, very few twin studies have been explicitly sampled for environmental risk (Burt et al., 2021). Because there is evidence that neighborhood disadvantage moderates the heritability of various phenotypes (e.g. Burt et al., 2016), it is possible that heritability is indeed lower within this

unique sampling frame, and higher in more advantaged neighborhoods, though genotype by environment interaction models would be necessary to test this hypothesis (and the current sample is under-powered to do so).

When employing the nuclear twin family model to decompose shared environmental effects into those shared by the whole family and those common to siblings, we found no evidence for family-level influences on child executive functioning. These influences could include anything that makes siblings and parents more similar to each other, such as shared culture or the general home environment. Using this model, we also found no evidence of passive genotype-environment correlation, or the correlation between the parents' genetic tendencies and the home environment they provide for their children. This finding is consistent with adoption work, which found an effect of parenting on executive functioning even when eliminating passive genotype-environment correlation (Bridgett et al., 2018). These findings add to a body of literature which implicates parenting practices as an important environmental driver of the development of child executive functioning (Cioffi et al., 2020; Cuevas, Deater-Deckard, Kim-Spoon, Watson, et al., 2014; Deater-Deckard, 2014; Distefano et al., 2018; Helm et al., 2020; Hughes & Devine, 2019; Towe-Goodman et al., 2014; Valcan et al., 2017), providing evidence that the association between parenting practices and child executive functioning are not likely to be due entirely to shared genetics between parents and their children.

To ensure the lack of family-level influences or passive genotype-environment correlation was not due to measurement differences between parents and children, we re-ran the nuclear twin family model separately for self-report of child executive functioning and for the behavioral measure of executive functioning. Even when using only self-report for both parents and children, we still found no evidence of family-level effects. Thus, it appears this finding is

not simply due to the inclusion of behavioral data for children and not for their parents. However, parents and children did not complete the exact same measures — parents self-reported their attention problems via the Adult Self Report and their inhibitory control via the Multidimensional Personality Questionnaire, while children self-reported attention, inhibitory control, and activation control via the Early Adolescent Temperament Questionnaire. Thus, measurement differences even within the self-report data could still play a role in the very small F (family-level influences). estimate, as the use of these different measures might artificially inflate differences between children and their parents. These measurement differences could also lead to our SEM results providing an underestimate of the association between parent and child executive functioning because these different measures may tap slightly different underlying constructs. Future work could re-test these questions using identical measures for parents and children.

Separate from measurement issues, it is important to note that executive functioning is still developing during middle childhood and adolescence (Kolb et al., 2012), and some differences between parents and children may also be due to the large difference in developmental stage between parents and their children. It would be interesting to examine whether the finding of very small family-level influences remains when twins reach adulthood.

We also employed bivariate twin modeling to examine the etiology of the association between parenting and child executive functioning, finding little evidence for evocative genotype-environment correlation (i.e., overlap of genetic components (A) of parenting and child executive functioning) and that harsh parenting had some non-shared environmental overlap with child executive functioning (rE=-.21). Harsh parenting has previously been shown to have a unique environmental impact on other child outcomes, such as callous-unemotional traits

(Tomlinson et al., 2021; Waller et al., 2018). Our finding in the present study further underscores the importance of reducing harsh parenting as an intervention target to improve child executive functioning and prevent related psychopathology.

We did find evidence of significant shared environmental (C) overlap between parenting and child executive functioning, which is difficult to interpret given very low C estimates for child executive functioning overall. Regardless, this finding reflects the fact that the association between executive functioning and parenting does not vary by zygosity or across co-twins, aligning with our finding of a main effect of parenting on child executive functioning. Notably, there were relatively strong correlations in parenting received between twins (for harsh parenting rMZ=.75 and rDZ=.64; for nurturing parenting rMZ=.81 and rDZ=.70). Reflecting these twin correlations, C estimates for both harsh and nurturing parenting were high (C=0.57 and 0.56, respectively in univariate ACE models; Table III.10). A child twin design indexes the extent to which influences on twins, not on parents, affect the parenting the twins receive (Klahr & Burt, 2014). Thus, in this child twin analysis of parenting, the large C estimates could reflect family-level environmental influences or passive genotype-environment correlation (Neiderhiser et al., 2004), or other influences that do not vary between twins, such as effects of parent personality, or even parent executive functioning, on the parenting they provide (Klahr & Burt, 2014)."

This study has several limitations. First, the current sample size is not sufficient for a GxE analysis. A GxE model could reveal that parenting affects the heritability of executive functioning, separate from any effects of genotype-environment correlation. For example, harsh parenting could moderate the heritability of executive functioning, "activating" genetic risk. Future work with larger sample sizes could address this question. Second, we designed our study to incorporate both self-report and computational measures of executive functioning in an effort

to use complimentary information about child executive functioning. Our factor score combining these metrics had relatively poor fit, and factor loadings for the computational measures were low. Based on these factor loadings, the extracted factor scores more closely reflected self-report. Thus, the factor contained less influence from our novel and promising computationally derived measures of executive functioning. These low loadings could reflect that the behavioral and self-report executive functioning measures were not very correlated, as expected given the growing body of literature questioning the cohesiveness of the executive functioning construct (Dang et al., 2020; Eisenberg et al., 2019; Karr et al., 2018; Weigard & Sripada, 2021). Third, our sample size was modest for these complex analyses. Studies have shown that shared environmental estimates (e.g., C) require relatively high sample sizes for adequate power; thus, our study may have underestimated these effects (Burt et al., 2020). Our sample may have also been underpowered to detect significant A overlap between nurturing parenting and executive functioning in in the bivariate model despite a relatively large proportion of variance explained (prA=0.31, Table III.10). Finally, internal consistency of the inhibitory control subscale of the Early Adolescent Temperament Questionnaire was poor ($\alpha = 0.355$), though our use of a factor score, which would only reflect shared variance with other constructs, may help to mitigate this issue.

In summary, we used a genetically informed design with multiple quantitative models to dig deeper into the role of parenting in the intergenerational transmission of executive functioning in adolescence. Like many others, we found that harsh parenting was associated with worse child executive functioning, while nurturing parenting was associated with better child executive functioning. We found little evidence of passive or evocative genotype-environment correlation, and instead found that the relationship between harsh parenting and child executive

functioning is due, at least in part, to non-shared environmental influences. These findings highlight that parenting may be one environmental mechanism through which executive functioning is passed across generations. More broadly, this work highlights that targeting harsh parenting through interventions is critical to improving adolescent executive functioning and preventing related psychopathology, and it underscores the importance of genetically-informed designs when studying parenting influences on executive functioning.

References

Achenbach, T. M., & Rescorla, L. A. (2001). *Manual for the Aseba School-Age Forms & Profiles*. University of Vermont, Research Center for Children, Youth, & Families.

Begolli, K. N., Richland, L. E., Jaeggi, S. M., Lyons, E. M., Klostermann, E. C., & Matlen, B. J. (2018). Executive Function in Learning Mathematics by Comparison: Incorporating Everyday Classrooms into the Science of Learning. *Thinking & Reasoning, 24*(2), 280-313. https://doi.org/10.1080/13546783.2018.1429306

Behrendt, S. (2014). *Lm.Beta: Add Standardized Regression Coefficients to Lm-Objects.* In (Version 1.5-1) https://CRAN.R-project.org/package=lm.beta

Bernier, A., Carlson, S. M., Deschenes, M., & Matte-Gagne, C. (2012). Social Factors in the Development of Early Executive Functioning: A Closer Look at the Caregiving Environment. *Developmental Science, 15*(1), 12-24. https://doi.org/10.1111/j.1467-7687.2011.01093.x

Bernier, A., Carlson, S. M., & Whipple, N. (2010). From External Regulation to Self-Regulation: Early Parenting Precursors of Young Children's Executive Functioning. *Child Development, 81*(1), 326-339. https://doi.org/10.1111/j.1467-8624.2009.01397.x

Bissett, P. G., & Logan, G. D. (2012). Post-Stop-Signal Slowing: Strategies Dominate Reflexes and Implicit Learning. *Journal of Experimental Psychology: Human Perception and Performance, 38*(3), 746-757. https://doi.org/10.1037/a0025429

Blair, C., Raver, C. C., Berry, D. J., & Family Life Project, I. (2014). Two Approaches to Estimating the Effect of Parenting on the Development of Executive Function in Early Childhood. *Developmental Psychology, 50*(2), 554-565. https://doi.org/10.1037/a0033647

Bloemen, A. J. P., Oldehinkel, A. J., Laceulle, O. M., Ormel, J., Rommelse, N. N. J., & Hartman, C. A. (2018). The Association between Executive Functioning and Psychopathology: General or Specific? *Psychological Medicine, 48*(11), 1787-1794. https://doi.org/10.1017/S0033291717003269

Bouchard, T. J., Jr., Lykken, D. T., McGue, M., Segal, N. L., & Tellegen, A. (1990). Sources of Human Psychological Differences: The Minnesota Study of Twins Reared Apart. *Science, 250*(4978), 223-228. https://doi.org/10.1126/science.2218526

Bridgett, D. J., Ganiban, J. M., Neiderhiser, J. M., Natsuaki, M. N., Shaw, D. S., Reiss, D., & Leve, L. D. (2018). Contributions of Mothers' and Fathers' Parenting to Children's Self-Regulation: Evidence from an Adoption Study. *Developmental Science, 21*(6), e12692. https://doi.org/10.1111/desc.12692

Bridgett, D. J., Kanya, M. J., Rutherford, H. J., & Mayes, L. C. (2017). Maternal Executive Functioning as a Mechanism in the Intergenerational Transmission of Parenting:

Preliminary Evidence. *Journal of Family Psychology, 31*(1), 19-29. https://doi.org/10.1037/fam0000264

Broomell, A. P. R., Smith, C. L., Calkins, S. D., & Bell, M. A. (2020). Context of Maternal Intrusiveness During Infancy and Associations with Preschool Executive Function. *Infant Child Development, 29*(1). https://doi.org/10.1002/icd.2162

Brown, S. D., & Heathcote, A. (2008). The Simplest Complete Model of Choice Response Time: Linear Ballistic Accumulation. *Cognitive Psychology, 57*(3), 153-178. https://doi.org/10.1016/j.cogpsych.2007.12.002

Burt, S. A., Clark, D. A., Pearson, A. L., Klump, K. L., & Neiderhiser, J. M. (2020). Do Neighborhood Social Processes Moderate the Etiology of Youth Conduct Problems? *Psychological Medicine, 50*(9), 1519-1529. https://doi.org/10.1017/S0033291719001521

Burt, S. A., & Klump, K. L. (2012). Etiological Distinctions between Aggressive and Non-Aggressive Antisocial Behavior: Results from a Nuclear Twin Family Model. *Journal of Abnormal Child Psychology, 40*(7), 1059-1071. https://doi.org/10.1007/s10802-012-9632-9

Burt, S. A., & Klump, K. L. (2019). The Michigan State University Twin Registry (Msutr): 15 Years of Twin and Family Research. *Twin Research and Human Genetics, 22*(6), 741-745. https://doi.org/10.1017/thg.2019.57

Burt, S. A., Klump, K. L., Gorman-Smith, D., & Neiderhiser, J. M. (2016). Neighborhood Disadvantage Alters the Origins of Children's Nonaggressive Conduct Problems. *Clinical Psychological Science, 4*(3), 511-526. https://doi.org/10.1177/2167702615618164

Burt, S. A., Klump, K. L., Vazquez, A. Y., Shewark, E. A., & Hyde, L. W. (2021). Identifying Patterns of Youth Resilience to Neighborhood Disadvantage. *Research in Human Development*, 1-16. https://doi.org/10.1080/15427609.2021.1935607

Burt, S. A., Krueger, R. F., McGue, M., & Iacono, W. (2003). Parent-Child Conflict and the Comorbidity among Childhood Externalizing Disorders. *Archives of General Psychiatry, 60*(5), 505-513. https://doi.org/10.1001/archpsyc.60.5.505

Capaldi, D. M., & Rothbart, M. K. (1992). Development and Validation of an Early Adolescent Temperament Measure. *The Journal of Early Adolescence, 12*(2), 153-173. https://doi.org/10.1177/0272431692012002002

Carroll, S. L., Clark, D. A., Hyde, L. W., Klump, K. L., & Burt, S. A. (2021). Continuity and Change in the Genetic and Environmental Etiology of Youth Antisocial Behavior. *Behavior Genetics, 51*(5), 580-591. https://doi.org/10.1007/s10519-021-10066-8

Casey, B. J., Trainor, R. J., Orendi, J. L., Schubert, A. B., Nystrom, L. E., Giedd, J. N., Castellanos, F. X., Haxby, J. V., Noll, D. C., Cohen, J. D., Forman, S. D., Dahl, R. E., & Rapoport, J. L. (1997). A Developmental Functional Mri Study of Prefrontal Activation

During Performance of a Go-No-Go Task. *Journal of Cognitive Neuroscience, 9*(6), 835-847. https://doi.org/10.1162/jocn.1997.9.6.835

Castellanos, F. X., Sonuga-Barke, E. J., Milham, M. P., & Tannock, R. (2006). Characterizing Cognition in Adhd: Beyond Executive Dysfunction. *Trends in Cognitive Sciences, 10*(3), 117-123. https://doi.org/10.1016/j.tics.2006.01.011

Cherkasova, M., Sulla, E. M., Dalena, K. L., Ponde, M. P., & Hechtman, L. (2013). Developmental Course of Attention Deficit Hyperactivity Disorder and Its Predictors. *Journal of the Canadian Academy of Child and Adolescent Psychiatry, 22*(1), 47-54. https://www.ncbi.nlm.nih.gov/pubmed/23390433

Cioffi, C. C., Leve, L. D., Natsuaki, M. N., Shaw, D. S., Reiss, D., & Neiderhiser, J. M. (2020). Does Maternal Warmth Moderate Longitudinal Associations between Infant Attention Control and Children's Inhibitory Control? *Infant Child Development, 29*(1). https://doi.org/10.1002/icd.2147

Cuevas, K., Deater-Deckard, K., Kim-Spoon, J., Wang, Z., Morasch, K. C., & Bell, M. A. (2014). A Longitudinal Intergenerational Analysis of Executive Functions During Early Childhood. *British Journal of Developmental Psychology, 32*(1), 50-64. https://doi.org/10.1111/bjdp.12021

Cuevas, K., Deater-Deckard, K., Kim-Spoon, J., Watson, A. J., Morasch, K. C., & Bell, M. A. (2014). What's Mom Got to Do with It? Contributions of Maternal Executive Function and Caregiving to the Development of Executive Function across Early Childhood. *Developmental Science, 17*(2), 224-238. https://doi.org/10.1111/desc.12073

Dang, J., King, K. M., & Inzlicht, M. (2020). Why Are Self-Report and Behavioral Measures Weakly Correlated? *Trends in Cognitive Sciences, 24*(4), 267-269. https://doi.org/10.1016/j.tics.2020.01.007

Deater-Deckard, K. (2014). Family Matters: Intergenerational and Interpersonal Processes of Executive Function and Attentive Behavior. *Current Directions in Psychological Science, 23*(3), 230-236. https://doi.org/10.1177/0963721414531597

Deater-Deckard, K., Sewell, M. D., Petrill, S. A., & Thompson, L. A. (2010). Maternal Working Memory and Reactive Negativity in Parenting. *Psychological Science, 21*(1), 75-79. https://doi.org/10.1177/0956797609354073

Diamond, A. (2013). Executive Functions. *Annual Review of Psychology, 64*, 135-168. https://doi.org/10.1146/annurev-psych-113011-143750

Distefano, R., Galinsky, E., McClelland, M. M., Zelazo, P. D., & Carlson, S. M. (2018). Autonomy-Supportive Parenting and Associations with Child and Parent Executive Function. *Journal of Applied Developmental Psychology, 58*, 77-85. https://doi.org/10.1016/j.appdev.2018.04.007

Eisenberg, I. W., Bissett, P. G., Zeynep Enkavi, A., Li, J., MacKinnon, D. P., Marsch, L. A., & Poldrack, R. A. (2019). Uncovering the Structure of Self-Regulation through Data-Driven Ontology Discovery. *Nature Communications, 10*(1), 2319. https://doi.org/10.1038/s41467-019-10301-1

Elkins, I. J., McGue, M., & Iacono, W. G. (1997). Genetic and Environmental Influences on Parent-Son Relationships: Evidence for Increasing Genetic Influence During Adolescence. *Developmental Psychology, 33*(2), 351-363. https://doi.org/10.1037//0012-1649.33.2.351

Engelhardt, L. E., Briley, D. A., Mann, F. D., Harden, K. P., & Tucker-Drob, E. M. (2015). Genes Unite Executive Functions in Childhood. *Psychological Science, 26*(8), 1151-1163. https://doi.org/10.1177/0956797615577209

Enkavi, A. Z., Eisenberg, I. W., Bissett, P. G., Mazza, G. L., MacKinnon, D. P., Marsch, L. A., & Poldrack, R. A. (2019). Large-Scale Analysis of Test-Retest Reliabilities of Self-Regulation Measures. *Proceedings of the National Academy of Sciences of the United States of America, 116*(12), 5472-5477. https://doi.org/10.1073/pnas.1818430116

Evans, N. J., & Wagenmakers, E.-J. (2020). Evidence Accumulation Models: Current Limitations and Future Directions. *The Quantitative Methods for Psychology, 16*(2), 73-90. https://doi.org/10.20982/tqmp.16.2.p073

Fay-Stammbach, T., Hawes, D. J., & Meredith, P. (2014). Parenting Influences on Executive Function in Early Childhood: A Review. *Child Developmentelopment Perspectives, 8*(4), 258-264. https://doi.org/10.1111/cdep.12095

Friedman, N. P., & Miyake, A. (2017). Unity and Diversity of Executive Functions: Individual Differences as a Window on Cognitive Structure. *Cortex, 86*, 186-204. https://doi.org/10.1016/j.cortex.2016.04.023

Friedman, N. P., Miyake, A., Young, S. E., DeFries, J. C., Corley, R. P., & Hewitt, J. K. (2008). Individual Differences in Executive Functions Are Almost Entirely Genetic in Origin. *Journal of Experimental Psychology: General, 137*(2), 201-225. https://doi.org/10.1037/0096-3445.137.2.201

Gagne, J. R., & Saudino, K. J. (2010). Wait for It! A Twin Study of Inhibitory Control in Early Childhood. *Behavior Genetics, 40*(3), 327-337. https://doi.org/10.1007/s10519-009-9316-6

Gee, D. G. (2016). Sensitive Periods of Emotion Regulation: Influences of Parental Care on Frontoamygdala Circuitry and Plasticity. *New Directions for Child and Adolescent Development, 2016*(153), 87-110. https://doi.org/10.1002/cad.20166

Gelman, A., Meng, X.-L., & Stern, H. (1996). Posterior Predictive Assessment of Model Fitness Via Realized Discrepancies. *Statistica Sinica*, 733-760.

Gotlib, I. H., & Joormann, J. (2010). Cognition and Depression: Current Status and Future Directions. *Annual Review of Clinical Psychology, 6*, 285-312. https://doi.org/10.1146/annurev.clinpsy.121208.131305

Hallquist, M. N., & Wiley, J. F. (2018). Mplusautomation: An R Package for Facilitating Large-Scale Latent Variable Analyses in Mplus. *Structural Equation Modeling, 25*(4), 621-638. https://doi.org/10.1080/10705511.2017.1402334

Hammond, S. I., Muller, U., Carpendale, J. I., Bibok, M. B., & Liebermann-Finestone, D. P. (2012). The Effects of Parental Scaffolding on Preschoolers' Executive Function. *Developmental Psychology, 48*(1), 271-281. https://doi.org/10.1037/a0025519

Heathcote, A. (2019). *Dynamic Models of Choice.* In https://osf.io/pbwx8/

Heathcote, A., Brown, S. D., & Wagenmakers, E.-J. (2015). An Introduction to Good Practices in Cognitive Modeling. In *An Introduction to Model-Based Cognitive Neuroscience* (pp. 25-48). https://doi.org/10.1007/978-1-4939-2236-9_2

Heathcote, A., Suraev, A., Curley, S., Gong, Q., Love, J., & Michie, P. T. (2015). Decision Processes and the Slowing of Simple Choices in Schizophrenia. *Journal of Abnormal Psychology, 124*(4), 961-974. https://doi.org/10.1037/abn0000117

Hedge, C., Powell, G., & Sumner, P. (2018). The Reliability Paradox: Why Robust Cognitive Tasks Do Not Produce Reliable Individual Differences. *Behavior Research Methods, 50*(3), 1166-1186. https://doi.org/10.3758/s13428-017-0935-1

Helm, A. F., McCormick, S. A., Deater-Deckard, K., Smith, C. L., Calkins, S. D., & Bell, M. A. (2020). Parenting and Children's Executive Function Stability across the Transition to School. *Infant Child Development, 29*(1). https://doi.org/10.1002/icd.2171

Huang-Pollock, C., Ratcliff, R., McKoon, G., Shapiro, Z., Weigard, A., & Galloway-Long, H. (2017). Using the Diffusion Model to Explain Cognitive Deficits in Attention Deficit Hyperactivity Disorder. *Journal of Abnormal Child Psychology, 45*(1), 57-68. https://doi.org/10.1007/s10802-016-0151-y

Hughes, C., & Devine, R. T. (2019). For Better or for Worse? Positive and Negative Parental Influences on Young Children's Executive Function. *Child Development, 90*(2), 593-609. https://doi.org/10.1111/cdev.12915

Iacono, W. G., Carlson, S. R., Taylor, J., Elkins, I. J., & McGue, M. (1999). Behavioral Disinhibition and the Development of Substance-Use Disorders: Findings from the Minnesota Twin Family Study. *Development and Psychopathology, 11*(4), 869-900. https://doi.org/10.1017/s0954579499002369

Jester, J. M., Nigg, J. T., Puttler, L. I., Long, J. C., Fitzgerald, H. E., & Zucker, R. A. (2009). Intergenerational Transmission of Neuropsychological Executive Functioning. *Brain and Cognition, 70*(1), 145-153. https://doi.org/10.1016/j.bandc.2009.01.005

Kao, K., Nayak, S., Doan, S. N., & Tarullo, A. R. (2018). Relations between Parent Ef and Child Ef: The Role of Socioeconomic Status and Parenting on Executive Functioning in Early Childhood. *Translational Issues in Psychological Science, 4*(2), 122-137. https://doi.org/10.1037/tps0000154

Karr, J. E., Areshenkoff, C. N., Rast, P., Hofer, S. M., Iverson, G. L., & Garcia-Barrera, M. A. (2018). The Unity and Diversity of Executive Functions: A Systematic Review and Re-Analysis of Latent Variable Studies. *Psychological Bulletin, 144*(11), 1147-1185. https://doi.org/10.1037/bul0000160

Keller, M. C., Medland, S. E., & Duncan, L. E. (2010). Are Extended Twin Family Designs Worth the Trouble? A Comparison of the Bias, Precision, and Accuracy of Parameters Estimated in Four Twin Family Models. *Behavior Genetics, 40*(3), 377-393. https://doi.org/10.1007/s10519-009-9320-x

Klahr, A. M., & Burt, S. A. (2014). Elucidating the Etiology of Individual Differences in Parenting: A Meta-Analysis of Behavioral Genetic Research. *Psychological Bulletin, 140*(2), 544-586. https://doi.org/10.1037/a0034205

Knopik, V. S., Neiderhiser, J. M., DeFries, J. C., & Plomin, R. (2017). *Behavioral Genetics* (7th ed.). Worth Publishers.

