

ESTIMATING THE MAGNITUDE OF GENE-ENVIRONMENT CORRELATION FOR
COGNITIVE ABILITY, PERSONALITY, PSYCHOPATHOLOGY, AND HEIGHT

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DISSERTATION

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ABSTRACT

Decades of quantitative genetics research have concluded that all human psychological differences are conditioned on genetic effects. However, most of the research has focused on the independent contribution of genetic and environmental effects on trait development. Much less is known about how genetic and environmental influence are intertwined to guide development, though process like genetic-environment correlation (rGE) has been identified as a central driver of development in theoretical models. It is imperative to understand how and to what extent do people passively receive, evoke, and select environmental experiences as a systematic function of their traits that are genetically conditioned. Studies on quantifying rGE are needed to better understand the association between environmental exposures and psychological, behavioral, and socio outcomes.

The current project seeks to investigate the role of genetic-environment correlation by using a range of phenotypes with multiple genetically informed designs. I used data drawn from the Adolescent Brain Cognition Development (ABCD) project, to explore the widespread covariation of measured environmental exposure and trait-specific genetic influences using between individual (singletons) and family-based designs (Chapter 6); and to investigate the effect of holistic passive, active/evocative genetic-environment correlation (rGE) across various trait domains by incorporating measured genetic effects (i.e., polygenic index) into twin models (Chapter 7). The results showed that i) trait specific PGI is associated with a wide range of putative environmental exposures, suggesting environmental mediation of polygenic prediction; 2); there is no evidence for passive, evocative, nor active rGE for cognitive and height; and 3) active/evocative rGE is accounting for large proportion of heritability for personality traits.

Leveraging a broad range of phenotypes, specific environmental exposures, and PGIs, this thesis provides a holistic perspective on the extent to which genetic and environmental influence entangled to influence characteristic development. It also illustrates the usefulness of incorporating PGI in research and detecting aggregated rGE using extended twin models. General implications, limitations, and future directions are discussed (Chapter 8).

To my family and friends, for their love and support.

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CHAPTER 1: BACKGROUND

1.1 OVERVIEW

All human psychological differences are conditioned by one's genes (Dobzhansky, 1962; Harden, 2021; Turkheimer, 2000). Behavioral genetic models quantify the extent to which genetic and environmental variance in the population is associated with variance in a phenotype (i.e., the observable state of an organism). More specifically, behavior genetic models estimate independent genetic and environmental variance components. Despite the obvious connection between biological organisms and their environment (Plomin et al., 1977), behavior genetic models have historically relied on the assumption of independent influences due to model identification requirements. Out of multiple competing gene-environment interplay processes (Briley et al., 2019), the current project will focus on gene-environment correlation (rGE). This process has been identified as a central driver of development in past theoretical, computational, and empirical models (e.g., Bakermans-Kranenburg & van IJzendoorn, 2015; Beam & Turkheimer, 2013; Dickens & Flynn, 2001; Scarr & McCartney, 1983). Gene-environment correlation occurs when partially genetically influenced characteristics are associated with environmental experiences. For example, more genetically related individuals tend to passively receive, evoke, or actively create more similar environmental experiences than less genetically related individuals (Kendler & Baker, 2007). Acknowledging that rGE exists and potentially has a causal impact on phenotype development complicates the standard interpretation of genetic influences in a behavior genetic model (Avinun, 2020; Gage et al., 2016; Harden, 2021). Rather than genetic influences being a purely “under-the-skin” phenomenon, “genetic” variance also includes causal environmental variance that is correlated with genotype.

Identifying the magnitude of rGE is critical for proper interpretation of behavior genetic models, which will become increasingly relevant as ethical arguments surrounding direct editing of the genome (CRISPR; Ledford, 2015) and other forms of control over reproduction (e.g., embryo selection; Karavani et al., 2019) are thrust into mainstream awareness. An appreciation of the dynamic transactions between partially genetically influenced traits and the environment is necessary for the lay public to make sense of behavior genetic results.

Given the importance of rGE , one might expect considerable empirical evidence concerning the magnitude of such effects on phenotype development. Considerable evidence exists that environments are non-randomly experienced (Kendler & Baker, 2007), but unfortunately, the propulsive nature of covariation among genes and environment has rarely been investigated systematically, partially due to limitations of data and modeling. A common problem is that environments with large effect sizes do not exist for most psychological phenotypes (Bleidorn et al., 2018). Searching for small correlations between specific environments and phenotypes is challenging due to the multiple testing problem and that it is often the psychological relevance of an experience, rather than simply the presence of an experience, that matters for psychological development (Roberts & Wood, 2006a).

Here, I use a state-of-the-art model (Dolan et al., 2021) of the classical twin design with polygenic scores to directly estimate the magnitude of rGE for four phenotypes (cognitive ability, sensation seeking, psychopathology, and height). In contrast to the traditional classical twin design, the model specification allows me to estimate rGE without violating any assumptions. Although I will be unable to identify any specific gene or environment associated with the target phenotypes, the model will consolidate all causal environmental effects into a single variance component. This feature allows me to estimate the aggregate correlation between genetic influences and the set of causal environmental effects. Using the Adolescent Brain and Cognitive Development dataset (Casey et al., 2018), I test the extent to which rGE plays a propulsive role in psychological development.

1.2 EVERYTHING IS HERITABLE

Heritability is commonly defined as the proportion of phenotypic variance associated with genetic variance (Visscher et al., 2008). Heritability estimates rely on comparing the phenotypic similarity of individuals with known levels of genetic similarity, such as monozygotic and dizygotic twins or traditionally unrelated individuals who have undergone genotyping. If monozygotic twins are more similar than dizygotic twins, or if individuals who happen to be 2% genetically similar are more phenotypically similar than those 1% similar, then that result implies some sort of genetic process at work. An exhaustive meta-analysis drawn from 3,000 articles across 50 years of twin and adoption studies of 15 million twin pairs found that all human traits are heritable and that effective environmental factors exist in all traits (Polderman et al., 2015). The constructs included in the meta-analysis were taken from across all forms of

human trait quantification, from height and weight to attitudes and beliefs to behaviors and abilities.

1.2.1 What heritability is and is not

Heritability is defined as the proportion of phenotypic variance accounted for by genetic variance. This statistical definition of heritability, in contrast to the lay or intuitive understanding of heritability, can be a barrier to interpreting the behavior genetic literature. A commonly held misconception states that the magnitude of heritability indicates the proportion of a phenotype that is inherited. For example, it is incorrect to say that because the heritability of personality is 40%, then it means that 40% of your traits are inherited from your parents and 60% comes from the environment. This is mismatching inheriting genes (which we do) with inheriting phenotypes (which we don't). It is also common to equate high heritability with genetic determination, that the phenotype is predestined and unchangeable through development and interventions (Jensen, 1969). Such misinterpretations overlook the fact that environmental factors—an influential factor in phenotypic variation—can and do change. For example, although cognitive ability is highly heritable, there were consistent cognitive ability gains across time (about 1.33 SD gained in a generation), reflecting changes in more nourishing environments resulting from societal, economic, and cultural development (Dickens & Flynn, 2001).

Another misconception is that genetic influences are static causal factors in development. Shifts in the sociocultural environment could alter the expression of genetic factors (Briley et al., 2015; Heath et al., 1985; Rimfeld et al., 2018). Heritability estimates are reflective of a certain context and population, and if relevant factors in the environment change, then the genetic architecture could also change. For example, a child may display genetically influenced tendencies toward high activity levels. These behavioral tendencies may conform to the demands of the environment fine when the child is in a certain classroom, but may inhibit learning as the child moves to a more strict teacher. In this case, novel genetic influences would emerge for learning that were not previously present. The stability of genetic effects may be imperfect. Taken together, heritability is a population index that is specific to a particular population in a particular environment and time. It can also vary as a function of the way heritability and environment is measured and how they changed over time.

1.2.2 Moving from description to explanation

Everything is heritable, and heritability serves more purposes in describing the genetic and environmental contribution as opposed to explaining those attributions. As such, in the postgenomic era, the resolution of the nature-nurture debate turns into the question of how genes and environments act, transact, and interact. Researchers largely fall into two competing camps in arguments over whether the association among genetic and environmental effects are additive or multiplicative.

A widely held belief among behavior geneticists is that the additive model is sufficient (Visscher et al., 2017; Yang et al., 2015; Zhu et al., 2015). The additive model implies that the effects of genes and environments are independent, and that summing up the individually tiny effects of specific genetic variants (Chabris et al., 2015) will yield something close to the full heritability estimate. This is the incentive of getting a larger sample size to obtain a better estimation of the heritability attributable to common genetic variation (Maher, 2008). In addition to increasing the number of assessed participants, another option is to increase the number of assessed genetic variants. Using full genome sequencing, essentially the full heritability of height can be found, pointing to the potential importance of rare variants for development (Wainschtein et al., 2019).

In contrast to the simple additive model, developmentally-oriented researchers have tended more toward interactionism (for a historical review of this tension, see Griffiths & Tabery, 2008). Under this model, all observable behavior results from the interaction of genes and their environments. The interaction is not merely a statistical parameter to be estimated, but rather genes and environments interact in a necessary and irreducible way which renders the effort of partitioning variance useless (Gottlieb, 2003; Lerner, 1978). Historically, interactionism has presented a limited view on the developing mechanisms of human characteristics as it largely conflates the terms mechanical dependence and statistical dependence (Plomin et al., 1977, p. 309).

Here, I propose a solution somewhere in the middle: I will combine additive and multiplicative models and treat rGE as an instrumental, propulsive mechanism underlying development. Broadly, I acknowledge the fact that it is an oversimplification to neatly decompose phenotypic variance into genetic and environmental components, and I want to make progress on understanding the complexity by tackling rGE . With rGE incorporated in the development framework of psychological traits, understanding dynamic interplay between

genetic and environmental pathways across social contexts can stimulate scientific discovery, evaluate intervention robustness, and improve policy-making (Raffington et al., 2020).

CHAPTER 2: BIOMETRIC STUDIES ON PSYCHOLOGICAL TRAITS

2.1 ESTIMATING THE RELATIVE ROLES OF GENES AND ENVIRONMENTS

Several methods have been used in the literature to perform biometric decompositions of phenotypic variance. In this section, I describe the role that twin and family designs, as well as more recent molecular genetic methods, have played in understanding the relative contribution of genetic and environmental influences on psychological development.

2.1.1 Basics of family-design behavior genetic study

Family, adoption, and twin structure provide the quasi-experimental design to split the phenotypic variation into genetic and environmental components. One of the most productive genetically informative designs involves the classical twin design (Martin et al., 1978). This design allows for identification of genetic and environmental components because twins share either all (monozygotic, MZ) or half (dizygotic, DZ) of their segregating genetic material, and share some of their environmental components which make siblings similar, such as the rearing environment, school, neighborhood (referred to as shared environmental factors) across the pairs. They also experience some aspects of their environments differently from each other (non-shared environmental factors). By comparing the differences in twin pair correlations across zygosity, the phenotypic variance can be decomposed into genetic and environmental sources of variance. The mathematical equation is defined below (Falconer, 1960):

$$\begin{aligned}h^2 + c^2 + e^2 &= 1 \\h^2 + c^2 &= r_{MZ} \\1/2h^2 + c^2 &= r_{DZ}\end{aligned}$$

Where h^2 stands for heritability, c^2 stands for shared environmental effects, and e^2 as non-shared environment/experience effects. Put differently, heritability is indicated if the MZ twins are more similar to one another than that of DZ twins. Shared environmental components are indicated if individuals sharing the same environment are more similar to one another, such as the similarity among MZ vs. DZ twins are close in magnitude. Finally, non-shared environmental components, sometimes termed as unique experience, are indicated as anything that differs MZ twins to be perfectly matched with each other, measurement error included.

2.1.2 Operationalization of environmental effects

It is important to note that, the environmental effects (shared and nonshared) refer to environmental influences, stimuli, experiences, events, and situations that causally make siblings more similar or different from one another (Turkheimer et al., 2005; Turkheimer & Waldron, 2000). Shared and nonshared environmental components would not capture the environmental background that are shared by all members of the population (e.g., culture). These universal features would not induce *variance* in the phenotype, and therefore would not be detectable even if highly influential (e.g., nutrition in populations with adequate food resources).

Additionally, it is important to distinguish between objective and effective environments. The shared environment as estimated in the classical twin design refers to effective shared events, rather than the objective assessment of environments. Environmental events that are commonly measured as shared across the family (e.g., family socioeconomic background, neighborhood environment) may nonetheless be sources of nonshared environment variation if they cause variation in siblings' phenotypes. Proper understanding of the environment definition is crucial to interpret the null findings for shared environmental components in classical twin designs across a wide range of phenotypes (Plomin et al., 2016; Polderman et al., 2015).

A striking case of the effective nonshared environment comes from findings on arising individuality among genetically identical animals reared under identical environmental conditions. Freund et al (2013) placed genetically identical mice in the same environment and found significant individual differences in behavior emerged over time as a result of plasticity. Similarly, Bierbach et al (2017) found divergent behavior patterns emerged in clonal fish when placed in near-identical environmental condition. In those studies, even though the objective environments were perfectly shared among the genetically identical 'siblings' (i.e., socioeconomic status), these shared environments nonetheless have differing effects on each individual (i.e., perception on family resources). As a result, the finding that the shared environment tends to explain very little variance in psychological phenotypes can be partially reconciled with the fact that meaningful and impactful common experience turn out to have individual effects on people (i.e., resulting in nonshared environmental variance; Turkheimer & Waldron, 2000).

2.1.3 Scope of twin design is limited by model assumptions

The classical twin design has important strengths and limitations. It provides a framework to estimate the boundary of genetic and environmental contributions to the

phenotypic variances, and it provides a nice overview on the total causal effect of an environment which is otherwise hard to consolidate. The limitation of the model stems from several key assumptions the classical twin design operates on, which potentially limit the scope to the extent it is realistic to the phenotypes. The assumptions include (Keller et al., 2010; Purcell, 2002; Rijdsdijk & Sham, 2002): equal environments (twins who are more genetically similar do not experience more similar trait-relevant environment just due to their genetic similarity), zero or small gene and environment interplay (absent genotype-environment correlation and interaction), no assortative mating (parental pairing are not based on phenotypic assortment), and twins are no different from the general population trait-wise. When the assumptions are met, the classical twin design is a powerful tool to decompose the phenotypic variance into genetic and environmental components. One alternative approach to get a holistic view on the environmental influence is to conduct large scale studies to account for all possible environmental factors (Environment-wide association studies; Patel et al., 2010). Though this design shows a promising future, collecting comprehensive exposure data for individual-level data is time-consuming and labor-intensive.

For more in-depth review of behavior genetic studies on the selected phenotypes of the proposal (i.e., cognitive ability, sensation seeking, psychopathology, height), see Appendix A.

2.2 MOLECULAR GENETIC RESEARCH

In twin studies, heritability is estimated based on the phenotypic resemblance among genetically similar family members (e.g., MZ and DZ twins). With rapid technological and methodological advances in measurement of human DNA, we can now estimate heritability based on aggregated genetic effects directly associated with phenotypes (Visscher et al., 2017). In fact, the paradigm of these genetic designs took place before the period when genotyping was cheap and scalable. For two decades, researchers embraced a candidate-gene study design, focusing on a selected sample of genetic variants that allegedly possess large effects according to a prior reasoning on their significance. However, a typical human trait is highly polygenic, as such it is associated with many genetic variants each contributing very small effect sizes (Chabris et al., 2015). Indeed, consistent replication failures of candidate gene findings urge the development of alternative methods (Colhoun et al., 2003; L. E. Duncan et al., 2014). As the cost of genotyping (assessment of million genetic variants instead of just a handful few) plummeted, candidate-gene studies were replaced by genome-wide association studies (GWAS). A GWAS

uses an brutal-force approach and scans across the genome containing hundreds of thousand genetic variants (e.g., single nucleotide polymorphisms, SNPs) that commonly vary between humans for possible association with a phenotype (Visscher et al., 2006). With the GWAS results, we can estimate the phenotypic variance explained by all SNPs in conventionally unrelated individuals, or so-called SNP-based heritability

2.2.1 Missing heritability problem in molecular genetic studies

A challenge in interpreting the heritability based on GWAS discoveries is the ‘missing heritability’ problem (Maher, 2008), that the SNP heritability based on GWAS discoveries was only a fraction of the h^2 based on twin and family studies—assuming that twin studies yield accurate heritability estimates (Manolio et al., 2009). For example, height—a trait with twin heritability of around 80% (Dubois et al., 2012), early GWAS identified ~ 40 variants that collectively explained only 5% of the variation (Weedon et al., 2008). One reason for the estimation gap is the small effect size of each variant, and the issue of the multiple testing burden as due to the polygenicity of human phenotype (i.e., multiple SNPs are associated; Chabris et al., 2015). Increasing genotyped sample size is one straightforward solution. A GWAS requires at least 100,000 participants to achieve adequate power (Plomin & von Stumm, 2018), and with growing power to detect tiny effects on highly polygenic phenotypes, we can yield better estimate on the SNP heritability (Visscher et al., 2017). Another reason is that heritability might be due to genetic variants besides SNPs. GWAS relies on genotyping of common variants that is commercially available, and it would not model signals coming from alternative variants (e.g., rare variants, insertions, deletions, etc.) due to the data constraint (McCarthy et al., 2008). Tackling either one or both considerations are effective way to recover the missing heritability, and with the emergence of large-scale biobanks, online repositories, and international collaboration, increasing sample sizes soon become a tangible solution. As a result, using a sample of over 600,000 participants, Yengo et al (2018) showed that the aggregated SNPs effect accounted for over 50% of the height variation. For cognitive ability, current SNP heritability is about 25%, recovering half of the twin heritability (Plomin & von Stumm, 2018).

Surprisingly, despite the rapid development in molecular genetic studies, the genetic underpinning of psychopathology and personality faced challenges in identifying specific genetic variants that were associated with complex traits (Benke et al., 2014; Karlsson Linnér et al., 2019; Nagel et al., 2018). Average twin heritability estimates for personality is 40%, yet GWAS

studies failed to reach 10% SNP heritability using a variety of personality instruments (Demange et al., 2020; Karlsson Linnér et al., 2019; Nagel et al., 2018). Personality assessments show the largest gap between twin and molecular estimates of heritability (Cheesman et al., 2017). This substantial gap in personality assessments and alike could be owing to non-additive genetic influence, rare variants, and gene-environment interplay captured by twin design but not by SNPs. One potentially overlooked possibility is that the *direction* of rGE differs across phenotypes. Positive rGE magnifies SNP associations, whereas negative rGE would reduce SNP associations. It may be the case that positive rGE is more common for phenotypes that have been more successfully analyzed using GWAS. This distinction is described in more detail below.

2.2.2 Molecular genetic variables and social science study

Even for researchers not interested in biological pathways per se, genetic propensity nonetheless could remain as an interesting property. Polygenic index (PGI)¹ serves the purpose of providing an overall prediction of a target phenotype by evaluating the effects of all genetic variants simultaneously. Simply put, PGI is an individual-specific sum of trait-associated genetic variants, with the strength of weights being the effect size associated with each SNPs in the discovery GWAS (Dudbridge, 2013). Through aggregation of large amounts of genetic markers each accounting for a tiny effect, PGI has the advantage of achieving adequate predictive power as a single predictor, bypassing the enormous sample size requirement for GWAS (Plomin & von Stumm, 2018). For example, a researcher who is interested in a PGI that captures 1% of the phenotypic variance would only need 1,500 participants to achieve 90% statistical power at $\alpha=.05$ to detect the PGI-phenotype association. The PGI with strongest predictive validity (Lee et al., 2018) is based on the largest GWAS to date ($N \sim 1$ million), accounts for over 15% of the variance in school performance, rivals some of the most important established predictors (e.g., family SES). While PGI is by no mean a perfect method to summarize an individual's genetic predisposition to a phenotype, as it does not account for all plausible genetic effects and it does

¹ Following Beck and colleagues (2021)' recommendation, in this dissertation, I use the term 'polygenic index (PGI)' as opposed to alternative terms 'polygenic score (PGS)' and 'polygenic risk score (PRS)'. The PGI is less likely to convey information relevant to value judgment than the others. Such preference of using 'indices' over 'score' has been accepted widely in agriculture and animal breeding, where heritability is considered the estimated breeding index.

account for some environmental confounds (for a more detailed review, see chapter 3 or Harden, 2021; Kong et al., 2018), it nevertheless opens up new revenues to study human psychological traits. One particularly useful aspect is that a person's DNA sequence is fixed at conception, and the majority of GWAS summary statistics are freely available in the public domain. As a result, once genetic data is collected in a sample, we can carry out analyses on constructs not measured in the sample (which GWAS statistics are available) using the PGI as a proxy (Harden & Koellinger, 2020).

CHAPTER 3: CAUSAL REASONING AND RGE

3.1 BEYOND HERITABILITY

Estimating heritability using a model where the variance can be neatly decomposed into genetic and environmental components is a necessary first step in getting a crude metric on the genetic effects of behavior. As aforementioned, my goal is to go beyond the all-for-one heritability estimate and focus more on identification of causal pathways among genotypes, environment, and phenotypes, specifically the rGE .

3.1.1 Socialization and selection effect in behavior genetic lens

A million-dollar question to psychologists who are interested in individual differences is whether life events shape human psychological traits in an enduring way. For example, being more invested in work and doing homework is associated with increasing conscientiousness (Göllner et al., 2017; Hudson et al., 2012). This way of understanding the effect of a life event is supported by socialization perspectives on development: that the environment has a causal impact on human psychological traits (Bleidorn et al., 2018; Lüdtke et al., 2011; Roberts & Wood, 2006b). On the other hand, the experience of events is not random, as often times people select themselves or are selected into experiences that are compatible with their psychological traits, a notion grounded in the selection perspective of development (e.g., Buss, 1987; Headey & Wearing, 1989; Roberts & Wood, 2006b). Individuals with higher neuroticism tend to encounter more negative experiences (Borghuis et al., 2020) and more secure individuals tend to experience more positive interpersonal events (Collins, 1996). Moreover, as all psychological traits are heritable, this transaction process between individual characteristics and experiences give rise to rGE : the notion that individuals' genetic propensities correlate with their environmental experiences (Beam & Turkheimer, 2013; Briley et al., 2019; Kandler et al., 2012; Plomin et al., 1977; Scarr & McCartney, 1983). Indeed, behavior genetic research has shown consistent non-zero heritability findings for measures of life events, such as in stressful life events, divorce, socioeconomic status, and working environments (Kendler & Baker, 2007; Polderman et al., 2015). An example of this is that parental educational expectations (i.e., parent-report educational expectation of each child) are moderately heritable (~23%), suggesting a child's genetic differences are associated with the parenting they experience (Avinun & Knafo,

2014). Parents form their expectations of the child on the basis of their genetically associated characteristics. Taken together, people differentially receive, evoke, and select their surrounding environment on the basis of genetically associated traits, and those experiences may in turn affect those traits. What seem to be environmental effects can reflect genetic influence as these experiences are correlated with genetic differences (Gage et al., 2016; Plomin et al., 2008), and what seem to be genetic effects can also reflect environmental contribution as genetic influence require matching and supportive environment to amplify its effect (Bronfenbrenner & Ceci, 1994; Dickens & Flynn, 2001). Take volitional change of personality traits as an example: people who have volitional goal of changing traits select themselves in environments inconsistent with their genetically associated personality traits and actually change their traits (Hudson et al., 2019, 2020), forming a negative rGE that support the development of personality traits and increased exposure to further relevant environmental experiences.

3.1.2 Different rGE developmental processes

There are two primary types of gene-environment interplay: gene $\times$ environment interaction (G $\times$ E), genetic sensitivity or susceptibility to environments; and gene-environment correlations (rGE), the nonrandom exposure of experience that associated with genetically influenced phenotype (Plomin et al., 1977; Purcell, 2002). Though theoretical arguments suggest both processes are pervasive (Scarr & McCartney, 1983), rGE are supported by more replicable, confirmed examples from both twin/family studies and molecular studies. Taken together, the current proposal aimed to provide an overarching estimate of rGE .

rGE s can emerge through passive, evocative, and active mechanisms (Plomin et al., 1977; Scarr & McCartney, 1983). Passive rGE occurs when children inherit genes that correlate with the rearing environment, such as parents who are skilled in musical ability are likely to provide their children with music enriched environment as well as genes that play a role in developing music ability. There can also be negative passive rGE , such that parents who have extensive exposure to music may provide a diverse and especially enriched environment for a child who did not play music well. Evocative rGE occurs when individuals evoke different reactions from other people on the basis of their genetically influenced phenotypes. An example is being selected as a music club member at school for one's musical ability or potential. Finally, active rGE occurs when individuals actively create, or seek out environments correlated with their genetically influenced phenotypes, a child who enjoyed music trying out for a musical club

or chorus and create music-related experience at school. Across different developmental stages, it is unlikely those types of rGE are equally influential. One of the ideas is that these processes are differentially influential at different points in life, such that passive rGE plays a more salient role in early childhood, and as people develop and gain agency and autonomy during development, active rGE begins to play a larger role (Scarr & McCartney, 1983). As people grow older, they become more capable in selecting and shaping their environment so that it will be in accordance with their genetically influenced characteristics (Avinun, 2020; Briley & Tucker-Drob, 2013).

