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This study investigated the potential moderating role of genetic predisposition for language performance on the association between various environmental variables and children's receptive and expressive language performance. Participants included 1150 preschool-aged monozygotic and dizygotic twins from the Early Childhood Longitudinal Study- Birth Cohort (ECLS-B). Hierarchical multiple regression analyses were conducted to explore interaction effect between genetic predisposition for language and singular environmental variables, genetic predisposition for language and indices of environmental risk and advantage, and genetic predisposition for language, indices of environmental risk and advantage, and gender. Findings provide tentative support for the interaction of genetic predisposition and environmental factors as a contributor to language performance. Three gene–environment interaction terms were found to be significant predictors of language performance. One two-way interaction and one three-way interaction were found to significantly contribute to their respective models' ability to account for variance in children's language performance.

INVESTIGATING GENE–ENVIRONMENT INTERACTION AS A CONTRIBUTOR TO LANGUAGE PERFORMANCE

by

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CHAPTER I

INTRODUCTION

Language ability, a distinctly human neurocognitive trait, emerges in children in a universal order of developmental milestones with little to no formal instruction and requires prolonged postnatal development characterized by notable susceptibility to environmental input, a duality suggesting the roles of both nature and nurture in language development (Sherwood, Subiaul, & Zawidzki, 2008; Pinker & Jackendoff, 2005; Noble, Norman, & Farah, 2005; Colledge, Bishop, Koeppen-Schomerus, Price, Happe´, Eley, et al., 2002; Chomsky, 1986). Research indicates that nearly all components of language ability are influenced by genetic factors to some extent (Stromswold, 2000), and environmental factors shown to yield a significant influence on language performance include household income, parental education, parental provision of cognitive stimulation, parental emotional supportiveness, and sex (Rowe, 2008; Kovas, Hayiou-Thomas, Oliver, Dale, Bishop, & Plomin, 2005; Pan, Rowe, Singer, & Snow, 2005; Raviv, Kessenich, & Morrison, 2004; Hart & Risley, 1999). Contemporary investigations into both genetic and environmental contributors to children's language performance largely employ the classical twin study analytic method, that of dividing and measuring the influence of genetic, shared environmental, and non-shared environmental factors. This analytic method implies that genetic and environmental forces function as separable

additive forces, rather than inseparable synergistic forces, a conceptualization incongruous with contemporary empirical evidence demonstrating inextricable gene–environment interaction as specifically delineated in the theory of probabilistic epigenesis (Goldhaber, 2000; Gottlieb, 2007).

The proposed study acknowledges two important criticisms of contemporary research in human development. First, O'Brien (2005) criticized contemporary research for lacking concordance between complex conceptualizations in human development theorizing and researchers' employed analytic strategies, especially that of focusing on main effects rather than moderating and mediating processes. O'Brien noted that contemporary research and theorizing in human development lays to rest the nature-nurture argument and promotes the conceptualization of complex, multilevel, interconnected biological and environmental processes of developmental change; thus researchers must seek to measure this complexity. Second, Moffit, Caspi, and Rutter (2006) noted that the majority of research into gene–environment interaction has been within the domain of psychopathology and that there exists a need to extend this research to other behaviors, including well-being, school achievement, and personality characteristics. The current study seeks to address these limitations by employing a contextualist theoretical frame and corresponding analytic methods to investigate inextricable gene–environment interaction as a contributor to children's language performance. Specifically, this study investigates whether genetic predisposition for language performance moderates the effect of various factors known to be associated with preschool children's language performance (i.e., income, parental education,

parental provision of cognitive stimulation, parental emotional supportiveness, indices of environmental risk and advantage, and child sex). This study is conceptualized similarly to those within psychopathology as it is assumed that individuals' sensitivity to environmental factors is moderated by genetic material.

CHAPTER II

THEORETICAL PERSPECTIVES

Theory of Human Development

Predetermined Epigenesis

Predetermined epigenesis, exemplifying the "central dogma" formerly promoted by molecular biologists and quantified behavioral geneticists, characterizes human development as a unidirectional process in which structural organization determines function (Gottlieb, 2000, p. 180). This deterministic theory views the genome as the blueprint for human development, incapable of interacting in the developmental trajectory. Scientists supporting this view describe DNA as "encapsulated," an exclusive entity protected by the nucleus's membrane from any environmental effects (Gottlieb, 2000, p. 182). Specifically, predetermined epigenesis asserts that, within the level of gene activity, information from DNA is transmitted to ribonucleic acid (RNA), in the process termed transcription, and information from RNA is transmitted to protein, in the process termed translation. Genetic activity then influences structural maturation, and structural maturation influences organisms' function, activity, and experience. Predetermined epigenesis allows for one exception to the rule of unidirectionality, which is that of RNA influencing DNA through retroviruses' capacity for reverse transcription. Otherwise, predetermined epigenesis maintains its assumption of unidirectionality and asserts that

three biological conditions are not possible: protein influencing protein, protein influencing DNA, and protein influencing RNA (Gottlieb, 2000).

Predetermined epigenesis, an unequivocally mechanistic theory, conceptualizes human development as a result of two independently functioning causes (biology and environment), maintains a reductionist view that both causes can be bifurcated and measured, and regards information derived through investigations as universally generalizable, capable of revealing characteristics about the human population across time and space (Goldhaber, 2000).

Probabilistic Epigenesis

The argument for unidirectionality is incongruent with contemporary evidence demonstrating interaction among genetic activity, structural maturation, and function, action, and experience. Although Gottlieb (2000) maintained the assertion within predetermined epigenesis that DNA is an inert entity, he presented the assertion and corresponding evidence that DNA operates at the lowest level of organismic organization and is thus incapable of determining the developmental outcomes of organisms. Thus, probabilistic epigenesis describes the course of organisms' development as arising through inextricable interactions between biology and environment, demonstrating bidirectional relationships between structure and function.