Kolb, B., Mychasiuk, R., Muhammad, A., Li, Y., Frost, D. O., & Gibb, R. (2012). Experience and the Developing Prefrontal Cortex. *Proceedings of the National Academy of Sciences of the United States of America, 109 Suppl 2*, 17186-17193. https://doi.org/10.1073/pnas.1121251109

Korucu, I., Litkowski, E., Purpura, D. J., & Schmitt, S. A. (2020). Parental Executive Function as a Predictor of Parenting Practices and Children's Executive Function. *Infant and Child Development, 29*(1). https://doi.org/10.1002/icd.2152

Lerche, V., von Krause, M., Voss, A., Frischkorn, G. T., Schubert, A. L., & Hagemann, D. (2020). Diffusion Modeling and Intelligence: Drift Rates Show Both Domain-General and Domain-Specific Relations with Intelligence. *Journal of Experimental Psychology: General, 149*(12), 2207-2249. https://doi.org/10.1037/xge0000774

Lerche, V., & Voss, A. (2017). Retest Reliability of the Parameters of the Ratcliff Diffusion Model. *Psychological Research, 81*(3), 629-652. https://doi.org/10.1007/s00426-016-0770-5

Li, J. B., Willems, Y. E., Stok, F. M., Dekovic, M., Bartels, M., & Finkenauer, C. (2019). Parenting and Self-Control across Early to Late Adolescence: A Three-Level Meta-Analysis. *Perspectives on Psychological Science, 14*(6), 967-1005. https://doi.org/10.1177/1745691619863046

Li, J. J., & Roberts, D. K. (2017). Genetic Influences on Executive Functions across the Life Span. In S. A. Wiebe & J. Karbach (Eds.), *Executive Function*. Routledge. https://doi.org/10.4324_9781315160719-8

Manuck, S. B., & McCaffery, J. M. (2014). Gene-Environment Interaction. *Annual Review of Psychology, 65*, 41-70. https://doi.org/10.1146/annurev-psych-010213-115100

Moffitt, T. E. (2018). Male Antisocial Behaviour in Adolescence and Beyond. *Nature Human Behaviour, 2*(3), 177-186. https://doi.org/10.1038/s41562-018-0309-4

Muthén, L. K., & Muthén, B. O. (1998-2017). *Mplus User's Guide* (Eighth Edition ed.). Muthén & Muthén.

Neiderhiser, J. M., Reiss, D., Pedersen, N. L., Lichtenstein, P., Spotts, E. L., Hansson, K., Cederblad, M., & Ellhammer, O. (2004). Genetic and Environmental Influences on Mothering of Adolescents: A Comparison of Two Samples. *Developmental Psychology, 40*(3), 335-351. https://doi.org/10.1037/0012-1649.40.3.335

Peeters, H., Van Gestel, S., Vlietinck, R., Derom, C., & Derom, R. (1998). Validation of a Telephone Zygosity Questionnaire in Twins of Known Zygosity. *Behavior Genetics, 28*(3), 159-163. https://doi.org/10.1023/a:1021416112215

Plomin, R., DeFries, J. C., Knopik, V. S., & Neiderhiser, J. M. (2012). *Behavioral Genetics*. Worth Publishers.

R Core Team. (2021). *R: A Language and Environment for Statistical Computing.* In R Foundation for Statistical Computing. https://www.R-project.org/

Ratcliff, R., Huang-Pollock, C., & McKoon, G. (2018). Modeling Individual Differences in the Go/No-Go Task with a Diffusion Model. *Decision (Wash D C), 5*(1), 42-62. https://doi.org/10.1037/dec0000065

Ratcliff, R., Smith, P. L., Brown, S. D., & McKoon, G. (2016). Diffusion Decision Model: Current Issues and History. *Trends in Cognitive Sciences, 20*(4), 260-281. https://doi.org/10.1016/j.tics.2016.01.007

Rouder, J. N., & Haaf, J. M. (2019). A Psychometrics of Individual Differences in Experimental Tasks. *Psychonomic Bulletin & Review, 26*(2), 452-467. https://doi.org/10.3758/s13423-018-1558-y

Schmiedek, F., Oberauer, K., Wilhelm, O., Suss, H. M., & Wittmann, W. W. (2007). Individual Differences in Components of Reaction Time Distributions and Their Relations to Working Memory and Intelligence. *Journal of Experimental Psychology: General, 136*(3), 414-429. https://doi.org/10.1037/0096-3445.136.3.414

Schubert, A.-L., Frischkorn, G., Hagemann, D., & Voss, A. (2016). Trait Characteristics of Diffusion Model Parameters. *Journal of Intelligence, 4*(3). https://doi.org/10.3390/jintelligence4030007

Shaw, D. S., & Bell, R. Q. (1993). Developmental Theories of Parental Contributors to Antisocial Behavior. *Journal of Abnormal Child Psychology, 21*(5), 493-518. https://doi.org/10.1007/BF00916316

Shelton, K. K., Frick, P. J., & Wootton, J. (1996). Assessment of Parenting Practices in Families of Elementary School-Age Children. *Journal of Clinical Child Psychology, 25*(3), 317-329. https://doi.org/10.1207/s15374424jccp2503_8

Smith, J. L., Mattick, R. P., Jamadar, S. D., & Iredale, J. M. (2014). Deficits in Behavioural Inhibition in Substance Abuse and Addiction: A Meta-Analysis. *Drug and Alcohol Dependence, 145*, 1-33. https://doi.org/10.1016/j.drugalcdep.2014.08.009

Snyder, H. R., Gulley, L. D., Bijttebier, P., Hartman, C. A., Oldehinkel, A. J., Mezulis, A., Young, J. F., & Hankin, B. L. (2015). Adolescent Emotionality and Effortful Control: Core Latent Constructs and Links to Psychopathology and Functioning. *Journal of Personality and Social Psychology, 109*(6), 1132-1149. https://doi.org/10.1037/pspp0000047

Sripada, C., & Weigard, A. (2021). Impaired Evidence Accumulation as a Transdiagnostic Vulnerability Factor in Psychopathology. *Frontiers in Psychiatry, 12*, 627179. https://doi.org/10.3389/fpsyt.2021.627179

Stanley, D. J., & Spence, J. R. (2018). Reproducible Tables in Psychology Using the Apatables Package. *Advances in Methods and Practices in Psychological Science, 1*(3), 415-431. https://doi.org/10.1177/2515245918773743

Tellegen, A., & Waller, N. G. (2008). Exploring Personality through Test Construction: Development of the Multidimensional Personality Questionnaire. *The SAGE handbook of personality theory and assessment, 2*, 261-292.

Tomlinson, R. C., Hyde, L. W., Dotterer, H. L., Klump, K. L., & Burt, S. A. (2021). Parenting Moderates the Etiology of Callous-Unemotional Traits in Middle Childhood. *Journal of Child Psychology and Psychiatry*. https://doi.org/10.1111/jcpp.13542

Towe-Goodman, N. R., Willoughby, M., Blair, C., Gustafsson, H. C., Mills-Koonce, W. R., Cox, M. J., & Family Life Project Key, I. (2014). Fathers' Sensitive Parenting and the Development of Early Executive Functioning. *Journal of Family Psychology, 28*(6), 867-876. https://doi.org/10.1037/a0038128

Turner, B. M., Sederberg, P. B., Brown, S. D., & Steyvers, M. (2013). A Method for Efficiently Sampling from Distributions with Correlated Dimensions. *Psychological Methods, 18*(3), 368-384. https://doi.org/10.1037/a0032222

Valcan, D. S., Davis, H., & Pino-Pasternak, D. (2017). Parental Behaviours Predicting Early Childhood Executive Functions: A Meta-Analysis. *Educational Psychology Review, 30*(3), 607-649. https://doi.org/10.1007/s10648-017-9411-9

Vasilopoulos, T., Franz, C. E., Panizzon, M. S., Xian, H., Grant, M. D., Lyons, M. J., Toomey, R., Jacobson, K. C., & Kremen, W. S. (2012). Genetic Architecture of the Delis-Kaplan Executive Function System Trail Making Test: Evidence for Distinct Genetic Influences on Executive Function. *Neuropsychology, 26*(2), 238-250. https://doi.org/10.1037/a0026768

Verdejo-Garcia, A., Lawrence, A. J., & Clark, L. (2008). Impulsivity as a Vulnerability Marker for Substance-Use Disorders: Review of Findings from High-Risk Research, Problem Gamblers and Genetic Association Studies. *Neuroscience & Biobehavioral Reviews, 32*(4), 777-810. https://doi.org/10.1016/j.neubiorev.2007.11.003

Waller, R., Hyde, L. W., Klump, K. L., & Burt, S. A. (2018). Parenting Is an Environmental Predictor of Callous-Unemotional Traits and Aggression: A Monozygotic Twin Differences Study. *Journal of the American Academy of Child and Adolescent Psychiatry, 57*(12), 955-963. https://doi.org/10.1016/j.jaac.2018.07.882

Weigard, A., Clark, D. A., & Sripada, C. (2021). Cognitive Efficiency Beats Top-Down Control as a Reliable Individual Difference Dimension Relevant to Self-Control. *Cognition, 215*, 104818. https://doi.org/10.1016/j.cognition.2021.104818

Weigard, A., & Sripada, C. (2021). Task-General Efficiency of Evidence Accumulation as a Computationally Defined Neurocognitive Trait: Implications for Clinical Neuroscience. *Biological Psychiatry Global Open Science, 1*(1), 5-15. https://doi.org/10.1016/j.bpsgos.2021.02.001

Weigard, A. S., Sathian, K., & Hampstead, B. M. (2020). Model-Based Assessment and Neural Correlates of Spatial Memory Deficits in Mild Cognitive Impairment. *Neuropsychologia, 136*, 107251. https://doi.org/10.1016/j.neuropsychologia.2019.107251

Wickham, H., Averick, M., Bryan, J., Chang, W., McGowan, L., François, R., Grolemund, G., Hayes, A., Henry, L., Hester, J., Kuhn, M., Pedersen, T., Miller, E., Bache, S., Müller, K., Ooms, J., Robinson, D., Seidel, D., Spinu, V., Takahashi, K., Vaughan, D., Wilke, C., Woo, K., & Yutani, H. (2019). Welcome to the Tidyverse. *Journal of Open Source Software, 4*(43). https://doi.org/10.21105/joss.01686

Wright, L., Lipszyc, J., Dupuis, A., Thayapararajah, S. W., & Schachar, R. (2014). Response Inhibition and Psychopathology: A Meta-Analysis of Go/No-Go Task Performance. *Journal of Abnormal Psychology, 123*(2), 429-439. https://doi.org/10.1037/a0036295

Zelazo, P. D., & Carlson, S. M. (2012). Hot and Cool Executive Function in Childhood and Adolescence: Development and Plasticity. *Child Developmentelopment Perspectives*, n/a-n/a. https://doi.org/10.1111/j.1750-8606.2012.00246.x

Zeytinoglu, S., Calkins, S. D., Swingler, M. M., & Leerkes, E. M. (2017). Pathways from Maternal Effortful Control to Child Self-Regulation: The Role of Maternal Emotional Support. *Journal of Family Psychology, 31*(2), 170-180. https://doi.org/10.1037/fam0000271

Table III.1

Means, standard deviations, and correlations with confidence intervals for all variables included in parent and child EF composites

Variable	*M*	*SD*	1	2	3	4	5	6	7	8	9
1. Mom MPS Ctrl	9.70	2.35									
2. Dad MPS Ctrl	9.35	2.28	-.02 [-.10, .07]								
3. Mom ASR Attn	5.24	4.30	-.36** [-.43, -.29]	-.14** [-.23, -.06]							
4. Dad ASR Attn	4.88	3.96	-.11** [-.20, -.03]	-.29** [-.36, -.21]	.24** [.16, .32]						
5. Twin EATQ Attn	3.46	0.65	.00 [-.07, .08]	.06 [-.02, .15]	-.14** [-.22, -.07]	-.18** [-.26, -.09]					
6. Twin EATQ Act Ctrl	3.39	0.81	.05 [-.03, .12]	.14** [.05, .22]	-.14** [-.21, -.06]	-.15** [-.24, -.07]	.56** [.50, .61]				
7. Twin EATQ Inh Ctrl	3.84	0.58	.06 [-.02, .13]	.04 [-.04, .13]	-.09* [-.16, -.01]	-.04 [-.12, .04]	.42** [.36, .48]	.31** [.24, .38]			
8. EEA (stop signal)	3.36	0.78	.11* [.00, .22]	.11 [-.01, .23]	-.08 [-.18, .03]	-.03 [-.15, .09]	.13* [.03, .24]	.06 [-.05, .17]	.12* [.01, .23]		
9. EEA (go)	2.63	0.53	-.00 [-.08, .08]	.08 [-.01, .17]	.02 [-.06, .10]	-.04 [-.13, .05]	.09* [.01, .17]	.01 [-.07, .09]	.09* [.01, .17]	.13* [.01, .24]	

10. EEA (no-go)	3.49	0.71	-.01 [-.09, .07]	.09* [.00, .18]	.01 [-.07, .09]	-.07 [-.16, .03]	.12** [.04, .20]	.05 [-.03, .13]	.11** [.03, .19]	.07 [-.04, .19]	.37** [.30, .44]

Note. M and *SD* are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation. Table generated with apaTables package in R (Stanley & Spence, 2018). * indicates $p < .05$. ** indicates $p < .01$.

Table III.2

Means, standard deviations, and correlations with confidence intervals for subscales included in harsh parenting composite

Variable	*M*	*SD*	1	2	3	4	5	6	7	8
1. PC APQ Inc Disc	12.68	3.72								
2. PC APQ Corporal	2.42	0.87	.45** [.39, .51]							
3. AC APQ Inc Disc	12.52	3.58	.22** [.13, .30]	.15** [.06, .23]						
4. AC APQ Corporal	2.32	0.90	.16** [.07, .24]	.24** [.16, .32]	.42** [.35, .49]					
5. Twin APQ Inc Disc	13.22	3.64	.29** [.22, .36]	.13** [.06, .21]	.26** [.18, .34]	.15** [.06, .23]				
6. Twin APQ Corporal	7.60	3.18	.19** [.11, .26]	.20** [.13, .27]	.14** [.06, .23]	.23** [.14, .31]	.31** [.24, .37]			
7. Twin PEQ Conflict	20.97	6.70	.30** [.23, .37]	.23** [.15, .30]	.08 [-.01, .17]	.15** [.06, .24]	.38** [.31, .45]	.46** [.40, .52]		
8. PC PEQ Conflict	20.31	5.98	.48** [.41, .53]	.49** [.43, .55]	.15** [.06, .24]	.23** [.14, .31]	.20** [.13, .28]	.22** [.15, .30]	.46** [.40, .52]	
9. AC PEQ Conflict	20.42	5.91	.29** [.22, .36]	.23** [.16, .31]	.48** [.41, .55]	.42** [.34, .49]	.19** [.11, .27]	.21** [.14, .29]	.28** [.21, .35]	.48** [.42, .54]

Note. M and *SD* are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation. Table generated with apaTables package in R (Stanley & Spence, 2018). * indicates $p < .05$. ** indicates $p < .01$.

Table III.3

Means, standard deviations, and correlations with confidence intervals of all subscales included in nurturing parenting composite

Variable	*M*	*SD*	1	2	3	4	5	6	7	8	9
1. PC APQ Involve	39.68	5.04									
2. PC APQ Positive	24.91	3.38	.60** [.55, .65]								
3. AC APQ Involve	36.31	5.94	.22** [.14, .30]	.04 [-.05, .13]							
4. AC APQ Positive	23.70	3.68	.24** [.15, .32]	.22** [.14, .30]	.63** [.57, .68]						
5. Twin APQ Involve (on mom)	34.73	6.56	.39** [.33, .45]	.19** [.12, .26]	.17** [.08, .26]	.10* [.02, .19]					
6. Twin APQ Involve (on dad)	30.06	8.42	.29** [.22, .36]	.09* [.02, .17]	.24** [.15, .32]	.10* [.01, .19]	.53** [.47, .58]				
7. Twin APQ Positive	21.27	4.33	.25** [.18, .32]	.30** [.23, .37]	.12** [.03, .21]	.19** [.11, .28]	.65** [.60, .69]	.41** [.34, .47]			
8. PC PEQ Involve	43.36	4.00	.59** [.54, .64]	.45** [.38, .51]	.07 [-.02, .17]	.18** [.09, .27]	.30** [.23, .37]	.22** [.15, .30]	.25** [.17, .32]		
9. AC PEQ Involve	41.03	5.33	.21** [.12, .29]	.07 [-.02, .17]	.68** [.63, .73]	.57** [.51, .63]	.09 [-.00, .18]	.21** [.12, .30]	.06 [-.03, .15]	.16** [.07, .25]	

10. Twin PEQ Involve	40.57	5.98	.23** [.15, .30]	.13** [.05, .20]	.19** [.10, .28]	.14** [.05, .23]	.59** [.53, .64]	.35** [.27, .42]	.53** [.46, .58]	.33** [.26, .40]	.17** [.08, .26]

Note. *M* and *SD* are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation. Table generated with apaTables package in R (Stanley & Spence, 2018). * indicates $p < .05$. ** indicates $p < .01$.

Table III.4

Means, standard deviations, and correlations with confidence intervals

Variable	*M*	*SD*	1	2	3
1. Child Executive Functioning	0.00	0.14			
2. Parent Executive Functioning	0.00	0.68	.15** [.08, .23]		
3. Harsh Parenting (Composite)	0.01	0.62	-.29** [-.35, -.22]	-.21** [-.28, -.14]	
4. Nurturing Parenting (Composite)	-0.02	0.63	.26** [.19, .33]	.12** [.04, .19]	-.33** [-.39, -.26]

Note. M and *SD* are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation. Table generated with apaTables package in R (Stanley & Spence, 2018). * indicates $p < .05$. ** indicates $p < .01$.

Table III.5

Cross-trait, cross-twin correlations with confidence intervals

Variable	1	2	3	4	5	6
1. Child Executive Functioning (T1)		**.23**** [.13, .31]	-.32** [-.40, -.23]	-.23** [-.31, -.13]	.25** [.16, .34]	.21** [.11, .29]
2. Child Executive Functioning (T2)	**.35**** [.24, .45]		-.23** [-.31, -.13]	-.32** [-.40, -.23]	.21** [.11, .29]	.25** [.16, .34]
3. Harsh Parenting (T1)	-.23** [-.34, -.12]	-.16** [-.27, -.04]		**.64**** [.58, .69]	-.32** [-.40, -.23]	-.20** [-.29, -.10]
4. Harsh Parenting (T2)	-.16** [-.27, -.04]	-.23** [-.34, -.12]	**.75**** [.70, .80]		-.20** [-.29, -.10]	-.32** [-.40, -.23]
5. Nurturing Parenting (T1)	.23** [.11, .34]	.21** [.10, .32]	-.36** [-.45, -.25]	-.31** [-.41, -.20]		**.70**** [.65, .74]
6. Nurturing Parenting (T2)	.21** [.10, .32]	.23** [.11, .34]	-.31** [-.41, -.20]	-.36** [-.45, -.25]	**.81**** [.77, .85]	

Note. Values in square brackets indicate the 95% confidence interval for each correlation. Correlations for monozygotic twins are included below the diagonal and dizygotic twins above the diagonal. T1 and T2 indicate twin 1 and twin 2. Correlations were generated from a double-entered dataset to eliminate any twin ordering effects. Correlations which represent ICCs for child executive functioning, harsh parenting, and nurturing parenting are bolded for clarity. * indicates $p < .05$. ** indicates $p < .01$.