3.2 IMPLICATION OF GENE-ENVIRONMENT CORRELATION

The impact of rGE on parameter estimates obtained from twin models is well acknowledged (Keller et al., 2010; Purcell, 2002). When genes and shared environmental factors correlate (e.g., passive rGE), shared environmental variance will increase, as everyone reared in the same household will increase in similarity. When genes and non-shared environmental factors correlate (e.g., evocative and/or active rGE), heritability estimate will increase as a result of observed widened differences in resemblance across more genetically similar individuals versus less genetically similar individuals. Siblings with greater genetic resemblance will evoke and select similar non-shared environments which magnify psychological resemblance, and genetically less similar siblings may select dissimilar environments that magnify their dissimilarity. Many efforts are devoted to accommodating rGE in twin designs, such as via simulation (Beam & Turkheimer, 2013; Purcell, 2002), extended family design (e.g., by including parents-of-twins or children-of-twins; Keller et al., 2009; Maes et al., 1997), longitudinal design (Dolan et al., 2014a; Kandler et al., 2012). The findings are consistent across designs.

Moving to the molecular level design, the implication of rGE on SNP heritability differs from that of twin and family design (increased shared environment variance). Passive rGE will inflate SNP heritability as the SNP-phenotype association includes both direct and indirect genetic mechanisms. Put differently, parents generate both family environments consistent with their genetically associated traits and pass on trait-relevant genotypes to their offspring. As children inherit half of their alleles from parents, including those trait-relevant variants as well as those related to their parents' dispositions to provide a rearing environment, the association between unrelated individual's SNP and phenotype will increase. One way to tackle this issue is

by looking at the association between non-transmitted parental alleles (i.e., alleles that the offspring had not inherited) with the children's outcome (Bates et al., 2018; Kong et al., 2018). For example, Kong et al (2018) used a sample of over 20,000 individuals, and found that the non-transmitted educational attainment PGI uniquely accounted for 3% of the phenotypic variation. The fact that non-transmitted PGI could also predict children's development demonstrated the impact of rGE , is referred to as genetic nurture (Kong et al., 2018). Another approach to estimate rGE is through within-family analysis of siblings. Since siblings have equal probability to inherit any given allele, they are matched on the indirect genetic mechanisms related to transmitted alleles. As the siblings living in the households are most likely to match on their rearing environment, within-family design can also account for the passive rGE . Indeed, several within-family analyses have demonstrated substantial attenuation of the PGI's predictive power compared to between-family analyses. In the most recent GWAS on educational attainment including over 1.1 million individuals, Lee et al (2018) found that within-family analysis showed a substantial reduction in SNP effect sizes of ~40% in comparison to that from unrelated individuals. Moreover, Trejo and Domingue (2019) found that the predictive power of educational attainment PGI within a family was reduced ~50% compared to between-family association in predicting number of years of schooling. These results imply that a substantial portion of the predictive validity of the PGI derive from the process of gene-environment correlation.

Incorporating rGE in the framework of causal reasoning provides a higher level of granularity in understanding the mechanisms of social inequalities and the way that social privilege is transmitted across generations. Activation of genetic potential is conditional on access to enriched and supportive environments which would allow children to seek out and evoke change on the basis of genetically associated characteristics (Bronfenbrenner & Ceci, 1994; Scarr & McCartney, 1983). Under disadvantage of socio-, economical-, cultural-conditions, even children with high genetic propensities are afforded only minimal opportunities to evoke enriched environments. Indeed, Belsky et al (2018) found that children from low-SES families with high education-related PGI ended up worse off relative to children from high-SES families with low education PGI.

3.3 TRACING CAUSAL PATHWAY IN TRAITS

The possibility of gene-environment interplay complicates clear causal pathways between genotypes and phenotypes or between genetically influenced phenotypes and consequential life outcomes. In this section, I describe the ways that the sociocultural environment as an institutional guide on development may interface with gene-environment interplay.

3.3.1 Social institutions, cognitive development, and rGE

In order to understand the causal pathway linking genotypes to phenotypes in human psychological development, the following section expands the literature on studying the rGE at single points in time to a longitudinal prospective. Take the heritability estimate of cognitive ability as an example. One of the most replicated findings in all behavior genetics is that the heritability of cognitive ability increases with age (Briley & Tucker-Drob, 2013; Plomin et al., 2016). Systematic reviews on longitudinal twin and adoption studies found that the heritability for cognitive ability increases dramatically from 20% in early childhood to nearly 70% in late adolescence (Briley & Tucker-Drob, 2013; Haworth et al., 2010; Tucker-Drob & Briley, 2014). Moreover, the genetic stability approaches 1 at middle adolescence, suggesting that early genetically influenced individual differences are aggregated and amplified as people move through genetically correlated environments. Such persistence and amplification of the genetic effect starting from early age may be specific to cognitive ability, with institutional opportunities and constraints serving as gateway mechanisms to reinforce positive outcomes (Bailey et al., 2020). Under such a framework, the compulsory education system provides ample opportunities allowing students to actively and evocatively select processes that promote upward changes and restrict opportunities to engage in deleterious behaviors that would widen the achievement gap.

3.3.2 Genetic transaction for personality and psychopathology

In comparison to cognitive ability, the potential mechanisms of development are likely different for personality, psychopathology, and height. At the phenotypic level, there are many shared developmental features across cognitive ability and personality, such as consistent pattern of mean-level change (McArdle et al., 2002; Roberts et al., 2006) and increasing rank-order stability (Roberts & DelVecchio, 2000; Sameroff et al., 1993; Tucker-Drob & Briley, 2014). The genetic stability for personality also increases from early childhood to late adolescence, suggesting strong predictive validity of genetic factors at one point to a later point in age (albeit not approaching perfect stability till midlife; Briley & Tucker-Drob, 2014). Yet unlike the

increasing heritability demonstrated in cognitive ability, the heritability estimate of personality appears to differ relatively little across the lifespan, with some evidence for minor increases of a few percentage points (Kandler & Papendick, 2017; Möttus et al., 2019), and the increasing stability primarily attributable to nonshared environmental factors (Briley & Tucker-Drob, 2014; Kandler et al., 2019). Simply put, genetically less related individuals would differentiate disproportionately while gravitating toward environments that best matched to their partially genetically influenced traits. This pattern is different from that of the cognitive ability, partly due to the capacity of accommodating broad ranges of behaviors with the sociocultural complexity of modern society, and the lack of institutionalized force that explicitly cultivate personality traits in certain direction (Briley & Tucker-Drob, 2017a; Smaldino et al., 2019). Importantly, this is not suggesting that there is no social pressure on how individuals navigate through life. In fact, the maturity principle of personality (Roberts & Wood, 2006b), which conceptualizes the socialization pressure that accounts for the population mean-level increases in socially desirable traits (agreeableness, emotional stability, and conscientiousness) may as well increase conformity on individual differences. Such an effect may counterbalance the person-environment transactional effects, but it may have a less stringent effect compared to the institutional gateway of cognitive ability, and may activate at a relatively later time point (e.g., when late adolescent transition into social roles; Roberts & Mroczek, 2008; Wrzus & Roberts, 2017a).

The mechanism for psychopathology likely lies in the middle ground of personality and cognitive ability. Consider the question of parental depression on a child's outcome. While children may inherit specific alleles that increase their susceptibility of depression, they may also be raised in an environment where the parents are unable to provide supportive, responsive care, which in turn elevate the risk (i.e., passive *rGE*; Plomin et al., 1977). On the other hand, negative *rGE* can work as a counterbalance. Parents with some previous experience of psychiatric disorders may be particularly aware of the detrimental effect of having psychopathological experience, and they may also acquire external resources that can work as a protection factor from developing psychopathology (e.g., intervention; Roberts et al., 2017).

To date, most evidence of *rGE* using molecular genetic designs was related to cognitive phenotypes, including educational attainment (Bates et al., 2018; Kong et al., 2018; Lee et al., 2018), and cognitive ability (Selzam et al., 2019). Unlike the promising role of *rGE* in phenotypic development inferred by twin and family designs, the evidence supporting *rGE* using

molecular genetic designs was rather mixed. For example, de Zeeuw et al (2020) found that childhood ADHD was only associated with transmitted PGI, and Selzam et al (2019) did not find effect size reduction among neuroticism, ADHD, and schizophrenia using within-family analyses across ~1000 twin pairs. On the other hand, Howe et al (2021) conducted GWAS using siblings (N=159,701) and observed reduction of effect size for depressive symptoms (~39%), albeit the confidence intervals of the estimates were still wide. These inconsistent findings compared to twin and family studies should not be interpreted as the evidence of lack of passive *rGE*. For starters, given the measured genetic variants for those phenotypes only explain very small (1~4%) proportions of the variance, the personality/psychopathology relevant GWAS may be underpowered to pick up any indirect genetic mechanisms. Additionally, it is likely that both evocative and active *rGE* acted in more prominent roles than passive *rGE*, which were not captured by the reviewed molecular genetic designs.

3.3.3 Height as a negative control

Unlike the three psychological traits above, the evidence of gene-environment interplay on height is limited, at least to the samples we have studied (e.g., sample with European ancestry, typically with sufficient access to nutrition, and WEIRD; Duncan et al., 2019). Cross-sectional and longitudinal studies (Dubois et al., 2012; Jelenkovic et al., 2016) on height have observed heritability increased from ~40% during infancy to over 80% during adolescence, but the increases in genetic variance is primarily due to novel activations of gene variants during developmental transitions (e.g., puberty; Silventoinen et al., 2007). Indeed, this observation on lack of G-E interplay is supported by findings from the molecular side, as height is markedly one of the best traits to recover the missing heritability to the studied samples. A study using whole-genome sequencing data from 21,620 individuals found variants (common and rare) that together explained 79% of variance in height (Wainschtein et al., 2019). This suggested that the missing heritability issue is negligible in human height and that additive genetic model can explain the majority of the heritable variation in human height (Yang et al., 2015). Moreover, the effect of genetic nurture was not found for height (Kong et al., 2018), and there was small deflation of effect sizes using within-family analysis (Lee et al., 2018) which can be fully attributed to the bias caused by population phenomenon such as assortative mating (i.e., partners are selected on the basis of height). Taken together, this suggests that height can serve as a negative control in

this study as I expect to find minimum r_{GE} in the current sample. If the estimated effect size is large, however, the statistical method is likely introducing potential confounders.

CHAPTER 4: SAMPLE DESCRIPTIONS AND MEASURES

In order to evaluate the magnitude of gene-environment correlation, I capitalized on primary data collection from the Adolescent Brain Cognitive Development (ABCD), a population based, diverse longitudinal sample of families with twins, siblings, and nonrelated singletons aged 9 years at the first wave. In this chapter, I describe the sample, measures, and provide basic descriptive statistics for the singleton and twin/sibling subsamples. In addition, in this thesis I used multiple quantitative genetic methods to investigate rGE . I gave a brief overview of the methods used for the following Chapter 6 and 7. These are family-based analyses and extended twin modeling.

4.1 SAMPLE

Participants were drawn from the Adolescent Brain Cognition Development (ABCD; Casey et al., 2018; Garavan et al., 2018; Uban et al., 2018) project, a longitudinal study of 9-10 years old and their parents collected at 21 sites across the US. The current sample included both singletons and twins across 2 years of data collection: the first assessment occurred in 2016-2018 (Wave 1), with subsequent follow-up assessment occurred 6-month later, one year later (2018-2019; Wave 2), and two year later (2019-2020; Wave 3). The 6-month follow-up measures did not cover assessments of the current phenotypes of interest and hence not included in the analyses. At the baseline, there were 11,875 children ages 9 to 10 (48% female), including over 1,600 twins or siblings. For follow-up studies, there are 11,235 children in the 1-year follow-up, and 6,571 in the 2-year follow-up. Both twins and singletons provide genetic (at Wave1), cognitive (Wave 1 and 3), personality (Wave 1 and 3), psychopathology (Wave 1-3) and behavioral (Wave 1-3) data. More detailed sample information and zygosity break down regarding each main variable is listed in Table 4.1.

To account for population structure that may act as spurious confounds for the genetic association, the current thesis restricts the sample to individuals of European ancestry ($N_{\text{euro}} = 7074$). Here, the ancestral status is defined as rating $>75\%$ of ABCD provided genetic European ancestry score. Specifically, the sample included 1477 twins or siblings and 5597 unrelated individuals.

4.2 PHENOTYPIC MEASURES

I selected phenotypes from four of the most heavily studied domains in behavior genetics: cognitive ability, personality, psychopathology, and height. The specific measured variables were selected on the basis of their respective PGI yield the highest predictive accuracy within their trait category, and that they were measured longitudinally in the ABCD study. Selected phenotypes were pre-registered² prior to current analysis. Several variables from the cognitive and personality domains were deviated from the pre-registered plan as: a) I excluded fluid intelligence, overall cognitive ability from the cognitive domains, as the longitudinal responses for those variables were not yet available by the time of the analyses; b) I additionally included planning and perseverance in the personality domains as recent study suggested better PGI predictability for relevant traits (Becker et al., 2021).

4.2.1 Cognitive domain

Crystallized Intelligence. Crystallized intelligence (Gc) was scored as an age-normed composite using the NIH Neurocognitive Assessments Toolbox (Hodes et al., 2013). The summary score of Gc included two tasks: picture vocabulary and oral reading recognition. In the picture vocabulary task, the participants were instructed to select one out of four pictures that best matched the auditorily presented words. In the oral reading recognition, the participants were instructed to read presented words aloud while a research assistant marked pronunciations as correct or incorrect. These data were available at the baseline and 2-year follow-up (Wave 1 and 3).

4.2.2 Personality domain

Constructs within the personality domain were measured using subscales of self-reported revised UPPS-P (for urgency, perseverance, premeditation, and sensation seeking; Cyders et al., 2007). For this thesis, I selected three subscales: sensation seeking, lack of planning, and lack of perseverance. Each subscale included 4 items. For sensation seeking scale, the items range from 1(not at all like me) to 4 (very much like me), and for lack of planning and perseverance, the items range from 1(very much like me) to 4 (not at all like me). I reverse coded items from subscale of lack of planning and perseverance to compose the planning and perseverance construct. These data were available at Wave 1 and 3.

² https://osf.io/ajvf8/?view_only=0768c576977545a9b46bc3e836108fa1

Sensation seeking. Sensation seeking refers to the tendency to pursue novel, or exciting experiences (Zapolski et al., 2009). An example item is: I would like to learn to fly an airplane.

Planning. Planning refers to the ability to plan ahead. An example item is: I tend to stop and think before doing things.

Perseverance. Perseverance refers to the tendency to maintain focus on a task. An example item is: I finish what I start.

4.2.3 Psychopathology domain

Constructs under the psychopathology domain were measured using the caregiver report of Child Behavior Checklist (CBCL, age 6-to-18 form; Achenbach et al., 2004). The CBCL contains 119 items describing a particular behavior (e.g., ‘Argues a lot’). Caregivers responded on a 3-point scale ranging from 0 (not true) to 2 (very true or often true) to rate the extent to which the item was characteristics of their child over the past 6 months. The items can be combined to create eight syndrome composites: anxious/depressed, withdrawn/depressed, somatic complaints, social problems, thought problems, attention problems, rule-breaking behavior, and aggressive behavior. The items can also combine to create two broadband problems: internalizing and externalizing problems. In this thesis, I focused on two broad categories of internalizing and externalizing problems. The CBCL was assessed yearly, and the following analyses included the first two waves (1 year apart) in the analyses.

Externalizing. Externalizing broadband incorporates a variety of symptoms that are characterized by externally focused or disinhibited behavior that tends to lead to discomfort in others. The delinquent and aggressive behavior scales are conceptualized as the externalizing problem composite.

Internalizing. Internalizing broadband, on the other hand, are characterized by internally focused or over-inhibited behaviors that tends to lead to self-discomfort. The withdrawn/depressed, somatic complaints, and anxious/depressed scales are conceptualized as the internalizing problem composite.

4.2.4 Anthropometric domain

Height. Height was measured following the procedures provided by CDC guidelines on the National Health and Nutrition Examination Survey (Johnson et al., 2013). A composite score was computed as the average of two out of three closest measures. The ABCD team measured height at each in-person session, and I included the first two waves in the analyses.

4.2.5 Environmental variables

I included 15 environments across four themes: family dynamics (including family socioeconomic status), school engagement (e.g., school involvement), child behavior (e.g., exercise and screen time), and neighborhood characteristics (e.g., lead exposure). Descriptions of those variables can be found in Figure 4.1. The environmental assessments were chosen based on findings of associations with neurocognitive performance and mental health outcomes. Specifically, family socioeconomic status (i.e., family income; Tomasi & Volkow, 2021), parent-children interaction (i.e., parental warmth; Modabbernia et al., 2020), neighborhood features (i.e., neighborhood disadvantage, lead exposure; Hackman et al., 2021; Marshall et al., 2021) and physical behaviors (i.e., screen time; Paulich et al., 2021) were related to cognitive and/or mental health outcomes in the ABCD sample of 9 years old. Past studies also found non-trivial associations between currently investigated phenotypes (e.g., crystallized intelligence, planning) and schooling (Richardson et al., 2012). Importantly, the past literature has not considered the possibility that genetic and environmental influences braid together across development. Evidence that these environmental variables are correlated with genetic differences across participants would be consistent with gene-environment correlation. Analyses including specific environmental variables were not pre-registered and should be considered exploratory.

4.2.6 Pre-processing data

All responses were included in the analyses, except for 124 responses (1% of observations) for the height composite at wave 2, which were marked as missing given that they were at least one standard deviation shorter than the baseline analyses (1 year ago), most likely reflecting data entry errors.

All manifest variables were residualized for age, sex, age^2 , $\text{age} \times \text{sex}$, $\text{age}^2 \times \text{sex}$, genotyping batch, and measurement site following established recommendations (McGue & Bouchard, 1984). I used the child's age at the time of the outcome measure collected as children from the same household can be interviewed at different time points. I then standardized the residuals relevant to the total sample (including nonrelated individuals and siblings) and used the residuals in all models. All analyses were conducted with Mplus (Muthén & Muthén, 2017) using full information maximum-likelihood to account for missingness and the MplusAutomation package in R (Hallquist & Wiley, 2018). For analyses involving twins, I used

the TYPE = COMPLEX and the cluster option of Mplus to account for nonindependence of observations and corrected the standard errors (McNeish et al., 2017).

Following the pre-registration, I used the ABCD pre-calculated composite scores for crystallized intelligence, externalizing, internalizing, and height. For constructs under the personality domain, however, instead of using the composite score as pre-registered, I used factor scores estimated via confirmatory factor analyses to achieve better measurement properties. The 3-factor solution using raw data at the first wave showed good model fit ($CFI \geq .95$, $RMSEA \leq .06$, $SRMR \leq .08$), and achieve partial scalar invariance across waves ($\chi^2_{(19)} = 11.575$, $p = .903$). I then saved the unstandardized factor scores of the three latent personality constructs at both waves for residualizing and future analyses. More details about the factor structure of sensation seeking, planning, and perseverance can be found in Appendix B.

4.3 POLYGENIC INDEX

Genotyping. Genotyping data for 11,826 participants were processed by the ABCD team using Affymetrix NIDA SmokeScreen Array (Uban et al., 2018) following the protocols of the manufacturer. Genotypes were imputed by the ABCD team on the Michigan Imputation Server pipeline v1.2.4, using the Haplotype Reference Consortium reference panel. Then I filtered by an $r^2 > 0.3$ and $MAF > 0.01$. I limited the set of variants to HapMap3 and filtered out variants with genotyping call rate $< .90$, genotyping missing rate $> 1\%$, Hardy-Weinberg equilibrium (p -value $< 10^{-6}$), and sex mismatch. Next, I calculated the genomic relatedness matrix and dropped 521 first-degree relatives from the non-sibling sample (with kinship coefficients $> .177$), and samples of less than 75% genetic European ancestry were excluded. This procedure was conducted in ABCD identified singletons and siblings separately. PLINK was used to handle genetic data and perform quality control (Purcell et al., 2007). The first 10 genetic ancestry principal components (PCs) were obtained as measures of population stratification. In the remaining sample for subsequent analyses, there were 1,051,274 SNPs, 5076 non-related singletons, and 1477 siblings.

Polygenic index. I constructed the indexes using LDpred2 (Privé et al., 2020; Vilhjálmsson et al., 2015) for 7 main phenotypes relevant to cognitive, personality, psychopathology and height. Summary statistics from GWAS discovery sample are listed in Table 4.2, and PGI that provides the highest predictive validity was retained for future biometric analyses. Following the pre-registration, I also computed PGIs using PRSice2 (see Appendix B

for more information). As results computed using LDpred2 showed better predictive power overall, I conducted subsequent analyses using PGIs from LDpred2.

For the LDpred2 pipeline, I used the bigsnpr R package (version 1.8.7; <https://github.com/privefl/bigsnpR>) and applied a grid mode for tuning parameters. LDpred2 infers posterior mean effect size for each variant by adjusting the beta of the SNP from the discovery sample using a prior on effect size and adjusted for LD with a reference panel to obtain an effect of each SNP that is statistically independent of other SNPs. The grid model contains a grid of hyper-parameters (i.e., 21 p-value thresholds and 3 heritability; total size of 63) assuming priors for the genetic architectures. Thus, I used the ABCD singleton sample as LD reference sample (i.e., compute sparse genome-wide correlation matrix), and I split the sample by 80/20 where I used the larger sample as training data to choose hyper-parameters and another part of the data to evaluate statistical properties. I then applied the LD pattern and tuned hyper-parameters to the sibling samples, where the predictability of the polygenic indexes in twin/sibling samples are reported in Table 4.2³.

PGI are known to be sensitive to arbitrary bias arising from differences in allele-frequency between populations of different ancestry (Hamer & Sirota, 2000), or differing estimations across subgroups within the population depending on whether the environment suppresses or nurtures individual's genetic predisposition (Tucker-Drob & Bates, 2016). For analyses examining covariation among measured environment and PGIs, all included PGIs were adjusted for the first 10 PCs derived from the genetic covariance matrix. For analyses using extended twin modeling, I followed Dolan et al (2021) guideline and used unadjusted PGI in the analyses

³ Here the table reported both the predictability PGI as the only exogenous variable, or the incremental predictability of PGI additional to standard covariates (sex and first 10 PCs).

4.4 FIGURE AND TABLES

		Description / item example	
Birth	Variable		
	Maternal age	Age of biological mother when the child was born.	
	Paternal age	Age of biological father when the child was born.	
Familial	Breastfed (month)	Length of child being breast fed.	
	Family total income	Total combined family income for the past 12 months; 1:< \$5,000; 10:>=200,000	
	Caregiver education	Primary caregiver education level. (1:1st grade; 21: doctoral degree)	
	Financial difficulty	Sum of 7 items (0/1) accessing family financial difficulty.	Needed food but couldn't afford it.
	Family conflict (child report)	Total number of family conflict event (0/1) occurred in the past 12 months	We fight a lot in our family.
	Parent monitor (child report)	Mean of 5 items (1-4) accessing parent's effort to keep track of child's condition.	How often do your parents know where you are?
	Family acceptance	Mean of 5 items (1-4) describing primary caregivers' warmth behavior.	(Primary caregiver) similes at me often'.
School	School environment	Sum of 6 items (1~4) reflecting positive school environment	I get along with my teachers
	School involvement	Sum of 4 items (1~4) reflecting positive involvement at school	I like school because I do well in class.
	School disengagement	Sum of 2 items (1~4) reflecting alienation from academic goals.	Usually, school bored me.
Physical	Physical exercise	Total number of days staying physically active for > 60mins/day in the past 7 days.	
	Screen time (Weekday)	Daily average of screen media usage during weekday.	
	Screen time (Weekend)	Daily average of screen media usage during weekend.	
Neighborhood	Neighborhood safety (child report)	Mean of 3 items (1-5) on safety of neighborhood.	My neighborhood is safe from crime.
	Area deprivation index	Derived from residential history, from national data; 0~100	
	Neighborhood crime	Uniform crime report from FBI (3 yrs average from 2010-2012)	
	Population density	Census tract level of population density (based on 2010)	
	Level of pollution (pm2.5)	Annual average of PM 25 from national data	
	Risk of lead exposure	Census tract level of estimated risk of lead (1~10)	

Figure 4.1 Descriptions on environmental variables used in the current study. A SES composite score is computed by leveraging family total income, caregiver education level, financial difficulty, and area deprivation index variables.

Table 4.1. Summary of measured variables used in the twin analyses

Theme	Variable name	Measurement	Items	Report	File name	Waves	Measures name	N MZ (pairs/wave)	N DZ (pairs)	N single (wave 1)
Cognitive	Crystalized intelligence	NIH toolbox	Composite; Age normed	Objective	abcd_tbss01	1,3	nihtbx_cryst_agecorrected	258	520	5452
	Sensation seeking							173	333	
	Lack of planning							259	521	
	Lack of perseverance							176	336	
Personality		UPPS-P	Raw items	Self	abcd_upps01	1,3	upps12, 18, 21, 27	259	521	5593
								176	336	
								259	521	
								176	336	
Psychopathology	Internalizing	CBCL	Composite	Informant	abcd_cbcls01	1,2	cbcl_scr_syn_internal_t	259	521	5597
								176	336	
								258	523	
								250	512	
Anthropometric	Height	phenX	Averaged of 3 measures	Objective	abcd_ant01	1,2	anthroheightcalc	258	523	5597
	Batch							250	512	
	Site							259	524	
								248	504	
Covariates	sex			Self	acspsw03	1	sex			

Note. I have also included age, age2, age×sex, and age2×sex as covariates in the model. Since siblings from the same household might be interviewed at different time point, I used the child's age at the time of the outcome measures were collected. MZ = monozygotic twins, DZ = dizygotic twins.