Refuting predetermined epigenesis by first addressing interaction within the level of genetic activity, Gottlieb acknowledged the unidirectional flow of transcription and translation, yet asserted that DNA receives information from RNA and that RNA receives information from protein. RNA influences DNA through retroviruses' capacity for

reverse transcription, as previously stated and acknowledged within predetermined epigenesis. Further expanding the argument for molecular demonstrations of bidirectionality, Gottlieb stated that a group of regulative proteins bind to DNA, serving to activate or inhibit DNA expression, asserting that proteins influence DNA. Proteins interact in that abnormally conformed proteins, or "prions," in neurodegenerative disorders, are capable of transferring their abnormal structure to other proteins. In addition, proteins interact when certain proteins activate or inactivate other proteins, for example, during the process of phosphorylation. During such events, the participation of DNA and RNA is entirely evaded. Last, DNA influences DNA in the case of alterations in gene expression depending on the genetic background of the organism. Within probabilistic epigenesis, the only pathway not yet proven is that of proteins directly influencing RNA structure, in a process of reverse translation; although proteins have been found to influence mRNA activity, wherein mRNA is activated by proteins as a consequence of phosphorylation (Gottlieb, 2000).

Gottlieb's Metatheoretical Model of Gene–Environment Interaction

Interaction with features outside the level of genetic activity introduces the fundamental and revolutionary assertion of probabilistic epigenesis: Genes are dependent upon environmental factors for their activation or inactivation and are thus highly interactive in the developmental trajectory. DNA, indeed being an inert molecule, cannot initiate or terminate its own activity and requires endogenous and exogenous stimulation for its expression (Gottlieb, 2007). Gottlieb (2000) presented his metatheoretical model to illustrate that genetic, neural, behavioral, and environmental influences inextricably

interact in bidirectional relationships over the course of individual development. Gottlieb asserted that genetic activity is influenced at each level of analysis in his metatheoretical model, emphasizing the ubiquitous interaction of genes with genetic and non-genetic factors up to and including the physical, social, and cultural components of the external environment. Further, Gottlieb and Halpern (2002) stated that phenotypic outcomes are a result of interaction between at least two factors belonging to the same or different levels of analysis in the model (as cited in Gottlieb, 2007). This concept of ubiquitous interaction provides an explanation for a lack of correlation between number of genes and complexity of structural organization and a lack of correlation between number of genes and number of neurons in the brains of different organisms (Gottlieb, 2000). As one of numerous examples, Gottlieb highlighted prominent phenotypic differences in a pair of monozygotic twins reared in two dissimilar family environments. Gottlieb's metatheoretical model provides the explanation that such divergent phenotypic outcomes can be attributed to differences in life experiences or variations in the interactions among the four levels of analysis over the course of individual development of each twin. Further, remarkable phenotypic differences in comparison to negligible variation in DNA across the human species demonstrate the powerful impact of interaction among the levels of analysis over time (Gottlieb, 2000).

The Influence of Normally Occurring Events on the Nervous System

Gottlieb emphasized the point that genetic expression is elicited from normally occurring events in organisms' internal and external environment. Gottlieb provided ample evidence regarding the influence of environment on structure, particularly,

neuroanatomical structure. First, given acoustic stimulation, mice experience augmented e-*fos* expression, neural activity, and neural organization of the auditory system. Second, given tactile stimulation, rats also experience augmented e-*fos* expression and an increase in somatosensory (sense of touch) cortical neurons. And last, given visual stimulation, cats experience an increase in visual cortex RNA complexity (Gottlieb, 2007). These findings are supported by G. Stent's (1981) assertion that research within molecular biology proves that genes are "at too many removes" from the actual events that construct neural cells and organize neural connectivity responsible for organisms' behavior (as cited in Gottlieb, 2000). Genes cannot singularly create neural and behavioral outcomes. Rather, scientists must evaluate gene–environment interaction to answer questions of typical and atypical development (Gottlieb, 2007).

Thus empirical support demonstrating probabilistic epigenesis serves as a justification for rejecting the classical twin study analytic strategy and for adopting an investigation into gene–environment interaction. The following section delineates theories of language acquisition emphasizing the importance of both biological and environmental contributions and culminates in language theory that acknowledges probabilistic epigenesis.

Theories of Language Acquisition

Early of theories of language acquisition demonstrate the importance of the contributions of biology and environment, particularly as earlier theorists posited that either biological or environmental factors were responsible for children's language acquisition. A researcher of the solely environmental perspective, Skinner (1957) sought

to understand how to control and predict verbal output by observing and manipulating input (as cited in Lust & Foley, 2004). Skinner asserted that external forces consisting of individuals' present stimulation and history of reinforcement (i.e., the frequency, arrangement, and dearth of reinforcing stimuli) are of utmost importance, that the contribution of individuals is diminutive and trivial, and that verbal behavior can be predicted with precision through the specification of external forces. Skinner limited his research to input-output relations, focusing solely on external input and failing to consider internal conditions that could influence humans' capacities to acquire and utilize language (as cited in Chomsky, 1986).

In opposition to this solely environmental characterization of language acquisition, Chomsky (1986) criticized Skinner's assertions for denying "the importance of the organism," and asserted that predicting verbal behavior of a complex organism requires an evaluation not only of external stimulation, but also knowledge of the internal structure of organisms and the ways in which organisms process external input and organize verbalization (as cited in Lust & Foley, 2004). Such characteristics, Chomsky argued, are a product of the amalgamation of inborn structure, the genetically determined course of maturation, and history of experience. Chomsky pointed to imprinting as evidence for an innate direction of learning. He considered children's intrinsic inquisitiveness and motivation and the fact that children do not learn language through the meticulous teaching of rules by adults; he concluded that there must be fundamental processes functioning independently of environmental feedback in the process of language acquisition. Chomsky supported the argument for "generative grammar," the

notion that there is an invariant substance to grammar, reflecting the mind, its functions, and the expression of universal forms of thought, i.e., thought believed to be the same across all languages. From this foundation, Chomsky constructed his theory of Universal Grammar (UG), which stated that the form and meaning of language are generated from a genetically determined “language faculty” or “language acquisition device.” This language faculty is a particular component of the human mind that produces language through interaction with presented experience. This innate language faculty enables children to acquire the nuanced rules of their particular native language.