Table III.6

Executive functioning factor score loadings and fit statistics

Item	Estimate (STDYX)	p
No-go Drift Rate	0.15	0.00
Go Drift Rate	0.12	0.04
Stop-Signal Drift Rate	0.16	0.01
EATQ Inhibitory Control	0.49	0.00
EATQ Attention	0.87	0.00
EATQ Activation Control	0.64	0.00

CFI = 0.83
TLI = 0.72
RMSEA = 0.12
Chi-sq = 94.01
df = 9
Chi-sq p = 0.00
SRMR = 0.08

Table III.7

More nurturing, less harsh parenting predicts better child executive functioning

Predictor	*b*	*b* 95% CI [LL, UL]	*beta*	sr^2	sr^2 95% CI [LL, UL]	Fit
(Intercept)	0.06	[-0.01, 0.12]	0.00			
Harsh Parenting	-0.05**	[-0.07, -0.03]	-0.23	.04	[.02, .07]	
Nurturing Parenting	0.04**	[0.02, 0.05]	0.17	.03	[.00, .05]	
Child Age	-0.00	[-0.01, 0.00]	-0.06	.00	[-.00, .01]	
Child Gender (Female)	-0.00	[-0.02, 0.02]	-0.02	.00	[-.00, .00]	
Child Ethnicity (Nonwhite)	-0.00	[-0.03, 0.02]	-0.01	.00	[-.00, .00]	
						R^2 = .116**
						95% CI[.07,.16]

Note. A significant *b*-weight indicates the semi-partial correlation is also significant. *b* represents unstandardized regression weights. sr^2 represents the semi-partial correlation squared. *LL* and *UL* indicate the lower and upper limits of a confidence interval, respectively. *Beta* indicates standardized regression coefficients. Table generated with apaTables package in R (Stanley & Spence, 2018). Beta coefficients generated with lm.beta package in R (Behrendt, 2014).
* indicates p < .05. ** indicates p < .01.

Table III.8

Better parent executive functioning predicts less harsh parenting

Predictor	*b*	*b* 95% CI [LL, UL]	*Beta*	sr^2	sr^2 95% CI [LL, UL]	Fit
(Intercept)	-0.09	[-0.40, 0.23]	0.00			
Parent Executive Functioning	-0.20**	[-0.27, -0.13]	-0.22	.05	[.02, .08]	
Child Age	0.01	[-0.02, 0.03]	0.02	.00	[-.00, .00]	
Child Gender (Female)	-0.06	[-0.15, 0.04]	-0.05	.00	[-.00, .01]	
Child Ethnicity (Nonwhite)	0.18**	[0.06, 0.29]	0.12	.02	[-.00, .03]	
						R^2 = .059**
						95% CI[.03, .09]

Note. A significant *b*-weight indicates the semi-partial correlation is also significant. *b* represents unstandardized regression weights. sr^2 represents the semi-partial correlation squared. *LL* and *UL* indicate the lower and upper limits of a confidence interval, respectively. *Beta* indicates standardized regression coefficients. Table generated with apaTables package in R (Stanley & Spence, 2018). Beta coefficients generated with lm.beta package in R (Behrendt, 2014).
* indicates $p < .05$. ** indicates $p < .01$.

Table III.9

Better parent executive functioning predicts more nurturing parenting

Predictor	*b*	*b* 95% CI [LL, UL]	*Beta*	sr^2	sr^2 95% CI [LL, UL]	Fit
(Intercept)	0.83**	[0.51, 1.15]	0.00			
Parent Executive Functioning	0.09**	[0.02, 0.16]	0.10	.01	[-.00, .02]	
Child Age	-0.06**	[-0.09, -0.04]	-0.22	.05	[.02, .08]	
Child Gender (Female)	0.19**	[0.10, 0.28]	0.15	.02	[.00, .04]	
Child Ethnicity (Nonwhite)	-0.02	[-0.14, 0.10]	-0.01	.00	[-.00, .00]	
						R^2 = .076**
						95% CI[.04, .11]

Note. A significant *b*-weight indicates the semi-partial correlation is also significant. *b* represents unstandardized regression weights. sr^2 represents the semi-partial correlation squared. *LL* and *UL* indicate the lower and upper limits of a confidence interval, respectively. *Beta* indicates standardized regression coefficients. Table generated with apaTables package in R (Stanley & Spence, 2018). Beta coefficients generated with lm.beta package in R (Behrendt, 2014).
* indicates $p < .05$. ** indicates $p < .01$.

Table III.10

Model estimates and model fit statistics for univariate and bivariate models

Nuclear Twin Family Models	A	S	F	E	AIC	BIC	ssBIC	RMSEA
Executive Functioning ASFE	0.26 [0 0.53]	0.07 [0 0.23]	0.00 [0 0.05]	0.68 *** [0.54 0.81]	3449.97	3473.19	3454.15	0.12
Executive Functioning ASE	0.22 ** [0.12 0.33]	0.09 [0 0.19]	-	0.69 *** [0.59 0.80]	3447.33	3466.67	3450.81	0.12
Classical Twin Models	**A**	**C**		**E**	**AIC**	**BIC**	**ssBIC**	**RMSEA**
Executive Functioning	0.29 [0 0.50]	0.08 [0 0.32]		0.63 *** [0.49 0.79]	1979.14	1994.62	1981.93	0.00
Harsh Parenting	0.17 [0 0.41]	0.57 *** [0.37 0.72]		0.26 *** [0.19 0.33]	1785.18	1800.66	1787.97	0.05
Nurturing Parenting	0.26 ** [0.07 0.47]	0.56 *** [0.37 0.72]		0.18 *** [0.13 0.24]	1725.03	1740.50	1727.81	0.02
Bivariate Models	**Axy**	**Cxy**		**Exy**	**CFI**	**TLI**	**SRMR**	**RMSEA**
Harsh Parenting / Executive Functioning	0.00 [0 0.32]	0.08 [0 0.19]		0.03 [0 0.09]	0.99	0.99	0.07	0.03
	rA = 0.02 prA = 0.01	rC = 1 *** prC = 0.70 ***		rE = 0.21 ** prE = 0.29 **				
Nurturing Parenting /	0.02	0.05		0.00	1	1	0.05	0.00

Executive Functioning	[0 0.32]	[0 0.19]	[0 0.09]
	rA = 0.27 prA = 0.31	rC = 0.78 * prC = 0.65 *	rE = 0.03 prE = 0.05

Note. This table depicts estimates and model fit statistics for the univariate Nuclear Twin Family Model, which provides a decomposition of shared environmental contributions into those shared between parents and children (F) and those shared between siblings (S). The reduced ASE model fit better than the full ASFE model. Additionally, this table depicts estimates and model fit statistics for the univariate ACE models, which provide a decomposition of genetic and environmental contributions to variance in executive functioning, harsh parenting, and nurturing parenting (N=354 pairs, 167 monozygotic). Finally, this table depicts relevant estimates from the bivariate ACE model, which provides a decomposition of genetic and environmental contributions to the phenotypic correlation between harsh parenting and executive functioning. Included are the ACE estimates for the overlap between harsh parenting and executive functioning, as well as the genetic (rA) and environmental (rC, rE) correlations, and the proportions of shared variance attributable to each (prA, prC, prE). High C correlations are likely driven by low C estimates overall. For estimates, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table III.11

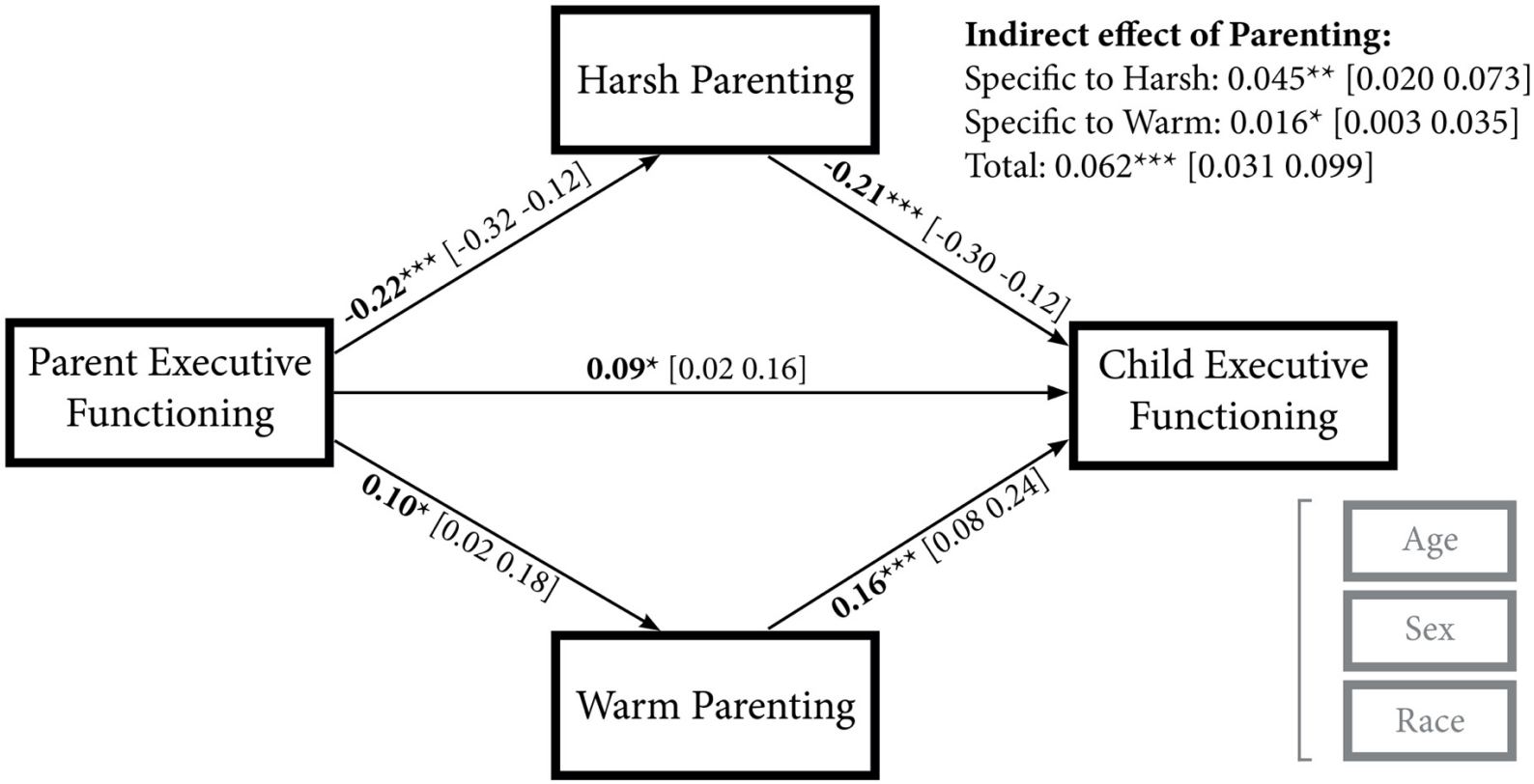
Model estimates and model fit statistics for supplemental ASFE models

Nuclear Twin Family Model	A	S	F	E	AIC	BIC	ssBIC	RMSEA
Executive Functioning (self-report only) ASFE	0.21 [0 0.49]	0.11 [0 0.27]	0.00 [0 0.05]	0.68 *** [0.54 0.82]	3408.31	3412.49	3412.49	0.12
Executive Functioning (behavioral only) ASFE	0.06 [0 0.38]	0.27 *** [0.08 0.38]	0.00 [0 0.04]	0.66 *** [0.52 0.75]	3241.11	3264.27	3245.24	0.13

Note. This table depicts estimates and model fit statistics for a supplemental Nuclear Twin Family Model using twin-report executive functioning data only (self-report only) and the behavioral executive functioning measure only (behavioral only) rather than the factor score which combined the two. The ASFE model provides a decomposition of shared environmental contributions (C in classical twin ACE model) into those shared between parents and children (F) and those shared between siblings (S). Note that, consistent with the results from the primary model using an executive functioning factor score, F is estimated at 0 regardless of the measurement strategy. For estimates, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Figure III.1

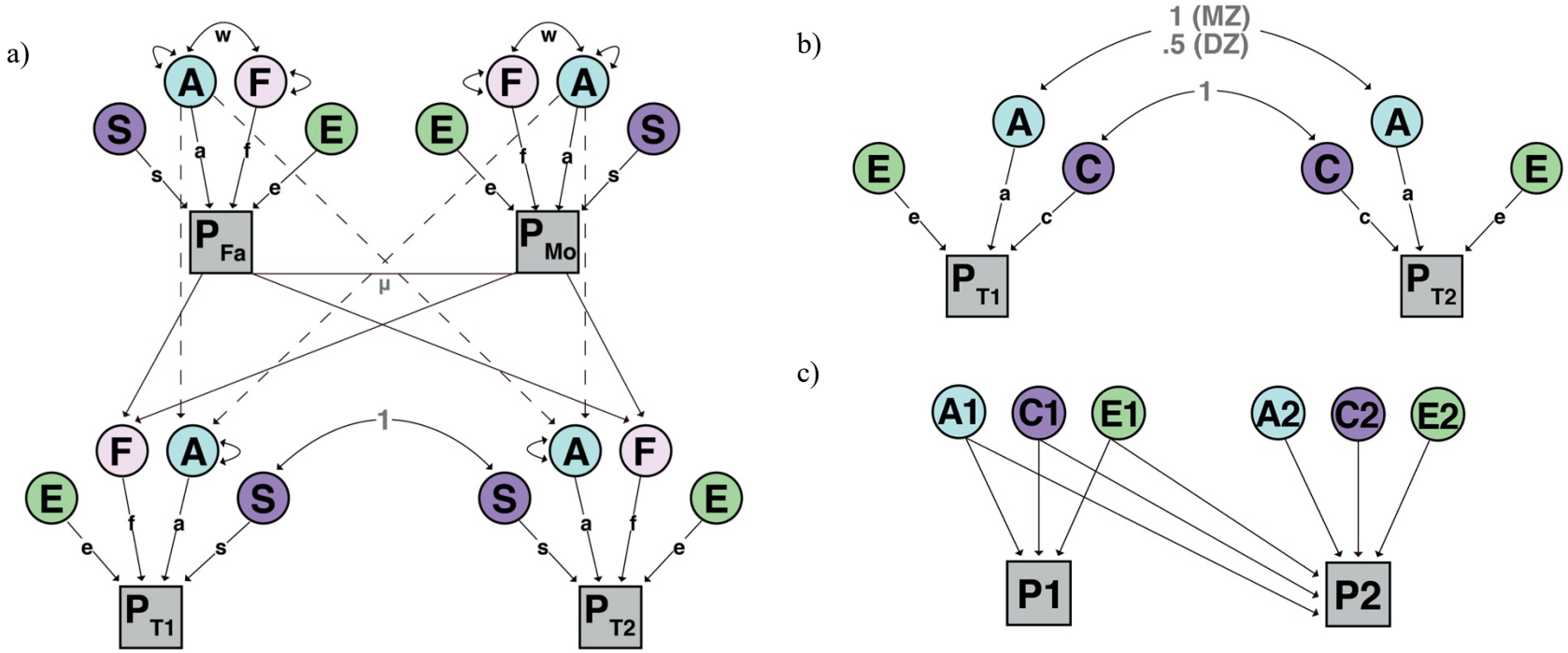
Parent executive functioning relates to child executive functioning via an effect on parenting



Note. Structural equation modeling using maximum likelihood estimation with bootstrapping and clustering for family in Mplus revealed an indirect effect of parent executive functioning on child executive functioning via both harsh and nurturing parenting behaviors. This figure depicts standardized estimates and bootstrapped 95% confidence intervals (bootstrap = 1000). For estimates, . p<0.1 * p <0.05, ** p<0.01, *** p<0.001

Figure III.2

Path diagrams of the nuclear twin family model, the classical univariate twin model, and the bivariate twin model



Note. This figure depicts path diagrams of the three twin models employed by the present study. **(a)** The nuclear twin family model incorporates parent measures of a phenotype of interest to allow for estimation of additional parameters. For the purposes of this study, an ASFE model was estimated. A represents genetic influences, S represents sibling-level shared environmental influences, F represents family-level shared environmental influences, and E represents nonshared environmental influences. P represents the phenotype of interest, measured for the father (Fa), mother (Mo), and twins (T1, T2). This model also takes into account assortative mating (μ) and calculates passive rGE (w). Adapted from Burt & Klump (2012). **(b)** The classical univariate twin model estimates genetic (A), shared environmental (C) and nonshared environmental (E) contributions to a given phenotype. Adapted from Burt & Klump (2012). **(c)** The bivariate twin model decomposes phenotypic covariance into genetic (A), shared environmental (C), and

nonshared environmental components (E). P1 and P2 represent the two phenotypes of interest. For simplicity, the path model for only one twin is depicted here. Adapted from Carroll et al. (2021).

CHAPTER IV

Parenting as a Buffer Against the Effects of Neighborhood Poverty on Executive Functioning

Socioeconomic disadvantage during childhood predicts a myriad of negative outcomes, including lower earnings, poorer mental and physical health, and higher rates of criminal behavior (Cohen et al., 2008; Duncan et al., 2010). One critical pathway linking socioeconomic disadvantage to these poorer outcomes involves executive functioning (Evans et al., 2021; Shameem & Hamid, 2014; Umbach et al., 2017). Indeed, meta-analyses support a robust link between socioeconomic disadvantage and reduced executive functioning performance (Lawson et al., 2018), and, in turn a constellation of evidence links executive functioning abilities to important life outcomes (Blair & Razza, 2007; Kubzansky et al., 2009; McClelland et al., 2007; Mischel et al., 2011; Moffitt et al., 2011; Seeyave et al., 2009). The mechanisms through which socioeconomic disadvantage undermines executive functioning performance remain elusive, though multiple theories posit that socioeconomic disadvantage may become biologically embedded via stress response systems that regulate executive functioning (Blair et al., 2011; Evans et al., 2021; Finegood & Blair, 2017; Ursache et al., 2015).

A growing literature points to neighborhood disadvantage as a particularly notable risk factor for executive functioning differences, over and above family-level socioeconomic variables. Neighborhood contexts confer additional risk beyond family-level factors because they increase exposure to additional adverse experiences (e.g., neighborhood danger, under-resourced schools, toxicant exposure; Evans, 2004; Leventhal & Brooks-Gunn, 2000). These

neighborhood-adversity-related stressors generate "wear" on stress regulatory systems, with implications for the brain circuitry underlying executive functioning (Finegood et al., 2017; McEwen, 2004). Indeed, there is a growing literature that finds effects of neighborhood context on brain structure and function. Recent work from our group demonstrated for the first time that children raised in neighborhoods with higher levels of poverty show reduced behavioral and brain measures of executive functioning (Tomlinson et al., 2020). Notably, this relationship held even when controlling for family-level socioeconomic variables (i.e., family income, maternal education), indicating that *where children live* is particularly important for their executive functioning development. There is also evidence of effects of neighborhood disadvantage on other corticolimbic structures and processes: for example, neighborhood disadvantage is associated with greater amygdala reactivity to faces (Gard et al., 2021), reduced prefrontal activation to working memory load (Murtha et al., 2021), changes in amygdala-prefrontal connectivity (Ramphal et al., 2020), reduced cortical thickness (Hackman et al., 2021; Hunt et al., 2020; Taylor et al., 2020), altered trajectories of brain age (Rakesh, Cropley, et al., 2021), and worse performance on neurocognitive tasks (Hackman et al., 2021; Taylor et al., 2020; Webb et al., 2021). Thus, considering the role of the neighborhood on EF-related brain development is of critical importance.

Though neighborhood disadvantage is a robust risk factor for negative outcomes, many children in this context do not show negative outcomes broadly, nor worse executive functioning proximally. One potential buffer supporting children's resilience to neighborhood stressors could the parenting they receive. In particular, nurturing, responsive parenting could act to buffer the effects of stress associated with living in a disadvantaged neighborhood. Indeed, there is substantial evidence that positive parenting helps children to acquire behavioral executive

functioning skills (Carlson, 2009; Fay-Stammbach et al., 2014; Valcan et al., 2017). There is also some evidence that the relationship between positive parenting and behavioral executive functioning persists in the context of poverty. For example, in families with low socioeconomic status, higher quality maternal behavior at age 1 predicted better performance on executive functioning tasks at age 3 (Rochette & Bernier, 2014). Similarly, positive parenting has been shown to buffer temperament-associated risk for lower executive functioning skills in the context of poverty (Song et al., 2018). Additionally, there is emerging evidence for positive parenting as a buffer for disadvantage-related brain differences: positive parenting behaviors were recently shown to moderate the relationship between neighborhood disadvantage and resting state functional connectivity (Rakesh, Seguin, et al., 2021). Thus, work is needed that investigates the moderating role of nurturing, responsive parenting on the relationship between neighborhood disadvantage and EF-related brain activation.