Table 4.2. GWAS summary and PGI predictability using LDpred2

Phenotype for PGI	Author (year)	N total	PGI R2 (free)	PGI R2 (10 PCs and sex)
<i>Crystal Intelligence</i>				
Educational attainment	Lee et al. (2018)	766,344	0.0931	
Intelligence	Savage et al. (2018)	269,867	0.0971	0.0856
<i>Externalizing</i>				
ever smoker	Liu et al. (2019)	632,802	0.0016	0.0009
Worry	Nagel et al. (2018)	348,219	0.0013	
<i>Internalizing</i>				
ever smoker	Liu et al. (2019)	632,802	0.001	
Worry	Nagel et al. (2018)	348,219	0.0085	0.0087
<i>Perseverance</i>				
Risk_Pc1	Linner et al. (2019)	315,894	0.0009	
ever smoker	Liu et al. (2019)	632,802	0.0064	0.0048
<i>Planning</i>				
Risk_Pc1	Linner et al. (2019)	315,894	1.66E-05	
ever smoker	Liu et al. (2019)	632,802	0.0049	0.0046
<i>Risk seeking</i>				
Risk_Pc1	Linner et al. (2019)	315,894	0.0059	0.0069
Risk tolerance	Linner et al. (2019)	466,571	0.0032	
ever smoker	Liu et al. (2019)	632,802	0.0009	
<i>Height</i>				
Height	Yengo et al. (2018)	693,529	0.1687	0.1672

Note. Bolded PGI were used in further analyses. PGI R2(free) is predictability, or R-squared, of PGI from models with just the polygenic index as the exogenous variable. PGI R2 (10 PCs and sex) is incremental predictability, or ΔR^2 of PGI additional to covariates (sex and top 10 PCs).

CHAPTER 5: PRELIMINARY ANALYSES

Here, I provide basic descriptive statistics at the phenotypic level for the subsamples of singletons, MZ twins, and DZ twins and siblings. I establish the distributional characteristics of the data, relations among the target phenotypes, the pattern of twin correlations and baseline ACE decompositions, and the extent to which the PGIs predict their target phenotype.

Specifically, I computed a family-level SES by extracting the first principal component from 4 variables: primary caregiver's educational level, past 12 months family total income, financial difficulty, and area deprivation index. The first component accounted for 53.1% of total variance.

5.1 DESCRIPTIVE STATISTICS

Descriptive statistics for seven main phenotypes can be found in Table 5.14. The test-retest reliabilities for objective measures (i.e., crystalized intelligence, height) and informant reports (i.e., externalizing, and internalizing problems) were adequate (r 's $> .6$) across groups. The stability for self-reported personality factor scores (i.e., perseverance, planning, and sensation seeking), on the other hand, were relatively weaker across groups (r 's $\sim .4$), consistent with previous findings on relatively low stability of personality traits in early life ($r = .40$; Roberts & DelVecchio, 2000).

Raw phenotypic correlations were consistent across singletons, MZ, and DZ twins (including siblings), suggested by the overlapping confidence intervals. The patterns of the associations suggested good convergent validity. For example, two facets of conscientiousness, perseverance, and planning, were positively correlated (r 's $\sim .5$). Externalizing problems were positively correlated with internalizing problems (r 's $\sim .5$). Externalizing also showed small negative associations with planning and perseverance, as expected (Barr & Dick, 2019). On the other hand, the lack of associations between crystalized intelligence (r 's $\sim .1$), height (r 's $\sim .05$) and the other phenotypes suggested good discriminant validity.

4 Mean, standard deviation, kurtosis and skewness of 13 environment variables for European sample can be found in Table B.3. Those descriptive statistics for non-European sample can be found in Table B.4.

It may come as a surprise for the null associations between sensation seeking and externalizing, as there were established associations between sensation seeking and delinquency behavior such as drug and alcohol use (Meier et al., 2008). However, given the developmental stage of the current participants (9 years old; pre-adolescent), the participants might be less likely to have the agency to transform from maladaptive behaviors to more severe forms of disruptive behaviors and risk-taking.

5.2 TWIN DESCRIPTIVE ANALYSES

To maximize power, I combined two waves worth of information in all twin modeling, following the pre-registered plan. Specifically, I used a correlated factor model, which estimate heritability, shared, and nonshared-environmental factor at wave 1 and wave 2, and the rank-order stabilities of genetic and environmental factors across waves (see Figure 5.1 for example). Put differently, test-retest stabilities of phenotypic variables were attributed to stabilities of genetic and environmental factors. Moreover, I fixed the parameters within wave (e.g., heritability for wave 1 and wave 2) to be equal across time. As a robustness check, I conducted sensitivity tests on whether fixing vs. freely estimating the parameters resulted in significantly worse model fit.

5.2.1 Twin correlation

Twin correlations are reported in Table 5.2. Fixing the within-wave correlation to be equal across wave did not result in significant worsen fit (p 's > .2). In all instances, the MZ twin correlation was larger than the DZ correlation, indicating the presence of additive genetic influences. For personality traits (i.e., planning, perseverance, sensation seeking), the MZ correlation was more than double the DZ correlation, indicating the possibility of nonadditive genetic effects and the inability of subsequent models to detect shared environmental influences. As such, these phenotypes are unlikely to find any evidence of gene-shared environmental correlations and excluded from that set of analyses to avoid model misfit.

5.2.2 Standard ACE/AE solution

To investigate the genetic and environmental contribution to the targeted phenotypes, I used the classical twin design (Neale & Cardon, 2013), which compares phenotypic similarity between twin pairs with known differences in genetic similarity (e.g., averaged of 1/.5 for MZ twins vs. DZ or siblings) to estimate the relative contribution of genetic and environmental factors to variation in that trait.

Both ACE and AE models using the correlated factors model were fit to the data. Rather than comparing model fits across the ACE and AE models to obtain the more parsimonious models, both models were retained as guides for the final modeling of genetic-shared environment covariances (covAC; using ACE model) and genetic-nonshared environment covariance (covAE; using AE model). However, for traits suggesting clear preferences for nonadditive genetic model (i.e., zero shared environmental contributions), I only based extensions on AE models to avoid substantial misfit.

The genetic and environmental contribution to phenotypic variances were reported in Table 5.3. Here, fixing the decomposed variance of A/C/E to equate across wave also did not result in significant worsen model fit (p 's > .1), I thus approached using the fixed parameter correlated model in analyses in Chapter 75.

Overall, the models suggested low shared environmental estimates, indicating that there are minimal effective shared environmental influences on the phenotypes, including crystallized ability which tends to have the largest estimates in the past literature. Note that this finding does not indicate that objectively shared environments had limited roles in psychological development, but rather objectively shared environments likely have differentiating impacts on development as twins do not experience the environment similarly. The shared environment estimates were virtually zero for planning, perseverance, and sensation seeking, thus I excluded those three traits from genetic-shared environment correlation models to avoid model misfit.

5.3 PGI DESCRIPTIVE ANALYSES

I calculated polygenic indexes for each of the phenotypes based on published GWAS summary statistics. As shown previously (see Chapter 4.3, Table 4.2), the PGIs for height and

5 ACE model suggested good model fits overall (CFI > .99; RMSEA < .02) Overall good fits except for height, which is driven by the misfit in fixed test-retest stability across zygosity groups, such that the stability among DZ twins were lower than that of MZ twins ($r_{MZ} = .93$ vs. $r_{DZ} = .83$). When the test-retest stability was freely estimated between zygosity groups, the model fitted the data well (CFI = .987, TLI = .991, RMSEA = .037, SRMR = .046). Given the cross-trait covariance is the less prominent parameters in the current project, I moved on with models fixing test-retest stability across twins with different zygosity in trade for extra degree of freedom.

crystallized ability accounted for a sizeable portion of variance, although much lower than previously reported estimates. Conversely, minimal associations were found for the remaining phenotypes, with the largest R^2 of less than 1%. More specifically, the phenotypic externalizing is negatively associated with computed polygenic score ($r = -.04$), but this association has been documented in previous study examining polygenic association of externalizing in the ABCD sample (Waszczuk et al., 2021). The predictive power of the polygenic indexes could be low for several reasons. First, polygenic indexes for self- or informant-report variables tends to be very weak (Cheesman et al., 2017). My estimates for the personality and CBCL-derived psychopathology phenotypes are not substantially lower than most other estimates. Second, most GWAS are based on adults, meaning the identified genetic variants may not be relevant to a child sample. Third, most GWAS are based on individuals born in other countries (e.g., UKBiobank) in much earlier cohorts (e.g., 1950s; Bycroft et al., 2018). Again, differences across the sociocultural environmental context flowing from chronological change may alter patterns of genetic association.

Table 5.4 presented genetic correlations between computed PGIs (see Figure 5.2 for phenotypic and PGI correlation). Genetic correlations mimic patterns from the phenotypic correlations except between externalizing and internalizing, where the largest correlation was between PGIs of planning and perseverance ($r = .74$). As a negative control, there were little observed correlation between phenotypes and height (r 's $\sim .02$). These results demonstrate the validity of the computed PGIs.

Next, I investigated the twin correlations for PGI, which acts as an estimate of alleles identical-by-descent (IBD) relevant to a specific phenotype. As shown in Table 5.4, the results suggested substantial assortative mating (e.g., DZ twin being more similar on genetic variants than the expected $r = .5$) for crystallized intelligence ($r = .61$) and height ($r = .59$), in line with past literature (e.g., Greenwood et al., 2014). To improve model fit, when incorporating the PGI in twin models (Chapter 7), I fixed the PGI similarity between DZ twins to the sample average.

5.4 SUMMARY

In sum, the selected phenotypes have generally acceptable psychometric properties, show reasonable test-retest stability across approximately a year, and twin models find estimates of genetic and environmental influences similar to past work, with the exception of relatively low estimates of shared environmental influences on crystallized ability. No phenotype displayed

substantial estimates of the shared environment. This result places a limit on the power to detect covariance between genetic and shared environmental factors, but covariance between genetic and nonshared environmental factors may be detectable. Next, I conduct exploratory analysis using constructed PGIs to correlate with a range of environmental exposure to estimate r_{GE} .

5.5 FIGURES AND TABLES

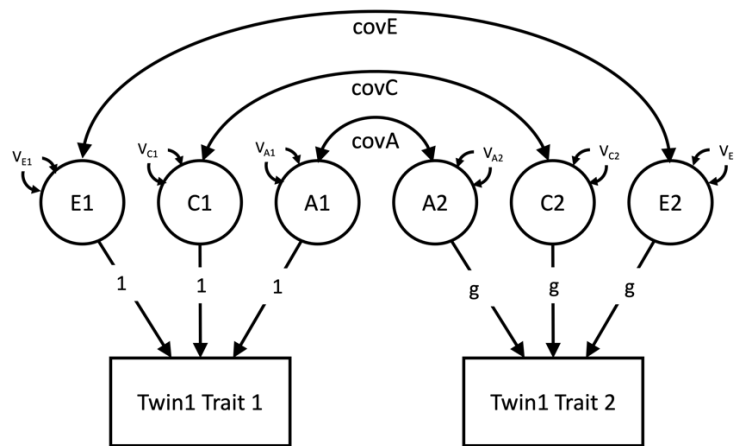


Figure 5.1 The correlated factors model. A = additive genetic factor; C = shared environmental factor; E = non-shared environment factor; covA = genetic covariance; covC = shared environmental covariance; covE = non-shared environmental covariance; g = scaling factor; VA, VC, VE are raw variances for additive genetic, shared, and nonshared environmental variance components.

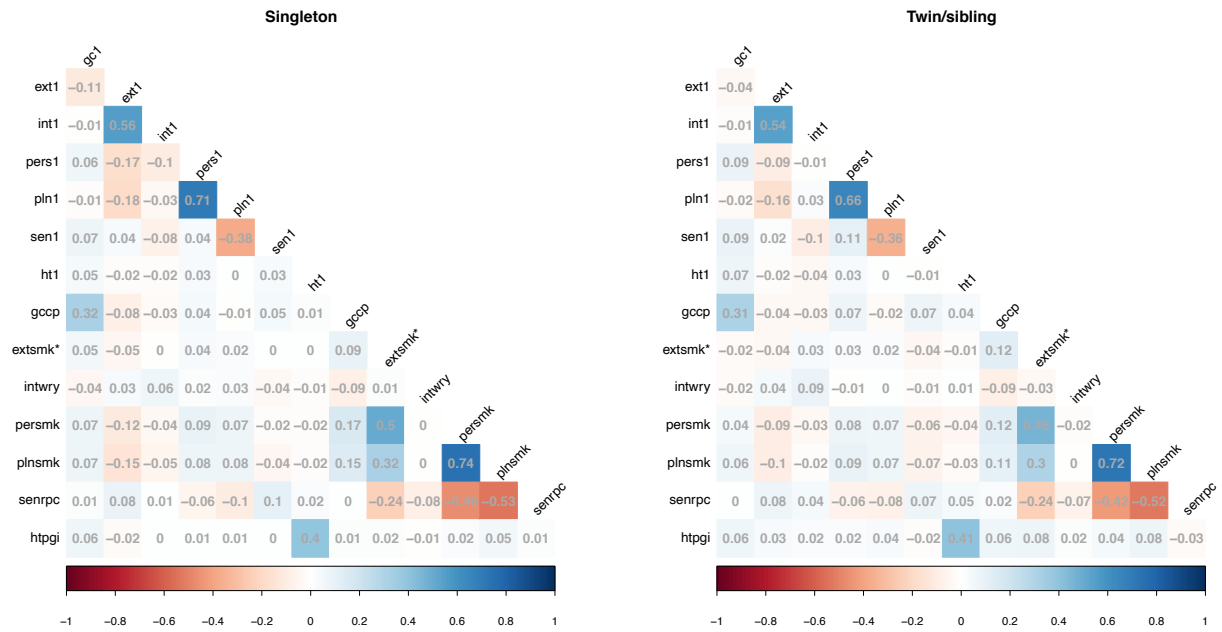


Figure 5.2 Phenotypic and PGI correlations across singleton and twin/sibling pairs.

Table 5.1. Descriptive statistics for the main variables included in the study

	Var	Wave 1 Mean(SD)	Descriptive			Standardized Phenotypic Correlation (SE)					
			Skewness	Kurtosis	Reliability	Crystallized intelligence	Externalizing	Internalizing	Perseverance*	Planning*	Sensation seeking*
Singleton (N = 5597)	Gc	110.655(17.816)	0.76	1.02	0.744						
	Ext	45.952(10.126)	0.57	-0.16	0.748	-.109(.014)					
	Int	49.257(10.485)	0.34	-0.41	0.689	-.006(.014)	.562(.01)				
	Pers*	-0.007(0.338)	-0.81	0.96	0.464	.064(.014)	-.166(.014)	-.102(.014)			
	Pln*	-0.004(0.33)	-0.7	0.77	0.437	-.013(.014)	-.184(.014)	-.039(.014)	.706(.008)		
	Sen*	0.003(0.478)	-0.15	-0.49	0.473	.082(.014)	.046(.014)	-.069(.014)	.041(.014)	-.378(.013)	
	Ht	55.087(3.073)	0	2.07	0.873	.081(.014)	-.02(.014)	.006(.013)	.047(.014)	-.001(.014)	.041(.013)
MZ twins (N = 518)	Gc	105.515(16.046)	0.63	1.34	0.673						
	Ext	43.349(9.383)	0.77	0.14	0.739	-.097(.046)					
	Int	46.711(10.55)	0.54	-0.43	0.649	-.013(.053)	.53(.042)				
	Pers*	0.038(0.308)	-0.83	1.15	0.358	.155(.048)	-.048(.054)	-.021(.054)			
	Pln*	0.038(0.313)	-0.77	1.02	0.378	.026(.049)	-.089(.059)	.027(.053)	.586(.031)		
	Sen*	-0.027(0.478)	-0.05	-0.58	0.441	.081(.047)	-.022(.047)	-.106(.046)	.143(.048)	-.396(.042)	
	Ht	55.198(3.004)	0.13	-0.12	0.953	.116(.055)	-.148(.05)	-.065(.048)	.158(.05)	.099(.049)	-.056(.048)
DZ twins/siblings (N = 959)	Gc	107.195(15.485)	0.86	1.17	0.694						
	Ext	43.991(9.72)	0.71	-0.14	0.678	-.014(.038)					
	Int	46.878(9.841)	0.49	-0.32	0.637	-.009(.035)	.554(.026)				
	Pers*	0.021(0.32)	-0.73	1.05	0.413	.051(.033)	-.144(.035)	-.017(.033)			
	Pln*	0.004(0.32)	-0.76	1.12	0.382	-.04(.034)	-.219(.034)	0(.034)	.711(.02)		
	Sen*	-0.005(0.477)	-0.13	-0.68	0.47	.093(.033)	.046(.034)	-.07(.033)	.078(.032)	-.333(.031)	
	Ht	55.457(2.909)	0.07	0.18	0.888	.052(.037)	-.005(.035)	-.03(.033)	.016(.034)	-.025(.031)	.044(.035)

Note. Reliability = test-retest reliability across 1 or 2 years. Variables with the * are latent factor scores. Gc = crystallized intelligence, ext=externalizing, int=internalizing, pers=perseverance, pln=planning, sen=sensation seeking, ht=height

Table 5.2. Wide format twin correlation estimations

Phenotype	rMZ (wave1)	rDZ (wave1)	rMZ (wave2)	rDZ (wave2)	Test-retest	CTCT (MZ)	CTCT (DZ)	CFI	TLI	RMSEA	SRMR	$\Delta\chi^2$ (2) scaled	p
Freely estimated													
Gc (wave1,3)	.672(.038)	.416(.044)	.746(.034)	.412(.047)	.699(.020)	.675(.033)	.341(.038)	0.999	1	0.007	0.038	-	-
Ext (wave1,2)	.698(.033)	.371(.047)	.700(.037)	.420(.043)	.700(.018)	.560(.031)	.312(.040)	0.996	0.997	0.023	0.045	-	-
Int (wave 1,2)	.537(.042)	.385(.045)	.607(.041)	.440(.042)	.636(.020)	.452(.034)	.327(.036)	1	1	0	0.044	-	-
Pers (wave 1,3)	.485(.047)	.095(.045)	.441(.061)	.097(.058)	.395(.027)	.326(.054)	.047(.039)	1	1	0.003	0.061	-	-
Pln (wave 1,3)	.325(.061)	.052(.042)	.354(.085)	.084(.055)	.388(.030)	.273(.054)	.047(.041)	0.954	0.967	0.034	0.073	-	-
Sen (wave 1,3)	.253(.059)	.034(.045)	.270(.073)	.144(.057)	.457(.026)	.206(.053)	.050(.043)	1	1	0	0.039	-	-
Ht (wave 1,2)	.917(.017)	.480(.037)	.926(.012)	.511(.037)	.911(.016)	.871(.017)	.485(.035)	0.936	0.955	0.077	0.05	-	-
Fixed parameters													
Gc (wave1,3)	.703(.028)	.414(.037)	-	-	.697(.020)	.672(.034)	.340(.038)	0.998	0.999	0.012	0.039	2.674	0.263
Ext (wave1,2)	.699(.028)	.394(.039)	-	-	.700(.018)	.560(.030)	.311(.040)	0.997	0.998	0.019	0.046	1.158	0.56
Int (wave 1,2)	.569(.033)	.413(.035)	-	-	.636(.020)	.450(.035)	.327(.036)	1	1	0	0.046	2.693	0.26
Pers (wave 1,3)	.467(.039)	.096(.037)	-	-	.395(.027)	.327(.053)	.047(.039)	1	1	0	0.062	0.359	0.836
Pln (wave 1,3)	.335(.052)	.065(.034)	-	-	.388(.030)	.271(.054)	.045(.040)	0.964	0.977	0.028	0.073	0.28	0.869
Sen (wave 1,3)	.259(.051)	.078(.039)	-	-	.456(.026)	.205(.053)	.041(.043)	1	1	0	0.044	2.885	0.236
Ht (wave 1,2)	.921(.011)	.496(.035)	-	-	.911(.016)	.871(.017)	.485(.035)	0.937	0.96	0.072	0.05	1.174	0.556

Note. CTCT = cross twin cross trait correlation. All variables are residualized for age, age2, age×sex, age2×sex, batch, and site prior to inclusion in the model. Gc = crystallized intelligence; Ext = externalizing; Int = internalizing; Ht = height; Per = perseverance; Pln = Planning; Sen = sensation seeking. $\Delta\chi^2$ (scaled) = Satorra-bentler adjusted chi-square difference tests. The scaled factor is estimated such that the twin variance at the subsequent wave is proportional to the baseline variance at the magnitude of g, such that the (standardized) twin correlations are equivalent across waves.

Table 5.3. Correlated factor ACE/AE model

	Corxy	H2 (Latent)	C2	E2	Scaling g	CFI	TLI	RMSEA	SRMR
Correlated factors-ACE									
Gc	.705(.020)	.581(.090)	.129(.078)	.290(.027)	.907(.027)	1	1	0	0.038
Ext	.702(.018)	.618(.093)	.088(.082)	.294(.028)	.945(.021)	0.997	0.998	0.021	0.045
Int	.637(.020)	.316(.093)	.256(.076)	.428(.032)	.975(.023)	1	1	0	0.048
Ht	.910(.015)	.845(.073)	.073(.071)	.082(.012)	1.088(.018)	0.941	0.963	0.069	0.05
Pers	.381(.027)	.405(.039)	.000(.000)	.595(.039)	1.180(.053)	0.911	0.944	0.053	0.092
Pln	.382(.030)	.274(.045)	.000(.000)	.726(.045)	1.071(.045)	0.944	0.965	0.035	0.08
Sen	.443(.026)	.247(.040)	.000(.000)	.753(.040)	1.164(.038)	0.935	0.959	0.047	0.071
Correlated factors-AE									
Gc	.702(.020)	.723(.026)		.277(.026)	.909(.028)	0.995	0.997	0.02	0.042
Ext	.700(.018)	.713(.025)		.287(.025)	.945(.021)	0.997	0.998	0.018	0.048
Int	.632(.020)	.600(.027)		.400(.027)	.974(.023)	0.991	0.995	0.029	0.062
Ht	.910(.015)	.910(.012)		.090(.012)	1.088(.018)	0.937	0.964	0.069	0.051
Pers	.386(.026)	.406(.040)		.594(.040)	1.182(.052)	0.907	0.947	0.052	0.094
Pln	.385(.030)	.275(.045)		.725(.045)	1.072(.045)	0.944	0.968	0.034	0.082
Sen	.444(.026)	.247(.040)		.753(.040)	1.166(.037)	0.936	0.964	0.044	0.071

Note. Gc = crystalized intelligence; Ext = externalizing; Int = internalizing; Ht = height; Per = perseverance; Pln = Planning; Sen = sensation seeking; varP = total phenotypic variance; varA=total genetic variance; H2 = heritability estimates; C2 = shared environmental influences; E2 = non-shared environmental influences

Table 5.4. PGI correlations across constructs

			Raw PGI Correlations (SE)					
	Var	Twin corr (IBD)	Gccp	Extsmk	Intwry	Persmk	Plnsmk	Senrpc
Singleton (N=5076)	Gccp							
	Extsmk		.096(.014)					
	Intwry		-.092(.014)	.013(.014)				
	Persmk		.172(.014)	.508(.016)	-.001(.014)			
	Plnsmk		.150(.014)	.327(.015)	.000(.014)	.756(.017)		
	Senrpc		.001(.014)	-.244(.015)	-.084(.014)	-.468(.015)	-.542(.016)	
	Htpgi		.017(.015)	.024(.014)	-.009(.014)	.026(.014)	.054(.014)	.013(.014)
MZ (N = 518)	Gccp	0.999						
	Extsmk	0.998	.129(.058)					
	Intwry	0.997	-.103(.054)	-.094(.050)				
	Persmk	0.996	.167(.060)	.470(.074)	-.063(.051)			
	Plnsmk	0.998	.207(.057)	.319(.063)	-.019(.052)	.671(.067)		
	Senrpc	0.997	-.050(.048)	-.170(.065)	-.048(.050)	-.315(.061)	-.427(.053)	
	Htpgi	0.998	.068(.064)	.059(.072)	.056(.056)	.058(.057)	.054(.057)	.017(.060)
DZ (N = 959)	Gccp	0.614						
	Extsmk	0.511	.089(.033)					
	Intwry	0.562	-.083(.036)	.006(.035)				
	Persmk	0.504	.079(.034)	.433(.038)	.012(.037)			
	Plnsmk	0.51	.037(.035)	.272(.035)	.017(.036)	.686(.042)		
	Senrpc	0.512	.071(.035)	-.247(.034)	-.071(.035)	-.435(.036)	-.512(.040)	
	Htpgi	0.594	.049(.037)	.066(.035)	.007(.036)	.020(.037)	.076(.034)	-.050(.038)

Note. Gccp = crystalized intelligence PGI computed using intelligence GWAS; Extsmk = externalizing PGI computed using smoking GWAS; Intwry = internalizing PGI computed using worry (Neuroticism subcluster) GWAS; Persmk = Perseverance PGI computed using smoking GWAS; Plnsmk = PGI computed using smoking GWAS; Senrpc = sensation seeking PGI computed using risk principle component GWAS; Htpgi = height PGI computed using Height GWAS;

CHAPTER 6: CORRELATIONS BETWEEN MEASURED GENETIC AND ENVIRONMENTAL FACTORS

6.1 INTRODUCTION

The rise of the polygenic index has resulted in a surge of studies investigating the mediating roles of environments in the association between genetic variants and phenotypes. Correlations between trait associated PGI and specific environmental exposures indicate r_{GE} . For example, PGIs have been linked with specific neighborhood features (i.e., population density) and health disparities (Colodro-Conde et al., 2018), as well as a wide range of environments using an environment-wide approach (e.g., Krapohl et al., 2017). This work indicates that genetic variants are distributed systematically across geographic regions (Abdellaoui et al., 2019) and environmental quality (von Stumm et al., 2020).