Modifying Chomsky’s theory of universal grammar but maintaining a biologically deterministic perspective, contemporary evolutionary and cognitive psychologist Pinker (1994) proposed his semantic bootstrapping hypothesis, postulating that language acquisition is a deductive process and a biological adaptation, the product of natural selection (as cited in Lust & Foley, 2004). The semantic bootstrapping hypothesis suggests that children are innately endowed with the intuition that grammatical categories and functions, such as nouns, verbs, subjects, and objects exist in language. For example, names of persons and things can be mapped onto the linguistic category of "noun," and actions can be mapped onto the linguistic category of "verb." Pinker asserted that children do not engage in a process of recording all perceptible characteristics and correlations among input and posited that children utilize these universal grammatical categories, what Chomsky termed a “rich deductive structure,” as the first premises for subsequent, more nuanced deductions of language rules. Pinker held that language development follows a “genetic blueprint” (Hoff, 2003, p. 1368). This

signifies a mechanistic worldview, which holds that development occurs as a result of independently functioning biological and environmental causes, maintains that both causes can be bifurcated and measured, and suggests that information derived from such investigations is universally generalizable, capable of revealing characteristics about the human population across time and space (Goldhaber, 2000).

Advances in research into biological and environmental contributions to development contradict the notion that genes serve as a genetic blueprint, incapable of interacting in the developmental trajectory (Gottlieb, 2007). Rather, contemporary research demonstrates that genes are dependent on environmental factors for their activation and inactivation and thus highly interactive in the developmental trajectory (Bennet et al., 2002; Caspi et al., 2002, Gottlieb, 2007). Such findings lead to the development of the theory of probabilistic epigenesis, which asserts that biological and environmental forces inextricably interact across genetic, neural, behavioral, and environmental (i.e., physical, cultural, and social) levels of analysis (Gottlieb, 2007). According to this view, environmental forces play a critical role; further, development is probabilistic and not genetically predetermined.

Such probablism is adopted by language theorists Werker and Tees (2005) who rejected the notion of critical periods (i.e., biologically determined durations of time during which linguistic input is critical to typical language acquisition) and articulated the notion of "optimal periods." Optimal periods are biologically *and experientially* determined intervals during which the timing and quality of input may extend the duration of such periods and subsequently expand the breadth and depth of language

development and competence. Werker and Tees's (1999) probabilistic epigenetic theory of optimal periods seeks to explain the initial state of infant speech perception and subsequent changes with age. The initial perceptual sensitivities in infants can be explained by a genetically activated brain composed of an overabundance of neural connections. Werker and Tees describe the human brain as (a) experience-expectant, wherein genetic potentials are elicited or inhibited and neuronal connections are strengthened or destroyed depending on early childhood experience, and (b) experience-dependent, wherein genetic potentials are elicited or inhibited and neuronal connections are strengthened or destroyed throughout the entirety of the lifespan. The experience-expectant brain is an evolved mechanism that allows rather ubiquitous human experiences (e.g., heard speech) to modify and sculpt neural connections and genetic components that facilitate language in an early optimal period, creating somewhat permanent neural structures early in development (Werker & Tees, 1999, p. 529; National Research Council and Institute of Medicine, 2000). The experience-dependent brain allows ubiquitous and individualistic human experiences to sculpt and re-sculpt neural connections and genetic components that facilitate language throughout life (Werker & Tees, 1999; National Research Council and Institute of Medicine, 2000). Werker and Tees (1999) noted that neural organization is not only influenced by heard speech, but also self-vocalizations, underscoring the contribution of individuals' behavior, motivations, and inquiry in language development. Thus Werker and Tees asserted that epigenetic processes, i.e., the perpetual interaction between the developing brain and environmental experience, facilitate language acquisition and subsequent

development. Werker and Tees's theory of optimal periods demonstrates the contextualist world view, which holds that biological and environmental forces inextricably interact, that is, that such forces cannot be bifurcated and measured, and that results from investigations are situation specific and cannot be generalized to the human population across time and space (Goldhaber, 2000).

In sum, two contemporary theories of language acquisition have emerged from the early nature versus nurture debate– Pinker's (1994) semantic bootstrapping hypothesis and Werker and Tees's (2005) theory of probabilistic optimal periods– which both provide articulate descriptions for different aspects of language acquisition. Pinker's (1994) semantic bootstrapping hypothesis provides an articulate account of evolutionary and cognitive processes involved in language acquisition, but the semantic bootstrapping hypothesis has been criticized for having limited explanatory value beyond children's innate linguistic categories. For example, many nouns do not refer to objects (e.g., the landing of the plane), many verbs do not indicate action (e.g., to desire), and many subjects are not always agents of action (e.g., John received a gift) (Rondal & Cession, 1990). One proposed solution to this problem is that children may learn to categorize atypical word types by observing their distribution within the known categories (Pinker, 2004). Additionally, although Pinker recognizes that environmental forces yield an influence, he focused primarily on evolutionary characteristics and genetic determinism. This compromises the utility of Pinker's hypothesis when striving to elucidate factors that maximize conditions for children's optimum language acquisition, in that evolutionary and genetic predispositions are unchangeable, whereas environmental factors that inhibit

or elicit genetic potentials are, to varying extents, malleable. Theories such as that of Gilbert Gottlieb's (2007) probabilistic epigenesis and Werker and Tee's (2005) optimal periods underscore the importance of developing children's language environment, espousing the contextualist worldview that environmental forces are capable of eliciting or inhibiting genetic potentials. The following literature review examines integral environmental variables whose influence on language performance may be moderated by genetic material.