Present Study

The present study examined the associations between neighborhood poverty, nurturing, supportive parenting, and EF-related brain activation in a large community sample of twins. We utilized go/no-go and stop signal data from the Michigan Twin Neurogenetics Study (MTwiNS), a unique longitudinal twin study (N=708) with oversampling for families living in low-income neighborhoods. First, we attempted to extend the results of Tomlinson et al. (2020) to a larger sample, testing whether neighborhood poverty is associated with executive functioning performance via EF-related inferior frontal gyrus (IFG) activation during a go/no-go task. Second, we investigated whether nurturing, supportive parenting, thoroughly assessed via child and parent report, correlated with EF-related brain activation. Because nurturing and harsh parenting are separable constructs (Power, 2013) we also examined effects of harsh parenting as

an exploratory analysis. Third, we examined whether nurturing, supportive parenting buffered, or harsh parenting exacerbated, the effect of neighborhood poverty on brain and behavioral measures of EF. In line with emerging evidence in the field (Rakesh, Seguin, et al., 2021), we hypothesized that nurturing, supportive parenting would buffer the effect of neighborhood poverty on EF-related brain activation.

Methods

Participants

The present study included data from the Michigan Twins Neurogenetics Study (MTwiNS), a longitudinal neuroimaging project within the large-scale Michigan State University Twin Registry (see Burt & Klump, 2019). The 354 families participating in MTwiNS were originally identified through birth records. Families were originally recruited into the TBED-C study in one of two cohorts — a population-based cohort that represented families living within 120 miles of Michigan State University and an at-risk cohort recruited from the same area, but only including families living in U.S. Census tracts where at least 10.5% of families lived below the poverty line (the mean for the state of Michigan at the time; Burt & Klump, 2019). MTwiNS participants were re-recruited from the original participant pool based upon criteria for the second, "at-risk" cohort. Thus, MTwiNS includes families from the "at-risk" cohort as well as those from the population-based cohort that would have qualified for the "at-risk" cohort. This re-recruitment strategy yielded a sample representative of families living in neighborhoods with above average levels of poverty, a unique sampling frame within both the behavioral genetics and neuroimaging literatures (Burt et al., 2021).

The 540 twins (from 306 pairs, 234 complete, 115 monozygotic) that were included in the present study completed at least part of the functional MRI scan at their MTwiNS visit, and therefore met basic MRI safety criteria (e.g. no unsafe metal in body; Table IV.1). The twins were 7 to 19 years old (Mean=14.7, SD=2.1; 55% male; 1.5% of the present sample was 10 or younger). The breakdown of twins' parent-reported ethnicity reflected the surrounding area (81% White, 10% Black, 5% Other, 1% Latino, 1% Pacific Islander, 1% Native American, 1% Asian). Median reported family annual income for this sample was $80,000 to $89,999 and ranged from less than $4,999 to greater than $90,000. 9% of included families reported an annual income below the 2020 federal poverty line of $26,246 per year and 48% reported annual income below the living wage for a family of 4 in Michigan with one parent working (http://livingwage.mit.edu/states/26). Zygosity was established using physical similarity questionnaires administered to the twins and/or their parents (Bouchard et al., 1990; Iacono et al., 1999; Peeters et al., 1998). Discrepancies were resolved through review of zygosity items or by DNA markers. Parents provided informed consent and children provided assent.

Procedure

Twins and their primary caregivers took part in a day-long visit to the University of Michigan. Each twin participated in a mock scan as well as a one-hour MRI scan. Twins completed several blood-oxygen-level-dependent (BOLD) fMRI tasks in the scanner, including the go/no-go task described below. Outside of the scanner, twins completed several additional computer tasks, including a stop signal task (SST), and a battery of child-report questionnaires. Primary caregivers completed a demographic interview with an examiner and a battery of self- and parent-report questionnaires. Return visits are ongoing and follow a nearly identical protocol.

Measures

Socioeconomic Disadvantage

Neighborhood poverty. Neighborhood poverty was defined using geocoding of family addresses to assess the proportion of neighborhood residents living below the poverty line in each family's census tract, (www.census.gov). Because MTwiNS study data collection has spanned several years, the available rolling five-year span ending closest to the participants' date of visit was used for analysis (e.g. for a participant run in 2015 the 2011-2015 estimate was used, for a participant run in 2018 the 2014-2018 estimate was used). N=4 individuals with usable fMRI data declined to provide neighborhood information.

Family income. Family income was defined via primary caregiver reported monthly household gross income, including outside additional sources such as government assistance or child support. Income-to-needs ratio was calculated by dividing each family's reported income by the federal poverty level for their reported family size during the year of their visit (via https://aspe.hhs.gov/topics/poverty-economic-mobility/poverty-guidelines/prior-hhs-poverty-guidelines-federal-register-references). N=9 individuals with usable fMRI data declined to provide family income information.

Maternal education. Maternal education was defined via the primary caregiver's highest completed level of education. Though most primary caregivers were mothers, a small percentage were fathers. In this case, the father's highest completed level of education was used.

Go/No-Go Task

The go/no-go task used in this study was adapted from Casey et al. (1997), in which neural reactivity during inhibition is elicited via a "whack-a-mole" game (stimuli courtesy of Sarah Getz and the Sackler Institute for Developmental Psychobiology; task downloaded from

http://fablab.yale.edu/page/assays-tools). In the present task, participants were instructed to press a button as quickly as possible in response to one stimulus ("go", a mole) and avoid responding to a less frequent non-target ("No-Go", a vegetable). The target stimuli (moles) were modified with various "disguises" to make the task more interesting and difficult given the relatively slow speed of stimuli used to accommodate fMRI data acquisition. The task consisted of four runs, each with approximately 55 trials, for a total of 255 trials of which 55 were no-go (21.6%). Each no-go trial was preceded by 1-5 go trials. Each trial lasted 2300ms, including a maximum of 1800ms stimulus presentation, 400ms feedback, and 100 – 1000ms fixation to account for individual differences in reaction time. Participants practiced the task briefly in an MRI simulator before the MRI scan.

Participants with overall below-chance performance (<55%) or below-chance performance on "go" trials (<55%) were not considered to be meaningfully participating in the task and were therefore dropped from all analyses (N=13), leaving N=589 individuals and N=267 complete twin pairs with viable go/no-go data.

Stop-Signal Task

The child-friendly stop-signal task used in this study was a 10 minute, 150 trial task adapted from Bissett & Logan (2012) as described previously (Begolli et al., 2018). Participants were presented with a fish for 850ms and told to push the "a" or "l" keys as quickly as possible based on the orientation of the fish, unless a visual stop-signal stimulus (Martin the Manta Ray) appeared on the screen, which occurred on 50 of the task trials (33%; "stop" trials). This stop-signal was presented following a stop signal delay (SSD) that was determined through a standard "staircase tracking" algorithm (Logan, 1994) designed to lead to a roughly 50% probability of inhibition on "stop" trials for each participant. This algorithm began with a 250ms SSD and was

thereafter increased or decreased by 50ms on each subsequent ”stop” trial based on whether the participant was able to successfully inhibit. Participants with below-chance performance (<55%) on go trials were excluded from stop-signal analyses (N=59 wave 1, N=63 wave 2) and a number of participants did not complete the task at wave 1 as it was added to the protocol partway through data collection (N=229 individuals). These participants were still included in go/no-go analyses given viable go/no-go data (N=304 individuals).

Parenting

Parental Environment Questionnaire. The Parental Environment Questionnaire (PEQ; Elkins et al., 1997) was administered to assess nurturance and conflict in each parent-child dyad. The involvement or nurturance subscale (12 items) assesses communication, closeness, and support in the parent-child relationship (e.g., “I praise my child when he/she does something well”). The conflict subscale (12 items) assesses disagreement, tension, and anger in the parent-child relationship (e.g., “I often criticize my child”). Mothers and fathers reported on their relationships with each twin, and each twin reported on their relationship with their parent(s). Within the MTwiNS sample, internal consistencies for the PEQ subscales were acceptable (for conflict, α = 0.901, 0.883, and 0.873 for twin, mother, and father report, respectively; for involvement, α = 0.859, 0.794, and 0.858 for twin, mother, and father report, respectively).

Alabama Parenting Questionnaire. The Alabama Parenting Questionnaire (APQ; Shelton et al., 1996) was administered to assess parenting in each parent-child dyad. The parental involvement or nurturance subscale (10 items) assesses closeness and communication in the parent-child relationship (e.g. “You have a friendly talk with your child”). The positive parenting subscale (6 items) assesses positive reinforcement behaviors (e.g. “You praise your child if he/she behaves well”). The inconsistent discipline subscale (6 items) assesses unpredictability in

parental discipline (e.g. “The punishment you give your child depends on your mood”). The corporal punishment subscale (3 items) assesses physical discipline (e.g. “You spank your child with your hand when he/she has done something wrong). Some participants received a shortened version of the corporal punishment subscale; these participants’ scores were prorated accordingly. Mothers and fathers reported on their relationships with each twin, and each twin reported on their relationship with their mother. Within the MTwiNS sample, internal consistencies for the APQ subscales were acceptable (for inconsistent discipline, $\alpha = 0.749$, and 0.701 for mother and father report, respectively; for positive parenting, $\alpha = 0.785$ and 0.801 for mother, and father report, respectively). Because some families received only one corporal punishment item, internal consistency was not computed for that scale.

Parenting Composite. Consistent with prior work (e.g. Burt et al., 2003), we created a composite of all reporters for each subscale of the PEQ and APQ to assess the overall parenting environment for each child, averaging z-scored twin reports, mother reports, and father reports, and then averaging these three reporters together. Using this method, we created a “harsh parenting” composite including data from all available reporters on the PEQ conflict, APQ inconsistent discipline, and APQ corporal punishment subscales; we created a “nurturing parenting” composite from all available reporters on the PEQ involvement, APQ parental involvement, and APQ positive parenting subscales. When any of these reporters were missing (N=144 (harsh) and N=132 (nurturing) individuals were missing at least 1 informant-report, primarily father), we calculated the composite from the available reports. Using this method, all twins with fMRI data had composite parenting data (N=540 individuals).

Functional neuroimaging

Functional imaging data were acquired using one of two GE Discovery MR750 3T scanners located at the University of Michigan Functional MRI Laboratory. To leverage improvements in MRI data acquisition and to be consistent with the Adolescent Brain Cognitive Development (ABCD) Study (Casey et al., 2018), we altered our acquisition protocol after the first 140 MTwiNS families. For the first 140 MTwiNS families, one run of 284 volumes was collected for each participant using an 8-channel head coil. BOLD functional images were acquired using a gradient-echo reverse spiral sequence (repetition time = 2000ms, echo time = 30ms, flip angle = 90°, FOV = 22cm). Images included 43 interleaved oblique slices of 3mm thickness with 3.44x3.44mm^2 in-plane resolution. High-resolution T1-weighted SPGR images (156 slices, slice thickness = 1mm, in plane resolution of 1x1mm^2) were aligned with the AC-PC plane and used during normalization of the functional images. For the remaining MTwiNS participants (families 141-354), one run of 685 volumes was collected for each participant. BOLD functional images were acquired using a gradient-echo multiband sequence (repetition time = 800ms, echo time = 30ms, flip angle = 52°, FOV = 21.6 cm) with a 32-channel head coil, which covered 697 interleaved axial slices of 2.4 mm thickness. High-resolution T1-weighted SPGR images (208 slices, slice thickness = 1 mm) were aligned with the AC-PC plane and used during normalization of the functional images. Scan protocol was included as a nuisance regressor in fMRI analyses.

Preprocessing for both acquisition sequences were identical unless otherwise specified. Functional data were preprocessed and analyzed using Statistical Parametric Mapping software version 12 (SPM12; Wellcome Centre for Human Neuroimaging). The first four volumes of each run were discarded to allow for stabilization of the MR signal. Raw k-space data were de-spiked before reconstruction to image space in reverse-spiral sequence acquisition. For gradient-echo

sequence data with multiband acquisition, task-specific field maps were constructed from volumes of both anterior-to-posterior and posterior-to-anterior phase encoding; field maps were applied after image construction to reduce spatial distortions and minimize movement artifacts. Slice timing correction was performed using the median slice as the reference slice. Functional data were then spatially realigned to the 10th slice of the volume. These spatially realigned data were coregistered to a high-resolution T1-weighted image, segmented, and spatially normalized into standard stereotactic space to the Montreal Neurological Institute (MNI) template. For participants with unusable high-resolution T1-weighted structural images (N=4), we spatially realigned using the T1-weighted overlay image (first 140 families: 43 slices, slice thickness=3mm, in plane resolution of 0.85x0.85mm^2; families 141-354: 60 slices, slice thickness = 2.4mm, in plane resolution of 0.9x0.9mm^2). Finally, functional data were smoothed using a 6mm Gaussian kernel.

After preprocessing, the Artifact detection Tools toolbox (ART; https://www.nitrc.org/projects/artifact_detect/) was used to detect translation or rotational motion outlier volumes that remained after earlier QA (>2mm movement or 3.5 rotation) and to scrub them from the dataset. Preprocessed images were also visually inspected for major artifacts. Coverage of the frontal lobe was checked using the WFU PickAtlas "frontal lobe" structural mask (Maldjian et al., 2003). A participant's fMRI data were considered unusable if they contained obvious prefrontal artifacts, had >5% of volumes labeled as artifacts, or had less than 90% coverage of the frontal mask (Table IV.1). After these checks, N=553 participants had usable fMRI data. Preprocessing was conducted in containerized versions of SPM12, and the standard pipeline is accessible via Github (https://github.com/UMich-Mind-Lab/pipeline-task-standard).

Functional data were modeled using the general linear model in SPM12. Three conditions were modeled: correct No-Go trials, in which a participant correctly withheld a response to a No-Go stimulus; incorrect No-Go trials, in which a participant incorrectly responded to a No-Go stimulus; and Go trials, in which a participant saw a Go stimulus. Incorrect Go trials were not modeled due to the expected high hit rates for Go trials (median 100%). For each participant, the main contrast of interest was all No-Go > Go.

Analytic Plan

Executive Functioning Performance

We used a model-based measure of task-general executive functioning, "Efficiency of Evidence Accumulation", as our executive functioning metric (Weigard & Sripada, 2021). Drift rate parameters from a drift diffusion model were calculated using Bayesian methods for each executive functioning task and condition (i.e. no-go, go, stop-signal) using the Dynamic Models of Choice functions (Heathcote, 2019), an adaptation of the differential evolution Markov chain Monte Carlo model (Turner et al., 2013), in R version 4.1.0 (R Core Team, 2021). Following previous work (Heathcote et al., 2015; Weigard et al., 2020), efficiency of evidence accumulation (hereafter, "EEA") was estimated as the difference in accumulation rates between the evidence accumulator for the correct choice (*vc*) and that for the incorrect choice (*ve*) scaled by the between-trial variability in accumulation rates pooled across both choices (*sv*): (*vc*-*ve*)/*sv*. Drift rate was calculated separately for "go" and "no-go" trial types within the go/no-go task due to previous evidence that it may systematically differ across the two conditions (Huang-Pollock et al., 2017; Ratcliff et al., 2018). Individual participants' EEA scores were calculated as latent factor scores from the three drift rate parameters (go, no-go, stop-signal) using maximum

likelihood estimation in Mplus version 8.6 (Muthén & Muthén, 1998-2017) via R, tidyverse (Wickham et al., 2019), and the *MplusAutomation* package (Hallquist & Wiley, 2018).

Aim 1: Replication of Tomlinson et al. (2020)

Using the same bilateral anatomically-defined IFG ROI from WFU PickAtlas (Maldjian et al., 2003) as in Tomlinson et al. (2020), we extracted IFG activation for a No-Go vs. Go F contrast (i.e., first eigenvariate in SPM12), controlling for scan protocol, for each participant for use in structural equation models (SEM) in Mplus version 8.6 (Muthén & Muthén, 1998-2017). Using Mplus, we first tested whether neighborhood poverty related to IFG activation over and above income-to-needs ratio and maternal education. We then re-tested the full SEM model from Tomlinson et al. (2020) to determine whether there was an indirect effect of neighborhood poverty on executive functioning performance via EF-related brain activation.

Contextual variables were also collected during twins' preliminary visit to Michigan State University upon recruitment to the TBED-C study (twin ages ranged from 6-11 at the time; Mean= 8.05; SD=1.43). As an exploratory analysis, we re-ran the full Tomlinson et al. (2020) model as a longitudinal model using contextual data (i.e. neighborhood poverty, income-to-needs ratio, maternal education) from this TBED-C childhood visit to predict brain and behavioral executive functioning.

Aim 2: Neural Correlates of Warm and Harsh Parenting

To identify brain regions associated with nurturing and harsh parenting behaviors, we ran two whole-brain regression models in SPM12, one with nurturing parenting and one with harsh parenting as a continuous regressor. Nuisance regressors were scan protocol, age, and sex. Due to the exploratory nature of this analysis, we employed a stringent, whole-brain approach, with cluster thresholding determined for each model by 3dttest++ within AFNI (Cox, 1996; Cox et al.,

2017a, 2017b) based on an overall error rate (alpha) of 0.05 with a voxel-wise threshold of $p<0.001$.

Aim 3: Parenting as a Moderator of Neighborhood Effects on Behavioral and Neural EF

To examine the role of parenting as a moderator of neighborhood effects, we ran a series of SEM models in MPlus using the IFG activation values extracted in Aim 1. First, we tested whether positive parenting buffered, or harsh parenting activated, neighborhood effects on behavioral executive functioning. Second, we tested whether positive parenting buffered, or harsh parenting activated, neighborhood effects on EF-related IFG activation. Third, we tested two full models in which nurturing and harsh parenting each moderated the path from neighborhood effects to IFG activation, which then predicted behavioral executive functioning. All models used Maximum Likelihood estimation (ML) and the CLUSTER command to account for nesting within families. Models controlled for all other SES-related variables, as well as age and sex.

Finally, as an exploratory analysis, we tested whether the interaction between positive parenting and neighborhood predicted activation in any brain regions. An interaction term (nurturing parenting * neighborhood poverty) was created and included as a continuous regressor in a whole-brain regression in SPM12. Additional regressors in this model were nurturing parenting, neighborhood poverty, scan protocol, age, and sex. We also ran an identical model for the interaction between harsh parenting and neighborhood poverty (harsh parenting * neighborhood poverty). We used a stringent, whole-brain approach for both models, with cluster thresholding determined for each model by 3dttest++ within AFNI (Cox, 1996; Cox et al., 2017a, 2017b) based on an overall error rate (alpha) of 0.05 with a voxel-wise threshold of $p<0.001$.

Results

Descriptive statistics and zero-order correlations for socioeconomic context variables, executive functioning, parenting composites, and extracted IFG activation are reported in Table IV.2. Contextual variables were all related to each other, with moderate correlations in the expected directions among neighborhood poverty, income-to-needs ratio, and maternal education (r's=-.17 to .40). At the zero-order level, the nurturing and harsh parenting composites were moderately negatively correlated (r=-.35, 95% CI [-.43, -.28]) and were not related to any of the contextual variables (Table IV.2). Harsh parenting was modestly negatively correlated with the behavioral executive functioning measure (r=-.10, 95% CI [-.19, -.02] and with extracted IFG activation (r=-.09, 95% CI [-.17, -.00]). Extracted IFG activation was also modestly negatively correlated with neighborhood poverty (r=-.14, 95% CI [-.22, -.06]) and positively correlated with behavioral executive functioning (r=.13, 95% CI [.05, .22]). As reported previously in a smaller subset of this sample, the child-friendly Go/No-Go task yielded robust IFG and ACC activation, among other regions, for the primary No-Go vs. Go F contrast (Figure IV.1; Tomlinson et al., 2020).

Neighborhood poverty relates to behavioral executive functioning via brain activation

Structural equation models accounting for age and relatedness within families revealed that neighborhood poverty did relate to IFG activation over and above income-to-needs ratio and maternal education (β=-0.12, 95% CI [-.21, -.03]; Figure IV.2a). An expanded model including behavioral executive functioning performance revealed that more positive IFG activation did relate to better executive functioning performance (β=0.09, 95% CI [0.01, 0.17]; Figure IV.2b) Consistent with Tomlinson et al. 2020, the expanded model also revealed a small indirect effect

of neighborhood poverty on executive functioning performance via inhibition-related IFG activation (β=-0.012, 95% CI [-0.025, -0.001]; Figure IV.2b).

An exploratory structural equation model revealed similar patterns when considering longitudinal, middle childhood contextual variables. Neighborhood poverty in childhood predicted IFG activation in adolescence over and above income-to-needs ratio and maternal education (β=-0.15, 95% CI [-.24, -.05]), and more positive IFG activation did relate to better executive functioning performance (β=0.133, 95% CI [0.04, 0.23]). There was again a small indirect effect of neighborhood poverty on executive functioning performance via inhibition-related IFG activation (β=-0.019, 95% CI [-0.04, -0.004]).