Interpreting associations between PGIs and the environment presents challenges. As with most correlational analyses, the direction of the potential causal association is not clear. It is possible that PGI-environment associations are due to individuals creating an environment on the basis of their genetically influenced characteristics (Scarr & McCartney, 1983). Or it is possible that environmental resources are distributed in line with historical inequities, such that individuals passively receive an environment correlated with their genotype. One methodological concern is the power and predictive accuracy of the PGI. Prediction estimates of PGI in samples of unrelated individuals can include effects of population stratification, assortative mating, or environmentally mediated genetic effects (i.e., passive r_{GE}). Recent work has shown that PGIs have limited portability across groups of different genetic ancestries, as well as limited generalizability within a single ancestry group when the predictive accuracy of the PGI depends on sample features such as age, sex, or socioeconomic status of the individuals (Morris et al., 2020; Mostafavi et al., 2020). Taken together, a variety of familial effects via either genetic, environmental, or both pathways could contribute to the association between environmental exposures and PGI calculated using samples of unrelated individuals.

When environmental effects causally impact phenotype development and correlate with PGIs due to r_{GE} , the causal effect appears as SNP associations in a traditional GWAS design. Within-family design can substantially reduce concerns about the causal interpretation as genes are randomized within sibling pairs conditional on parental genotype, and siblings are matched

for their objective environment (Trejo & Domingue, 2019). For education attainment, the within-family effect size of PGI is roughly half of the between-family effect sizes, reflecting a variety of environmentally mediated genetic effects (Lee et al., 2018; Selzam et al., 2019). Recently, Howe and colleagues (2021) conducted a large scale within-family GWAS across 25 traits of ~160,000 siblings, and they observed considerable within-sibship shrinkage ranging from 10~49% for the polygenic signal on relevant traits, where shrinkage for educational attainment is one of the strongest (46%).

In this chapter, I investigate the magnitude of *r*GE by testing associations between measured PGIs and range of specific environments in the singleton sample, and then I test for within-family associations in the twin and sibling sample.

6.2 ANALYTICAL PLAN

6.2.1 Trait prediction in unrelated samples

A single-score regression model assessed associations between each of seven phenotypes and one of 15 environmental experiences. I then replicated the procedure for corresponding PGIs. To account for population stratification and genotyping method, PGIs in these analyses were residualized for the first ten principal components and the variables capturing genotyping information (i.e, batch, site). I then used the standardized residuals in the following analyses. I used the first wave of the ABCD data to maximize power. The environmental experiences were specified as dependent variables. All variables were standardized prior to the analyses such that the β coefficient from the linear regression models was equivalent to Pearson's correlation coefficients. I followed Cohen's (1992) threshold to interpret the reported effect size.

6.2.2 Within family analyses

I used a two-level regression model with a random intercept on DZ twins to investigate the within- and between- family effects. I used DZ twins as they share all objective rearing environment but remain distinguishable at the measured genetic level:

$$Y_{ij} = \beta_{0j} + \beta_1(PGI_{ij} - \overline{PGI_j}) + \beta_2\overline{PGI_j} + \varepsilon_{ij}$$

Here, *i* corresponds to the individual twins clustered in the family *j*, β_1 is the fixed within-family effect and β_2 is the fixed between-family effect. Here, the within-family coefficients refer to the rate of change in outcome *Y* given one unit change in the difference between one sibling's PGI and the family average PGI (while holding other parameters constant). Put differently, β_1 quantifies the extent to which a sibling (within a family) with

higher planning related PGI tend to rate higher on tendency to stop and think before doing things.

Given the use of mixed-effects model, the relationship between PGI and outcome Y, which is equivalent to those estimated in singleton models described in 6.2.1, can then be specified as:

$$\beta_{PGI} = \beta_1(1 - ICC) + \beta_2 ICC$$

Where ICC stands for the intraclass correlation coefficient (Genser et al., 2015; Selzam et al., 2019). Using this equation, the proportion of total effect accounted for by within family effect can be specified the ICC adjusted fixed effect.

For the within-family analyses, trait-relevant PGI of DZ twins and siblings were used to predict their intended phenotype. For the purposes of this analysis, the siblings must vary on the environmental outcome, and therefore, I was able to perform the analysis on 9 out of 15 environmental variables taken from the family dynamics (self-reported by the child), personal behavior (screen time and exercise assessments), and school (self-reported by the child). I accounted for the non-independence of observations and corrected the standard errors by using TYPE = COMPLEX and the cluster option in Mplus (McNeish et al., 2017).

6.3 RESULTS

Multiple testing correction of the α significance threshold was performed using the effective number of tests (Meff) correction (Derringer, 2018). Among the 15 environmental factors explored, the estimated effective number of tests was 14.53 for singletons, translating to $\alpha = 0.0034$ for adjusted threshold. With the current sample size ($N = 5076$), I have 90% to detect $\beta = .06$ in the singleton sample. Following presentation focused primarily on interpreting the effect size of the point estimates.

6.3.1 Phenotypic correlations

At the phenotypic level, family socioeconomic background (i.e., total income, parental education, and financial difficulties) showed a widespread association with singletons' behavioral and psychological characteristics, except for sensation seeking (see Figure 6.1 Phenotypic estimations were presented as dots). Similar to past literature, the results suggested nontrivial positive associations between screen usage and externalizing, and negative associations with crystallized intelligence, perseverance, and planning ($\beta_s \sim -.15$). I also found positive association with (positive) school experience and two facets of conscientiousness traits.

Alternatively, I found null effects (β s $< .05$) between height and 15 environmental exposures, supporting its role as a negative control.

6.3.2 PGI-Environment association in unrelated samples

Mirroring phenotypic results, cognitive related PGI, and only cognitive PGI, was positively correlated with parental characteristics, family SES and negatively related to area deprivation index and screen time ($|\beta|$ s $\sim .15$; see Figure c.1). Height PGI was not associated with the environmental features, again consistent with the phenotypic results. For the remaining PGIs, associations were much smaller or in the opposite direction from the phenotypic results, most likely due to the weak predictive power of the PGIs. The strongest associations were between screen hours per day and PGIs of perseverance and planning.

6.3.3 Within-family analyses

Next, I used a within-family design using sibling pairs matched for environmental backgrounds to ensure that associations are not due to population stratification or other family-level confounds. Figure 6.2 depicts the within- and between-family PGI predictions of its intended traits, and Figure 6.3 shows the total PGI-environment effects decomposed to within- and between-family effect (ICC adjusted). Overall, the model did not suggest much reduction in within-family effects of PGI compared to total effects, such that the magnitude of within-family estimation of crystallized intelligence PGI recovered 73% of the PGI-phenotype correlations reported in unrelated individuals. I find null between-family effects for personality and psychopathology-related PGIs, and that for crystalized intelligence and height was comparable to within-family effects. As for PGI-environment correlation, I found null within-family effects across most pairs.

As within-family estimates accounted for shared environmental effects, including passive r GE, the lack of attenuation in within-family prediction provides limited evidence of genetic nurturing (i.e., that genetic effects are mediated through rearing environment contexts shared across twins). Importantly, this does not mean that within-family design is free from any r GE. Genetic influences may emerge as children become more entangled with the environment as a systematic function of their genetically influenced traits, showing active/evocative r GE.

6.4 SUMMARY

In this investigation, I found limited evidence of passive r GE, indicating that these data are inconsistent with the family environment mediating the association between genetic effect

and phenotype. However, family-based designs are not sufficient to account for all possible r_{GE} as environmental exposures can expand beyond familial environments (e.g., school, peer group, social community). I used an extended twin analyses in Chapter 7 to examine the overall r_{GE} by investigating the covariation between aggregated environmental effects and genetic effects to address this issue.

6.5 FIGURES

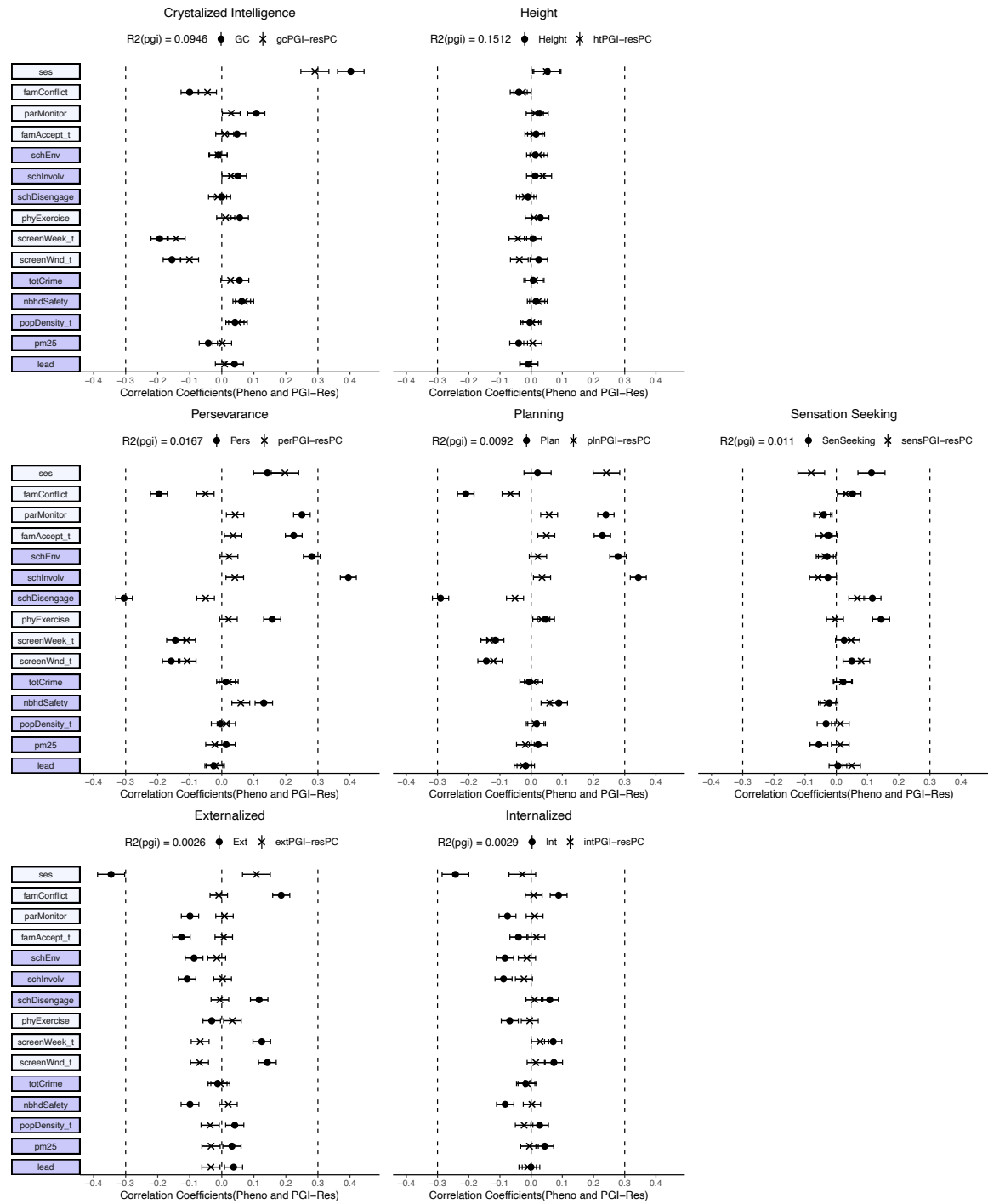


Figure 6.1. Associations between environmental variables with each trait and trait-relevant PGI in singletons. Here the PGI is adjusted for the top 10 PCs. Error bars are standard error for the point estimates. The dotted lines represented the beta coefficients of -.3, 0, and .3.

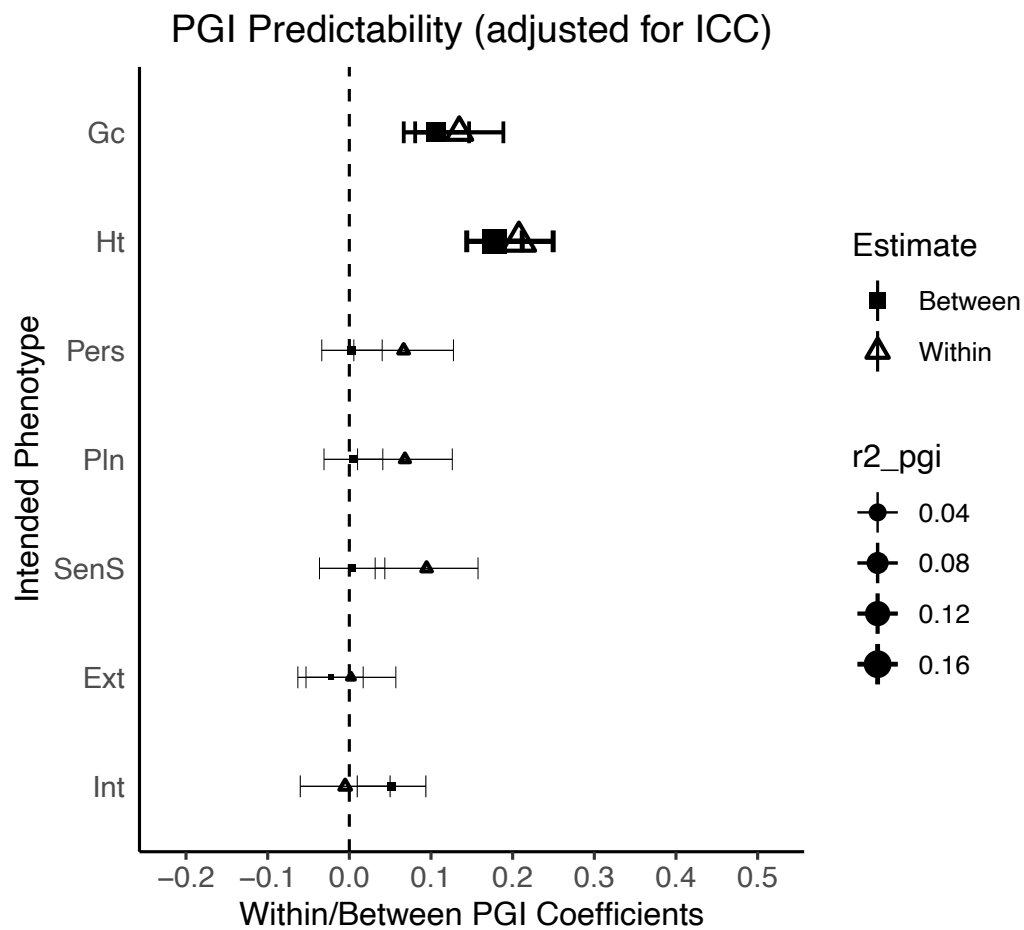


Figure 6.2. Within- and Between-Family predication estimates of seven PGIs. Each row in represents the within/between-family predictive power of PGI of intended trait. The size of the estimates were scaled relative to the PGI predictability.

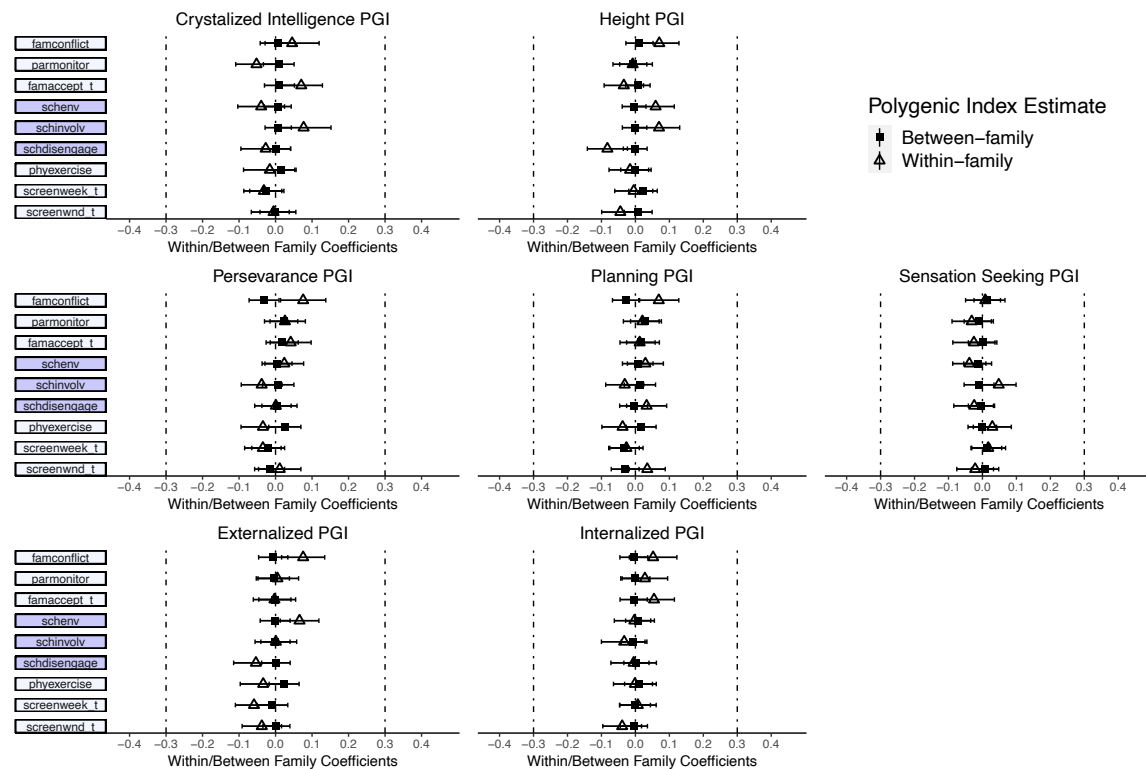


Figure 6.3. Within- and Between-Family cross-trait predication estimates of seven PGIs

CHAPTER 7: CORRELATIONS BETWEEN LATENT GENETIC AND ENVIRONMENTAL FACTORS

7.1 INTRODUCTION

As family-based designs can still be biased and not sufficient to eliminate genetic confounds, it remains necessary to triangulate evidence using different methods. In addition, the above within-family correlations between measured genetic variants and environmental exposures ranging from $-.1$ to $.1$, providing limited information on the magnitude of the aggregated effects of those nonrandom exposures to environment (or environmental mediating of genetic effects). Given that, the following chapter investigated the overall extent to which phenotypic variation is attributable to r_{GE} using an extended classical twin analyses incorporating PGI. Specifically, this approach consolidates all causal environmental effects into a single latent component to examine its association with genetic factors. Incorporating PGI into the classical twin model also provides a unique opportunity to examine the magnitude of gene-nonspecific environment correlation (r_{AE}), likely capturing active and evocative r_{GE} . Whereas all other currently established methods are limited to examine passive r_{GE} (e.g., genetic nurturing; r_{AC}), the current method attempts to develop a holistic perspective of how people evoke and select into environments in correspondence with their traits.

7.2 ANALYTICAL PLAN

The hypotheses, measures, and analytical plan were pre-registered with the Open Science Framework (https://osf.io/ajvf8/?view_only=0768c576977545a9b46bc3e836108fa1), except when indicated. The non-preregistered analyses should be considered exploratory. In the following report, I used r_{GE} to indicate theoretical specification of gene-environment correlation, r_{AC} and r_{AE} refers to latent correlation between genetic-shared, and genetic-nonspecific environmental influence.

I conducted a power simulation as part of the pre-registration plan. Simply put, the power estimations for the ACE model were delicate and sensitive to different parameters during different phases (see Appendix C for power simulation parameters and estimates). The power simulation estimates (given $\alpha = .05$) was organized in a decision flowchart (see Figure 7.1)

7.2.1 Incorporating PGI to estimate r_{GE}

The idea to directly estimate r_{GE} relies upon adding measured genetic components (i.e., PGI) to the classical twin model (Dolan et al., 2021). This model operationalized total additive genetic variance as a composite of PGI variance (e.g., A_{p1}) and the latent residual variation due to heritability not captured by the PGI (e.g., A_{q1} ; See Figure 7.2 (A)). As our model incorporated a wide-format design, the PGI was included as a time-invariant variable at both waves, whereas the residual genetic components were estimated at both wave identical to which described in Chapter 5.2. For model identification purposes, I fixed the predictability of PGI to the average estimation from the overall twin sample. For polygenic similarity among DZ twins (i.e., IBD), I also fixed the parameter to empirical estimates as reported in Table 5.4. Examples of model specifications for r_{AC} and r_{AE} was shown in Figure 7.2.

Inclusion of r_{GE} can have important ramification for the definition of heritability, especially under the classical twin modeling framework. Take r_{AE} as an example. In the classical twin design, the variance in a phenotype is decomposed as $Var(P) = Var(A) + Var(C) + Var(E)$, where the genetic factor (A) captures components that makes MZ twins more similar than DZ twins on a specific phenotype, including environmental mediated effects such that similarity among MZ twins was magnified as they selected into more congruent environment. Alternatively, with the explicitly modeled genetic-environment covariation (covAE), such that: $Var(P) = Var(A) + Var(C) + Var(E) + 2 * covAE^6$, the $Var(A)$ would then be estimated most closely to its definition as variance due to genetic effects. Note that it is not yet possible to estimate both cov(AC) and cov(AE) in the model, I therefore set one of the covariation to equal zero when estimating the alternative parameter.

It is important to differentiate the effect of unmodeled r_{GE} on parameter estimates across different methods. Passive r_{GE} (or r_{AC} in the current model) would contribute to C estimate in biometric model, but it may contribute to the PGI predictability in model incorporating molecular genetic variants.

6 For model identification purposes, I used AE model to estimate contribution of covAE in all phenotypes.

7.2.2 Incorporating SES to estimate rAC

For specific contexts contributing to shared environment component (C) for cognitive ability and socioecological contexts (i.e., family SES) accounted for large proportion of variance in C (~70%; Engelhardt et al., 2019). Given the large contribution of family SES to shared environment estimates, I conducted an exploratory analysis incorporating family SES (as opposed to PGI) in the twin model to estimate rAC (see Figure 7.1 (C)). Here, the measured family SES was the same as that described in Chapter 5, and the predictability of SES was fixed to the average estimates from the overall twin sample.

7.3 RESULTS

Augmented twin models incorporating PGI and SES, without modeling rGE , showed overall good model fit (CFIs > .97, TLIs > .98, RMSEA < .06; see Table 7.1). Point estimates for the A/C/E parameters fluctuate slightly across augmented models and standard solution, such that the C estimates for Gc was .123, .090, .134 in the standard, PGI incorporated, and SES incorporated model, respectively. However, the overlapping confidence intervals for those estimates suggested they were likely consistent across models.

7.3.1 Estimating rAC

Direct estimates of rAC by incorporating PGI or SES can be found in Table 7.2 and Table 7.37. I did not find consistent rAC across the phenotypes. Surprisingly, the covAC in crystalized intelligence did not contribute significantly to the phenotypic variance, where I expected to find the largest covariation. Both rAC for externalizing and internalizing were negative in the model with SES, suggesting possible mechanism for compensatory processes that work as protective factors against magnifying phenotypic differences. However, the confidence interval of those point estimates crossed zero, so we should take care when interpreting those results. The intended negative control of height (prorAC = -4.3% with PGI and 2.0% with SES) was not consistently weaker than that in other traits.

The null effect of rAC suggests either the absence of rGE or that the design suffered from a lack of power. Based on power simulation of a model incorporating PGI conducted prior to the validation on real data (see Appendix C), I did not have enough power to detect even large effects. For crystalized intelligence and height, where I have the best predictive power of PGI,

7 The model estimating covAC of externalizing (with PGI) did not converge.

the current design have $< 70\%$ power (at $\alpha = .05$) to detect passive gene-environment correlation even if the proportion of rAC contribution is 18%, which is roughly equivalent to correlations of .35 between latent genetic and shared environmental component. For externalizing and internalizing, where the PGIs explain less than 1% of the indicated phenotype's variance, the sample have less than 20% power to detect rAC as large as contributing to 18% of the total variance.

For our exploratory analyses incorporating SES in twin model, a post-hoc power analysis indicated that with our current sample size and alpha, the sample could have detect proportion of $rAC = 16\%$ when $C = .2$, however, the power to detect negative rAC was relatively low across parameter settings $< 20\%$ (see Appendix C).

7.3.2 Estimating rAE

Estimates of rAE on augmented AE model for all seven phenotypes were presented in Table 7.4. The data suggested positive signals for rAE in planning ($r = .159$) and perseverance ($r = .200$), in line with our hypothesis. Each contributing to $\sim 13\%$ of the total variance and accounting for 31% and 48% percent of the heritability obtained from standard solution. Residualized heritability now accounted for 28% and 13% of total variance. Power analysis suggested that with current sample size and $R^2_{PGI} > 0.5\%$, I would be 80% powered to detect $rAE = .1$, and 90% powered to detect $rAE = .3$ (when heritability $> 30\%$).