CHAPTER III

EMPIRICAL REVIEW

Environmental Contributors to Early Language Performance

The following section reviews key environmental variables hypothesized to influence children's language performance and development. Findings from previous studies are provided to support the validity of these hypotheses. Environmental variables reviewed include income, parental education, parental provision of cognitive stimulation, parental emotional supportiveness, and child sex. In addition, a review of the impact of indices of environmental risk and advantage is provided. Hypotheses include positive associations between income, parental education, parental provision of cognitive stimulation, and parental emotional supportiveness and children's language performance. Female sex is hypothesized to be associated positively with children's language performance. The index of environmental risk is hypothesized to be negatively associated with children's language performance. The index of environmental advantage is hypothesized to be positively associated with children's language performance.

Income

Household income is hypothesized to influence children's language performance in that income reflects families' abilities to provide cognitively stimulating materials and

experiences that facilitate children's language development; further, income reflects families' ability to provide for children's biological needs, i.e., proper nutrition, to facilitate proper cognitive and language development. The link between income and children's developmental outcomes is well-established (Brooks-Gunn & Duncan, 1997; Dearing et al., 2001; NICHD Early Child Care Research Network, 2001a, 2002; Mistry et al., 2004, Tamis-LeMonda, 2008). Various studies find an association between low-income and children's language delays: Children from poor families have lower language abilities at younger ages and construct vocabularies at slower rates as compared to their wealthier peers (Hart & Risley, 1995; Nord, Lennon, Liu, Chandler, 2000; Hoff, 2003). Characteristics of low-income such as lack of nutrition and exposure to pollutants are capable of influencing brain development and function (Farah, Shera, Savage, Betancourt, Giannetta, Brodsky, et al., 2006). Further, the association between poverty and children's lower language performance can in part be attributed to the finding that poverty, especially persistent poverty, is linked to less cognitively stimulating home environments (Brooks-Gunn & Duncan, 1997; Smith, Brooks-Gunn, Klebanov, 1997).

Income indicates, in part, the extent to which parents are capable of providing learning environments that facilitate children's language development. Parents with more resources, e.g., higher income, are able to better provide positive language learning experiences for their children (Tamis-LeMonda & Rodriguez, 2008). Poor families have less money for basic resources such as food, clothing, and shelter, and as a result are often unable to afford various cognitively stimulating toys, materials, and experiences to facilitate their children's cognitive development (Votruba-Drzal, 2003). Further,

materials in the home present the opportunity for parent-child communication, children's symbolic play, and development of children's receptive skills and positive approaches to learning (Tomopoulos, Dreyer, Tamis-LeMonda, Flynn, Rovira, Tineo, et al., 2006; Gottfried, Fleming, Gottfried, 1998). For example, research demonstrates an association between children's familiarity with storybooks and children's receptive and expressive vocabularies (Payne, Whitehurst, Angell, 1994; Senechal, LeFevre, Hudson, Lawson, 1998).

More scientifically advanced research has begun to examine the influence of income on the functioning of various neurocognitive systems. Noble, Norman, and Farah (2005) used a sample of kindergarten children to investigate the influence of low- and middle-socioeconomic factors on five neurocognitive systems, the occipitotemporal/visual cognition, the parietal/spatial cognition, the medial temporal/memory, the left perisylvian/language, and the prefrontal/executive system. Results reveal that socioeconomic status (SES) was disproportionately associated with the left perisylvian/language system and the prefrontal/executive system. Specifically, SES was associated with vocabulary, syntactic ability, and phonological awareness. Farah, Shera, Savage, Betancourt, Giannetta, Brodsky, et al. (2006) further investigated underlying neurocognitive systems implicated in older children's experience of poverty, adding the medial temporal/memory system to their analysis. In relation to language, comprehension of single word lexical–semantics and sentence-level syntax were examined. Farah et al. found that SES was associated with the left perisylvian/language and the medial temporal/memory systems, and, similar to Noble et al.'s (2005) findings,

differences for other neurocognitive systems, such as the occipitotemporal/pattern vision and parietal/spatial cognition, were nonsignificant. These findings indicate that the left perisylvian/language system may be more sensitive to the influence of income as compared to other neurocognitive systems.

Thus it is hypothesized that household income will be positively associated with children's language performance, and this association will be moderated by children's genetic predisposition for language performance.

Parental Education

Measuring the contribution of parental education to children's language performance is important in two respects. First, level of parental education serves as a close estimate of the child's genetic endowment of intelligence from parents, as parental level of education can roughly reflect parents' level of intelligence potentially passed on to their children. Second, level of parental education can indicate the quality of the child's language-learning environment (i.e., the quality of language used with the child). Previous research indicates that parents with lower levels of education demonstrate less sophisticated language and literacy abilities themselves, and parents with higher levels of education have greater quality of language used when interacting with their children (Rowe, Pan, & Ayoub, 2005). Research also shows that less educated parents read less often to their children (Raikes, Pan, Luze, Tamis-LeMonda, Brooks-Gunn, Tarullo, et al., 2006; Scarborough, Dobrich, 1994).

Thus it is hypothesized that parental education will be positively associated with children's language performance, and this association will be moderated by children's genetic predisposition for language performance.

Parental Provision of Cognitive Stimulation

Parental provision of cognitive stimulation is hypothesized to influence children's language performance in that parents who provide children with higher levels of cognitive stimulation may be using greater quantity and quality of language, thus also promoting their children's language development. Indeed, previous research demonstrates the unique influence of parental provision of cognitive stimulation on children's language performance. Raviv, Kessenich, and Morrison (2004) used a sample of 1,016 families from the NICHD Study of Early Child Care and Youth Development to investigate influences on three-year olds' receptive and expressive language skills. Results revealed that the relation between SES and children's language development was mediated by the role of parent-child interaction, specifically that of maternal cognitive stimulation.