Neither Nurturing nor Harsh Parenting Correlate with Inhibition-Related Brain Activation

Though harsh parenting negatively correlated with extracted IFG activation at the zero-order level (r=-.09, 95% CI [-.17, -.00]; Table IV.2), neither the nurturing nor the harsh parenting composites correlated with brain activation in any region when controlling for age, sex, and scan protocol, and when correcting for multiple comparisons across the whole brain (i.e. no clusters passed threshold of $p_{unc}<0.001$, alpha<0.05, k=153). Even at more lenient thresholds (i.e. $p_{unc}<0.005$, alpha<0.05; and $p_{unc}<0.01$, alpha<0.1) no clusters passed thresholding. However, at very lenient thresholds (i.e. $p_{unc}<0.01$, no cluster thresholding) harsher parenting was associated with less activation in one moderately sized inferior frontal gyrus cluster (k=197), which is likely the driver of the zero-order correlation between harsh parenting and extracted IFG activation. This cluster was too modest to pass any rigorous whole-brain correction.

Concurrent Parenting Does Not Moderate Neighborhood Effects on Executive Functioning

Structural equation models revealed that neither nurturing nor harsh parenting moderated neighborhood effects on executive functioning performance (for nurturing x neighborhood, β=-0.07, 95% CI [-0.21, 0.08]; for harsh x neighborhood, β=-0.15, 95% CI [-0.78, 0.48]; Figure IV.3a). Similarly, neither nurturing nor harsh parenting moderated neighborhood effects on extracted IFG activation (for nurturing x neighborhood, β=0.02, 95% CI [-0.09, 0.13]; for harsh x neighborhood, β=0.01, 95% CI [-0.09, 0.11]; Figure IV.3b). Full models in which nurturing and harsh parenting each moderated the path from neighborhood effects to extracted IFG activation, which then predicted behavioral executive functioning, yielded similar results. Scatterplots of the association between neighborhood poverty and brain and behavioral executive functioning at different levels of nurturing and harsh parenting are provided in Figure IV.4.

Exploratory functional neuroimaging analyses revealed that neither the interaction between neighborhood poverty and nurturing parenting nor the interaction between neighborhood poverty and harsh parenting correlated with brain activation after controlling for multiple comparisons across the whole brain (i.e. no clusters passed threshold of $p_{unc}<0.001$, alpha<0.05, k=153).

Discussion

Within a well-sampled cohort of adolescents with oversampling for families living in low-income neighborhoods, we extended the results of Tomlinson et al. (2020) to a much larger sample, again finding that neighborhood poverty was associated with executive functioning performance via inhibition-related inferior frontal gyrus (IFG) activation during a go/no-go task, over and above effects of income-to-needs ratio and maternal education. Moreover, we extended this previous finding further to show that neighborhood poverty during childhood also longitudinally predicted IFG activation and executive functioning during adolescence. Contrary

to hypotheses, we found that our broad nurturing and harsh parenting composites, calculated from child, mother, and father report, did not correlate with EF-related brain activation when controlling for multiple comparisons across the whole brain. Similarly, we did not find that nurturing, supportive parenting buffered the effect of neighborhood poverty on brain and behavioral measures of executive functioning, nor that harsh parenting activated risk. These findings again highlight the role of the neighborhood context as an important predictor of EF-related brain activation, but also indicate that the parenting environment in adolescence, at least as measured here, does not promote nor undermine resilience to these concurrent neighborhood effects.

In line with a growing constellation of evidence implicating neighborhood disadvantage as an important predictor of neurocognitive performance (Hackman et al., 2021; Taylor et al., 2020; Webb et al., 2021) and measures of corticolimbic brain structure and function (Gard et al., 2021; Hackman et al., 2021; Hunt et al., 2020; Murtha et al., 2021; Rakesh, Cropley, et al., 2021; Ramphal et al., 2020; Taylor et al., 2020), we found that higher levels of neighborhood poverty were associated with reduced inhibition-related IFG activation during a go/no-go task, over and above effects of income-to-needs ratio or maternal education. We also found that more positive inhibition-related IFG activation was associated with better behavioral executive functioning performance, and that neighborhood poverty related to executive functioning performance via IFG activation, extending the results of Tomlinson et al. (2020) to a larger sample.

Notably, there were several updates to the structural equation models used in the present study that led to slight differences from Tomlinson et al. (2020). For one, the present study measured behavioral executive functioning via a novel, computationally derived, task-general executive functioning measure, including performance on an additional task performed outside

the scanner, rather than a simple summary of go/no-go performance. This update broadens the scope of the model, implicating neighborhood poverty as a predictor of broader executive functioning rather than inhibition performance alone. Additionally, because data collection spanned several years, the present study transformed family income into an income-to-needs ratio reflecting each family's size and the year of their visit. This updated method should much more accurately capture the extent to which each family's income was meeting their family's needs at the time given their individual circumstances. Even with these updates, structural equation models revealed a very similar pattern of results to Tomlinson et al. (2020) with more than double the sample size (N=527 vs. N=185). Notably, an exploratory longitudinal model utilizing contextual data from middle childhood also yielded similar results, indicating that neighborhood poverty in middle childhood relates longitudinally to brain and behavioral executive functioning in adolescence.

Contrary to our hypotheses, we did not find any associations between the broad parenting environment and inhibition-related brain activation when controlling for multiple comparisons across the whole brain. This null result was surprising, given relatively robust and consistent findings that nurturing, supportive parenting behaviors promote better executive functioning development, while harsh, controlling parenting behaviors undermine executive functioning development (Fay-Stammbach et al., 2014; Hughes & Devine, 2019; Li et al., 2019). This result also runs in contrast to other parenting and brain work finding parenting effects on amygdala activation during emotional tasks (Gard et al., 2017; Romund et al., 2016) and on inhibition-related activation during an activation control task (Dandash et al., 2021). However, there are several differences between the present study and previous work that may explain this discrepancy. For one, there are many different ways to measure parenting. For example, previous

work on parenting and executive functioning measured specific and highly relevant parenting behaviors like scaffolding or intrusiveness (Broomell et al., 2020; Distefano et al., 2018; Fay-Stammbach et al., 2014; Hammond et al., 2012; Hughes & Devine, 2019; Valcan et al., 2017). On the other hand, Dandash et al. (2021) collected an observational measure of positive and aggressive parenting, finding in an exploratory analysis that more positive parenting was associated with less inhibition-related brain activation in relevant regions. The parenting composites used in the present study differed from both of these methods, instead indexing a broad informant-report picture of the nurturance and harshness of the parent-child relationship, incorporating multiple reporters and multiple questionnaire measures. It may be that our parenting measures were too broad to capture an association between more specific behaviors, like scaffolding, and brain activation.

A second difference between this study and extant work regarding parenting and executive functioning is our focus on adolescence rather than early childhood (i.e. age 0-5; see Valcan et al., 2017 for a meta-analysis). Though adolescence is a time when symptoms of psychopathology related to deficits in executive functioning begin to emerge (Cherkasova et al., 2013; Moffitt, 2018), early childhood represents a particularly interesting timepoint for both executive functioning development and for parenting effects. Executive functioning skills and related brain architecure are developing very rapidly during early childhood (Diamond, 2013; Kolb et al., 2012; Zelazo & Carlson, 2012), setting the stage for future pruning and refinement later in adolescence (Kolb et al., 2012). Early childhood is also a period when children spend much of their time in the home, often with parents, and thus parenting effects may be more apparent (Shaw & Bell, 1993). Future work could investigate the longitudinal role of parenting,

ideally measured in early childhood, on EF-related brain activation in childhood and adolescence.

Also, in contrast to our hypotheses, we found that neither nurturing nor harsh parenting moderated neighborhood effects on brain or behavioral measures of executive functioning, and that there were no brain regions in which EF-related activation correlated with the interaction between either parenting composite and neighborhood poverty. These results are surprising given emerging evidence in the field that positive parenting buffers the effect of neighborhood disadvantage on resting state functional connectivity in EF-relevant regions (ventral attention network, visual network, default mode network; Rakesh, Seguin, et al., 2021). However, the present study design differed substantially from this previous work. For one, our parenting composites incorporated multiple measures of mother-, father-, and child-reported parenting, compared to a five-item, child-report measure of specific positive parenting behaviors used in Rakesh, Seguin et al., 2021. Relatedly, neither our nurturing nor our harsh parenting composites correlated with any contextual measure, including neighborhood poverty or income-to-needs ratio (Table IV.2, Figure IV.5). It is possible that our comprehensive, multi-informant composites are too broad to capture relevant parenting behaviors. It is also possible that the use of multiple informants constrains variation, such that reports from one reporter are negating those of another. Additionally, children in our sample were older on average than in Rakesh, Seguin et al., 2021 (M=14.7 vs. M=10.0 years). Finally, and most notably, we measured task-related activation as our brain variable rather than resting state connectivity. It is possible the present results might differ if we were to consider connectivity between regions rather than task-related activation within them.

Though this study extends the Tomlinson et al. (2020) findings to a larger sample, it does not move us closer to understanding why the more distal neighborhood context, and nor the more proximal context as measured by income-to-needs ratio and maternal education, relates to brain and behavioral measures of executive functioning. Future work could further extend the model presented here and in Tomlinson et al. (2020) by investigating *what about* the neighborhood may be associated with executive functioning and related brain activation. There are a multitude of variables that covary with neighborhood poverty, but not necessarily with family income or maternal education, that may contribute. For example, neighborhood disadvantage is associated with exposure to additional adverse experiences such as exposure to community violence, under-resourced schools, lower home quality, poorer municipal services, and increased toxicant exposure (Evans, 2004; Leventhal & Brooks-Gunn, 2000). These stressors are thought to generate "wear" on stress regulatory systems, affecting brain development (Finegood et al., 2017; McEwen, 2004), though it is not clear which stressors may be the "active ingredients". Studies of emotion processing and related corticolimbic circuitry point to exposure to community violence as a key factor: exposure to community violence is associated with heightened amygdala reactivity to angry faces (White et al., 2019) and smaller hippocampal volumes (Saxbe et al., 2018) in youth. However, recent meta-analytic evidence points to early-life deprivation (e.g. food insecurity, neglect) as a stronger predictor of executive functioning performance than early-life threat (e.g. exposure to violence in or out of the home; Johnson et al., 2021). Future work could assess whether neighborhood-related stressors such as neighborhood deprivation or danger predict executive functioning performance or related brain activation.

In summary, the present study extended the results of Tomlinson et al. (2020) to a larger sample, again finding that neighborhood poverty was associated with executive functioning

performance via IFG activation during a go/no-go task, over and above effects of income-to-needs ratio and maternal education, both concurrently and longitudinally. Contrary to hypotheses, we did not find evidence for nurturing, supportive parenting as a buffer nor for harsh parenting as an activator of neighborhood risk. These findings again highlight that *where children live* is critical for executive functioning and related brain activity. However, the broad parenting environment in adolescence does not appear to promote nor undermine resilience to the effects of neighborhood poverty.

REFERENCES

Begolli, K. N., Richland, L. E., Jaeggi, S. M., Lyons, E. M., Klostermann, E. C., & Matlen, B. J. (2018). Executive Function in Learning Mathematics by Comparison: Incorporating Everyday Classrooms into the Science of Learning. *Thinking & Reasoning, 24*(2), 280-313. https://doi.org/10.1080/13546783.2018.1429306

Bissett, P. G., & Logan, G. D. (2012). Post-Stop-Signal Slowing: Strategies Dominate Reflexes and Implicit Learning. *J Exp Psychol Hum Percept Perform, 38*(3), 746-757. https://doi.org/10.1037/a0025429

Blair, C., Raver, C. C., Granger, D., Mills-Koonce, R., Hibel, L., & Family Life Project Key, I. (2011). Allostasis and Allostatic Load in the Context of Poverty in Early Childhood. *Dev Psychopathol, 23*(3), 845-857. https://doi.org/10.1017/S0954579411000344

Blair, C., & Razza, R. P. (2007). Relating Effortful Control, Executive Function, and False Belief Understanding to Emerging Math and Literacy Ability in Kindergarten. *Child Dev, 78*(2), 647-663. https://doi.org/10.1111/j.1467-8624.2007.01019.x

Bouchard, T. J., Jr., Lykken, D. T., McGue, M., Segal, N. L., & Tellegen, A. (1990). Sources of Human Psychological Differences: The Minnesota Study of Twins Reared Apart. *Science, 250*(4978), 223-228. https://doi.org/10.1126/science.2218526

Burt, S. A., & Klump, K. L. (2019). The Michigan State University Twin Registry (Msutr): 15 Years of Twin and Family Research. *Twin Res Hum Genet, 22*(6), 741-745. https://doi.org/10.1017/thg.2019.57

Burt, S. A., Klump, K. L., Vazquez, A. Y., Shewark, E. A., & Hyde, L. W. (2021). Identifying Patterns of Youth Resilience to Neighborhood Disadvantage. *Research in Human Development*, 1-16. https://doi.org/10.1080/15427609.2021.1935607

Burt, S. A., Krueger, R. F., McGue, M., & Iacono, W. (2003). Parent-Child Conflict and the Comorbidity among Childhood Externalizing Disorders. *Arch Gen Psychiatry, 60*(5), 505-513. https://doi.org/10.1001/archpsyc.60.5.505

Carlson, S. M. (2009). Social Origins of Executive Function Development. *New Dir Child Adolesc Dev, 2009*(123), 87-98. https://doi.org/10.1002/cd.237

Casey, B. J., Cannonier, T., Conley, M. I., Cohen, A. O., Barch, D. M., Heitzeg, M. M., Soules, M. E., Teslovich, T., Dellarco, D. V., Garavan, H., Orr, C. A., Wager, T. D., Banich, M. T., Speer, N. K., Sutherland, M. T., Riedel, M. C., Dick, A. S., Bjork, J. M., Thomas, K. M., Chaarani, B., Mejia, M. H., Hagler, D. J., Jr., Daniela Cornejo, M., Sicat, C. S., Harms, M. P., Dosenbach, N. U. F., Rosenberg, M., Earl, E., Bartsch, H., Watts, R., Polimeni, J. R., Kuperman, J. M., Fair, D. A., Dale, A. M., & Workgroup, A. I. A. (2018). The Adolescent Brain Cognitive Development (Abcd) Study: Imaging Acquisition across 21 Sites. *Dev Cogn Neurosci, 32*, 43-54. https://doi.org/10.1016/j.dcn.2018.03.001

Casey, B. J., Trainor, R. J., Orendi, J. L., Schubert, A. B., Nystrom, L. E., Giedd, J. N., Castellanos, F. X., Haxby, J. V., Noll, D. C., Cohen, J. D., Forman, S. D., Dahl, R. E., & Rapoport, J. L. (1997). A Developmental Functional Mri Study of Prefrontal Activation During Performance of a Go-No-Go Task. *Journal of Cognitive Neuroscience, 9*(6), 835-847. https://doi.org/10.1162/jocn.1997.9.6.835

Cherkasova, M., Sulla, E. M., Dalena, K. L., Ponde, M. P., & Hechtman, L. (2013). Developmental Course of Attention Deficit Hyperactivity Disorder and Its Predictors. *J Can Acad Child Adolesc Psychiatry, 22*(1), 47-54. https://www.ncbi.nlm.nih.gov/pubmed/23390433

Cohen, P., Chen, H., Gordon, K., Johnson, J., Brook, J., & Kasen, S. (2008). Socioeconomic Background and the Developmental Course of Schizotypal and Borderline Personality Disorder Symptoms. *Dev Psychopathol, 20*(2), 633-650. https://doi.org/10.1017/S095457940800031X

Cox, R. W. (1996). Afni: Software for Analysis and Visualization of Functional Magnetic Resonance Neuroimages. *Comput Biomed Res, 29*(3), 162-173. https://doi.org/10.1006/cbmr.1996.0014

Cox, R. W., Chen, G., Glen, D. R., Reynolds, R. C., & Taylor, P. A. (2017a). Fmri Clustering and False-Positive Rates. *Proc Natl Acad Sci U S A, 114*(17), E3370-E3371. https://doi.org/10.1073/pnas.1614961114

Cox, R. W., Chen, G., Glen, D. R., Reynolds, R. C., & Taylor, P. A. (2017b). Fmri Clustering in Afni: False-Positive Rates Redux. *Brain Connect, 7*(3), 152-171. https://doi.org/10.1089/brain.2016.0475

Cumming, G. (2014). The New Statistics: Why and How. *Psychol Sci, 25*(1), 7-29. https://doi.org/10.1177/0956797613504966

Dandash, O., Cherbuin, N., Schwartz, O., Allen, N. B., & Whittle, S. (2021). The Long-Term Associations between Parental Behaviors, Cognitive Function and Brain Activation in Adolescence. *Sci Rep, 11*(1), 11120. https://doi.org/10.1038/s41598-021-90474-2

Diamond, A. (2013). Executive Functions. *Annu Rev Psychol, 64*, 135-168. https://doi.org/10.1146/annurev-psych-113011-143750

Duncan, G. J., Ziol-Guest, K. M., & Kalil, A. (2010). Early-Childhood Poverty and Adult Attainment, Behavior, and Health. *Child Dev, 81*(1), 306-325. https://doi.org/10.1111/j.1467-8624.2009.01396.x

Elkins, I. J., McGue, M., & Iacono, W. G. (1997). Genetic and Environmental Influences on Parent-Son Relationships: Evidence for Increasing Genetic Influence During Adolescence. *Dev Psychol, 33*(2), 351-363. https://doi.org/10.1037//0012-1649.33.2.351

Evans, G. W. (2004). The Environment of Childhood Poverty. *Am Psychol, 59*(2), 77-92. https://doi.org/10.1037/0003-066X.59.2.77

Evans, G. W., Farah, M. J., & Hackman, D. A. (2021). Early Childhood Poverty and Adult Executive Functioning: Distinct, Mediating Pathways for Different Domains of Executive Functioning. *Dev Sci*, e13084. https://doi.org/10.1111/desc.13084

Fay-Stammbach, T., Hawes, D. J., & Meredith, P. (2014). Parenting Influences on Executive Function in Early Childhood: A Review. *Child Development Perspectives, 8*(4), 258-264. https://doi.org/10.1111/cdep.12095

Finegood, E. D., & Blair, C. (2017). Poverty, Parent Stress, and Emerging Executive Functions in Young Children. In K. Deater-Deckard & R. Panneton (Eds.), *Parental Stress and Early Child Development*. Springer.