For crystalized intelligence and psychopathological traits, the rAE was virtually null. The seemingly modest rAE in externalizing ($r = .118$) and internalizing ($r = -.340$) was qualified by large confidence intervals. The model also had lower power to detect negative rAE , such that I would be $\sim 50\%$ powered to detect $rAE = -.3$ with $R^2_{PGI} \sim 2\%$ and residualized heritability = 60%.

7.4 SUMMARY

Overall, the results showed limited support that standard twin heritability/shared environmental estimates across a range of psychological and behavior characteristics are upwardly biased by failing to address the covariance between genes and environment. However, the current sample with the design is subjected to high Type I error, that I have relatively low power ($< 70\%$) to detect such dynamic interplay in this current design, especially for latent genetic-shared environment correlation. Thus, additional data is necessary to better inform models of gene-environment interplay.

Whether or not more accurate PGI would lead to different results is an open question. However, empirical evidence has emerged in support of *r*GE playing a role in human development, especially in cognitive relevant traits. Specifically, a recent meta-analysis on genetic nurture effect of child educational outcome (Wang et al., 2021) found that the indirect genetic effects (e.g., passive *r*GE; $\beta = .08$) was roughly half the size of direct genetic effects (e.g., SNP heritability; $\beta = .21$). Moreover, adoption studies comparing the associations of parental PGI with bio/adopted children's educational outcome also found that the predictability of PGI in adoption sample was about half the size in biological offspring samples (Domingue & Fletcher, 2020). Those results also triangulated with the family-based designs (Selzam et al., 2019) to suggest that genetic nurturing accounted for roughly half of the measured heritability.

Importantly, the scope of current investigation is largely limited by the extent that the PGI accurately captures heritability and the magnitude of shared environment factor. The latter issue, the downstream consequences of low C estimates, became pronounced in the case of crystalized intelligence. For example, past research finds that estimates of shared environment influences in cognitive ability ranged from 10%-40% (Briley & Tucker-Drob, 2013; Tucker-Drob & Briley, 2014; Vogt et al., 2021), yet the C estimate in current study hit the lower bound at 13%. One possibility for the relatively low C estimates may have to do with our homogenous sample. If participants do not vary on relevant shared environmental factors, then those potential causal factors would not be identified in a twin model. To maximize the predictive power of the PGI, the analyses were run on a racially homogenous group. In a study investigating the specific source of contexts contributing to C estimates in cognitive ability, Engelhardt and colleagues (2019) found that racial differences itself accounted for ~40% of shared environmental variance for crystalized intelligence. The remaining C components ranged from 13%~19% for crystalized intelligence, concordant with the point estimate of C (13%) in the current study.

The limitation of PGI predictability on accurately estimating *r*GE highlights the need for more powerful molecular genetic study designs specifically targeted at behavioral and psychological traits. The within-family study suggested there are trait-environment correlations guiding trait development. However, the results of our extended twin model did not support that conclusion with few exceptions. Future studies should use PGIs with greater predictive accuracy. Currently, GWAS are rapidly increasing in sample size and advancement in estimating joint genetic architecture of complex traits (e.g., Genomic SEM; Multi-polygenic score; Allegrini et

al., 2020; Grotzinger et al., 2019). The predictive accuracy of psychological and psychopathological related traits is likely to increase as well. For example, a recent published GWAS on externalizing in 1.5 million people found that the predictability of computed PGI was $\sim 9\%$ (Linnér et al., 2019), 45 times of what was the best predictive externalizing PGI in the current report.

7.5 FIGURES AND TABLES

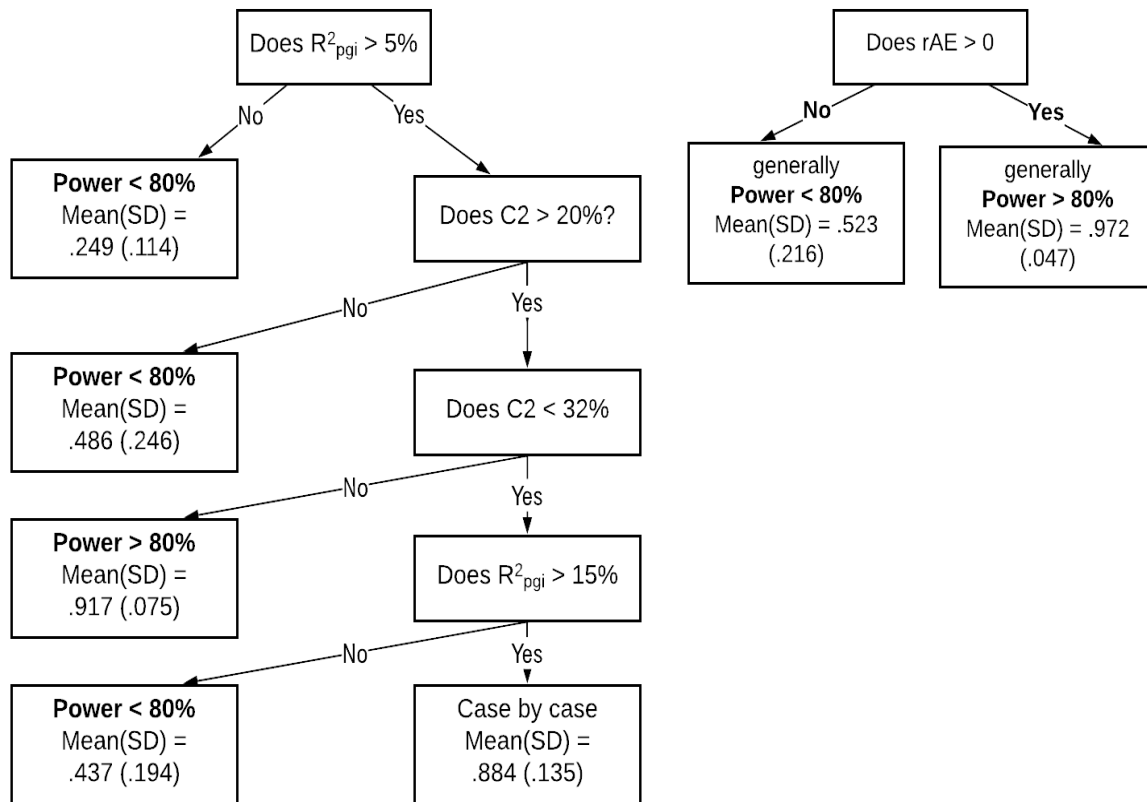


Figure 7.1. Power decision flowchart based on simulated results for ACE and AE model. The mean and standard deviation for the correspondingly filtered power estimations is presented in each box.

R^2_{PGI} =predictive validity of PGI, C2 refers to the shared environmentality in model without adjusting for rGE.

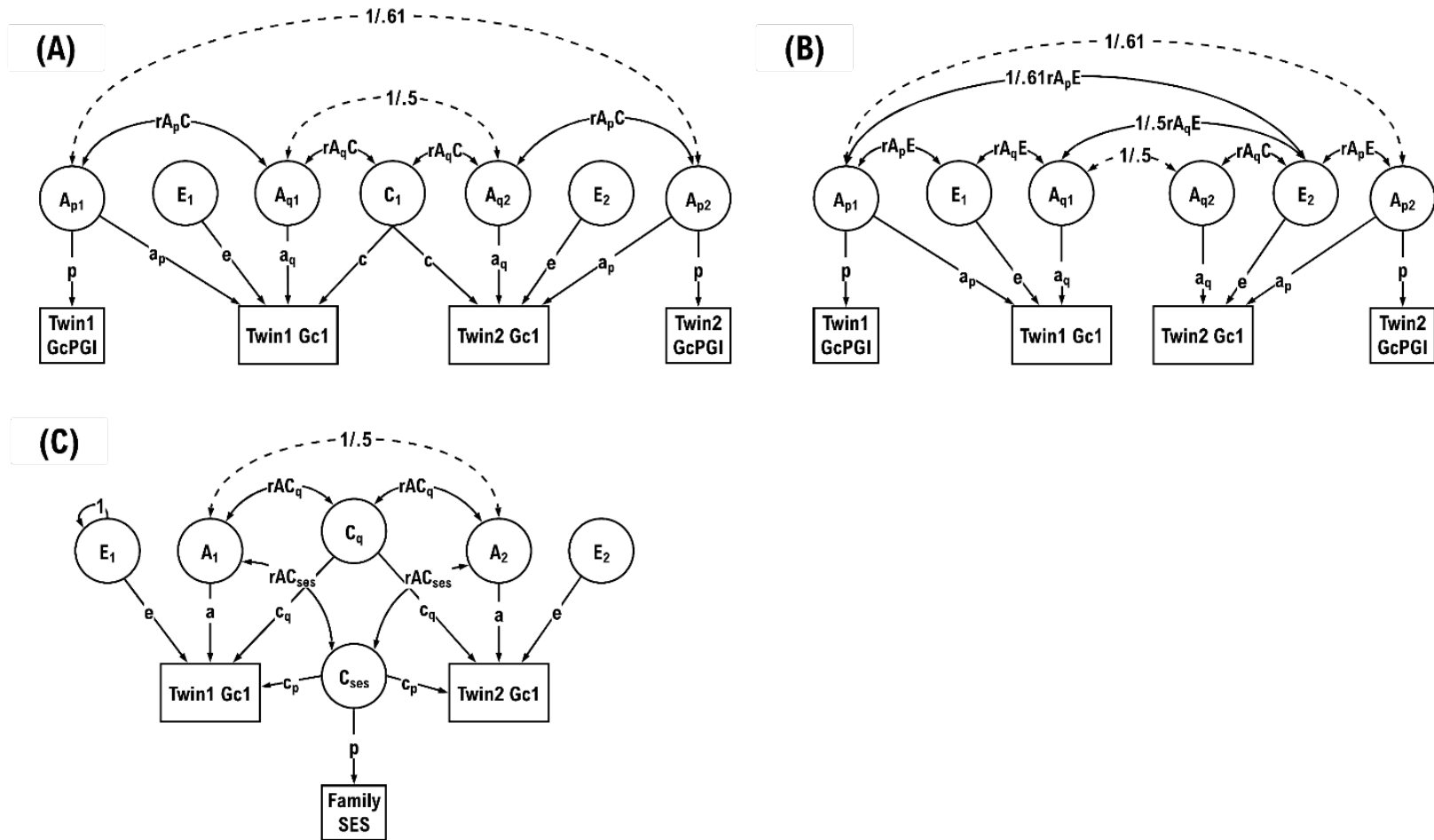


Figure 7.2. Extended twin model incorporating measured genetic effects and shared environmental effects. All models were displaying the first wave of data for simplicity. (A) shows the ACE model with PGI, (B) shows the AE model with PGI, and (C) shows the ACE model with SES.

Table 7.1. Augmented ACE/AE model

Variable	R2 (measured var)	IBD (DZ twin)	H2 (total)	C2	E2	CFI	TLI	RMSEA	SRMR	BIC
ACE with PGI										
Gc	0.0971	0.6139	.619(.093)	.090(.080)	.291(.029)	1	1	0	0.034	21332.68
Ext	0.0013	0.5113	.623(.092)	.083(.081)	.294(.027)	1	1	0	0.041	22893.233
Int	0.0085	0.5619	.328(.095)	.245(.078)	.426(.033)	0.994	0.996	0.028	0.059	23537.516
Ht	0.1687	0.5936	.863(.311)	.055(.296)	.082(.057)	0.973	0.981	0.054	0.043	13428.876
ACE with SES										
Gc	0.0788	-	.575(.161)	.134(.141)	.291(.058)	1	1	0	0.035	20613.498
Ext	0.0544	-	.613(.093)	.093(.081)	.294(.028)	1	1	0	0.042	21910.616
Int	0.0249	-	.315(.093)	.257(.076)	.428(.033)	1	1	0	0.04	22550.727
Ht	0.0015	-	.844(.073)	.074(.071)	.082(.012)	0.994	0.996	0.02	0.044	12586.776
AE with PGI										
Gc	0.0971	0.6139	.714(.029)	-	.286(.029)	1	1	0	0.036	21330.007
Ext	0.0013	0.5113	.706(.025)	-	.294(.025)	1	1	0	0.043	22881.105
Int	0.0085	0.5619	.598(.028)	-	.402(.028)	0.99	0.993	0.034	0.065	23534.896
Ht	0.1687	0.5936	.915(.015)	-	.085(.015)	0.968	0.979	0.056	0.043	13435.324
Pers	0.0064	0.5041	.406(.042)	-	.594(.042)	1	1	0	0.061	15551.964
Pln	0.0049	0.5099	.278(.046)	-	.722(.046)	0.991	0.994	0.028	0.065	15206.898
Sen	0.0059	0.5122	.228(.044)	-	.772(.044)	1	1	0	0.051	17494.794

Note. H2 displayed the total heritability attributable to both PGI (Ap) and residualized latent genetic effects (Aq).

Table 7.2. *r*AC estimated from ACE model including PGI

Phenotype	prcovAC	H2	C2	E2	H2 (ACE+PGI)	C2 (ACE+PGI)	E2 (ACE+PGI)	CFI	TLI	RMSEA	SRMR	BIC
Gc	-.024(.094)	.617(.086)	.107(.107)	.300(.028)	.619(.093)	.090(.080)	.291(.029)	0.998	0.999	0.016	0.043	28233.848
Int	-.005(.212)	.296(.097)	.273(.190)	.437(.034)	.292(.088)	.268(.073)	.439(.032)	0.994	0.996	0.027	0.058	23535.74
Ht	-.043(1.744)	.850(.581)	.112(1.853)	.080(.189)	.863(.311)	.055(.296)	.082(.057)	0.968	0.978	0.057	0.047	13439.344

Note. prcovAC = contribution of overall genetic-shared environment covariance to the total phenotype; H2(ACE+PGI) = heritability estimates without incorporating covAC

Table 7.3. *r*AC estimated with ACE model including SES

Phenotype	prcovAC	H2	C2	E2	H2 (ACE+SES)	C2 (ACE+SES)	E2 (ACE+SES)	CFI	TLI	RMSEA	SRMR	BIC
Gc	.046(.544)	.574(.089)	.096(.456)	.297(.028)	.575(.161)	.134(.141)	.291(.058)	1	1	0	0.039	20612.546
Ext	-.211(.280)	.656(.064)	.255(.254)	.299(.029)	.613(.093)	.093(.081)	.294(.028)	1	1	0	0.043	21897.282
Int	-.085(2.325)	.311(.093)	.343(2.304)	.431(.033)	.315(.093)	.257(.076)	.428(.033)	1	1	0	0.039	22542.526
Ht	.020(.446)	.850(.073)	.052(.492)	.079(.011)	.844(.073)	.074(.071)	.082(.012)	0.992	0.995	0.022	0.044	12587.661

Note. prcovAC = contribution of overall genetic-shared environment covariance to the total phenotype;

Table 7.4. *r*AE estimated from AE model including PGI

	R2					H2	E2					
	PGI (PGI)	<i>r</i>AE	prcovAE	H2	E2	(AE+PGI)	(AE+PGI)	CFI	TLI	RMSEA	SRMR	BIC
Gc	0.0971	-.030(.137)	-.027(.127)	.750(.127)	.277(.023)	.714(.029)	.286(.029)	1	1	0	0.036	21327.075
Ext	0.0013	.118(.989)	.100(.773)	.601(.773)	.299(.027)	.706(.025)	.294(.025)	1	1	0	0.042	22886.358
Int	0.0085	-.340(7.845)	-.446(1.437)	1.029(.066)	.417(1.495)	.598(.028)	.402(.028)	0.992	0.994	0.032	0.062	23536.085
Ht	0.1687	-.001(5.952)	.000(.026)	.915(.014)	.085(.026)	.915(.015)	.085(.015)	0.969	0.979	0.056	0.043	13435.324
Pers	0.0064	.159(.178)	.129(.117)	.279(.121)	.592(.042)	.406(.042)	.594(.042)	1	1	0	0.061	15551.389
Pln	0.0049	.200(.108)	.126(.049)	.134(.051)	.741(.043)	.278(.046)	.722(.046)	0.992	0.995	0.027	0.064	1520.406
Sen	0.0059	-.025(.192)	-.021(.170)	.225(.176)	.796(.041)	.228(.044)	.772(.044)	1	1	0	0.052	17491.403

Note. *r*AE = genetic and non-shared environment correlation; prcovAE = contribution of covAE to total phenotypic variance

CHAPTER 8: GENERAL DISCUSSION

The current project used two genetically informed methods (family-based analyses, extended classical twin analyses) to investigate the magnitude of gene-environment correlation in a sample of 9-year-olds. Triangulation of evidence showed little support for genetic-environment correlations (neither passive, evocative, active) for cognitive ability and height, mixed effects for psychopathological traits, and positive evocative/active r_{GE} for two out of three personality traits.

8.1 LACK OF RAC AND SOCIAL SAFETY NET

Contradicting past findings of genetic nurturing on cognitive ability and educational outcomes, which indicate positive genetic-shared environment correlation, both studies in this dissertation found null effects of r_{AC} . For within-between family design, the predictability of within-family effects of G_c recovered ~70% of that in unrelated individuals, showing only ~10% of reduction of that in between-family effects. For augmented twin analyses, I found null effect of latent genetic-shared environment correlation on variation of G_c . The latter analyses, though benefit from aggregating effects to maximize power, is constricted by high type I errors.

What could we learn from lack of evidence of genetic-shared environment correlation? To navigate through this question, let's first consider one of the obstacles hindering statistical power for the augmented twin analyses: relatively low C estimates for crystallized intelligence. Meta-analytical estimate of C for cognitive ability suggested 30~40% during early adolescent, whereas the point estimate in the current 9-year-olds is 13%. Theoretically, even when the objective shared environment is shared among siblings reared together, those shared environments could nonetheless have differing effects on each individual when choices and tangible outcomes are available. Alternatively, when social and economic barrier exists such that life opportunities depend more on familial or neighborhood's socioecological contexts, then siblings from the same background could end up more similar, with equally repressive environments depriving cognitive development. As such leveling down process could function through both environmental and parental pathway, suggesting passive r_{GE} , then the null finding of r_{AC} on cognitive ability could point to the possible counterbalancing mechanisms that interfere with the intergenerational cycle of educational underachievement. For example, a cross-country study examining moderating effect of economic resources on how closely students

interested in science pursued science knowledge found substantial reduction in the moderating effect of national economical level ($N > 500,000$; Zheng et al., 2019) over a decade, which is associated with the increase of resource accessibility over time. Thus, it may be the case that the connection between economic disparities and educational opportunities has weakened somewhat over the past several decades, which could act to reduce estimates of shared environmental influences.

Relating to the possibility that institutional opportunities and social safety net serve as gateway mechanisms to reinforce positive outcomes in cognitive traits, it is interesting that the extended twin model suggested null active/evocative rGE in sibling differentiation. The lack of rAE in crystallized intelligence may suggest an age-onset later than 9-11 years, such that active/evocative rGE may play a more salient role when children obtain agency to make education-relevant choices (e.g., match course-taking) that are supported by the social and institutional structure. Moreover, though a social system like compulsory education may serve as social scaffolds promoting positive socioeconomic and health outcomes, whether the current design also provides diversified environment that could enrich active person-environment and promote social mobility need further investigation.

8.2 WINDING PATH TO CONSOLIDATE PERSONALITY TRAITS

With augmented twin design, I found positive genetic-nonshared environment correlation (rAE) for two personality traits (i.e., perseverance and planning; $r_s \sim .17$), accounting for $\sim 40\%$ of the heritability estimates when rGE was unaccounted. These results served as the first direct estimates of active/evocative rGE for personality dimensions, indicating that individuals select or evoke environmental experiences as a systematic function of their genetically influenced traits (Plomin et al., 1977; Scarr & McCartney, 1983). Individuals follow diverse pathways to adulthood to consolidate personality traits. Put differently, rather than genetic influences being a purely “under-the-skin” phenomenon, “genetic” variance could also flow through environmental pathways.

Unlike the null finding of rAE in cognitive traits, the pattern of rAE in personality traits may be due to the societal and cultural capacity to accommodate broad ranges of environmental exposures that enrich positive gene-environment transactions (Briley & Tucker-Drob, 2013, 2017b; Dickens & Flynn, 2001). Importantly, these transactions likely happened frequently and repetitively to establish the accumulation process (Wrzus & Roberts, 2017). Otherwise, if the

experiences selected into or evoked from others are transient and wear off in a short time, then the accumulation is unlikely to manifest as trait-level change.

Note that the positive rAE during early adolescence did not necessarily contradict the meta-analytical finding where the phenotypic stability is primarily attributable to the nonshared environmental process. On the one hand, rAE could largely maintain, rather than reinforce or magnify, genetic influences, such that heritability would not increase as individuals navigate through environmental exposure. On the other hand, people may habituate to environmental stimuli, with self-selected environments serving to stabilize personality instead of evoking cumulative change (i.e., Niche-picking; Roberts & Nickel, 2017). Whatsmore, given the lack of institutionalized force that cultivates personality traits in a uniform direction (though socializing pressure such as maturation may be at play), personality development likely unfolded with intertwined mechanisms playing significant roles across different life stages.

For psychopathology, the model suggests the possibility that negative gene-shared environmental effects are working as protection factors from developing psychopathology. Parents with previous experience of psychopathology may be particularly aware of the detrimental effect of having psychopathological experience, and perhaps also the beneficial effects of therapy (Roberts et al., 2017). Note that the results are subject to speculation due to the limited power to detect likely effects.

8.3 FUTURE DIRECTION

Gene-environment interplay can have crucial consequences for researchers interested in drawing causal inferences from associations between environmental exposures and psychological, behavioral, and sociological outcomes. On the practical side, the extent to which rGE is associated with the phenotype could provide guidance on the target of intervention. Modeling rGE effect could provide strong evidence on the environmental effects, such as advance our knowledge on the way that social privilege is transmitted across generations. While the presented work in my thesis provided a holistic perspective on the extent to which genetic and environmental influence entangled to influence development, there are still many unanswered questions for future research.

Theoretically, rGE unfolds and iterates across development, rather than functioning as a static process as modeled here. Longitudinal twin models can advance our understanding of the phenotype-environment transaction by decomposing the E component as occasional-specific

effects and environment-mediated genetic pathways (Dolan et al., 2014b). In addition, future work can extend the augmented twin model on longitudinal design and examine the contribution of r_{GE} on stability and change of traits. Another avenue of research concerns the direction of causal association for a genetic-environment transaction. In my within-family studies, it is equally possible that PGI-environment associations are due to nonrandom exposure of environment functioning on the basis of genetically dependent traits, or, that environmental resources are distributed in line with societal inequity. Designs such as the direction-of-causation model using cross-sectional twin/sibling design (Heath et al., 1993) or Mendelian randomization using instrumental variables (Davey Smith & Ebrahim, 2003) could better understand possible mechanisms linking environmental exposures and outcomes.

It would be helpful to increase the analyses' power to build on the work I present here. For example, as I do not detect large shared environmental influences of crystallized intelligence, future studies could utilize a larger sample or family from more diverse socioeconomic backgrounds to increase power. In addition, the scope of the current investigation is substantially limited by PGI predictability on accurately estimating r_{GE} , in both within-family and extended twin model design. This challenge highlights the need for a more powerful molecular genetic study design targeted explicitly at behavioral and psychological traits. Multivariate approaches to polygenic prediction are continuously developed to improve PGI predictive power by leveraging pleiotropy across complex traits. For example, genomic structural equation modeling (e.g., Genomic SEM; Grotzinger et al., 2019) estimate joint SNP effects for multiple related traits. A recent GWAS using genomic SEM on externalizing in 1.5 million people found that the predictive power of PGI was $\sim 9\%$ (Linnér et al., 2019), 45 times of what was the best predictive externalizing PGI in the current report.

8.4 CONCLUSION

In conclusion, the work presented addressed different forms of genetic-environment correlations using novel approaches and multiple phenotypes. The findings of this thesis illustrate the potential role of genetic-environment correlation, especially active/evocative genetic-environment correlations across a range of complex traits. Many questions remain unanswered. Incorporating polygenic index in biometric model represents a promising area for future research.

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APPENDIX A: HERITABILITY ESTIMATES FOR COGNITIVE ABILITY, PERSONALITY, PSYCHOPATHOLOGY, AND HEIGHT

Behavior Genetic Research on Height

As a classical anthropometric trait with relative ease of measurement, height has been a target of extensive research. In fact, human height was among the earliest phenotypes studied by quantitative genetics as early as the 19th century. Galton published data in the 19th century on the strong relationship between parent and offspring height (Galton, 1886). Since then, lines of evidence from differing designs (e.g., twins, families and adoption studies) have demonstrated the important roles of genetic factors on the determination of height. Heritability estimates obtained from twin studies are between .70 to .90 of the variation in height at the population level, with an average estimation of .8 (Dubois et al., 2012; Jelenkovic et al., 2016; Polderman et al., 2015). The rest of the variance was split between the shared environmental factor ($\sim .1$) and the non-shared environmental factor ($\sim .1$). Here, the active ingredient of the shared environment may coincide with the objective environment shared among family members, such as level of nutrition, dietary protein, and infection history in the region. Strong evidence links nutrition with height (Prentice et al., 2013), and as such, it may be surprising that relatively little variance is associated with the shared (or nonshared) environment. In part, the result is likely due to the majority of families in the relevant studies having sufficient nutrition. For example, the common environmental estimates of height were higher ($\sim .24$) and the heritability estimates were lower in less-developed East-Asian countries (Hur et al., 2008). Again, this result highlights the fact that heritability is not a fixed property of a phenotype but can vary corresponding to population-level differences in both genetic background and environmental variation (Visscher et al., 2008). Similar to the discovery timeline where quantitative genetics start off with studying height, GWAS on height also took off early and has identified the significant role played by genetic variants for adult height. The most recent GWAS meta-analyses in 693,529 individuals of European ancestry identified 3,290 genome-wide significant SNPs associated with height that together explained over 50% of the variance in height (Yengo et al., 2018). Additionally, a study using whole-genome sequencing data from 44,126 individuals found genetic variants (common and rare) that together explained 79% of variance in height (Wainschtein et al., 2019). These converging lines of evidence for the high heritability of height from the molecular genetic studies suggested that the missing heritability issue is negligible in human height and that additive

genetic model can explain the majority of the heritable variation in human height (Yang et al., 2015).