Hoff (2003) conducted a longitudinal study with naturalistic data collection at two points, ten weeks apart, and found that differences in vocabulary development in children of higher and lower SES were fully mediated by maternal speech. Hoff observed that mothers of higher SES spoke with longer utterances, used richer vocabulary, and created more complex sentences than mothers of lower SES. These are particularly beneficial practices, as children who hear longer utterances build larger expressive vocabularies at a more rapid pace than children who hear shorter utterances (Rowe, 2008). Hoff (2003)

proposed that, in addition to biological disparities related to genetic endowment or health, such language disparities in children of different SES could result from global effects of disparities in family interactions and home environments or specific effects of disparities in language learning experiences. Notably, Hoff (2003) concluded that, "aspects of experience that support vocabulary acquisition are not equally available to children across socioeconomic strata" (p. 1375).

Hart and Risley (1999) conducted a comprehensive qualitative investigation into the everyday lives of young children as they acquired language. Researchers' rich qualitative data unearthed various important aspects of parent-child relations that were shown to facilitate or hinder children's language performance and development. Hart and Risley studied 42 one- and two- year old children and their parents interacting in their homes every month for 2 ½ years. Results revealed that quantity of talk was generally correlated with SES, in that parents on welfare were typically characterized as taciturn, and parents with professional jobs typically exhibited talkativeness. Irrespective of SES, however, the more parents talked with their children on a daily basis, the more rapidly their children's vocabulary expanded and the higher their children's IQ score was likely to be by the age of three. Results show that the sequence of acquisition of particular words, language structures, categories of speech, and grammatical markers was immensely similar across all children assessed, but the chronological age at which these children achieved such milestones varied tremendously. "Extra talk" (i.e., optional talk other than directives for obedience and routines of daily life, which serve the purpose of sharing ideas and solidifying relationships) was more prevalent among professional

families and the most talkative working class families. Extra talk in these families consisted of more varied vocabulary, complex ideas, subtle guidance, and positive feedback, all believed to be integral to children's cognitive development. These optional conversations occurred primarily when parents and children participated in mutual or parallel activities in which engagement was rewarding and not necessary. Vocabulary and concepts embedded in these conversations lacked planning or effort; this contributed to children's accrual of language and cognitive accomplishments. During such interactions, children were more cooperative, parents were more approving, and both parents and children were more likely to comment or elaborate on what the other stated. Linguistic abilities were observed and children were dichotomized into talkative and taciturn groups. Children of the talkative groups employed three times as many utterances per hour and utilized an average of twice as many different words, which were drawn from vocabularies on average twice as large as those of children in the taciturn group.

Thus it is hypothesized that parental provision of cognitive stimulation will be positively associated with children's language performance, and this association will be moderated by children's genetic predisposition for language performance.

Parental Emotional Supportiveness

Parental emotional supportiveness is characterized by parents' provision of experiences that facilitate children's engagement in reciprocal verbal and nonverbal exchanges that are stimulating and rewarding for children (Pungello, Iruka, Dotterer, Mills-Koonce, & Reznick, 2009). Parental emotional supportiveness is hypothesized to influence children's language performance in that parents who engage in positive

exchanges, both verbally and nonverbally, with their children may create and support greater language-learning experiences, and this may be more facilitative of children's language performance.

Research into parental emotional supportiveness repeatedly shows its association with multiple important developmental outcomes such as emotional security, behavioral independence, social ability, intellectual achievement, and verbal ability (Bornstein, 1989; Bornstein, Tamis-LeMonda, & Haynes, 1999; Bradley, 1989; Goldberg, Lojkasek, Gartner, & Corter, 1989; Landry, Smith, Swank, Assel, & Vellet, 2001; Riksen-Walraven, 1978; Tamis-LeMonda, Bornstein, & Baumwell, 2001). Research demonstrates the positive association between parents' contingent response to their children's verbal initiatives and children's receptive and expressive vocabularies (Beals & DeTemple, 1993; Hann & Osofsky, 1996; Silven, Niemi, & Voeten, 2002; Tamis-LeMonda, Bornstein, & Baumwell, 2001). Additional research demonstrates that parental emotional supportiveness is associated positively with children's early language knowledge and literacy development (Dodici, Draper, & Peterson, 2003). Maternal responsiveness has been shown to be associated positively with achievement of language milestones in infancy and early childhood (Tamis-LeMonda, 2001).

Thus it is hypothesized that parental emotional supportiveness will be positively associated with children's language performance, and this association will be moderated by children's genetic predisposition for language performance.

Sex

Genetic studies with same- and opposite-sex twins repeatedly reveal a male disadvantage for various components of language ability (Kovas, Hayiou-Thomas, Oliver, Dale, Bishop, & Plomin, 2005). Girls more often produce language earlier than boys (Karmiloff & Karmiloff-Smith, 2001), and boys are more likely to have delays in vocabulary acquisition at age 2, but are almost as likely as girls to demonstrate typical levels of language abilities by age 4 (Dale, Price, Bishop, & Plomin, 2003). Additionally, the average ratio of males to females with Specific Language Impairment is approximately 2.8:1 (Robinson, 1987 as cited in Kovas et al., 2005).

Kovas et al. (2005) delineated three possible causes of individual differences – apart from mean differences– in language performance in males and females. First, termed qualitative differences, different genetic and environmental factors facilitate individual language disparities for males and females. One example of qualitative sex differences is that of sex-specific genetic influences that create differences for one sex and not the other, such as genes interacting with sex hormones. Second, termed quantitative differences and not mutually exclusive from qualitative differences, disparate genetic and environmental influences affect individual differences in males and females to varying extents. One example of quantitative sex differences is that of the same gene yielding a greater influence in males and thus facilitating more individual differences for males than females. Last, even though mean differences between the sexes exist, there is no difference between the genetic or environmental factors that contribute to individual differences for both males and females. That is, although males demonstrate disadvantage

in language performance, genetic and environmental factors that differentiate one male from another are the same as those that differentiate one female from another.

Thus it is hypothesized that female children will have higher language performance, and this association will be moderated by children's genetic predisposition for language performance.