Finegood, E. D., Rarick, J. R. D., Blair, C., & Family Life Project, I. (2017). Exploring Longitudinal Associations between Neighborhood Disadvantage and Cortisol Levels in Early Childhood. *Dev Psychopathol, 29*(5), 1649-1662. https://doi.org/10.1017/S0954579417001304

Gard, A. M., Maxwell, A. M., Shaw, D. S., Mitchell, C., Brooks-Gunn, J., McLanahan, S. S., Forbes, E. E., Monk, C. S., & Hyde, L. W. (2021). Beyond Family-Level Adversities: Exploring the Developmental Timing of Neighborhood Disadvantage Effects on the Brain. *Dev Sci, 24*(1), e12985. https://doi.org/10.1111/desc.12985

Gard, A. M., Waller, R., Shaw, D. S., Forbes, E. E., Hariri, A. R., & Hyde, L. W. (2017). The Long Reach of Early Adversity: Parenting, Stress, and Neural Pathways to Antisocial Behavior in Adulthood. *Biol Psychiatry Cogn Neurosci Neuroimaging, 2*(7), 582-590. https://doi.org/10.1016/j.bpsc.2017.06.005

Hackman, D. A., Cserbik, D., Chen, J. C., Berhane, K., Minaravesh, B., McConnell, R., & Herting, M. M. (2021). Association of Local Variation in Neighborhood Disadvantage in Metropolitan Areas with Youth Neurocognition and Brain Structure. *JAMA Pediatr, 175*(8), e210426. https://doi.org/10.1001/jamapediatrics.2021.0426

Hallquist, M. N., & Wiley, J. F. (2018). Mplusautomation: An R Package for Facilitating Large-Scale Latent Variable Analyses in Mplus. *Struct Equ Modeling, 25*(4), 621-638. https://doi.org/10.1080/10705511.2017.1402334

Heathcote, A. (2019). *Dynamic Models of Choice.* In https://osf.io/pbwx8/

Heathcote, A., Suraev, A., Curley, S., Gong, Q., Love, J., & Michie, P. T. (2015). Decision Processes and the Slowing of Simple Choices in Schizophrenia. *J Abnorm Psychol, 124*(4), 961-974. https://doi.org/10.1037/abn0000117

Huang-Pollock, C., Ratcliff, R., McKoon, G., Shapiro, Z., Weigard, A., & Galloway-Long, H. (2017). Using the Diffusion Model to Explain Cognitive Deficits in Attention Deficit Hyperactivity Disorder. *J Abnorm Child Psychol, 45*(1), 57-68. https://doi.org/10.1007/s10802-016-0151-y

Hughes, C., & Devine, R. T. (2019). For Better or for Worse? Positive and Negative Parental Influences on Young Children's Executive Function. *Child Dev, 90*(2), 593-609. https://doi.org/10.1111/cdev.12915

Hunt, J. F. V., Vogt, N. M., Jonaitis, E. M., Buckingham, W. R., Koscik, R. L., Zuelsdorff, M., Okonkwo, O. C., Clark, L. R., Johnson, S. C., Asthana, S., Bendlin, B. B., & Kind, A. J. (2020). Neighborhood Disadvantage Is Associated with Accelerated Cortical Thinning and Cognitive Decline in Cognitively Unimpaired Adults. *Alzheimer's & Dementia, 16*(S10). https://doi.org/10.1002/alz.043170

Iacono, W. G., Carlson, S. R., Taylor, J., Elkins, I. J., & McGue, M. (1999). Behavioral Disinhibition and the Development of Substance-Use Disorders: Findings from the Minnesota Twin Family Study. *Dev Psychopathol, 11*(4), 869-900. https://doi.org/10.1017/s0954579499002369

Johnson, D., Policelli, J., Li, M., Dharamsi, A., Hu, Q., Sheridan, M. A., McLaughlin, K. A., & Wade, M. (2021). Associations of Early-Life Threat and Deprivation with Executive Functioning in Childhood and Adolescence: A Systematic Review and Meta-Analysis. *JAMA Pediatr, 175*(11), e212511. https://doi.org/10.1001/jamapediatrics.2021.2511

Kassambara, A. (2020). *Ggpubr: 'Ggplot2' Based Publication Ready Plots.* In https://CRAN.R-project.org/package=ggpubr

Kolb, B., Mychasiuk, R., Muhammad, A., Li, Y., Frost, D. O., & Gibb, R. (2012). Experience and the Developing Prefrontal Cortex. *Proc Natl Acad Sci U S A, 109 Suppl 2*, 17186-17193. https://doi.org/10.1073/pnas.1121251109

Kubzansky, L. D., Martin, L. T., & Buka, S. L. (2009). Early Manifestations of Personality and Adult Health: A Life Course Perspective. *Health Psychol, 28*(3), 364-372. https://doi.org/10.1037/a0014428

Lawson, G. M., Hook, C. J., & Farah, M. J. (2018). A Meta-Analysis of the Relationship between Socioeconomic Status and Executive Function Performance among Children. *Dev Sci, 21*(2). https://doi.org/10.1111/desc.12529

Leventhal, T., & Brooks-Gunn, J. (2000). The Neighborhoods They Live In: The Effects of Neighborhood Residence on Child and Adolescent Outcomes. *Psychological Bulletin, 126*(2), 309-337. https://doi.org/10.1037/0033-2909.126.2.309

Li, J. B., Willems, Y. E., Stok, F. M., Dekovic, M., Bartels, M., & Finkenauer, C. (2019). Parenting and Self-Control across Early to Late Adolescence: A Three-Level Meta-Analysis. *Perspect Psychol Sci, 14*(6), 967-1005. https://doi.org/10.1177/1745691619863046

Maldjian, J. A., Laurienti, P. J., Kraft, R. A., & Burdette, J. H. (2003). An Automated Method for Neuroanatomic and Cytoarchitectonic Atlas-Based Interrogation of Fmri Data Sets. *Neuroimage, 19*(3), 1233-1239. https://www.ncbi.nlm.nih.gov/pubmed/12880848

McClelland, M. M., Cameron, C. E., Connor, C. M., Farris, C. L., Jewkes, A. M., & Morrison, F. J. (2007). Links between Behavioral Regulation and Preschoolers' Literacy, Vocabulary, and Math Skills. *Dev Psychol, 43*(4), 947-959. https://doi.org/10.1037/0012-1649.43.4.947

McEwen, B. S. (2004). Protection and Damage from Acute and Chronic Stress: Allostasis and Allostatic Overload and Relevance to the Pathophysiology of Psychiatric Disorders. *Ann N Y Acad Sci, 1032*, 1-7. https://doi.org/10.1196/annals.1314.001

Mischel, W., Ayduk, O., Berman, M. G., Casey, B. J., Gotlib, I. H., Jonides, J., Kross, E., Teslovich, T., Wilson, N. L., Zayas, V., & Shoda, Y. (2011). 'Willpower' over the Life Span: Decomposing Self-Regulation. *Soc Cogn Affect Neurosci, 6*(2), 252-256. https://doi.org/10.1093/scan/nsq081

Moffitt, T. E. (2018). Male Antisocial Behaviour in Adolescence and Beyond. *Nature Human Behaviour, 2*(3), 177-186. https://doi.org/10.1038/s41562-018-0309-4

Moffitt, T. E., Arseneault, L., Belsky, D., Dickson, N., Hancox, R. J., Harrington, H., Houts, R., Poulton, R., Roberts, B. W., Ross, S., Sears, M. R., Thomson, W. M., & Caspi, A. (2011). A Gradient of Childhood Self-Control Predicts Health, Wealth, and Public Safety. *Proc Natl Acad Sci U S A, 108*(7), 2693-2698. https://doi.org/10.1073/pnas.1010076108

Murtha, K., Larsen, B., Pines, A., Parkes, L., Moore, T. M., Adebimpe, A., Alexander-Bloch, A., Calkins, M. E., Davila, D. G., Lindquist, M. A., Mackey, A. P., Roalf, D. R., Scott, J. C., Wolf, D. H., Gur, R. C., Gur, R. E., Barzilay, R., & Satterthwaite, T. D. (2021). https://doi.org/10.1101/2021.08.12.454903

Muthén, L. K., & Muthén, B. O. (1998-2017). *Mplus User's Guide* (Eighth Edition ed.). Muthén & Muthén.

Peeters, H., Van Gestel, S., Vlietinck, R., Derom, C., & Derom, R. (1998). Validation of a Telephone Zygosity Questionnaire in Twins of Known Zygosity. *Behav Genet, 28*(3), 159-163. https://doi.org/10.1023/a:1021416112215

Power, T. G. (2013). Parenting Dimensions and Styles: A Brief History and Recommendations for Future Research. *Child Obes, 9 Suppl*, S14-21. https://doi.org/10.1089/chi.2013.0034

R Core Team. (2021). *R: A Language and Environment for Statistical Computing.* In R Foundation for Statistical Computing. https://www.R-project.org/

Rakesh, D., Cropley, V., Zalesky, A., Vijayakumar, N., Allen, N. B., & Whittle, S. (2021). Neighborhood Disadvantage and Longitudinal Brain-Predicted-Age Trajectory During Adolescence. *Dev Cogn Neurosci, 51*, 101002. https://doi.org/10.1016/j.dcn.2021.101002

Rakesh, D., Seguin, C., Zalesky, A., Cropley, V., & Whittle, S. (2021). Associations between Neighborhood Disadvantage, Resting-State Functional Connectivity, and Behavior in the Adolescent Brain Cognitive Development Study: The Moderating Role of Positive

Family and School Environments. *Biol Psychiatry Cogn Neurosci Neuroimaging*. https://doi.org/10.1016/j.bpsc.2021.03.008

Ramphal, B., DeSerisy, M., Pagliaccio, D., Raffanello, E., Rauh, V., Tau, G., Posner, J., Marsh, R., & Margolis, A. E. (2020). Associations between Amygdala-Prefrontal Functional Connectivity and Age Depend on Neighborhood Socioeconomic Status. *Cereb Cortex Commun, 1*(1), tgaa033. https://doi.org/10.1093/texcom/tgaa033

Ratcliff, R., Huang-Pollock, C., & McKoon, G. (2018). Modeling Individual Differences in the Go/No-Go Task with a Diffusion Model. *Decision (Wash D C), 5*(1), 42-62. https://doi.org/10.1037/dec0000065

Rochette, É., & Bernier, A. (2014). Parenting, Family Socioeconomic Status, and Child Executive Functioning: A Longitudinal Study. *Merrill-Palmer Quarterly, 60*(4). https://doi.org/10.13110/merrpalmquar1982.60.4.0431

Romund, L., Raufelder, D., Flemming, E., Lorenz, R. C., Pelz, P., Gleich, T., Heinz, A., & Beck, A. (2016). Maternal Parenting Behavior and Emotion Processing in Adolescents-an Fmri Study. *Biol Psychol, 120*, 120-125. https://doi.org/10.1016/j.biopsycho.2016.09.003

Rorden, C., & Brett, M. (2000). Stereotaxic Display of Brain Lesions. *Behav Neurol, 12*(4), 191-200. https://doi.org/10.1155/2000/421719

Saxbe, D., Khoddam, H., Piero, L. D., Stoycos, S. A., Gimbel, S. I., Margolin, G., & Kaplan, J. T. (2018). Community Violence Exposure in Early Adolescence: Longitudinal Associations with Hippocampal and Amygdala Volume and Resting State Connectivity. *Dev Sci, 21*(6), e12686. https://doi.org/10.1111/desc.12686

Seeyave, D. M., Coleman, S., Appugliese, D., Corwyn, R. F., Bradley, R. H., Davidson, N. S., Kaciroti, N., & Lumeng, J. C. (2009). Ability to Delay Gratification at Age 4 Years and Risk of Overweight at Age 11 Years. *Arch Pediatr Adolesc Med, 163*(4), 303-308. https://doi.org/10.1001/archpediatrics.2009.12

Shameem, F., & Hamid, S. (2014). Socioeconomic Status and Adolescent Aggression: The Role of Executive Functioning as a Mediator. *Am J Psychol, 127*(4), 419-430. https://doi.org/10.5406/amerjpsyc.127.4.0419

Shaw, D. S., & Bell, R. Q. (1993). Developmental Theories of Parental Contributors to Antisocial Behavior. *J Abnorm Child Psychol, 21*(5), 493-518. https://doi.org/10.1007/BF00916316

Shelton, K. K., Frick, P. J., & Wootton, J. (1996). Assessment of Parenting Practices in Families of Elementary School-Age Children. *Journal of Clinical Child Psychology, 25*(3), 317-329. https://doi.org/10.1207/s15374424jccp2503_8

Song, J.-H., Miller, A. L., Leung, C. Y. Y., Lumeng, J. C., & Rosenblum, K. L. (2018). Positive Parenting Moderates the Association between Temperament and Self-Regulation in Low-

Income Toddlers. *Journal of Child and Family Studies, 27*(7), 2354-2364. https://doi.org/10.1007/s10826-018-1066-8

Taylor, R. L., Cooper, S. R., Jackson, J. J., & Barch, D. M. (2020). Assessment of Neighborhood Poverty, Cognitive Function, and Prefrontal and Hippocampal Volumes in Children. *JAMA Netw Open, 3*(11), e2023774. https://doi.org/10.1001/jamanetworkopen.2020.23774

Tomlinson, R. C., Burt, S. A., Waller, R., Jonides, J., Miller, A. L., Gearhardt, A. N., Peltier, S. J., Klump, K. L., Lumeng, J. C., & Hyde, L. W. (2020). Neighborhood Poverty Predicts Altered Neural and Behavioral Response Inhibition. *Neuroimage, 209*, 116536. https://doi.org/10.1016/j.neuroimage.2020.116536

Turner, B. M., Sederberg, P. B., Brown, S. D., & Steyvers, M. (2013). A Method for Efficiently Sampling from Distributions with Correlated Dimensions. *Psychol Methods, 18*(3), 368-384. https://doi.org/10.1037/a0032222

Umbach, R., Raine, A., Gur, R. C., & Portnoy, J. (2017). Neighborhood Disadvantage and Neuropsychological Functioning as Part Mediators of the Race–Antisocial Relationship: A Serial Mediation Model. *Journal of Quantitative Criminology, 34*(2), 481-512. https://doi.org/10.1007/s10940-017-9343-z

Ursache, A., Noble, K. G., & Blair, C. (2015). Socioeconomic Status, Subjective Social Status, and Perceived Stress: Associations with Stress Physiology and Executive Functioning. *Behav Med, 41*(3), 145-154. https://doi.org/10.1080/08964289.2015.1024604

Valcan, D. S., Davis, H., & Pino-Pasternak, D. (2017). Parental Behaviours Predicting Early Childhood Executive Functions: A Meta-Analysis. *Educational Psychology Review, 30*(3), 607-649. https://doi.org/10.1007/s10648-017-9411-9

Webb, E. K., Weis, C. N., Huggins, A. A., Parisi, E. A., Bennett, K. P., Miskovich, T., Krukowski, J., deRoon-Cassini, T. A., & Larson, C. L. (2021). Neighborhood Disadvantage Is Associated with Stable Deficits in Neurocognitive Functioning in Traumatically-Injured Adults. *Health Place, 67*, 102493. https://doi.org/10.1016/j.healthplace.2020.102493

Weigard, A., & Sripada, C. (2021). Task-General Efficiency of Evidence Accumulation as a Computationally Defined Neurocognitive Trait: Implications for Clinical Neuroscience. *Biological Psychiatry Global Open Science, 1*(1), 5-15. https://doi.org/10.1016/j.bpsgos.2021.02.001

Weigard, A. S., Sathian, K., & Hampstead, B. M. (2020). Model-Based Assessment and Neural Correlates of Spatial Memory Deficits in Mild Cognitive Impairment. *Neuropsychologia, 136*, 107251. https://doi.org/10.1016/j.neuropsychologia.2019.107251

Wellcome Centre for Human Neuroimaging. *Spm12*. https://www.fil.ion.ucl.ac.uk/spm/

White, S. F., Voss, J. L., Chiang, J. J., Wang, L., McLaughlin, K. A., & Miller, G. E. (2019). Exposure to Violence and Low Family Income Are Associated with Heightened Amygdala Responsiveness to Threat among Adolescents. *Dev Cogn Neurosci, 40*, 100709. https://doi.org/10.1016/j.dcn.2019.100709

Wickham, H., Averick, M., Bryan, J., Chang, W., McGowan, L., François, R., Grolemund, G., Hayes, A., Henry, L., Hester, J., Kuhn, M., Pedersen, T., Miller, E., Bache, S., Müller, K., Ooms, J., Robinson, D., Seidel, D., Spinu, V., Takahashi, K., Vaughan, D., Wilke, C., Woo, K., & Yutani, H. (2019). Welcome to the Tidyverse. *Journal of Open Source Software, 4*(43). https://doi.org/10.21105/joss.01686

Zelazo, P. D., & Carlson, S. M. (2012). Hot and Cool Executive Function in Childhood and Adolescence: Development and Plasticity. *Child Development Perspectives*, n/a-n/a. https://doi.org/10.1111/j.1750-8606.2012.00246.x

Table IV.1

Summary of data included in fMRI analyses

	Number Lost	Participants With Usable Data
Participated in First Visit		**708** (354 complete pairs)
Sample with Any Go/No-Go fMRI Data		**600**
• Incomplete MRI scan	16	
• Software malfunction	3	
• Declined MRI scan	27	
• Uncomfortable with scan	18	
• Dental (e.g. braces, retainer)	17	
• Metal in/on the body	13	
• Exceeded scanner size restrictions	5	
• Major medical/neurological disorder	9	
Total Lost	*108*	
Sample with Usable fMRI Data		**540**
• Frontal lobe coverage <90%	(40)	
• ART Outliers >5%	(7)	
• Behavioral data reflected poor attention to task	(13)	
Total Lost	*60*	

Note. Breakdown of available fMRI data based on lab standard fMRI quality checks.

Table IV.2

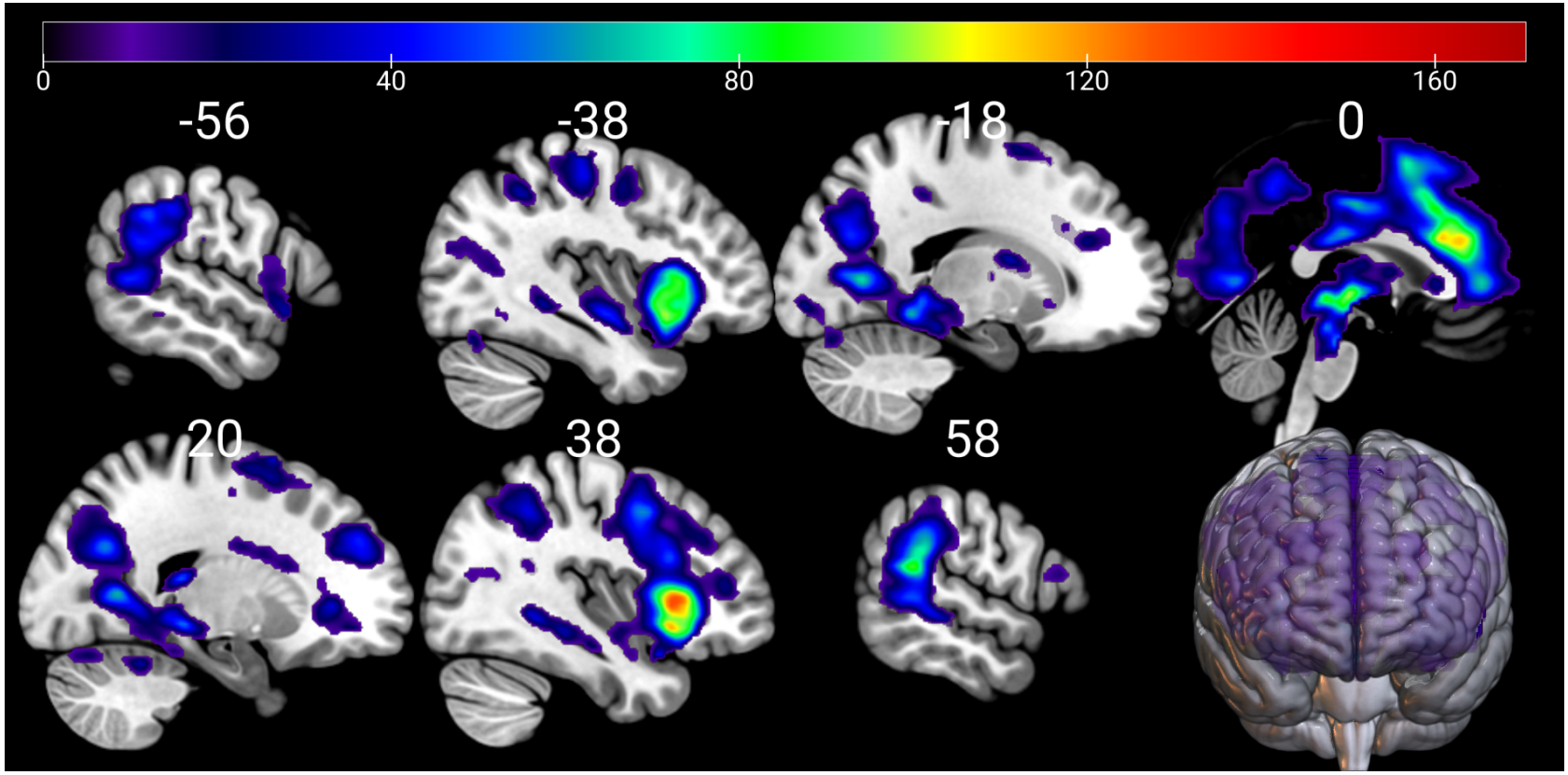
Means, standard deviations, and correlations with confidence intervals

Variable	*M*	*SD*	1	2	3	4	5	6
1. Neighborhood Poverty	0.18	0.16						
2. Income-to-Needs Ratio	2.29	0.94	-.30** [-.37, -.22]					
3. Maternal Education (Years)	15.02	2.05	-.17** [-.26, -.09]	.40** [.33, .47]				
4. Executive Functioning	0.02	0.68	-.21** [-.29, -.12]	.18** [.10, .26]	.16** [.08, .24]			
5. Nurturing Parenting	-0.02	0.63	.03 [-.06, .11]	.05 [-.03, .14]	-.01 [-.10, .07]	-.08 [-.16, .01]		
6. Harsh Parenting	-0.01	0.58	.02 [-.06, .11]	-.03 [-.12, .05]	-.03 [-.11, .06]	-.10* [-.19, -.02]	-.35** [-.43, -.28]	
7. IFG Activation	1.12	1.49	-.14** [-.22, -.06]	.03 [-.05, .12]	.03 [-.05, .12]	.13** [.05, .22]	-.01 [-.09, .08]	-.09* [-.17, -.00]

Note. M and *SD* are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014). * indicates $p < .05$. ** indicates $p < .01$.