Behavior Genetic Research on Cognitive Ability

Cognitive ability is one of the most studied areas of behavioral genetics, in part motivated by its association with wide range of life outcomes such as interpersonal relationships, income, educational achievement, physical and mental health (Deary et al., 2010; Mollon et al., 2018; Richardson et al., 2012; Roberts et al., 2007). Cognitive ability is hierarchically organized and defined as the ‘mental capability... to reason, plan, solve problems, think abstractly, comprehend complex ideas, learn quickly and learn from experience (Gottfredson, 1997). General cognitive ability reflects the commonality among specific abilities such as verbal ability, spatial ability, memory, and abstract reasoning (Spearman, 1904).

The modern history of genetic studies of cognitive ability also traced back to Galton. As a man stimulated in studying eminence, Galton published the first article on heredity of human behavior in his study of mental ability (Galton, 1865). Following his advocacy on twin design and adoption design (Galton, 1876), Burks (1928) found the association of *g* in adoptive families were smaller than that in nonadoptive family. Research on twins reared apart also added evidence to this, as MZ twins reared apart were nearly as similar as MZ reared together (Burt, 1966). Moreover, MZ twins reared apart were about as similar on cognitive ability as the same individual measured twice. The evidence that *g* was heritable soon reached consensus based on studies involving over 10,000 twin pairs, 25,000 sibling pairs and 8,000 parent-offspring pairs (Bouchard & McGue, 1981; Plomin et al., 2008). Heritability estimates obtained on average 50% of the variation in cognitive ability, and the role of shared environmental factor is about 30%, leaving the rest of variance attributed to nonshared environment, including measurement errors. The phenotypic similarity of cognitive ability was also found among genetically similar unrelated individual, where SNP based heritability estimates on cognitive ability are generally half the twin-based heritability (~25%, Plomin & von Stumm, 2018a).

The heritability of cognitive ability has brought in some controversial claims, starting in 1969, with Jensen’s publication (Jensen, 1969) reasoned the ethnic differences observed in cognitive performance test scores involve genetic differences. Similar argument resurfaced in the publication of *The Bell Curve* (Herrnstein & Murray, 1994), which drew much criticism to behavioral genetic research as a field. Yet even in an impossible world where the genetic effects

and environmental effects are truly independent, and the cognitive performance test scores are perfectly equivalent between groups, such claims would not hold as predictors of individual differences within groups have little implications to the factors mattered for differences between groups. Heritability, measuring the proportion of phenotypic variance accounted for by genetic effects in a specific group, is looking at within-group differences.

Behavior Genetic Research on Personality

Personality refers to the characteristics that reflect patterns of thinking, feeling, motives, and behaviors that are relatively enduring across time and context (McAdams & Pals, 2006; McCrae & Costa, 2008). Those enduring patterns include general disposition (e.g., personality traits), motivational traits (e.g., goal orientation), and narratives (e.g., life narratives; Sackett et al., 2017; Wrzus & Roberts, 2017b). The following proposal focuses on studies of personality traits for simplicity, but the framework also applies equally well to other personality domains. Personality traits are thought to be organized hierarchically, with the most widely endorsed personality model capturing five broad personality dimensions (e.g., Extraversion, Agreeableness, Conscientiousness, Neuroticism, Openness) operating at the highest level (Goldberg, 1990; McCrae & John, 1992). Those overarching traits are thought to be subdivided into narrower facets or aspects (Costa & McCrae, 2008), which can be further divided into nuances (McCrae, 2015). Though most of the behavioral genetic research on personality focused on the broad domains, the magnitude of heritability estimates are consistent across structures at all levels of the personality hierarchy, especially when reliabilities were accounted for (Möttus, Sinick, et al., 2019; Turkheimer et al., 2014). Cross-sectional genetic research on personality traits across adoption, family, and twin studies provide a consistent pattern of results: heritability estimates and the non-shared environmental factor each account for approximately 50% of the variance in personality (Plomin et al., 2008). Shared environmental factors appear to make little or no contribution to individual differences in personality. Two recent systematic reviews of heritable contributions to individual differences in personality returned similar results. (Vukasović & Bratko, 2015) meta-analyzed 62 twin, adoption, and family studies of personality traits and found the heritability estimate of approximately 40%. In a meta-analysis of twin studies of all human traits, (Polderman et al., 2015) derived averaged heritability estimates of personality at 47%. Though the majority of twin research is based on self-report assessments of

personality, there are numbers of studies using other-rated personality traits and the results are consistent with those based on self-report (e.g., (Möttus, Briley, et al., 2019).

As aforementioned, the evidence on genetic structure of personality remains far behind other phenotypes even though there is ample evidence of its heritability. Neuroticism was the first personality-related GWAS target (Shifman et al., 2008) and remained as the most frequently tested personality trait, likely due to its relevance with common psychiatric disorders. (Okbay et al., 2016) conducted the first GWAS of personality (neuroticism) to identify SNP variants with extremely small effect size in a sample of 170,911 individuals. They identified 11 variants associated with neuroticism, and the SNP heritability was .91%. (Nagel et al., 2018) (2018) almost tripled the sample size to almost 450k and identified 136 variants associated with neuroticism, yet the aggregate SNP based heritability only accounted for 4% of the variance. The largest GWAS on personality to date investigated risk tolerance (N = 975,353) and adventurousness (N = 557,923) with SNP heritability at 4.6% and 9.8% respectively (Karlsson Linnér et al., 2019), where the vast majority of heritability still remained missing (Manolio et al., 2009). One possibility contributing to missing heritability, is again the inability to detect small-effect SNPs with given sample size. Indeed, using statistical methods to extrapolate SNP heritability indicates that the aggregated effect of all of the SNPs could account for approximately 10%~15% of the personality trait variance (Sanchez-Roige et al., 2018). Other alternatives are that personality dimensions as constructed phenotypically do not match the genetic structure, that dominant genetic effects play an important role (e.g., MZ twins tend to be more than twice as similar as DZ twins), and complex patterns of gene-environment interplay.

Behavior Genetic Research on Psychopathology

Psychopathology is one of the leading causes of worldwide disability and self-harm behaviors (Vos et al., 2020). One extraordinary challenge in psychiatric diagnosing and intervention is the prevalence of comorbidity, or the presence of multiple disorders concurrently (Newman et al., 1998). Roughly 50% of respondents met criteria for one diagnosis would also meet a criteria for a second disorder (Bijl et al., 1998). Such high comorbidity has added on to the surging debates surrounding the current practice of diagnosing psychopathology (Kotov et al., 2017; Markon et al., 2011; Zimmerman et al., 2015). Indeed, empirical evidence has accrued to suggest that psychopathology is organized hierarchically and one's liability to mental disorder

can be measured by a single dimension—p factor (Caspi et al., 2014; Caspi & Moffitt, 2018; Kotov et al., 2017).

Twin and family studies added to this line of research showing that the heritability estimates were shared among different disorders (Kendler et al., 2011; Smoller et al., 2019). For example, Pettersson et al (2016) used a national register data from over 1 million sibling pairs, and found a general genetic factor accounted for 15%~36% of the observed phenotypic variance among 8 psychiatric disorders (depression, anxiety, ADHD, alcohol use disorder, drug abuse, violent crime, schizophrenia, and schizoaffective disorder). Across the psychiatric disorder, shared environmental factors were largely absent (Bornovalova et al., 2010; Kato et al., 2009). Moving to the molecular level, genetic effects of most phenotypes are both polygenic and pleiotropic, meaning that a trait is affected by many genetic variants (with small effects), and that a genetic variant may affect multiple traits (Visscher et al., 2017). GWAS sought to capitalize on this shared genetic architecture and growing body of evidence indicating that different mental disorders indeed shared common genetic variants. Lee et al (2019) meta-analyzed over 700k participants and identified 109 variants associated with the commonality among 8 psychiatric disorders. The SNP heritability of the general psychopathology accounted for 14.6% of the phenotypic variance.

APPENDIX B. ADDITIONAL INFORMATION ON MEASURES

Factor score solution for personality constructs

To test the structural model, I used a CFA model including three latent factors representing each of the personality constructs on the first wave of data. Each latent factor was indicated by four corresponding items. The final CFA model, which allowed for two correlations within subscale items (i.e., item 1, 4 from planning, and item 3,4 from sensation-seeking subscale) following modification indices suggestion, fitted the data well (wave1 CFI = .964, wave2 CFI = .970). Table B.1 reported the factor loadings and phenotypic correlations among latent factors at wave 1. Factor correlations were modest between sensation seeking and the other two factors, and moderate between planning and perseverance ($r = .580$). This correlation implies that children who tend to plan ahead also tend to maintain their focus on tasks.

Table B.1. Factor loadings for the three personality constructs (N = 7067)

Construct	Item	Sensation	Planning	Perseverance
sensation seeking	I enjoy taking risks.	0.633		
	I like new, thrilling things, even if they are a little scary.	0.468		
	I would like to learn to fly an airplane. *	0.13		
	I would like to ski very fast down a high mountain slope. *	0.409		
perseverance	I finish what I start.		0.532	
	I tend to get things done on time.		0.661	
	I am a person who always gets the job done.		0.733	
	I almost always finish projects that I start.		0.577	
planning	I try to take a careful approach to things.*			0.534
	I am very careful.			0.64
	I tend to stop and think before doing things.			0.683
	I like to stop and think about things before I do it.*			0.623
Sensation seeking		1		
perseverance		0.065	1	
planning		-0.288	0.58	1

Note. Item with '*' indicates they were allowed to correlate with the other item from the same subscale.

Next I tested for configural, metric, and scalar variance across two waves. The model suggested good configural invariance (CFI = .967, TLI = .955, RMSEA = .037, SRMR = .035). For metric structure, the full measurement invariance did not hold, as indicated by the significant result of chi-square difference testing ($\chi^2_{(9)} = 38.020$, $p = .000$) using the Satorra-Bentler scaled

chi-square (Satorra & Bentler, 2010). Lack of invariance was largely due to two items from the perseverance subscales, “I finish what I start” and “I almost always finish projects that I start”, both showed stronger loading at Wave 3. I then freed the loading of those two items in the third wave, leading to comparable model fit in metric ($\chi^2_{(7)} = 9.674$, $p = .208$) and scalar level of analysis ($\chi^2_{(19)} = 11.575$, $p = .903$). After partial measurement invariance had been established across two waves, I saved the factor scores for each one of the three personality constructs and included them in following biometric analyses.

After establishing the structural model of the perseverance, planning, and sensation seeking at the first wave, I then tested for measurement invariance across Waves 1 and 3 by subsequently testing the configural, metric, and scalar structure of the tested factor model. After establishing measurement invariance or partial invariance across waves, I saved the factor scores of the three latent constructs at both waves for future analyses.

Polygenic index using PRSice2

For the PRSice2 pipeline, I used the LD clumping window of 250kb and $r^2 = .01$. The polygenic indexes were scored for each phenotype first with the singleton sample by summing the number of effect alleles present for each SNP (0,1 or 2) weighted by the beta of that SNP from the GWAS discovery sample. For the varying p-value thresholds, I called for ‘high resolution scoring’ in PRSices2, which automatically calculated polygenic indexes across a large number of thresholds and returned the best-fitted (i.e., with the highest predictability) threshold. Then I restricted scoring in the sibling sample to the specified best-fitted threshold and the corresponding SNPs with command --bar-level and --extract. Table B.2 reported the selected threshold and the polygenic R2 in the sibling sample.

Table B.2. GWAS summary and PGI predictability using LDpred2

Phenotype for PGI	Author (year)	N total	PGI R2 (twin; LDpred2)
<i>Crystal Intelligence</i>			
Educational attainment	Lee et al. (2018)	766,344	0.09309468247
Intelligence	Savage et al. (2018)	269,867	0.09711929298
<i>Externalizing</i>			
ever smoker	Liu et al. (2019)	632,802	0.00158410632
Worry	Nagel et al. (2018)	348,219	0.00125146148

Table B.2. (cont.)

<i>Internalizing</i>			
ever smoker	Liu et al. (2019)	632,802	0.00097975873
Worry	Nagel et al. (2018)	348,219	0.00853246414
<i>Perseverance</i>			
Risk_Pc1	Linner et al. (2019)	315,894	0.00086097383
ever smoker	Liu et al. (2019)	632,802	0.00635709124
<i>Planning</i>			
Risk_Pc1	Linner et al. (2019)	315,894	1.66E-05
ever smoker	Liu et al. (2019)	632,802	0.00485458602
<i>Risk seeking</i>			
Risk_Pc1	Linner et al. (2019)	315,894	0.00586030697
Risk tolerance	Linner et al. (2019)	466,571	0.00315432323
ever smoker	Liu et al. (2019)	632,802	0.00089471059
<i>Height</i>			
Height	Yengo et al. (2018)	693,529	0.1686532182

Note. Bolded PGI were used in further analyses. Estimates were from models with just the polygenic index as the exogenous variable, and no other covariates were included.

Table B.3. Descriptive statistics for environment variables included in the study (European)

Variables	Dataset	Index	Acronym	N	Mean (SD)	Median	Range	Skew	Kurtosis	Description	Note
Caregiver education	abcd_lpds01	demo_prnt_ed_v2_1	parEdu	5367	17.49 (2.04)	18	6-21	-0.99	1.36	What is the highest grade or level of school you have completed or the highest degree you have received?	Range 0-21
Family total income		demo_comb_income_v2_1	totIncome	5139	8.09 (1.79)	9	1-10	-1.63	2.91	What is your total combined family income for the past 12 months?	Range 1-10
Financial difficulty		demo_fam_exp1_v2_1 (~exp7)	sesDfcl*	5391	0.31 (0.95)	0	0-7	3.89	17.29	7 questions describing significant financial difficulty associated with being able to afford living functionality	Needed food but couldn't afford it
Family conflict	abcd_sscey01	fes_y_ss_fc_pr	famConflict	5591	1.93 (1.95)	1	0-9	1.05	0.49	Total number of family conflict event (0/1) occurred in the past 12 months	We fight a lot in our family.
Parent monitor		pmq_y_ss_mean	parMonitor	5594	4.42 (0.48)	4.6	1.8-5	-1.11	1.6	Mean of 5 items (1-4) accessing parent's effort to keep track of child's condition	How often do your parents know who you are with when you are not at school and away from home?
Family acceptance		crpbi_y_ss_parent	famAccept+	5588	2.79 (0.29)	3	1-3	-1.82	3.75	Mean of 5 items (1-4) describing primary caregivers' warmth behavior	[First caregiver] makes me feel better after talking over my worries with him/her
School environment	abcd_sscey01	srpf_y_ss_ses	schEnv	5591	19.87 (2.72)	20	6-24	-0.82	1.03	Sum of 6 items (1-4) reflecting positive school environment	In my school, students have lots of chances to help decide things like class activities and rules.

Table B.3. (cont.)

School involvement		srpf_y_ss_iiss	schInvolv	5591	12.99 (2.35)	13	4-16	- 0.85	0.6	Sum of 4 items (1-4) reflecting positive involvement at school	I like school because I do well in class.
School disengagement		srpf_y_ss_dfs	schDisengage	5591	3.79 (1.43)	4	2-8	0.61 0.07	-	Sum of 2 items (1-4) reflecting alienation from academic goals	Usually, school bores me.
Physical exercise	abcd_yrb01	physical_activity1_y	phyExercise	5591	3.73 (2.29)	4	0-7	- 0.03	- 1.21	Total number of days staying physically active for > 60 mins/day in the past 7 days	During the past 7 days, on how many days were you physically active for a total of at least 60 minutes per day?
Screen time- Weekday	abcd_ssmt01	stq_y_ss_weekday	screenWeek*	5593	2.94 (2.66)	2	0-24	1.93	5.73	Self-report of 14 question divided by weekdays and weekends; Questions asked how many hours on a typical weekday/weekend day the child uses screens for different types of media (not for educational purpose), with responses ranging from 0h (0) to 4+h (4). There are 6 forms of recreational media use: TV shows or movies, videos (i.e., youtube), video games, texting, social media, or video chat. Total screen time per weekend/weekday is used.	
Screen time- Weeknd		stq_y_ss_weekend	screenWnd*	5592	4.17 (3.23)	3.25	0-24	1.68	4.23		
Child-report neighborhood crime	abcd_nsc01	neighborhood_crime_y	nbhdCrime	5593	4.16 (0.99)	4	1-5	-1.1	0.7	Child self-report on safety of neighborhood	My neighborhood is safe from crime (1-5)
Area deprivation index	abcd_rhds01	reshist_addr1_adi_perc	API	5376	33.78 (23.45)	29	0-100	0.8	0.05	National percentiles for area deprivation, higher means higher value of ADI	
Neighborhood crime-linked		reshist_addr1_grndtot	totCrime*	5376	27676.27 (28043.47)	20421.33	0-159207	1.46	1.85	Uniform crime report from FBI (3yrs average from 2010-2012)	
Population density		reshist_addr1_popdensity	popDensity*	5376	1689.02 (1398.04)	1412.85	0-10252.17	1.6	4.33	Census tract level of population density (based on 2010)	
Level of pollution (pm25)		reshist_addr1_pm252016aa	pm25	5374	7.42 (1.5)	7.52	2.26-15.9	- 0.06	0.39	Residential history derived - annual average of PM 2.5 in 2016 at primary residential address at 1x1km2	
Risk of exposure (lead)		reshist_addr1_leadrisk	lead	5374	4.54 (2.9)	4	0-10	0.44	- 1.11	Estimated lead risk in census tract of primary residential address (1-10 scale) (1-10)	

Note. Variables with * were square rooted, and variable with + was squared to transform the variables

Table B.4. Descriptive statistics for environment variables included in the study (Non-European)

Variables	Acronym	N	Mean (SD)	Median	Range	Skew	Kurtosis
Familial							
Caregiver education	parEdu	3161	15.35 (3.06)	15	1-21	-0.79	1.03
Family total income	totIncome	2761	5.84 (2.69)	6	1-10	-0.3	-0.97
Financial difficulty	sesDfcl*	3191	0.74 (1.33)	0	0-7	2.01	3.68
Family conflict	famConflict	3490	2.22 (1.98)	2	0-9	0.78	-0.08
Parent monitor	parMonitor	3491	4.33 (0.57)	4.4	1-5	-1.08	1.34
Family acceptance	famAccept+	3483	2.76 (0.32)	2.8	1-3	-1.71	3.23
School							
School environment	schEnv	3490	19.99 (3.05)	21	6-24	-0.97	1.12
School involvement	schInvolv	3490	13.19 (2.41)	14	4-16	-0.91	0.55
School disengagement	schDisengage	3490	3.69 (1.51)	4	2-8	0.7	0.03
Physical							
Physical exercise	phyExercise	3485	3.12 (2.32)	3	0-7	0.27	-1.12
Screen time-Weekday	screenWeek*	3489	5.51 (4.1)	4.5	0-24	1.31	2.01
Screen time-Weeknd	screenWnd*	3490	4.32 (3.59)	3.25	0-24	1.51	2.79
Neighborhood							
Child-report neighborhood crime	nbhdCrime	3488	3.76 (1.24)	4	1-5	-0.72	-0.52
Area deprivation index	API	3294	50.86 (31.24)	47	0-100	0.1	-1.27
Neighborhood crime-linked	totCrime*	3294	39837.36 (35183.12)	31626.67	0-163414.33	0.78	-0.38
Population density	popDensity*	3294	2859.93 (2091.79)	2386.04	0-10261.62	1.12	0.96
Level of pollution (pm25)	pm25	3293	8.33 (1.49)	8.34	1.72-15.9	0.21	0.8
Risk of exposure (lead)	lead	3293	6.52 (3.02)	7	0-10	-0.39	-1.2

APPENDIX C: POWER SIMULATION

Table C.1. Power to reject the null hypothesis that proportion of rAC contribution is zero in ACE model with PGI ($\alpha = .05$)

power	vA	vC	vE	rAEnv	covAC	prcov	Average covAC	average_se	g	vt	r2
0.204	0.4	0.2	0.4	-0.3	-0.0849	-0.2044	-0.1001	0.0712	0.05	0.8303	0.0241
0.173	0.4	0.2	0.4	-0.2	-0.0566	-0.1276	-0.0608	0.05	0.1	0.8869	0.0451
0.854	0.5	0.2	0.3	0.25	0.0791	0.1365	0.0781	0.0269	0.4	1.1581	0.1727
0.196	0.5	0.2	0.3	0.3	0.0949	0.1595	0.0868	0.0828	0.05	1.1897	0.021
0.308	0.5	0.2	0.3	0.3	0.0949	0.1595	0.0914	0.0604	0.1	1.1897	0.042
0.622	0.5	0.2	0.3	0.3	0.0949	0.1595	0.0932	0.042	0.2	1.1897	0.0841
0.955	0.5	0.2	0.3	0.3	0.0949	0.1595	0.094	0.0277	0.4	1.1897	0.1681
0.226	0.5	0.2	0.3	0.35	0.1107	0.1812	0.102	0.0839	0.05	1.2214	0.0205
0.408	0.5	0.2	0.3	0.35	0.1107	0.1812	0.1072	0.061	0.1	1.2214	0.0409
0.734	0.5	0.2	0.3	0.35	0.1107	0.1812	0.1092	0.0429	0.2	1.2214	0.0819
0.982	0.5	0.2	0.3	0.35	0.1107	0.1812	0.1095	0.0287	0.4	1.2214	0.1638
0.264	0.5	0.2	0.3	0.4	0.1265	0.2019	0.1156	0.0842	0.05	1.253	0.02
0.326	0.4	0.2	0.4	-0.2	-0.0566	-0.1276	-0.0576	0.035	0.2	0.8869	0.0902
0.506	0.5	0.2	0.3	0.4	0.1265	0.2019	0.1227	0.0619	0.1	1.253	0.0399
0.831	0.5	0.2	0.3	0.4	0.1265	0.2019	0.1242	0.0437	0.2	1.253	0.0798
0.998	0.5	0.2	0.3	0.4	0.1265	0.2019	0.1255	0.0298	0.4	1.253	0.1596
0.312	0.5	0.2	0.3	0.45	0.1423	0.2216	0.1319	0.0853	0.05	1.2846	0.0195
0.594	0.5	0.2	0.3	0.45	0.1423	0.2216	0.1384	0.0624	0.1	1.2846	0.0389
0.904	0.5	0.2	0.3	0.45	0.1423	0.2216	0.1402	0.0446	0.2	1.2846	0.0778
0.998	0.5	0.2	0.3	0.45	0.1423	0.2216	0.1409	0.0309	0.4	1.2846	0.1557
0.719	0.4	0.2	0.4	-0.2	-0.0566	-0.1276	-0.0569	0.0239	0.4	0.8869	0.1804
0.986	0.5	0.3	0.2	-0.25	-0.0968	-0.2402	-0.0978	0.0256	0.4	0.8064	0.248
0.254	0.5	0.3	0.2	-0.2	-0.0775	-0.1833	-0.0822	0.0574	0.1	0.8451	0.0592
0.525	0.5	0.3	0.2	-0.2	-0.0775	-0.1833	-0.0787	0.0391	0.2	0.8451	0.1183
0.911	0.5	0.3	0.2	-0.2	-0.0775	-0.1833	-0.0783	0.0254	0.4	0.8451	0.2367
0.121	0.5	0.3	0.2	0.2	0.0775	0.1341	0.074	0.0896	0.05	1.1549	0.0216
0.12	0.4	0.2	0.4	0.2	0.0566	0.1016	0.0501	0.0729	0.05	1.1131	0.018
0.216	0.5	0.3	0.2	0.2	0.0775	0.1341	0.0767	0.0635	0.1	1.1549	0.0433
0.437	0.5	0.3	0.2	0.2	0.0775	0.1341	0.0764	0.0428	0.2	1.1549	0.0866
0.819	0.5	0.3	0.2	0.2	0.0775	0.1341	0.0768	0.0271	0.4	1.1549	0.1732
0.162	0.5	0.3	0.2	0.25	0.0968	0.1622	0.0919	0.0911	0.05	1.1936	0.0209
0.304	0.5	0.3	0.2	0.25	0.0968	0.1622	0.0948	0.0645	0.1	1.1936	0.0419
0.627	0.5	0.3	0.2	0.25	0.0968	0.1622	0.0962	0.0436	0.2	1.1936	0.0838
0.952	0.5	0.3	0.2	0.25	0.0968	0.1622	0.0961	0.0278	0.4	1.1936	0.1676
0.146	0.4	0.2	0.4	0.2	0.0566	0.1016	0.0543	0.0525	0.1	1.1131	0.0359
0.961	0.5	0.4	0.1	-0.2	-0.0894	-0.2179	-0.0905	0.0258	0.4	0.8211	0.2436
0.1	0.6	0.1	0.3	-0.2	-0.049	-0.1086	-0.0777	0.0784	0.05	0.902	0.0333
0.294	0.4	0.2	0.4	0.2	0.0566	0.1016	0.0554	0.0368	0.2	1.1131	0.0719
0.149	0.6	0.1	0.3	-0.2	-0.049	-0.1086	-0.0635	0.0566	0.1	0.902	0.0665

Table C.1. (cont.)