Indices of Environmental Risk and Advantage

Environmental factors do not exist in isolation. Thus measuring the influence of one particular environmental factor on a developmental outcome and failing to acknowledge the multiplicity of environmental forces that ineluctably influence this outcome may not convey the most accurate conceptualization of human experience. Measuring environmental factors that exist concurrently, however, acknowledges that individuals are situated in contexts of multiple interactive internal and external forces. This approach seeks to capture a more holistic picture of developmental milieu that may indeed be closer to "real life." For example, O'Brien (2005) stated that disadvantages tend to occur in tandem (e.g., families experiencing poverty tend to have little education, live in dangerous neighborhoods, have poor health care, and work at demanding jobs with inconvenient hours); the same co-occurrence tends to hold true for advantages as well. Measuring the presence of multiple factors of environmental risk and advantage operates according to the assumption that singular environmental factors alone may not yield a strong influence on development, yet when positive or negative environmental factors accumulate in the developmental milieu, the strength of such factors is multiplicative, rather than additive.

Indeed, employing indices of environmental effects is recommended over measuring singular environmental factors for gene–environment interaction studies, as it provides a more accurate, sensitive, and reliable measurement of the environmental forces that interact with genes to elicit genetic potentials (Moffitt, Caspi, & Rutter, 2006). Gene–environment interaction studies indicate that the influence of a singular environmental factor can yield a small effect, whereas the influence of multiple environmental factors can have an effect that is quite considerable (G.W. Evans, 2003; Rutter & Quintin, 1977; Sameroff & Bartko, 1997). To use an example from the psychopathology literature, Caspi et al. (2003) found that a multiplicity of negative life events (e.g., unemployment, divorce, experience of abuse) interacted more strongly with genetic predisposition for depression as compared to a singular life event, even when any singular life event was severely traumatic (Caspi et al., 2003; Kendler et al., 2005). In the language literature, researchers examining the influence of cumulative risk on low-income children's language performance found that boys consistently had lower language performance at every level of cumulative risk as compared to girls, suggesting that boys' language abilities are more vulnerable to cumulative environmental risk than those of girls' (Stanton-Chapman, Chapman, Kaiser, and Hancock, 2004).

For this study, indices of environmental risk and advantage variables will be composed of lower and higher levels of household income, parental education, and parental provision of cognitive stimulation. It is hypothesized that environmental risk and advantage will interact with children's genetic predisposition for language performance, and this interaction may be more detectable than the interactions between genetic

predispositions and singular environmental factors. Additionally, it is hypothesized that males will perform worse in the context of environmental risk, and females will perform better in the context of environmental advantage.

Potential Moderating Genes and Their Involvement in Language Ability

This study is founded on the notion that genes enable the human capacity for language. Research into specific genes– and gene variants, or polymorphisms– demonstrates how genes may influence the development of certain areas of the brain pertaining to language ability and language learning (Bishop, 2002). The following serves as a brief review of research into specific genes and polymorphisms that have been found to be implicated in language ability.

FOXP2 is part of a larger class of genes called the FORKHEAD or FOX genes (Gontier, 2008). All FOX genes are regulators of embryogenesis. The FOXP2 gene is considered to be responsible for language impairments in the famous KE family, an extended family of 30 members, 15 of whom have a mutation on a particular segment of the FOXP2 gene and have severe language impairments (Kovas et al., 2005). FOXP2 was discovered through karyotype analysis; a translocation present in an individual with specific language disorder (SLD) was found to be located within FOXP2. Impairments associated with the FOXP2 polymorphism include difficulty producing intelligible speech and moving certain facial muscles, characteristics unaltered even after intensive speech therapy. Functional magnetic resonance imaging (fMRI) reveals that, as compared to individuals without this point mutation, the affected members process speech in incredibly disparate ways, such as notable underactivation in various brain regions

important for language processing (Liégeois, Baldeweg, Connelly, Gadian, Mishkin & Vargha-Khadem, 2003). FOXP2 has been thought to play a critical role in the development of neural systems involved in language and speech (Liégeois et al., 2003). The aggregate of findings related to FOXP2 indicates the broad role of this gene in the formation of words and language. The FOXP2 gene, however, has not been found in larger populations of children with language impairments, and thus it is unlikely that this gene is involved in more common instances of language impairment (Kovas et al., 2005). RNAi knockdown (i.e., modification in DNA or RNA expression) of FOXP2 in songbirds impeded their ability to correctly imitate songs of other songbirds (Haesler et al., 2007). This may indicate an evolutionary role of FOXP2 in the development of communication and language.

DCDC2 was first discovered in a genetic association study of 220 families with reading disabilities from Colorado (Meng, Smith, Hager, Held, Liu, Olson, et al., 2005). A separate study conducted in Germany later verified the role of this gene (Schumacher, Anthoni, Dahdouh, Konig, Hillmer, Kluck, et al., 2006). DCDC2 messenger RNA (mRNA) is expressed in the temporal cortex and cingulate gyrus, two regions of the brain specifically involved in reading. Research indicates that known susceptibility polymorphisms of DCDC2 may influence location (e.g., particular regions of the brain), developmental timing (e.g., brain development), and the amount of its protein production (Gibson & Gruen, 2008). DCDC2 may be necessary for neurons' typical movement– from the region around the brain ventricles where neurons are produced during early

embryogenesis, to the furthest layer of the cerebral cortex where they then remain throughout the rest of development (Meng et al., 2005).

DYX1C1 was first discovered by karyotype analysis in a family with reading disabilities in Finland (Nopola-Hemmi, Taipale, Haltia, Lehesjoki, Voutilainen, & Kere, 2000). DYX1C1 has been shown to be both highly expressed in the brain and to be crucially involved in neuronal migration; thus this gene is likely involved in early brain development (Wang, Paramasivam, Thomas, Bai, Kaminen-Ahola, & Kere,2006). One genetic study in Finland demonstrated an association between EKN1 (thought to be a susceptibility gene for dyslexia) and DYX1C1, yet studies using samples from the U.S. and Italy failed to replicate this finding. This illustrates that the role of DYX1C1 as a susceptibility gene for reading disability may be evident only in particular populations.