Figure IV.1

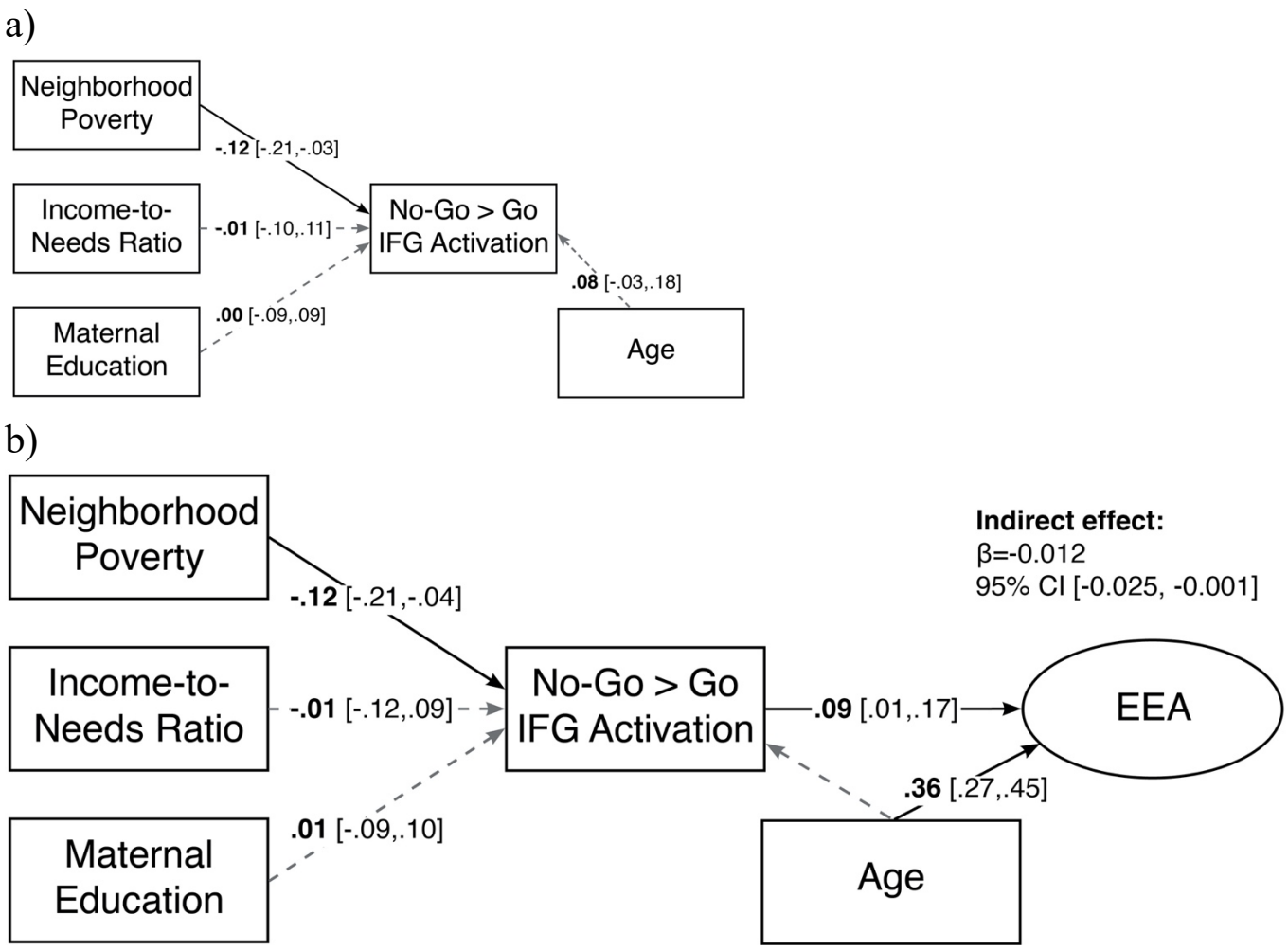
Brain activation associated with No-go Vs. Go F Contrast (N=540)



Note. This image depicts brain activation associated with a No-go vs. Go F contrast after whole-brain, cluster-level thresholding (α=0.05, p_{unc}<0.001, k=153). Sagittal slice locations are indicated in white above each slice. Image generated in MRIcroGL (Rorden & Brett, 2000) using a thresholded activation map from SPM12 (Wellcome Centre for Human Neuroimaging).

Figure IV.2

Neighborhood poverty is associated with brain and behavioral executive functioning (N=527)

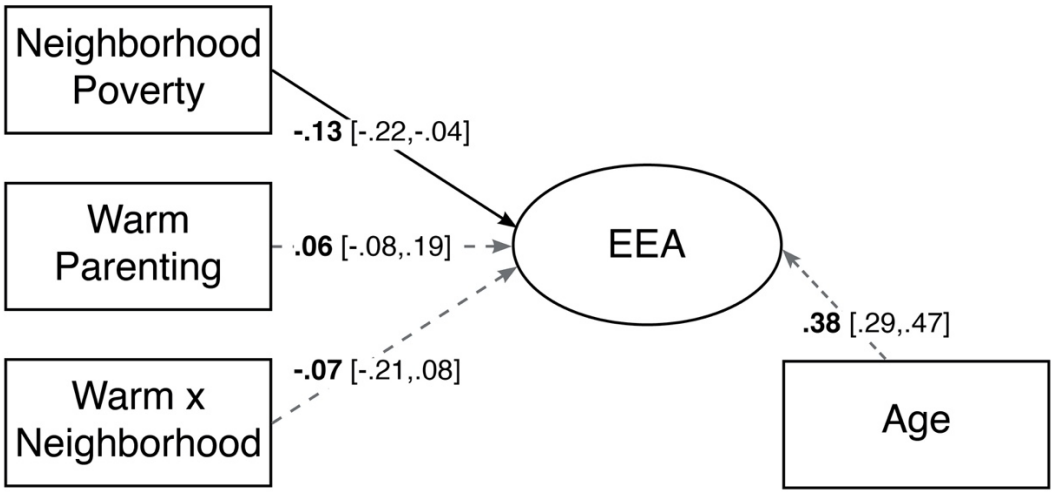


Note. This figure depicts structural equation models in which (a) the three socioeconomic context variables predict extracted inferior frontal gyrus activation, which (b) then predicts behavioral executive functioning. N=527. Neighborhood poverty predicts IFG activation even when accounting for income-to-needs ratio, maternal education, age, and relatedness within families. Standardized estimates are depicted in bold and 95% confidence intervals (bootstrap=1000) are depicted in brackets. Significant paths are indicated with solid lines and non-significant with dashed lines. In (b), direct paths from the context variables were also modeled, but they were not significant and are not pictured here for simplicity.

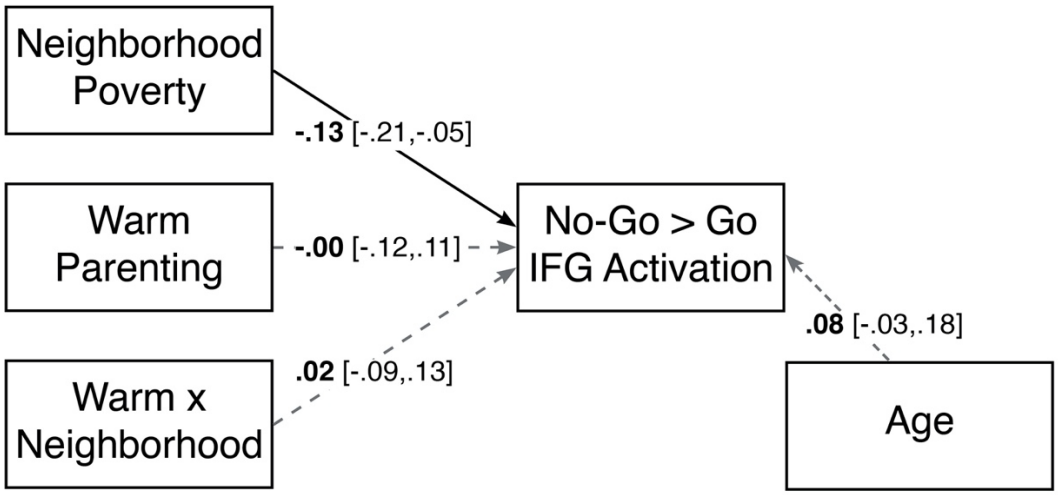
Figure IV.3

Parenting does not moderate neighborhood effects on brain or behavioral executive functioning

a)



b)

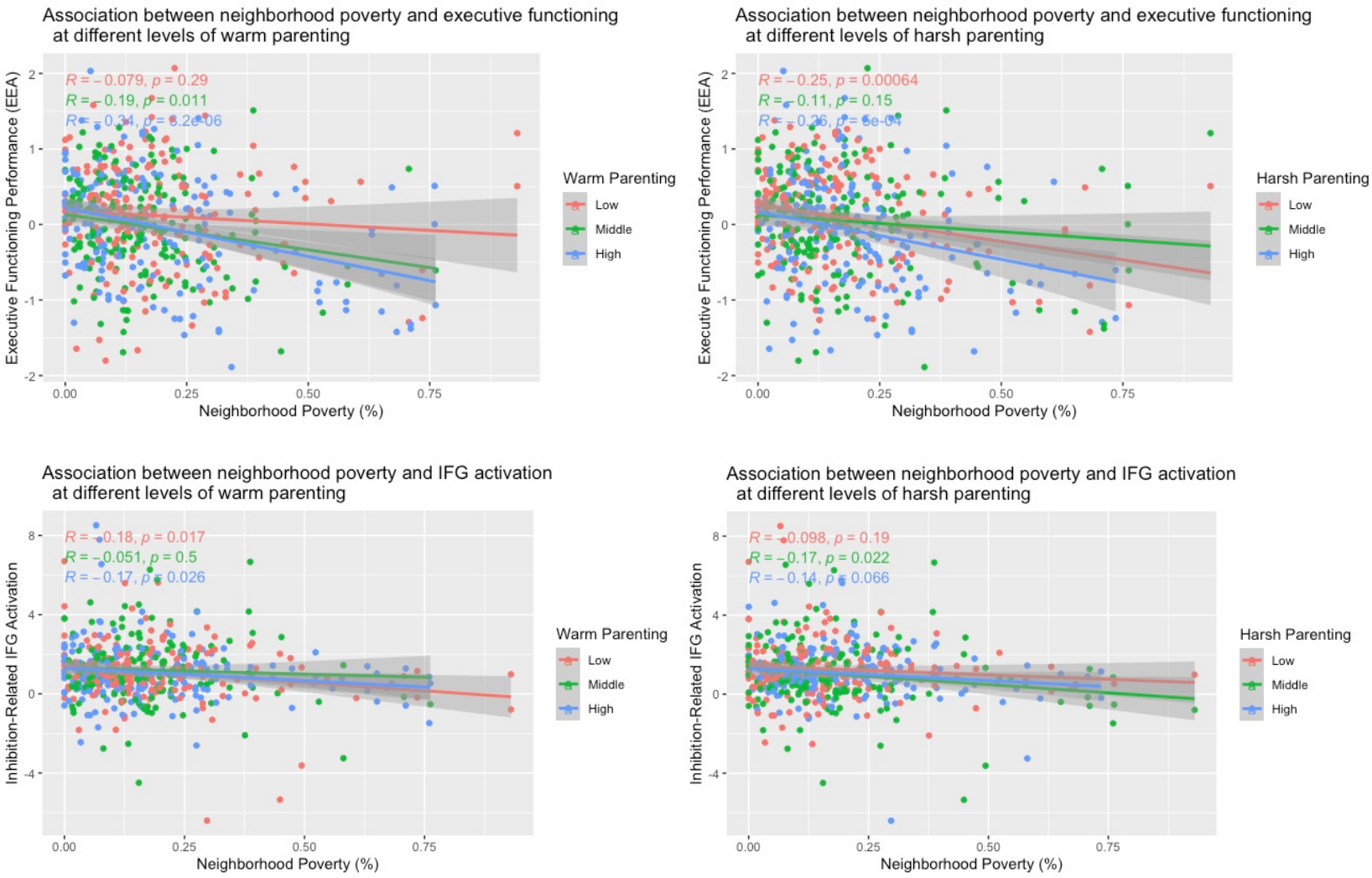


Note. This image depicts the structural equation models used to test whether parenting moderated neighborhood effects on brain or behavioral (EEA) measures of executive functioning (N=536). We used the cluster command in Mplus (Muthén & Muthén, 1998-2017) to account for relatedness within families. Standardized estimates are pictured in bold, with 95% confidence

intervals in brackets. We ran identical models for harsh parenting, yielding similar null results (for harsh x neighborhood on EEA , β=-0.15, 95% CI [-0.78, 0.48]; for harsh x neighborhood on IFG, β=0.01, 95% CI [-0.09, 0.11]).

Figure IV.4

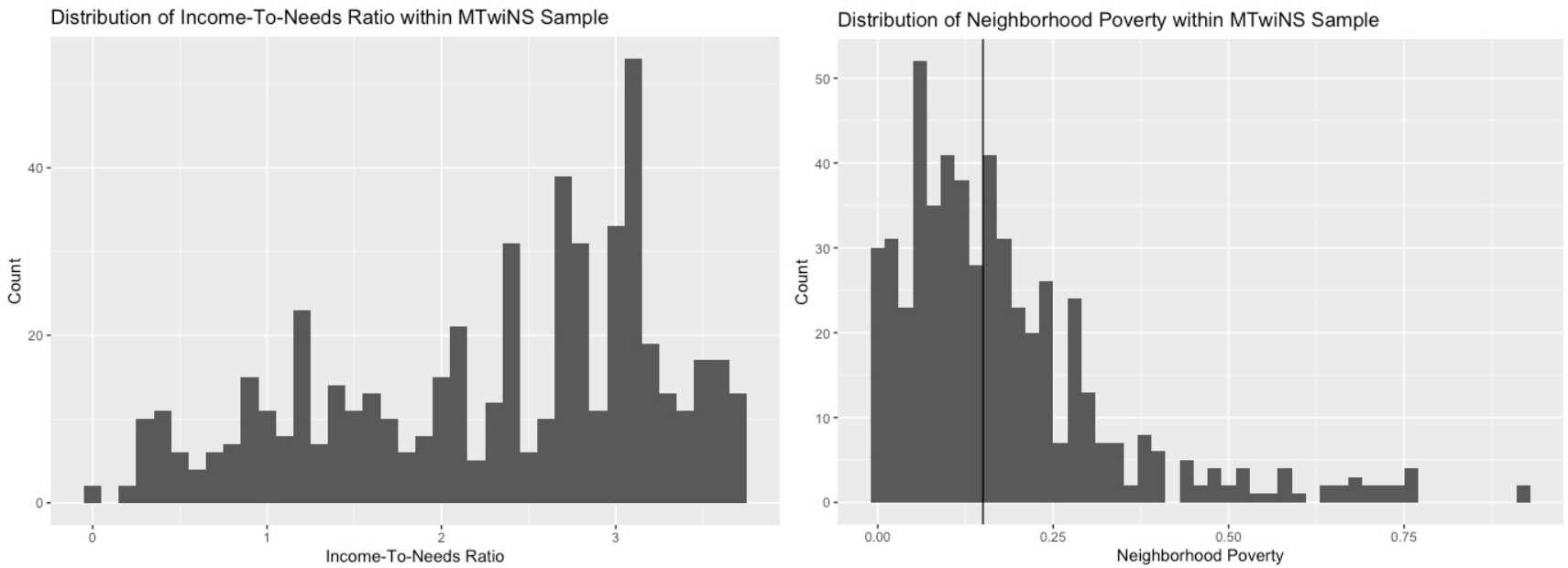
Parenting does not moderate the association between neighborhood poverty and brain or behavioral executive functioning



Note. This figure depicts scatterplots of the associations between neighborhood poverty and executive functioning performance (top) and between neighborhood poverty and extracted IFG activation (bottom) at the lowest, middle, and highest levels of nurturing and harsh parenting (N=180 per group). Contrary to hypotheses, there were no moderating effects of either nurturing or harsh parenting. Figures generated with the *tidyverse* and *ggpubr* packages in R (Kassambara, 2020; R Core Team, 2021; Wickham et al., 2019).

Figure IV.5

Distributions of Income-to-Needs Ratio and Neighborhood Poverty within MTwiNS Sample



Note. This figure depicts the distributions of income-to-needs ratio (left) and neighborhood poverty (right) in the MTwiNS sample. The mean neighborhood poverty level in the state of Michigan at the time of original recruitment was 10.5%, indicated here with a vertical line.

CHAPTER V

General Discussion

As discussed in the General Introduction (Chapter I), executive functioning abilities measured early in life are known to predict important outcomes later in life, including academic outcomes like literacy, vocabulary, and math skills (Blair & Razza, 2007; McClelland et al., 2007), learning (Marcovitch et al., 2008), and even physical health (Kubzansky et al., 2009; Mischel et al., 2011; Seeyave et al., 2009). Deficits in executive functioning abilities have also been linked to various forms of psychopathology (Bloemen et al., 2018; Castellanos et al., 2006; ADHD; Cherkasova et al., 2013; Gotlib & Joormann, 2010; Moffitt, 2018; Smith et al., 2014; Verdejo-Garcia et al., 2008; Wright et al., 2014). Unfortunately, a growing literature identifies a consistent relationship between socioeconomic disadvantage in childhood and worse executive functioning performance (Evans et al., 2021; Hackman et al., 2014; Last et al., 2018; Lawson et al., 2018; Raver et al., 2013; Rochette & Bernier, 2014; Shameem & Hamid, 2014). The mechanisms underlying this troubling relationship between disadvantage and executive functioning are not well understood. Thus, the present dissertation addressed several complications muddying our understanding of executive functioning and its relationship with disadvantage, including (1) how we measure executive functioning, (2) how we can disentangle non-genetic from genetic effects of parenting on executive functioning, and (3) how multiple levels of context may become biologically embedded to affect executive functioning.

Summary of Results

Study 1. In Study 1, we found encouraging evidence for a novel computational measure of task-general executive functioning, Efficiency of Evidence Accumulation (EEA), as a transdiagnostic risk factor associated with externalizing psychopathology in adolescents. EEA demonstrated reasonable test-retest reliability, and it was the only performance metric in Study 1 that related to both concurrent and prospective externalizing psychopathology when accounting for age, sex, and relatedness within families. Functional neuroimaging analyses also revealed that EEA had robust brain correlates, relating to inhibition-related brain activation in two relevant regions, the anterior cingulate cortex and the right superior temporal gyrus. Notably, however, several other traditional summary metrics correlated with brain activation in similar regions. These findings highlight that computational methods may provide some advantages over more traditional summary metrics of executive functioning performance in adolescents, while also revealing that some traditional metrics (i.e., Efficiency, Standard Deviation of Reaction Time) may provide a reasonable alternative when computational modeling is not possible.

Study 2. In Study 2, we employed a genetically informed design with multiple quantitative models to dig deeper into the role of parenting in the intergenerational transmission of executive functioning in adolescence. Like many others (see Valcan et al., 2017 for a meta-analysis), we found that harsh parenting was associated with worse child executive functioning, while nurturing parenting was associated with better child executive functioning. We found little evidence of passive or evocative genotype-environment correlation, and instead found that the relationship between harsh parenting and child executive functioning is due, at least in part, to non-shared environmental influences. We also found substantial shared environmental overlap between parenting and executive functioning. This overlap could reflect family-level environmental influences, or it could reflect other influences that do not vary between twins,

such as effects of parent personality or parent executive functioning on the parenting they provide (Klahr & Burt, 2014). These findings highlight that parenting may be one environmental mechanism through which executive functioning is passed across generations. More broadly, this work highlights that targeting harsh parenting through interventions is critical to improving adolescent executive functioning and preventing related psychopathology, and underscores the importance of genetically-informed designs when studying parenting influences on executive functioning.

Study 3. In Study 3, we found that neighborhood poverty was associated with executive functioning performance via inferior frontal gyrus activation during a go/no-go task, over and above effects of income-to-needs ratio and maternal education, extending the results of Tomlinson et al. (2020) to a larger sample. Contrary to hypotheses, we did not find evidence for nurturing, supportive parenting as a buffer, nor for harsh parenting as an activator of neighborhood risk. These findings again highlight that *where children live* is critical for executive functioning and related brain activity. However, the broad parenting environment in adolescence did not appear to promote nor undermine resilience to the effects of neighborhood poverty.

Implications

Taken together, the results of the three studies provide an interesting picture regarding the complex relationships between parenting, neighborhood, and brain and behavioral measures of executive functioning. Study 1 establishes a novel computational metric, EEA, as a useful measure of task-general executive functioning in children, with robust brain correlates. Studies 2 and 3 then investigate how executive functioning relates to parenting and the neighborhood, finding that parenting does relate to a broad measure of executive functioning, and that the

neighborhood context does relate to executive functioning via brain activation, but that parenting does not moderate neighborhood effects on executive functioning.

` Studies 1 and 3 are particularly interesting in combination. Both studies consider EEA alone as an outcome measure, and both investigate relationships between EEA and brain activation. Notably, in Study 1 we employed an exploratory, whole-brain approach to identify brain regions in which activation related to EEA scores, finding robust correlates in the anterior cingulate cortex and the superior temporal gyrus, but not the inferior frontal gyrus at the whole-brain level. On the other hand, in Study 3 we identified the inferior frontal gyrus as a region of interest a priori based on previous work which identified neighborhood poverty as a robust predictor of inferior frontal gyrus activation (Tomlinson et al., 2020). Contrary to the findings from the more stringent whole-brain approach in Study 1, we did find evidence in Study 3 that extracted activation in the inferior frontal gyrus correlated with EEA scores, and that neighborhood poverty predicted EEA scores via an effect on inferior frontal gyrus activation. Though these findings may seem contradictory, there are strong connections between the anterior cingulate cortex and the inferior frontal gyrus, and these two regions are widely considered to be major hubs of the salience network (Seeley et al., 2007). Thus, it is not surprising that the robust correlation seen in Study 1 between EEA and anterior cingulate cortex activation might be accompanied by a weaker, but present, correlation between EEA and inferior frontal gyrus activation. Taken together, the results of these two studies may reflect that neighborhood effects on task-general executive functioning are unlikely to be so simple as to be limited to effects on activation in one brain region. There may instead be a network-level effect, perhaps within the salience network. Future work could investigate the effects of neighborhood poverty on task-

related connectivity within the salience network, investigating the possibility of an anterior cingulate cortex – inferior frontal gyrus decoupling.