0.231	0.6	0.1	0.3	-0.2	-0.049	-0.1086	-0.0546	0.0391	0.2	0.902	0.133
0.538	0.6	0.1	0.3	-0.2	-0.049	-0.1086	-0.0505	0.0246	0.4	0.902	0.2661
0.172	0.6	0.1	0.3	0.2	0.049	0.0892	0.0267	0.0799	0.05	1.098	0.0273
0.147	0.6	0.1	0.3	0.2	0.049	0.0892	0.0372	0.058	0.1	1.098	0.0546
0.221	0.6	0.1	0.3	0.2	0.049	0.0892	0.0454	0.0401	0.2	1.098	0.1093
0.483	0.6	0.1	0.3	0.2	0.049	0.0892	0.0474	0.0254	0.4	1.098	0.2186
0.195	0.6	0.1	0.3	0.25	0.0612	0.1091	0.0383	0.0794	0.05	1.1225	0.0267
0.207	0.6	0.1	0.3	0.25	0.0612	0.1091	0.0511	0.0577	0.1	1.1225	0.0535
0.296	0.6	0.1	0.3	0.25	0.0612	0.1091	0.0562	0.0403	0.2	1.1225	0.1069
0.649	0.4	0.2	0.4	0.2	0.0566	0.1016	0.0559	0.0249	0.4	1.1131	0.1437
0.653	0.6	0.1	0.3	0.25	0.0612	0.1091	0.0595	0.0258	0.4	1.1225	0.2138
0.229	0.6	0.1	0.3	0.3	0.0735	0.1281	0.0506	0.0797	0.05	1.147	0.0262
0.247	0.6	0.1	0.3	0.3	0.0735	0.1281	0.0628	0.058	0.1	1.147	0.0523
0.419	0.6	0.1	0.3	0.3	0.0735	0.1281	0.0688	0.0406	0.2	1.147	0.1046
0.788	0.6	0.1	0.3	0.3	0.0735	0.1281	0.0716	0.0262	0.4	1.147	0.2092
0.253	0.6	0.1	0.3	0.35	0.0857	0.1464	0.0633	0.08	0.05	1.1715	0.0256
0.293	0.6	0.1	0.3	0.35	0.0857	0.1464	0.0757	0.0582	0.1	1.1715	0.0512
0.51	0.6	0.1	0.3	0.35	0.0857	0.1464	0.0806	0.041	0.2	1.1715	0.1024
0.887	0.6	0.1	0.3	0.35	0.0857	0.1464	0.0838	0.0266	0.4	1.1715	0.2049
0.269	0.6	0.1	0.3	0.4	0.098	0.1639	0.0759	0.0802	0.05	1.196	0.0251
0.14	0.4	0.2	0.4	0.25	0.0707	0.1239	0.0627	0.0735	0.05	1.1414	0.0175
0.342	0.6	0.1	0.3	0.4	0.098	0.1639	0.0867	0.0587	0.1	1.196	0.0502
0.618	0.6	0.1	0.3	0.4	0.098	0.1639	0.0924	0.0413	0.2	1.196	0.1003
0.952	0.6	0.1	0.3	0.4	0.098	0.1639	0.0957	0.0271	0.4	1.196	0.2007
0.298	0.6	0.1	0.3	0.45	0.1102	0.1806	0.0882	0.0803	0.05	1.2205	0.0246
0.406	0.6	0.1	0.3	0.45	0.1102	0.1806	0.0993	0.0589	0.1	1.2205	0.0492
0.692	0.6	0.1	0.3	0.45	0.1102	0.1806	0.1041	0.0418	0.2	1.2205	0.0983
0.976	0.6	0.1	0.3	0.45	0.1102	0.1806	0.1076	0.0276	0.4	1.2205	0.1966
0.205	0.4	0.2	0.4	0.25	0.0707	0.1239	0.067	0.0534	0.1	1.1414	0.035
0.958	0.6	0.2	0.2	-0.25	-0.0866	-0.2095	-0.0877	0.0257	0.4	0.8268	0.2903
0.196	0.6	0.2	0.2	-0.2	-0.0693	-0.1609	-0.1119	0.0904	0.05	0.8614	0.0348
0.213	0.6	0.2	0.2	-0.2	-0.0693	-0.1609	-0.0813	0.0614	0.1	0.8614	0.0697
0.389	0.6	0.2	0.2	-0.2	-0.0693	-0.1609	-0.0729	0.0411	0.2	0.8614	0.1393
0.826	0.6	0.2	0.2	-0.2	-0.0693	-0.1609	-0.0703	0.0256	0.4	0.8614	0.2786
0.141	0.6	0.2	0.2	0.2	0.0693	0.1217	0.0593	0.0916	0.05	1.1386	0.0263
0.169	0.6	0.2	0.2	0.2	0.0693	0.1217	0.0661	0.0653	0.1	1.1386	0.0527
0.327	0.6	0.2	0.2	0.2	0.0693	0.1217	0.0684	0.0443	0.2	1.1386	0.1054
0.708	0.6	0.2	0.2	0.2	0.0693	0.1217	0.0685	0.0275	0.4	1.1386	0.2108
0.168	0.6	0.2	0.2	0.25	0.0866	0.1476	0.0756	0.0914	0.05	1.1732	0.0256
0.437	0.4	0.2	0.4	0.25	0.0707	0.1239	0.069	0.0376	0.2	1.1414	0.0701
0.23	0.6	0.2	0.2	0.25	0.0866	0.1476	0.0824	0.0661	0.1	1.1732	0.0511
0.481	0.6	0.2	0.2	0.25	0.0866	0.1476	0.0845	0.0451	0.2	1.1732	0.1023
0.865	0.6	0.2	0.2	0.25	0.0866	0.1476	0.0857	0.0281	0.4	1.1732	0.2046
0.195	0.6	0.2	0.2	0.3	0.1039	0.1721	0.0917	0.0924	0.05	1.2078	0.0248

Table C.1. (cont.)

0.306	0.6	0.2	0.2	0.3	0.1039	0.1721	0.0998	0.0668	0.1	1.2078	0.0497
0.629	0.6	0.2	0.2	0.3	0.1039	0.1721	0.1021	0.0456	0.2	1.2078	0.0994
0.96	0.6	0.2	0.2	0.3	0.1039	0.1721	0.103	0.0287	0.4	1.2078	0.1987
0.22	0.6	0.2	0.2	0.35	0.1212	0.1952	0.1097	0.0927	0.05	1.2425	0.0241
0.394	0.6	0.2	0.2	0.35	0.1212	0.1952	0.1157	0.0675	0.1	1.2425	0.0483
0.736	0.6	0.2	0.2	0.35	0.1212	0.1952	0.1194	0.0463	0.2	1.2425	0.0966
0.369	0.4	0.2	0.4	-0.3	-0.0849	-0.2044	-0.0892	0.0505	0.1	0.8303	0.0482
0.82	0.4	0.2	0.4	0.25	0.0707	0.1239	0.0698	0.026	0.4	1.1414	0.1402
0.985	0.6	0.2	0.2	0.35	0.1212	0.1952	0.1204	0.0293	0.4	1.2425	0.1932
0.255	0.6	0.2	0.2	0.4	0.1386	0.217	0.1261	0.0939	0.05	1.2771	0.0235
0.492	0.6	0.2	0.2	0.4	0.1386	0.217	0.1338	0.0681	0.1	1.2771	0.047
0.831	0.6	0.2	0.2	0.4	0.1386	0.217	0.1364	0.047	0.2	1.2771	0.094
0.998	0.6	0.2	0.2	0.4	0.1386	0.217	0.1376	0.03	0.4	1.2771	0.1879
0.303	0.6	0.2	0.2	0.45	0.1559	0.2377	0.1434	0.0947	0.05	1.3118	0.0229
0.591	0.6	0.2	0.2	0.45	0.1559	0.2377	0.1518	0.069	0.1	1.3118	0.0457
0.9	0.6	0.2	0.2	0.45	0.1559	0.2377	0.1531	0.0478	0.2	1.3118	0.0915
0.999	0.6	0.2	0.2	0.45	0.1559	0.2377	0.155	0.0307	0.4	1.3118	0.183
0.173	0.4	0.2	0.4	0.3	0.0849	0.1451	0.0778	0.0741	0.05	1.1697	0.0171
0.29	0.4	0.2	0.4	0.3	0.0849	0.1451	0.0814	0.0541	0.1	1.1697	0.0342
0.936	0.6	0.3	0.1	-0.2	-0.0849	-0.2044	-0.0861	0.0262	0.4	0.8303	0.2891
0.112	0.6	0.3	0.1	0.2	0.0849	0.1451	0.0788	0.1001	0.05	1.1697	0.0256
0.216	0.6	0.3	0.1	0.2	0.0849	0.1451	0.0843	0.0701	0.1	1.1697	0.0513
0.448	0.6	0.3	0.1	0.2	0.0849	0.1451	0.0839	0.0468	0.2	1.1697	0.1026
0.829	0.6	0.3	0.1	0.2	0.0849	0.1451	0.0841	0.0287	0.4	1.1697	0.2052
0.151	0.6	0.3	0.1	0.25	0.1061	0.175	0.0999	0.1018	0.05	1.2121	0.0248
0.299	0.6	0.3	0.1	0.25	0.1061	0.175	0.1045	0.0711	0.1	1.2121	0.0495
0.625	0.6	0.3	0.1	0.25	0.1061	0.175	0.1051	0.0474	0.2	1.2121	0.099
0.957	0.6	0.3	0.1	0.25	0.1061	0.175	0.1054	0.0291	0.4	1.2121	0.198
0.201	0.6	0.3	0.1	0.3	0.1273	0.2029	0.1222	0.1019	0.05	1.2546	0.0239
0.591	0.4	0.2	0.4	0.3	0.0849	0.1451	0.0829	0.0388	0.2	1.1697	0.0684
0.423	0.6	0.3	0.1	0.3	0.1273	0.2029	0.1261	0.072	0.1	1.2546	0.0478
0.747	0.6	0.3	0.1	0.3	0.1273	0.2029	0.1261	0.0481	0.2	1.2546	0.0957
0.988	0.6	0.3	0.1	0.3	0.1273	0.2029	0.1266	0.0296	0.4	1.2546	0.1913
0.263	0.6	0.3	0.1	0.35	0.1485	0.229	0.1429	0.1036	0.05	1.297	0.0231
0.538	0.6	0.3	0.1	0.35	0.1485	0.229	0.147	0.0729	0.1	1.297	0.0463
0.858	0.6	0.3	0.1	0.35	0.1485	0.229	0.1473	0.0488	0.2	1.297	0.0925
0.998	0.6	0.3	0.1	0.35	0.1485	0.229	0.1478	0.0302	0.4	1.297	0.185
0.933	0.4	0.2	0.4	0.3	0.0849	0.1451	0.0838	0.0274	0.4	1.1697	0.1368
0.196	0.4	0.2	0.4	0.35	0.099	0.1653	0.0888	0.075	0.05	1.198	0.0167
0.376	0.4	0.2	0.4	0.35	0.099	0.1653	0.0956	0.0551	0.1	1.198	0.0334
0.713	0.4	0.2	0.4	0.35	0.099	0.1653	0.0972	0.04	0.2	1.198	0.0668
0.976	0.4	0.2	0.4	0.35	0.099	0.1653	0.0977	0.0289	0.4	1.198	0.1336
0.718	0.4	0.2	0.4	-0.3	-0.0849	-0.2044	-0.0855	0.0362	0.2	0.8303	0.0964
0.996	0.4	0.3	0.3	-0.3	-0.1039	-0.2624	-0.1045	0.0265	0.4	0.7922	0.202

Table C.1. (cont.)

0.376	0.4	0.3	0.3	-0.25	-0.0866	-0.2095	-0.0889	0.0516	0.1	0.8268	0.0484
0.721	0.4	0.3	0.3	-0.25	-0.0866	-0.2095	-0.0872	0.0365	0.2	0.8268	0.0968
0.984	0.4	0.3	0.3	-0.25	-0.0866	-0.2095	-0.0871	0.0255	0.4	0.8268	0.1935
0.144	0.4	0.3	0.3	-0.2	-0.0693	-0.1609	-0.078	0.0738	0.05	0.8614	0.0232
0.238	0.4	0.3	0.3	-0.2	-0.0693	-0.1609	-0.0704	0.0517	0.1	0.8614	0.0464
0.486	0.4	0.3	0.3	-0.2	-0.0693	-0.1609	-0.0702	0.0361	0.2	0.8614	0.0929
0.988	0.4	0.2	0.4	-0.3	-0.0849	-0.2044	-0.0852	0.026	0.4	0.8303	0.1927
0.883	0.4	0.3	0.3	-0.2	-0.0693	-0.1609	-0.0697	0.0246	0.4	0.8614	0.1857
1	0.4	0.4	0.2	-0.3	-0.12	-0.3158	-0.1208	0.026	0.4	0.76	0.2105
0.994	0.4	0.4	0.2	-0.25	-0.1	-0.25	-0.1007	0.0253	0.4	0.8	0.2
0.161	0.4	0.4	0.2	-0.2	-0.08	-0.1905	-0.0825	0.0758	0.05	0.84	0.0238
0.154	0.4	0.2	0.4	-0.25	-0.0707	-0.1647	-0.0815	0.0703	0.05	0.8586	0.0233
0.317	0.4	0.4	0.2	-0.2	-0.08	-0.1905	-0.081	0.0529	0.1	0.84	0.0476
0.606	0.4	0.4	0.2	-0.2	-0.08	-0.1905	-0.081	0.0367	0.2	0.84	0.0952
0.949	0.4	0.4	0.2	-0.2	-0.08	-0.1905	-0.0806	0.0248	0.4	0.84	0.1905
0.094	0.5	0.1	0.4	-0.2	-0.0447	-0.0982	-0.0669	0.0709	0.05	0.9106	0.0275
0.132	0.5	0.1	0.4	-0.2	-0.0447	-0.0982	-0.0544	0.0511	0.1	0.9106	0.0549
0.229	0.5	0.1	0.4	-0.2	-0.0447	-0.0982	-0.0492	0.036	0.2	0.9106	0.1098
0.498	0.5	0.1	0.4	-0.2	-0.0447	-0.0982	-0.0462	0.0235	0.4	0.9106	0.2196
0.144	0.5	0.1	0.4	0.2	0.0447	0.0821	0.0265	0.0721	0.05	1.0894	0.0229
0.155	0.5	0.1	0.4	0.2	0.0447	0.0821	0.0368	0.0517	0.1	1.0894	0.0459
0.211	0.5	0.1	0.4	0.2	0.0447	0.0821	0.0416	0.0362	0.2	1.0894	0.0918
0.254	0.4	0.2	0.4	-0.25	-0.0707	-0.1647	-0.0741	0.0501	0.1	0.8586	0.0466
0.447	0.5	0.1	0.4	0.2	0.0447	0.0821	0.0431	0.0238	0.4	1.0894	0.1836
0.176	0.5	0.1	0.4	0.25	0.0559	0.1006	0.0368	0.0725	0.05	1.1118	0.0225
0.188	0.5	0.1	0.4	0.25	0.0559	0.1006	0.0472	0.052	0.1	1.1118	0.045
0.304	0.5	0.1	0.4	0.25	0.0559	0.1006	0.0518	0.0366	0.2	1.1118	0.0899
0.624	0.5	0.1	0.4	0.25	0.0559	0.1006	0.0539	0.0243	0.4	1.1118	0.1799
0.206	0.5	0.1	0.4	0.3	0.0671	0.1183	0.0488	0.0719	0.05	1.1342	0.022
0.237	0.5	0.1	0.4	0.3	0.0671	0.1183	0.0582	0.0523	0.1	1.1342	0.0441
0.404	0.5	0.1	0.4	0.3	0.0671	0.1183	0.0624	0.037	0.2	1.1342	0.0882
0.763	0.5	0.1	0.4	0.3	0.0671	0.1183	0.065	0.0248	0.4	1.1342	0.1763
0.221	0.5	0.1	0.4	0.35	0.0783	0.1353	0.058	0.0731	0.05	1.1565	0.0216
0.517	0.4	0.2	0.4	-0.25	-0.0707	-0.1647	-0.0717	0.0356	0.2	0.8586	0.0932
0.286	0.5	0.1	0.4	0.35	0.0783	0.1353	0.0687	0.0527	0.1	1.1565	0.0432
0.499	0.5	0.1	0.4	0.35	0.0783	0.1353	0.0732	0.0375	0.2	1.1565	0.0865
0.871	0.5	0.1	0.4	0.35	0.0783	0.1353	0.0759	0.0254	0.4	1.1565	0.1729
0.258	0.5	0.1	0.4	0.4	0.0894	0.1517	0.0706	0.0723	0.05	1.1789	0.0212
0.342	0.5	0.1	0.4	0.4	0.0894	0.1517	0.0795	0.0529	0.1	1.1789	0.0424
0.615	0.5	0.1	0.4	0.4	0.0894	0.1517	0.0842	0.0378	0.2	1.1789	0.0848
0.94	0.5	0.1	0.4	0.4	0.0894	0.1517	0.0867	0.0261	0.4	1.1789	0.1697
0.28	0.5	0.1	0.4	0.45	0.1006	0.1675	0.0812	0.0733	0.05	1.2012	0.0208
0.397	0.5	0.1	0.4	0.45	0.1006	0.1675	0.0901	0.0536	0.1	1.2012	0.0416
0.692	0.5	0.1	0.4	0.45	0.1006	0.1675	0.0946	0.0385	0.2	1.2012	0.0832

Table C.1. (cont.)

0.907	0.4	0.2	0.4	-0.25	-0.0707	-0.1647	-0.0711	0.0249	0.4	0.8586	0.1864
0.974	0.5	0.1	0.4	0.45	0.1006	0.1675	0.0975	0.0269	0.4	1.2012	0.1665
0.991	0.5	0.2	0.3	-0.3	-0.0949	-0.2342	-0.0957	0.0257	0.4	0.8103	0.2468
0.167	0.5	0.2	0.3	-0.25	-0.0791	-0.1878	-0.0932	0.0763	0.05	0.8419	0.0297
0.287	0.5	0.2	0.3	-0.25	-0.0791	-0.1878	-0.0852	0.0554	0.1	0.8419	0.0594
0.578	0.5	0.2	0.3	-0.25	-0.0791	-0.1878	-0.0811	0.0381	0.2	0.8419	0.1188
0.942	0.5	0.2	0.3	-0.25	-0.0791	-0.1878	-0.0798	0.0252	0.4	0.8419	0.2376
0.137	0.5	0.2	0.3	-0.2	-0.0633	-0.1448	-0.0815	0.0794	0.05	0.8735	0.0286
0.114	0.4	0.2	0.4	-0.2	-0.0566	-0.1276	-0.0653	0.0702	0.05	0.8869	0.0226
0.195	0.5	0.2	0.3	-0.2	-0.0633	-0.1448	-0.0697	0.0555	0.1	0.8735	0.0572
0.372	0.5	0.2	0.3	-0.2	-0.0633	-0.1448	-0.0653	0.038	0.2	0.8735	0.1145
0.779	0.5	0.2	0.3	-0.2	-0.0633	-0.1448	-0.0639	0.0247	0.4	0.8735	0.229
0.14	0.5	0.2	0.3	0.2	0.0632	0.1123	0.0543	0.0823	0.05	1.1265	0.0222
0.152	0.5	0.2	0.3	0.2	0.0632	0.1123	0.0597	0.0592	0.1	1.1265	0.0444
0.318	0.5	0.2	0.3	0.2	0.0632	0.1123	0.0617	0.0406	0.2	1.1265	0.0888
0.69	0.5	0.2	0.3	0.2	0.0632	0.1123	0.0626	0.0261	0.4	1.1265	0.1775
0.164	0.5	0.2	0.3	0.25	0.0791	0.1365	0.0704	0.0825	0.05	1.1581	0.0216
0.23	0.5	0.2	0.3	0.25	0.0791	0.1365	0.0761	0.0598	0.1	1.1581	0.0432
0.472	0.5	0.2	0.3	0.25	0.0791	0.1365	0.0779	0.0412	0.2	1.1581	0.0863

Table C.2. Power to reject the null hypothesis that proportion of *r*AC contribution is zero in ACE model with SES (alpha = .05)

power	vA	vC	vE	rAEnv	covAC	pcac	average prcovAC	average_se	g	vt	r2
0	0.4	0.2	0.4	-0.3	-0.0849	-0.2044	-0.0564	0.1283	0.15	0.8303	0.0361
0	0.4	0.2	0.4	-0.2	-0.0566	-0.1276	-0.0881	0.335	0.1	0.8869	0.0226
0	0.6	0.1	0.3	-0.2	-0.049	-0.1086	-0.1544	0.6777	0.3	0.902	0.0333
1	0.6	0.1	0.3	0.2	0.049	0.0892	0.065	0.0255	0.15	1.098	0.0137
0.36	0.6	0.1	0.3	0.2	0.049	0.0892	0.0395	0.0228	0.2	1.098	0.0182
0.391	0.6	0.1	0.3	0.2	0.049	0.0892	0.0426	0.0204	0.3	1.098	0.0273
1	0.6	0.1	0.3	0.25	0.0612	0.1091	0.0791	0.0261	0.15	1.1225	0.0134
0.655	0.6	0.1	0.3	0.25	0.0612	0.1091	0.0582	0.0236	0.2	1.1225	0.0178
0.718	0.6	0.1	0.3	0.25	0.0612	0.1091	0.0591	0.0226	0.3	1.1225	0.0267
0.811	0.6	0.1	0.3	0.3	0.0735	0.1281	0.0705	0.0258	0.15	1.147	0.0131
0.857	0.6	0.1	0.3	0.3	0.0735	0.1281	0.071	0.0247	0.2	1.147	0.0174
0.812	0.6	0.1	0.3	0.3	0.0735	0.1281	0.0662	0.0235	0.3	1.147	0.0262
0	0.4	0.2	0.4	-0.2	-0.0566	-0.1276	-0.2441	1.165	0.2	0.8869	0.0451
0.667	0.6	0.1	0.3	0.35	0.0857	0.1464	0.0801	0.0262	0.15	1.1715	0.0128
0.87	0.6	0.1	0.3	0.35	0.0857	0.1464	0.075	0.0253	0.2	1.1715	0.0171
0.9	0.6	0.1	0.3	0.35	0.0857	0.1464	0.0816	0.0251	0.3	1.1715	0.0256
0	0.6	0.1	0.3	-0.1	-0.0245	-0.0515	-0.0586	0.0897	0.15	0.951	0.0158
0	0.6	0.1	0.3	-0.1	-0.0245	-0.0515	-0.0558	0.0995	0.1	0.951	0.0105
0	0.6	0.1	0.3	-0.1	-0.0245	-0.0515	-0.0781	0.2912	0.2	0.951	0.021
0	0.6	0.1	0.3	-0.1	-0.0245	-0.0515	-0.0709	0.2291	0.3	0.951	0.0315
1	0.6	0.1	0.3	0.15	0.0367	0.0685	0.0506	0.0253	0.15	1.0735	0.014
0.389	0.6	0.1	0.3	0.15	0.0367	0.0685	0.0326	0.0226	0.2	1.0735	0.0186
0.667	0.6	0.1	0.3	0.15	0.0367	0.0685	0.0385	0.0198	0.3	1.0735	0.0279
0	0.4	0.2	0.4	-0.2	-0.0566	-0.1276	-0.1242	0.2056	0.3	0.8869	0.0677
1	0.4	0.2	0.4	0.2	0.0566	0.1016	0.0706	0.03	0.15	1.1131	0.027
0.353	0.4	0.2	0.4	0.2	0.0566	0.1016	0.0446	0.039	0.1	1.1131	0.018
0.5	0.4	0.2	0.4	0.2	0.0566	0.1016	0.0445	0.0267	0.2	1.1131	0.0359
0.714	0.4	0.2	0.4	0.2	0.0566	0.1016	0.0514	0.0227	0.3	1.1131	0.0539
0.667	0.4	0.2	0.4	0.25	0.0707	0.1239	0.0573	0.0321	0.15	1.1414	0.0263
1	0.4	0.2	0.4	0.25	0.0707	0.1239	0.0847	0.0346	0.1	1.1414	0.0175
0.79	0.4	0.2	0.4	0.25	0.0707	0.1239	0.069	0.0255	0.2	1.1414	0.035
0	0.4	0.2	0.4	-0.3	-0.0849	-0.2044	-0.113	0.2419	0.1	0.8303	0.0241
0.86	0.4	0.2	0.4	0.25	0.0707	0.1239	0.0687	0.0226	0.3	1.1414	0.0526
0.791	0.4	0.2	0.4	0.3	0.0849	0.1451	0.0794	0.0289	0.15	1.1697	0.0256
1	0.4	0.2	0.4	0.3	0.0849	0.1451	0.1005	0.0338	0.1	1.1697	0.0171
0.91	0.4	0.2	0.4	0.3	0.0849	0.1451	0.083	0.0258	0.2	1.1697	0.0342
0.867	0.4	0.2	0.4	0.3	0.0849	0.1451	0.0742	0.0229	0.3	1.1697	0.0513
0.915	0.4	0.2	0.4	0.35	0.099	0.1653	0.0952	0.0285	0.15	1.198	0.025
0.75	0.4	0.2	0.4	0.35	0.099	0.1653	0.0904	0.0358	0.1	1.198	0.0167
0.857	0.4	0.2	0.4	0.35	0.099	0.1653	0.0937	0.0271	0.2	1.198	0.0334
0.967	0.4	0.2	0.4	0.35	0.099	0.1653	0.0904	0.024	0.3	1.198	0.0501
0	0.4	0.2	0.4	-0.3	-0.0849	-0.2044	-0.3094	0.9209	0.2	0.8303	0.0482
0	0.4	0.3	0.3	-0.25	-0.0866	-0.2095	-0.0621	0.0654	0.15	0.8268	0.0544
0	0.4	0.3	0.3	-0.25	-0.0866	-0.2095	-0.0499	0.0683	0.1	0.8268	0.0363
0	0.4	0.3	0.3	-0.25	-0.0866	-0.2095	-0.1255	0.138	0.2	0.8268	0.0726

Table C.2. (cont.)