KIAA0319 was first identified in a study of 223 siblings with reading disabilities in the United Kingdom (Cope, Harold, Hill, Moskvina, Stevenson, Holmans, et al., 2005). This study revealed an association with an area adjacent to the KIAA0319 transcription start site; such areas control the timing and extent of gene transcription. Researchers found that single nucleotide polymorphisms (SNPs), or variations in single bases that occur at the rate of one per 100 bases of DNA, that are associated with the development of reading disability seem to decrease the transcription of KIAA0319. Extant in the brain more so than DCDC2, KIAA0319 is expressed specifically in the visual and parietal cortices.

ROBO1 was first identified through karyotype analysis by detecting a translocation in a family with reading disabilities (Nopola-Hemmi, Taipale, Haltia,

Lehesjoki, Voutilainen, & Kere, 2000). Researchers provided the caveat that this finding has not yet been validated in a separate cohort with reading disabilities (Gibson and Gruen, 2008). ROBO1 is unlike the aforementioned genes in that it does not influence neuronal migration. This gene encodes an axonal guidance receptor, a protein involved in receiving signals to direct the projection of axons, which transport electrical signals from the neuron (Hannula-Jouppi, Kaminen-Ahola, Taipale, Eklund, Nopola-Hemmi, Kaariainen, et al., 2005).

The five aforementioned genes have been shown to enable or influence language ability. It is the assumption of this study that these genes may be implicated in the language performance of the assessed twin sample. Consistent with probabilistic epigenetic theory, it is not the sole influence of genes that enable the human capacity for language; rather, inextricable gene–environment interaction facilitates language development. Thus, the following section reviews literature on gene–environment interaction.

Twin Studies in Language Performance

Contemporary language researchers often employ DeFries-Fulker (DF) regression to analyze twin data to estimate genetic and environmental influences on group membership in the extreme tails of continuous, normally distributed language characteristics. Both Rowe, Jacobson, Van den Oord (1999) and Friend, DeFries, Olson, Pennington, Harlaar, Samuelsson et al. (2009) employed DF regression to investigate the moderating role of the environment, specifically parental education, on genetic contributions to language performance. Rowe et al. (1999) investigated the moderating

role of parental education on separate genetic and environmental contributions to verbal IQ in a twin sample with a mean age of 16. Findings revealed that both heritability and the shared environment were moderated by level of parental education. Researchers found that among parents with higher levels of education, heritability of verbal IQ was higher and the contribution of shared environmental factors was lower as compared to parents with lower levels of education.

Friend et al. (2009) performed their study based on previous research revealing that group heritability for low (DeFries and Alarco´n 1996; Gaya´n and Olson 2001; Harlaar et al. 2005) and high (Boada et al. 2002) reading ability was large, accounting for over 50% of extreme group membership. Friend et al. (2009) sought to determine if the heritability of high reading ability was moderated by parental education in a longitudinal study with a sample of kindergarten and second grade twins. Results revealed that the heritability of reading performance significantly increased with lower levels of parental education. Researchers found that resilience, i.e., high reading ability in the context of low environmental support, was more strongly influenced by heritability than was high reading ability in the context of high environmental support.

The aforementioned twin studies analyzed environmental variables as moderators of genetic influence. This approach differs from the current study, however, which analyzes genetic predisposition as a moderator of various environmental variables. Further, the aforementioned studies employed analytic strategies incongruous with the stipulations of inextricable gene–environment interaction. It is the assertion of the author

that a different, more appropriate analytic strategy is needed in order to examine the role of inextricable gene–environment interaction.

Research into Gene–Environment Interaction

Research into gene–environment interaction is most prevalent in the field of psychopathology (Moffit, Caspi, & Rutter, 2006). It is thought that the probabilism of gene–environment interaction may help to explain instances in which environmental risks or pathogens do not determine developmental outcomes. For example, various researchers have observed that not all children who experience maltreatment develop conduct disorders, and some maltreated children demonstrate adaptive behavior that persists into adulthood (Cicchetti, Rogosch, Lynch, & Holt, 1993; McGloin & Widom, 2001).

Caspi et al. (2002) found that a polymorphism in the promoter region of the gene that encodes monoamine oxidase A (MAOA), a neurotransmitter-metabolizing enzyme, moderated the influence of childhood maltreatment on children's later engagement in violence. Individuals with low levels of MAOA expression developed conduct disorder and antisocial personality more often and committed violent crimes as adults more often as well, as compared to individuals with high levels of MAOA expression. Caspi et al. (2003) found that a polymorphism in the promoter region of the gene that transports serotonin (5-HTTLPR) moderated the effect of stressful life events on the occurrence of depression. Individuals with one or two copies of the short allele for 5-HTTLPR demonstrated more depressive symptoms and diagnosable depression after a stressful life event as compared to individuals with the long allele for this gene.

These studies had the benefit of access to individuals' genetic characteristics, e.g., polymorphisms in specified genes' regions. This information is not easily available to all researchers, nor is it common practice (as of yet) to collect such information in large national datasets. Given such constraints, researchers have developed alternative ways of investigating gene–environment interaction using twin samples.

In the absence of specified allelic characteristics, various configurations of twin studies have been employed. Seeking to examine the moderating role of genetic risk on the relationship between maltreatment and conduct disorder, Jaffee, Caspi, Moffitt, Dodge, Rutter, Taylor, et al. (2005) used 1,116 five-year-old British twin pairs from the E-Risk Longitudinal Twin Study, a sample frame of the Twins' Early Development Study (TEDS). Researchers estimated children's genetic risk as a function of their co-twins diagnosis of conduct disorder and the twin pairs' zygosity. This method was previously employed to estimate genetic risk for depression and negative life events on genetic risk for major depressive disorder (Kendler and Kessler, 1995). Jaffee et al. (2005) ranked twins' genetic risk on a continuum, classifying genetic risk as highest for monozygotic (MZ) twins if their co-twin has the diagnosis for conduct disorder, high for dizygotic (DZ) twins if their co-twin has the diagnosis for the disorder, low for DZ twins whose co-twin does not have the diagnosis for the disorder, and lowest for MZ twins whose co-twin does not have the diagnosis for the disorder. Consistent with the DSM-IV, children were diagnosed with a conduct disorder if they had a count of 3 or more symptoms on a range of scores from 0-11.