Studies 2 and 3 are also interesting in combination. Study 2 implicated parenting, and particularly harsh parenting, as an important environmental pathway linking parent executive functioning to child executive functioning. On the other hand, in Study 3 we did not find effects of nurturing or harsh parenting on brain measures of executive functioning, and did not find evidence for nurturing or harsh parenting as moderators of neighborhood effects on executive functioning. Though these seemingly conflicting findings are surprising on the surface, differences in study design may explain the discrepancy. For example, though both studies employed the same methods for calculating the broad parenting composites, their executive functioning measures differed. In Study 2 we tested main effects of parenting on a broad picture of child executive functioning, incorporating both EEA and self-report measures, which may relate better to real-world outcomes (Eisenberg et al., 2019). On the other hand, in Study 3 we considered main effects of parenting on EF-related brain activation. Thus, though we know from Study 2 and from extant literature that parenting matters for child executive functioning (see Valcan et al., 2017 for a meta-analysis), this effect may not be mediated by concurrent parenting effects on brain activation during inhibitory control in adolescence, as reflected by the null brain activation findings in Study 3. Indeed, it is important to note that at the zero-order level, harsh parenting did correlate with behavioral EEA (Study 3, Table 2). It is important to note that in Study 3 we measured task-related brain activation during an inhibitory control task. This is only one brain measure of countless possibilities. There could be a parenting effect on task-related activation for a different task, or on connectivity between brain regions, or on brain structure. Additionally, we measured parenting and brain activation concurrently, in adolescence. It is

entirely possible that the parenting environment in early childhood, when the neural architecture underlying executive functioning is growing rapidly (Kolb et al., 2012), may have main effects on EF-related brain structure and functioning concurrently in early childhood or lasting into adolescence. Future work could consider longitudinal effects of early childhood parenting on the brain, perhaps considering effects on connectivity or on the integrity of brain structures important for executive functioning.

Finally, though these three studies attempt to disentangle some of the major roadblocks to our understanding of neighborhood effects on executive functioning, they do not address *what about the neighborhood* is affecting children in this way. As discussed in Study 3, there are many neighborhood-related stressors that covary with neighborhood poverty. For example, neighborhood disadvantage is associated with exposure to additional adverse experiences such as neighborhood danger, under-resourced schools, lower home quality, poorer municipal services, and increased toxicant exposure (Evans, 2004; Leventhal & Brooks-Gunn, 2000). Recent meta-analytic evidence points to early-life deprivation (e.g. food insecurity, neglect) as a stronger predictor of executive functioning performance than early-life threat (e.g. exposure to violence in or out of the home; Johnson et al., 2021). Interestingly, emerging evidence also points to neighborhood deprivation as a stronger predictor of parenting behaviors than neighborhood danger (Burt et al., 2022). Taken together with the results from Study 2, in which harsh parenting affected child executive functioning separate from any genetic confounds, it seems possible that considering parenting as a *moderator* of neighborhood effects on executive functioning in Study 3 was misguided. Instead, a constellation of factors related to neighborhood deprivation, including differences in parenting behaviors, may directly affect the development of executive functioning, especially during early childhood. Consistent with the family stress model (Conger

et al., 2010; Morrison Gutman et al., 2005), lack of access to critical resources in an impoverished neighborhood context may stress parents, affecting parenting quality and subsequently affecting their young child's executive functioning development. Poorer preschool and grade school quality in these neighborhoods may further exacerbate these effects. Thus, future work may consider parenting as one *mediator* of the complex relationship between neighborhood stress and executive functioning.

Future directions

The results of these three studies represent advances in our understanding of the complex relationships between parenting, neighborhood, and executive functioning. However, they also raise many additional questions, highlighting important avenues for future research.

Considering age. The three studies comprising this dissertation all utilize data from the first visit of the MTwiNS study, with participants ranging in age from 7 to 19 years old (M=14.7). Studying this age group was incredibly informative for the purposes of this dissertation. For one, symptoms of externalizing psychopathology emerge during this time period (Cherkasova et al., 2013; Moffitt, 2018), which allowed us to confirm that our computationally-derived EEA measure did in fact relate to externalizing symptoms. Additionally, adolescents are capable of reporting on their own executive functioning and the parenting environment, adding rich self-report data to our executive functioning and parenting composites. However, the association between parenting and child executive functioning has been most widely studied in early childhood (see Valcan et al., 2017 for a meta-analysis), and for good reason. Early childhood is a time when children spend the most time with their parents (Shaw & Bell, 1993) and when executive functioning, and its underlying brain circuitry, are rapidly developing (Diamond, 2013; Kolb et al., 2012; Zelazo & Carlson, 2012). Thus, though the questions and models employed in

this dissertation add to our understanding of the complex relationships between neighborhood, parenting, and child executive functioning, much could be gained by exploring similar questions and methods in a younger sample. Future work could consider the associations between neighborhood poverty, parenting, and child executive functioning during early childhood. Additionally, future work could consider the effects of chronicity and timing of neighborhood poverty early in life on brain and behavioral measures of executive functioning in adolescence.

Considering racism. "Race" is a social construct, and one that comes with implications regarding where families live. The historical practice of "redlining" in the United States limited the flow of resources into communities labeled as "hazardous", limiting the housing and economic opportunities for families in those neighborhoods (Mitchell & Franco, 2018). This practice primarily affected neighborhoods in which minoritized families lived, and over 60% of those neighborhoods are still primarily inhabited by minoritized families today (Mitchell & Franco, 2018). Ongoing issues of structural racism further promote segregation and limit opportunities for minoritized families, with direct consequences for health (see Adkins-Jackson et al., 2022 for an overview). Thus, no research program investigating the effects of neighborhood poverty would be complete without considering the role of structural racism. Though the present dissertation does not address this issue, future work should consider how racism affects *who* is experiencing neighborhood poverty, and how racism might play a role in *how* families are affected by neighborhood poverty. Regarding the question of *who* is experiencing neighborhood poverty, socially-defined race is associated with neighborhood poverty, such that black and brown families are more likely to live in impoverished neighborhoods than white families (Mitchell & Franco, 2018; Osypuk & Acevedo-Garcia, 2010; Pastor, 2001). Future models might consider including socially defined race as a predictor of

neighborhood poverty to explicitly acknowledge and include this structural issue. Regarding *how* neighborhood poverty is experienced, it is likely that minoritized families have different experiences within their neighborhoods than their white neighbors. For example, a black family might be more likely to be directly affected by police brutality (Alang et al., 2017), or at the least to be more keenly aware of, and stressed by, over-policing in impoverished neighborhoods. Thus, future work regarding the mechanisms underlying the effects of neighborhood poverty should consider that some neighborhood-related stressors may disproportionately affect black and brown families.

Expanding "task-general". The present dissertation considered a novel method for calculating "task-general" executive functioning via computational modeling. The EEA metric employed in the three studies is intended to capture a biologically-relevant process that underlies performance on any forced-choice task (Weigard & Sripada, 2021). Such an approach provides an interesting contrast to extant work on executive functioning, which has largely focused on splitting up higher-order functions into their component parts, with limited success (Karr et al., 2018; Lee et al., 2013; McKenna et al., 2017; Packwood et al., 2011; Weigard & Sripada, 2021). However, for the present dissertation there were only two tasks available for modeling, and both tasks were designed to measure "inhibition", one of several proposed subcomponents of executive functioning (Miyake et al., 2000). Future work should apply this modeling approach across a much wider selection of tasks to both improve the latent factor score and to expand the generalizability of findings.

Conclusions

The associations between neighborhood poverty, parenting, and child executive functioning are complex and not well understood. The present dissertation employed a variety of

methods and modeling approaches to shed some light on the way we measure task-general executive functioning, non-genetic versus genetic effects of parenting on executive functioning, and the biological embedding of context to affect executive functioning. We found that a novel computational method for measuring executive functioning was associated with concurrent and prospective symptoms of externalizing psychopathology, indicating promise for this biologically-relevant metric. We also found evidence of a unique, non-genetic effect of harsh parenting on executive functioning. On the other hand, we did not find evidence of a parenting effect on EF-related brain activation, or of parenting promoting nor undermining resilience to neighborhood poverty. Future work is needed that takes a similar multi-method approach in an early childhood sample, with additional consideration regarding what aspects of the neighborhood may contribute to parenting stress.

REFERENCES

Adkins-Jackson, P. B., Chantarat, T., Bailey, Z. D., & Ponce, N. A. (2022). Measuring Structural Racism: A Guide for Epidemiologists and Other Health Researchers. *Am J Epidemiol, 191*(4), 539-547. https://doi.org/10.1093/aje/kwab239

Alang, S., McAlpine, D., McCreedy, E., & Hardeman, R. (2017). Police Brutality and Black Health: Setting the Agenda for Public Health Scholars. *Am J Public Health, 107*(5), 662-665. https://doi.org/10.2105/AJPH.2017.303691

Blair, C., & Razza, R. P. (2007). Relating Effortful Control, Executive Function, and False Belief Understanding to Emerging Math and Literacy Ability in Kindergarten. *Child Dev, 78*(2), 647-663. https://doi.org/10.1111/j.1467-8624.2007.01019.x

Bloemen, A. J. P., Oldehinkel, A. J., Laceulle, O. M., Ormel, J., Rommelse, N. N. J., & Hartman, C. A. (2018). The Association between Executive Functioning and Psychopathology: General or Specific? *Psychol Med, 48*(11), 1787-1794. https://doi.org/10.1017/S0033291717003269

Burt, S. A., Thaler, D., Shewark, E. A., Pearson, A. M., Anaya, C., Tomlinson, R. C., Neiderhiser, J. M., Klump, K. L., & Lonstein, J. S. (2022). Illuminating Associations between Parenting and Deleterious Neighborhood Characteristics Via an Exhaustive Modeling Approach. *Journal of Marriage and Family, In press*.

Castellanos, F. X., Sonuga-Barke, E. J., Milham, M. P., & Tannock, R. (2006). Characterizing Cognition in Adhd: Beyond Executive Dysfunction. *Trends Cogn Sci, 10*(3), 117-123. https://doi.org/10.1016/j.tics.2006.01.011

Cherkasova, M., Sulla, E. M., Dalena, K. L., Ponde, M. P., & Hechtman, L. (2013). Developmental Course of Attention Deficit Hyperactivity Disorder and Its Predictors. *J Can Acad Child Adolesc Psychiatry, 22*(1), 47-54. https://www.ncbi.nlm.nih.gov/pubmed/23390433

Conger, R. D., Conger, K. J., & Martin, M. J. (2010). Socioeconomic Status, Family Processes, and Individual Development. *J Marriage Fam, 72*(3), 685-704. https://doi.org/10.1111/j.1741-3737.2010.00725.x

Diamond, A. (2013). Executive Functions. *Annu Rev Psychol, 64*, 135-168. https://doi.org/10.1146/annurev-psych-113011-143750

Eisenberg, I. W., Bissett, P. G., Zeynep Enkavi, A., Li, J., MacKinnon, D. P., Marsch, L. A., & Poldrack, R. A. (2019). Uncovering the Structure of Self-Regulation through Data-Driven Ontology Discovery. *Nat Commun, 10*(1), 2319. https://doi.org/10.1038/s41467-019-10301-1

Evans, G. W. (2004). The Environment of Childhood Poverty. *Am Psychol, 59*(2), 77-92. https://doi.org/10.1037/0003-066X.59.2.77

Evans, G. W., Farah, M. J., & Hackman, D. A. (2021). Early Childhood Poverty and Adult Executive Functioning: Distinct, Mediating Pathways for Different Domains of Executive Functioning. *Dev Sci*, e13084. https://doi.org/10.1111/desc.13084

Gotlib, I. H., & Joormann, J. (2010). Cognition and Depression: Current Status and Future Directions. *Annu Rev Clin Psychol, 6*, 285-312. https://doi.org/10.1146/annurev.clinpsy.121208.131305

Hackman, D. A., Betancourt, L. M., Gallop, R., Romer, D., Brodsky, N. L., Hurt, H., & Farah, M. J. (2014). Mapping the Trajectory of Socioeconomic Disparity in Working Memory: Parental and Neighborhood Factors. *Child Dev, 85*(4), 1433-1445. https://doi.org/10.1111/cdev.12242

Johnson, D., Policelli, J., Li, M., Dharamsi, A., Hu, Q., Sheridan, M. A., McLaughlin, K. A., & Wade, M. (2021). Associations of Early-Life Threat and Deprivation with Executive Functioning in Childhood and Adolescence: A Systematic Review and Meta-Analysis. *JAMA Pediatr, 175*(11), e212511. https://doi.org/10.1001/jamapediatrics.2021.2511

Karr, J. E., Areshenkoff, C. N., Rast, P., Hofer, S. M., Iverson, G. L., & Garcia-Barrera, M. A. (2018). The Unity and Diversity of Executive Functions: A Systematic Review and Re-Analysis of Latent Variable Studies. *Psychol Bull, 144*(11), 1147-1185. https://doi.org/10.1037/bul0000160

Kolb, B., Mychasiuk, R., Muhammad, A., Li, Y., Frost, D. O., & Gibb, R. (2012). Experience and the Developing Prefrontal Cortex. *Proc Natl Acad Sci U S A, 109 Suppl 2*, 17186-17193. https://doi.org/10.1073/pnas.1121251109

Kubzansky, L. D., Martin, L. T., & Buka, S. L. (2009). Early Manifestations of Personality and Adult Health: A Life Course Perspective. *Health Psychol, 28*(3), 364-372. https://doi.org/10.1037/a0014428

Last, B. S., Lawson, G. M., Breiner, K., Steinberg, L., & Farah, M. J. (2018). Childhood Socioeconomic Status and Executive Function in Childhood and Beyond. *PLoS One, 13*(8), e0202964. https://doi.org/10.1371/journal.pone.0202964

Lawson, G. M., Hook, C. J., & Farah, M. J. (2018). A Meta-Analysis of the Relationship between Socioeconomic Status and Executive Function Performance among Children. *Dev Sci, 21*(2). https://doi.org/10.1111/desc.12529

Lee, K., Bull, R., & Ho, R. M. (2013). Developmental Changes in Executive Functioning. *Child Dev, 84*(6), 1933-1953. https://doi.org/10.1111/cdev.12096

Leventhal, T., & Brooks-Gunn, J. (2000). The Neighborhoods They Live In: The Effects of Neighborhood Residence on Child and Adolescent Outcomes. *Psychological Bulletin, 126*(2), 309-337. https://doi.org/10.1037/0033-2909.126.2.309

Marcovitch, S., Jacques, S., Boseovski, J. J., & Zelazo, P. D. (2008). Self-Reflection and the Cognitive Control of Behavior: Implications for Learning. *Mind, Brain, and Education, 2*(3), 136-141. https://doi.org/10.1111/j.1751-228X.2008.00044.x

McClelland, M. M., Cameron, C. E., Connor, C. M., Farris, C. L., Jewkes, A. M., & Morrison, F. J. (2007). Links between Behavioral Regulation and Preschoolers' Literacy, Vocabulary, and Math Skills. *Dev Psychol, 43*(4), 947-959. https://doi.org/10.1037/0012-1649.43.4.947

McKenna, R., Rushe, T., & Woodcock, K. A. (2017). Informing the Structure of Executive Function in Children: A Meta-Analysis of Functional Neuroimaging Data. *Front Hum Neurosci, 11*, 154. https://doi.org/10.3389/fnhum.2017.00154

Mischel, W., Ayduk, O., Berman, M. G., Casey, B. J., Gotlib, I. H., Jonides, J., Kross, E., Teslovich, T., Wilson, N. L., Zayas, V., & Shoda, Y. (2011). 'Willpower' over the Life Span: Decomposing Self-Regulation. *Soc Cogn Affect Neurosci, 6*(2), 252-256. https://doi.org/10.1093/scan/nsq081

Mitchell, B., & Franco, J. (2018). *Holc "Redlining" Maps: The Persistent Structure of Segregation and Economic Inequality*. http://arks.princeton.edu/ark:/88435/dsp01dj52w776n

Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The Unity and Diversity of Executive Functions and Their Contributions to Complex "Frontal Lobe" Tasks: A Latent Variable Analysis. *Cogn Psychol, 41*(1), 49-100. https://doi.org/10.1006/cogp.1999.0734

Moffitt, T. E. (2018). Male Antisocial Behaviour in Adolescence and Beyond. *Nature Human Behaviour, 2*(3), 177-186. https://doi.org/10.1038/s41562-018-0309-4

Morrison Gutman, L., McLoyd, V. C., & Tokoyawa, T. (2005). Financial Strain, Neighborhood Stress, Parenting Behaviors, and Adolescent Adjustment in Urban African American Families. *Journal of Research on Adolescence, 15*(4), 425-449. https://doi.org/10.1111/j.1532-7795.2005.00106.x

Osypuk, T. L., & Acevedo-Garcia, D. (2010). Beyond Individual Neighborhoods: A Geography of Opportunity Perspective for Understanding Racial/Ethnic Health Disparities. *Health Place, 16*(6), 1113-1123. https://doi.org/10.1016/j.healthplace.2010.07.002

Packwood, S., Hodgetts, H. M., & Tremblay, S. (2011). A Multiperspective Approach to the Conceptualization of Executive Functions. *J Clin Exp Neuropsychol, 33*(4), 456-470. https://doi.org/10.1080/13803395.2010.533157

Pastor, M. (2001). Geography and Opportunity. In *America Becoming: Racial Trrends and Their Consequences* (Vol. 1, pp. 435-468).

Raver, C. C., Blair, C., & Willoughby, M. (2013). Poverty as a Predictor of 4-Year-Olds' Executive Function: New Perspectives on Models of Differential Susceptibility. *Dev Psychol, 49*(2), 292-304. https://doi.org/10.1037/a0028343

Rochette, É., & Bernier, A. (2014). Parenting, Family Socioeconomic Status, and Child Executive Functioning: A Longitudinal Study. *Merrill-Palmer Quarterly, 60*(4). https://doi.org/10.13110/merrpalmquar1982.60.4.0431

Seeley, W. W., Menon, V., Schatzberg, A. F., Keller, J., Glover, G. H., Kenna, H., Reiss, A. L., & Greicius, M. D. (2007). Dissociable Intrinsic Connectivity Networks for Salience Processing and Executive Control. *J Neurosci, 27*(9), 2349-2356. https://doi.org/10.1523/JNEUROSCI.5587-06.2007

Seeyave, D. M., Coleman, S., Appugliese, D., Corwyn, R. F., Bradley, R. H., Davidson, N. S., Kaciroti, N., & Lumeng, J. C. (2009). Ability to Delay Gratification at Age 4 Years and Risk of Overweight at Age 11 Years. *Arch Pediatr Adolesc Med, 163*(4), 303-308. https://doi.org/10.1001/archpediatrics.2009.12

Shameem, F., & Hamid, S. (2014). Socioeconomic Status and Adolescent Aggression: The Role of Executive Functioning as a Mediator. *Am J Psychol, 127*(4), 419-430. https://doi.org/10.5406/amerjpsyc.127.4.0419

Shaw, D. S., & Bell, R. Q. (1993). Developmental Theories of Parental Contributors to Antisocial Behavior. *J Abnorm Child Psychol, 21*(5), 493-518. https://doi.org/10.1007/BF00916316

Smith, J. L., Mattick, R. P., Jamadar, S. D., & Iredale, J. M. (2014). Deficits in Behavioural Inhibition in Substance Abuse and Addiction: A Meta-Analysis. *Drug Alcohol Depend, 145*, 1-33. https://doi.org/10.1016/j.drugalcdep.2014.08.009

Tomlinson, R. C., Burt, S. A., Waller, R., Jonides, J., Miller, A. L., Gearhardt, A. N., Peltier, S. J., Klump, K. L., Lumeng, J. C., & Hyde, L. W. (2020). Neighborhood Poverty Predicts Altered Neural and Behavioral Response Inhibition. *Neuroimage, 209*, 116536. https://doi.org/10.1016/j.neuroimage.2020.116536

Valcan, D. S., Davis, H., & Pino-Pasternak, D. (2017). Parental Behaviours Predicting Early Childhood Executive Functions: A Meta-Analysis. *Educational Psychology Review, 30*(3), 607-649. https://doi.org/10.1007/s10648-017-9411-9

Verdejo-Garcia, A., Lawrence, A. J., & Clark, L. (2008). Impulsivity as a Vulnerability Marker for Substance-Use Disorders: Review of Findings from High-Risk Research, Problem Gamblers and Genetic Association Studies. *Neurosci Biobehav Rev, 32*(4), 777-810. https://doi.org/10.1016/j.neubiorev.2007.11.003

Weigard, A., & Sripada, C. (2021). Task-General Efficiency of Evidence Accumulation as a Computationally Defined Neurocognitive Trait: Implications for Clinical Neuroscience. *Biological Psychiatry Global Open Science, 1*(1), 5-15. https://doi.org/10.1016/j.bpsgos.2021.02.001

Wright, L., Lipszyc, J., Dupuis, A., Thayapararajah, S. W., & Schachar, R. (2014). Response Inhibition and Psychopathology: A Meta-Analysis of Go/No-Go Task Performance. *J Abnorm Psychol, 123*(2), 429-439. https://doi.org/10.1037/a0036295

Zelazo, P. D., & Carlson, S. M. (2012). Hot and Cool Executive Function in Childhood and Adolescence: Development and Plasticity. *Child Development Perspectives*, n/a-n/a. https://doi.org/10.1111/j.1750-8606.2012.00246.x