0	0.4	0.3	0.3	-0.2	-0.0693	-0.1609	-0.043	0.0519	0.15	0.8614	0.0522
0	0.4	0.3	0.3	-0.2	-0.0693	-0.1609	-0.1036	0.2222	0.1	0.8614	0.0348
0	0.4	0.3	0.3	-0.2	-0.0693	-0.1609	-0.0589	0.0561	0.2	0.8614	0.0697
0	0.5	0.1	0.4	-0.2	-0.0447	-0.0982	-0.6812	10.4501	0.15	0.9106	0.0165
0	0.5	0.1	0.4	-0.2	-0.0447	-0.0982	-0.1309	0.0918	0.1	0.9106	0.011
0	0.4	0.2	0.4	-0.3	-0.0849	-0.2044	-0.1248	0.1713	0.3	0.8303	0.0723
0	0.5	0.1	0.4	-0.2	-0.0447	-0.0982	-0.1678	0.4851	0.2	0.9106	0.022
0	0.5	0.1	0.4	-0.2	-0.0447	-0.0982	-0.1335	0.5971	0.3	0.9106	0.0329
1	0.5	0.1	0.4	0.2	0.0447	0.0821	0.0595	0.026	0.15	1.0894	0.0138
0.333	0.5	0.1	0.4	0.2	0.0447	0.0821	0.0374	0.0239	0.2	1.0894	0.0184
0.45	0.5	0.1	0.4	0.2	0.0447	0.0821	0.0393	0.0207	0.3	1.0894	0.0275
1	0.5	0.1	0.4	0.25	0.0559	0.1006	0.0726	0.0265	0.15	1.1118	0.0135
0.667	0.5	0.1	0.4	0.25	0.0559	0.1006	0.0563	0.024	0.2	1.1118	0.018
0.671	0.5	0.1	0.4	0.25	0.0559	0.1006	0.0545	0.0228	0.3	1.1118	0.027
1	0.5	0.1	0.4	0.3	0.0671	0.1183	0.0855	0.0272	0.15	1.1342	0.0132
0.69	0.5	0.1	0.4	0.3	0.0671	0.1183	0.0611	0.0247	0.2	1.1342	0.0176
0	0.4	0.2	0.4	-0.25	-0.0707	-0.1647	-0.1084	0.149	0.15	0.8586	0.0349
0.737	0.5	0.1	0.4	0.3	0.0671	0.1183	0.066	0.024	0.3	1.1342	0.0265
0.769	0.5	0.1	0.4	0.35	0.0783	0.1353	0.067	0.0261	0.15	1.1565	0.013
0.8	0.5	0.1	0.4	0.35	0.0783	0.1353	0.0697	0.0255	0.2	1.1565	0.0173
0.8	0.5	0.1	0.4	0.35	0.0783	0.1353	0.0685	0.0248	0.3	1.1565	0.0259
0	0.5	0.1	0.4	-0.1	-0.0224	-0.0468	-0.0724	0.2215	0.15	0.9553	0.0157
0	0.5	0.1	0.4	-0.1	-0.0224	-0.0468	-0.0387	0.0927	0.1	0.9553	0.0105
0	0.5	0.1	0.4	-0.1	-0.0224	-0.0468	-0.064	0.2298	0.2	0.9553	0.0209
0	0.5	0.1	0.4	-0.1	-0.0224	-0.0468	-0.2821	1.616	0.3	0.9553	0.0314
0	0.5	0.1	0.4	0.15	0.0335	0.0629	0.0459	0.026	0.15	1.0671	0.0141
0.333	0.5	0.1	0.4	0.15	0.0335	0.0629	0.0338	0.0213	0.2	1.0671	0.0187
0	0.4	0.2	0.4	-0.25	-0.0707	-0.1647	-0.0969	0.3051	0.1	0.8586	0.0233
0.375	0.5	0.1	0.4	0.15	0.0335	0.0629	0.0333	0.0199	0.3	1.0671	0.0281
0	0.5	0.2	0.3	-0.3	-0.0949	-0.2342	-0.1432	0.2729	0.3	0.8103	0.0741
0	0.5	0.2	0.3	-0.25	-0.0791	-0.1878	-0.1087	0.1629	0.15	0.8419	0.0356
0	0.5	0.2	0.3	-0.25	-0.0791	-0.1878	-0.1265	0.3905	0.1	0.8419	0.0238
0	0.5	0.2	0.3	-0.25	-0.0791	-0.1878	-0.1202	0.1636	0.2	0.8419	0.0475
0	0.5	0.2	0.3	-0.25	-0.0791	-0.1878	-0.5157	9.8804	0.3	0.8419	0.0713
0	0.5	0.2	0.3	-0.2	-0.0632	-0.1448	-0.0659	0.0894	0.15	0.8735	0.0343
0	0.4	0.2	0.4	-0.25	-0.0707	-0.1647	-0.0933	0.1415	0.2	0.8586	0.0466
0	0.5	0.2	0.3	-0.2	-0.0632	-0.1448	-0.1089	0.3375	0.1	0.8735	0.0229
0	0.5	0.2	0.3	-0.2	-0.0632	-0.1448	-0.3309	1.2675	0.2	0.8735	0.0458
0	0.5	0.2	0.3	-0.2	-0.0632	-0.1448	-0.1123	0.2515	0.3	0.8735	0.0687
1	0.5	0.2	0.3	0.2	0.0632	0.1123	0.0794	0.0293	0.15	1.1265	0.0266
1	0.5	0.2	0.3	0.2	0.0632	0.1123	0.0781	0.0346	0.1	1.1265	0.0178
0.607	0.5	0.2	0.3	0.2	0.0632	0.1123	0.0531	0.0262	0.2	1.1265	0.0355
0.776	0.5	0.2	0.3	0.2	0.0632	0.1123	0.0605	0.0221	0.3	1.1265	0.0533
0.75	0.5	0.2	0.3	0.25	0.0791	0.1365	0.0732	0.0301	0.15	1.1581	0.0259
1	0.5	0.2	0.3	0.25	0.0791	0.1365	0.0954	0.0335	0.1	1.1581	0.0173
0.643	0.5	0.2	0.3	0.25	0.0791	0.1365	0.0665	0.0259	0.2	1.1581	0.0345
0	0.4	0.2	0.4	-0.25	-0.0707	-0.1647	-0.1331	0.3208	0.3	0.8586	0.0699
0.9	0.5	0.2	0.3	0.25	0.0791	0.1365	0.0702	0.0223	0.3	1.1581	0.0518

Table C.2. (cont.)

0.7	0.5	0.2	0.3	0.3	0.0949	0.1595	0.0847	0.0294	0.15	1.1897	0.0252
1	0.5	0.2	0.3	0.3	0.0949	0.1595	0.1127	0.0328	0.1	1.1897	0.0168
0.95	0.5	0.2	0.3	0.3	0.0949	0.1595	0.0842	0.0256	0.2	1.1897	0.0336
0.98	0.5	0.2	0.3	0.3	0.0949	0.1595	0.0938	0.0236	0.3	1.1897	0.0504
0.909	0.5	0.2	0.3	0.35	0.1107	0.1812	0.1011	0.0294	0.15	1.2214	0.0246
1	0.5	0.2	0.3	0.35	0.1107	0.1812	0.1297	0.0324	0.1	1.2214	0.0164
0.964	0.5	0.2	0.3	0.35	0.1107	0.1812	0.1078	0.0263	0.2	1.2214	0.0328
1	0.5	0.2	0.3	0.35	0.1107	0.1812	0.108	0.0243	0.3	1.2214	0.0491
0	0.5	0.2	0.3	-0.1	-0.0316	-0.0675	-0.085	0.183	0.15	0.9368	0.032
0	0.4	0.2	0.4	-0.2	-0.0566	-0.1276	-0.0679	0.0886	0.15	0.8869	0.0338
0	0.5	0.2	0.3	-0.1	-0.0316	-0.0675	-0.034	0.0672	0.1	0.9368	0.0214
0.015	0.5	0.2	0.3	-0.1	-0.0316	-0.0675	-0.0471	0.0827	0.2	0.9368	0.0427
0.015	0.5	0.2	0.3	-0.1	-0.0316	-0.0675	-0.0362	0.0448	0.3	0.9368	0.0641
1	0.5	0.2	0.3	0.15	0.0474	0.0866	0.062	0.0301	0.15	1.0949	0.0274
0	0.5	0.2	0.3	0.15	0.0474	0.0866	0.0605	0.0363	0.1	1.0949	0.0183
1	0.5	0.2	0.3	0.15	0.0474	0.0866	0.0626	0.0267	0.2	1.0949	0.0365
0.5	0.5	0.2	0.3	0.15	0.0474	0.0866	0.0382	0.0224	0.3	1.0949	0.0548
0	0.6	0.1	0.3	-0.2	-0.049	-0.1086	-0.1582	0.5489	0.15	0.902	0.0166
0	0.6	0.1	0.3	-0.2	-0.049	-0.1086	-0.0871	0.848	0.1	0.902	0.0111
0	0.6	0.1	0.3	-0.2	-0.049	-0.1086	-0.1971	0.3924	0.2	0.902	0.0222

Table C.3. Power to reject the null hypothesis that proportion of *r*AE contribution is zero in AE model (alpha = .05)

power	vA	vE	rAE	covAE	pcae	average prcovAE	average se	g	vt	r2
1	0.6	0.4	0.45	0.2205	0.306	0.2718	0.0342	0.05	1.4409	0.0208
1	0.6	0.4	0.45	0.2205	0.306	0.2792	0.0268	0.1	1.4409	0.0416
1	0.2	0.8	0.05	0.02	0.0385	0.1128	0.0228	0.2	1.04	0.0385
1	0.3	0.7	0.3	0.1375	0.2157	0.1727	0.0298	0.05	1.275	0.0118
1	0.2	0.8	0.05	0.02	0.0385	0.128	0.0203	0.4	1.04	0.0769
1	0.2	0.8	0.1	0.04	0.0741	0.1235	0.0226	0.2	1.08	0.037
1	0.2	0.8	0.1	0.04	0.0741	0.1412	0.0204	0.4	1.08	0.0741
1	0.2	0.8	0.15	0.06	0.1071	0.1218	0.0252	0.1	1.12	0.0179
1	0.2	0.8	0.15	0.06	0.1071	0.133	0.0226	0.2	1.12	0.0357
1	0.2	0.8	0.15	0.06	0.1071	0.1532	0.0206	0.4	1.12	0.0714
1	0.3	0.7	0.3	0.1375	0.2157	0.1816	0.0253	0.1	1.275	0.0235
1	0.7	0.3	-0.1	-0.0458	-0.1009	0.245	0.0387	0.2	0.9083	0.1541
1	0.7	0.3	0.05	0.0229	0.0438	0.2885	0.0423	0.1	1.0458	0.0669
1	0.7	0.3	0.05	0.0229	0.0438	0.2756	0.0304	0.2	1.0458	0.1339
1	0.7	0.3	0.05	0.0229	0.0438	0.2438	0.0215	0.4	1.0458	0.2677
1	0.7	0.3	0.1	0.0458	0.084	0.2921	0.0394	0.1	1.0917	0.0641
1	0.7	0.3	0.1	0.0458	0.084	0.2823	0.0286	0.2	1.0917	0.1282
1	0.7	0.3	0.15	0.0687	0.1209	0.2949	0.0371	0.1	1.1375	0.0615
1	0.7	0.3	0.15	0.0687	0.1209	0.288	0.0271	0.2	1.1375	0.1231
1	0.3	0.7	0.35	0.1604	0.2429	0.1772	0.0288	0.05	1.3208	0.0114
1	0.3	0.7	0.35	0.1604	0.2429	0.1864	0.025	0.1	1.3208	0.0227
1	0.3	0.7	0.4	0.1833	0.2683	0.1811	0.0281	0.05	1.3666	0.011
1	0.3	0.7	0.4	0.1833	0.2683	0.1889	0.0248	0.1	1.3666	0.022
1	0.3	0.7	0.45	0.2062	0.292	0.1847	0.0276	0.05	1.4124	0.0106
1	0.3	0.7	0.45	0.2062	0.292	0.193	0.0246	0.1	1.4124	0.0212
1	0.4	0.6	0.2	0.098	0.1639	0.2061	0.0282	0.1	1.196	0.0334
1	0.4	0.6	0.25	0.1225	0.1968	0.2111	0.0272	0.1	1.2449	0.0321
1	0.4	0.6	0.3	0.147	0.2272	0.207	0.0325	0.05	1.2939	0.0155
1	0.4	0.6	0.3	0.147	0.2272	0.2155	0.0264	0.1	1.2939	0.0309
1	0.4	0.6	0.35	0.1715	0.2554	0.2105	0.0312	0.05	1.3429	0.0149
1	0.4	0.6	0.35	0.1715	0.2554	0.2194	0.0258	0.1	1.3429	0.0298
1	0.4	0.6	0.4	0.196	0.2816	0.2135	0.0301	0.05	1.3919	0.0144
1	0.4	0.6	0.4	0.196	0.2816	0.2229	0.0253	0.1	1.3919	0.0287
1	0.4	0.6	0.45	0.2205	0.306	0.2162	0.0292	0.05	1.4409	0.0139
1	0.4	0.6	0.45	0.2205	0.306	0.2252	0.0249	0.1	1.4409	0.0278
1	0.3	0.7	0.2	0.0917	0.1549	0.1702	0.0264	0.1	1.1833	0.0254
1	0.5	0.5	0.2	0.1	0.1667	0.2386	0.0302	0.1	1.2	0.0417
1	0.5	0.5	0.25	0.125	0.2	0.2357	0.0377	0.05	1.25	0.02
1	0.5	0.5	0.25	0.125	0.2	0.2425	0.0289	0.1	1.25	0.04
1	0.5	0.5	0.3	0.15	0.2308	0.2385	0.0357	0.05	1.3	0.0192
1	0.5	0.5	0.3	0.15	0.2308	0.2459	0.0278	0.1	1.3	0.0385
1	0.5	0.5	0.35	0.175	0.2593	0.2409	0.034	0.05	1.35	0.0185
1	0.5	0.5	0.35	0.175	0.2593	0.2489	0.0269	0.1	1.35	0.037
1	0.5	0.5	0.4	0.2	0.2857	0.243	0.0326	0.05	1.4	0.0179
1	0.5	0.5	0.4	0.2	0.2857	0.2515	0.0262	0.1	1.4	0.0357
1	0.5	0.5	0.45	0.225	0.3103	0.2448	0.0314	0.05	1.45	0.0172

Table C.3. (cont.)

1	0.5	0.5	0.45	0.225	0.3103	0.2537	0.0256	0.1	1.45	0.0345
1	0.6	0.4	0.2	0.098	0.1639	0.2688	0.0324	0.1	1.196	0.0502
1	0.6	0.4	0.25	0.1225	0.1968	0.2669	0.0417	0.05	1.2449	0.0241
1	0.3	0.7	0.25	0.1146	0.1864	0.1762	0.0258	0.1	1.2291	0.0244
1	0.6	0.4	0.25	0.1225	0.1968	0.2716	0.0309	0.1	1.2449	0.0482
1	0.6	0.4	0.3	0.147	0.2272	0.2685	0.0394	0.05	1.2939	0.0232
1	0.6	0.4	0.3	0.147	0.2272	0.2739	0.0296	0.1	1.2939	0.0464
1	0.6	0.4	0.35	0.1715	0.2554	0.2698	0.0374	0.05	1.3429	0.0223
1	0.6	0.4	0.35	0.1715	0.2554	0.276	0.0285	0.1	1.3429	0.0447
1	0.6	0.4	0.4	0.196	0.2816	0.2709	0.0357	0.05	1.3919	0.0216
1	0.6	0.4	0.4	0.196	0.2816	0.2777	0.0276	0.1	1.3919	0.0431
0.99	0.2	0.8	0.1	0.04	0.0741	0.1133	0.0258	0.1	1.08	0.0185
0.99	0.4	0.6	0.25	0.1225	0.1968	0.2031	0.0342	0.05	1.2449	0.0161
0.99	0.3	0.7	0.25	0.1146	0.1864	0.1677	0.031	0.05	1.2291	0.0122
0.99	0.6	0.4	0.2	0.098	0.1639	0.2648	0.0443	0.05	1.196	0.0251
0.98	0.3	0.7	0.45	0.2062	0.292	0.1772	0.0341	0.025	1.4124	0.0053
0.98	0.4	0.6	0.2	0.098	0.1639	0.1985	0.0362	0.05	1.196	0.0167
0.98	0.3	0.7	0.2	0.0917	0.1549	0.162	0.0325	0.05	1.1833	0.0127
0.98	0.5	0.5	0.2	0.1	0.1667	0.2324	0.0401	0.05	1.2	0.0208
0.97	0.6	0.4	0.45	0.2205	0.306	0.2644	0.0478	0.025	1.4409	0.0104
0.97	0.7	0.3	0.15	0.0687	0.1209	0.2954	0.0524	0.05	1.1375	0.0308
0.97	0.3	0.7	0.4	0.1833	0.2683	0.1736	0.0356	0.025	1.3666	0.0055
0.97	0.4	0.6	0.4	0.196	0.2816	0.2057	0.0399	0.025	1.3919	0.0072
0.97	0.4	0.6	0.45	0.2205	0.306	0.2084	0.0379	0.025	1.4409	0.0069
0.97	0.5	0.5	0.45	0.225	0.3103	0.237	0.0424	0.025	1.45	0.0086
0.96	0.2	0.8	0.05	0.02	0.0385	0.1036	0.0269	0.1	1.04	0.0192
0.96	0.5	0.5	0.4	0.2	0.2857	0.2352	0.0448	0.025	1.4	0.0089
0.95	0.7	0.3	-0.1	-0.0458	-0.1009	0.27	0.0549	0.1	0.9083	0.0771
0.95	0.7	0.3	0.1	0.0458	0.084	0.2938	0.0562	0.05	1.0917	0.0321
0.95	0.6	0.4	0.4	0.196	0.2816	0.2635	0.0506	0.025	1.3919	0.0108
0.94	0.2	0.8	-0.1	-0.04	-0.087	0.0768	0.0216	0.4	0.92	0.087
0.94	0.3	0.7	0.35	0.1604	0.2429	0.1695	0.0375	0.025	1.3208	0.0057
0.94	0.4	0.6	0.35	0.1715	0.2554	0.2026	0.0423	0.025	1.3429	0.0074
0.93	0.3	0.7	0.3	0.1375	0.2157	0.1649	0.0399	0.025	1.275	0.0059
0.93	0.2	0.8	0.15	0.06	0.1071	0.1143	0.0306	0.05	1.12	0.0089
0.93	0.7	0.3	0.05	0.0229	0.0438	0.2915	0.0607	0.05	1.0458	0.0335
0.93	0.5	0.5	0.35	0.175	0.2593	0.2331	0.0476	0.025	1.35	0.0093
0.92	0.6	0.4	0.35	0.1715	0.2554	0.2624	0.0537	0.025	1.3429	0.0112
0.91	0.4	0.6	0.3	0.147	0.2272	0.1989	0.0451	0.025	1.2939	0.0077
0.91	0.5	0.5	0.3	0.15	0.2308	0.2306	0.0508	0.025	1.3	0.0096
0.91	0.6	0.4	0.3	0.147	0.2272	0.261	0.0572	0.025	1.2939	0.0116
0.89	0.6	0.4	0.25	0.1225	0.1968	0.2593	0.0614	0.025	1.2449	0.012
0.88	0.4	0.6	0.25	0.1225	0.1968	0.1947	0.0485	0.025	1.2449	0.008
0.88	0.5	0.5	0.25	0.125	0.2	0.2276	0.0546	0.025	1.25	0.01
0.87	0.3	0.7	0.25	0.1146	0.1864	0.1596	0.0429	0.025	1.2291	0.0061
0.84	0.5	0.5	0.2	0.1	0.1667	0.224	0.0592	0.025	1.2	0.0104
0.84	0.6	0.4	0.2	0.098	0.1639	0.2571	0.0661	0.025	1.196	0.0125
0.83	0.2	0.8	0.1	0.04	0.0741	0.106	0.0326	0.05	1.08	0.0093

Table C.3. (cont.)

0.82	0.4	0.6	0.2	0.098	0.1639	0.1898	0.0527	0.025	1.196	0.0084
0.82	0.3	0.7	0.2	0.0917	0.1549	0.1534	0.0467	0.025	1.1833	0.0063
0.798	0.7	0.3	-0.1	-0.0458	-0.1009	0.2771	0.0805	0.05	0.9083	0.0385
0.78	0.2	0.8	0.05	0.02	0.0385	0.0963	0.0356	0.05	1.04	0.0096
0.77	0.2	0.8	-0.1	-0.04	-0.087	0.0694	0.0265	0.2	0.92	0.0435
0.76	0.6	0.4	-0.2	-0.098	-0.2437	0.203	0.0668	0.1	0.804	0.0746
0.7	0.5	0.5	-0.2	-0.1	-0.25	0.1587	0.0633	0.1	0.8	0.0625
0.65	0.6	0.4	-0.2	-0.098	-0.2437	0.2091	0.0987	0.05	0.804	0.0373
0.616	0.2	0.8	-0.1	-0.04	-0.087	0.0636	0.0354	0.1	0.92	0.0217
0.61	0.4	0.6	-0.2	-0.098	-0.2437	0.1121	0.0592	0.1	0.804	0.0497
0.6	0.5	0.5	-0.2	-0.1	-0.25	0.1582	0.0945	0.05	0.8	0.0313
0.576	0.6	0.4	-0.2	-0.098	-0.2437	0.1946	0.1628	0.025	0.804	0.0187
0.57	0.6	0.4	-0.25	-0.1225	-0.3244	0.1878	0.1156	0.05	0.7551	0.0397
0.51	0.5	0.5	-0.25	-0.125	-0.3333	0.1328	0.1128	0.05	0.75	0.0333
0.51	0.6	0.4	-0.3	-0.147	-0.4163	0.1576	0.1384	0.05	0.7061	0.0425
0.51	0.6	0.4	-0.25	-0.1225	-0.3244	0.1718	0.1931	0.025	0.7551	0.0199
0.49	0.4	0.6	-0.2	-0.098	-0.2437	0.1061	0.0902	0.05	0.804	0.0249
0.49	0.5	0.5	-0.2	-0.1	-0.25	0.1383	0.1637	0.025	0.8	0.0156
0.475	0.2	0.8	-0.1	-0.04	-0.087	0.0534	0.058	0.05	0.92	0.0109
0.44	0.4	0.6	-0.25	-0.1225	-0.3244	0.0833	0.0711	0.1	0.7551	0.053
0.429	0.6	0.4	-0.3	-0.147	-0.4163	0.135	0.2377	0.025	0.7061	0.0212
0.42	0.3	0.7	-0.2	-0.0917	-0.2244	0.0644	0.0552	0.1	0.8167	0.0367
0.41	0.4	0.6	-0.2	-0.098	-0.2437	0.0776	0.1769	0.025	0.804	0.0124
0.4	0.5	0.5	-0.25	-0.125	-0.3333	0.1075	0.2023	0.025	0.75	0.0167
0.35	0.4	0.6	-0.25	-0.1225	-0.3244	0.0763	0.1107	0.05	0.7551	0.0265
0.34	0.5	0.5	-0.3	-0.15	-0.4286	0.0972	0.1385	0.05	0.7	0.0357
0.333	0.3	0.7	-0.2	-0.0917	-0.2244	0.055	0.0884	0.05	0.8167	0.0184
0.28	0.5	0.5	-0.3	-0.15	-0.4286	0.0632	0.2603	0.025	0.7	0.0179
0.26	0.4	0.6	-0.25	-0.1225	-0.3244	0.0356	0.2422	0.025	0.7551	0.0132
0.23	0.3	0.7	-0.2	-0.0917	-0.2244	-0.0092	0.3851	0.025	0.8167	0.0092
0.153	0.4	0.6	-0.3	-0.147	-0.4163	0.0395	0.1372	0.05	0.7061	0.0283
0.152	0.4	0.6	-0.3	-0.147	-0.4163	-0.0278	0.3861	0.025	0.7061	0.0142