To measure the influence of gene–environment interaction on children's continuous conduct scores, Jaffee et al. (2005) conducted OLS regression analysis in which physical maltreatment and genetic risk were entered in the first step, and the interaction of maltreatment and genetic risk was entered in the second step. Results revealed that both experience of maltreatment and high genetic risk were associated with conduct disorder and that the interaction between maltreatment and genetic risk was significant. Among children at high genetic risk, maltreatment was associated with an increase of 24% in the probability of a diagnosed conduct disorder, whereas among children at low genetic risk, maltreatment was associated with an increase of 2% in the probability of a diagnosed conduct disorder. This study, relevant for its investigation of genetic predisposition in the absence of specific genetic material (through twin zygosity and co-twin scores), serves as a model for the proposed study.

Hypotheses: Bioecological and Diathesis-Stress

McGrath, Pennington, Willcutt, Boada, Shriberg, and Smith (2007) asserted that research into gene–environment interaction in psychopathology finds evidence for the diathesis-stress model, whereas research into gene–environment interaction in cognitive characteristics finds more evidence for the bioecological model (Kremen et al., 2005; Rowe, Jacobson, & Van den Oord, 1999; Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003). The diathesis-stress model predicts that diathesis, i.e., genetic vulnerability, in the context of environmental stress will increase the probability of aberrant behavior (Rende & Plomin, 1992). As such, this model suggests that the heritability of traits will be higher under conditions of risk (Rutter et al., 2006).

Conversely, the bioecological model posits that genetic predispositions are actualized in the context of environmental support and suppressed in the context of risky environments (Bronfenbrenner & Ceci, 1994; Gottesman, 1963; Scarr, 1992). From this perspective, the heritability of traits is expected to be higher for individuals exposed to enriched environments (Rutter et al., 2006).

The assertion by McGrath et al. (2007) is controversial because evidence for the diathesis-stress model in cognitive studies has been found as well (Asbury, Wachs, & Plomin, 2005; van den Oord & Rowe, 1998). Further, Price and Jaffee (2008) recently found support for the diathesis-stress model when investigating children's language performance. This study utilized a new analytic strategy to detect and quantify the effect of passive gene–environment correlation in the context of gene–environment interaction, revealing that genetic predisposition for poor verbal performance in children four years of age was more strongly expressed in high risk, i.e., more chaotic, home environments. It appears that there is not sufficient evidence to select either the diathesis-stress or bioecological models for their applicability to language investigations, and thus this study will test both models.

Research into Gene–Environment Interaction in Language Performance

McGrath, Pennington, Willcutt, Boada, Shriberg, and Smith (2007) asserted that research into the role of gene–environment interaction in speech, language, and reading research has been relatively neglected. McGrath et al. (2007) were the first researchers to investigate specific allelic characteristics activated in language outcomes. This study investigated the influence of gene–environment interaction in two Specific Speech Delay

(SSD)/Reading Disability (RD) linkage peaks on chromosomes 6 (6p22) and 15 (15q21) and used continuous measures of SES, home language/literacy environment, and frequency of ear infections to examine environmental influence. DNA was collected from children and all available biological parents. Gene–environment interaction was investigated by using the extended DF model. It was predicted that all interactions would demonstrate bioecological gene–environment interaction.

Results revealed that four of the five significant interactions operated in the bioecological direction, and one interaction between the 15th chromosome and number of ear infections operated in a diathesis-stress direction. Authors plotted these interactions with the x-axis indicating genetic predisposition, the y-axis indicating language performance, and lines designating higher and lower environmental support. Authors conducted a simple slopes analysis to test if the slopes of the environmental group lines were significantly different from zero (Aiken and West, 1991). This study found that in three out of the five cases, the slope of the more enriched environment line was significantly different from zero, and in one of the five cases, the slope was trending toward significance. In one of the five cases, the slope of the less optimal environmental line was significantly different from zero. Authors followed up this unexpected finding by examining interactions that showed nonsignificance but a trend toward significance ($p<.15$); these follow-up analyses on three trending interactions revealed support in the diathesis-stress direction. Authors state that in support of the bioecological model, the first four slopes demonstrate that genetic factors played a stronger role in environments of support and less of a role in environments of less support. The trend-level support for

diathesis-stress gene–environment interaction, however, highlights the need for further research into gene–environment interaction as a contributor to children's language.

This initial study demonstrated support for both the diathesis-stress and bioecological model. To the knowledge of the author, this is the only study that examined gene–environment interaction in language performance. For these reasons, further research into the role of gene–environment interaction in language performance is gravely warranted.

CHAPTER IV

THE CURRENT STUDY

As noted in the above review, contemporary research demonstrates associations between multiple environmental variables and children's language performance. Much less is known, however, about the moderating role of genetic predisposition for language performance and its influence on these associations. Extant literature demonstrates a lack of investigation into the role of gene–environment interaction in both language ability and disability. Further, more research is needed in order to provide support for a diathesis-stress or bioecological model of gene–environment interaction in language development.

The current study seeks to extend gene–environment research into the area of language by examining whether genetic predisposition for language performance moderates the influence of environmental variables on children's receptive and expressive language performance in a sample of MZ and DZ twins. Both distal and proximal predictors of children's language are investigated. Specifically of interest are the relations of income, parental education, parental provision of cognitive stimulation, parental emotional supportiveness, child sex, and indices of environmental advantage and risk to language performance and the extent to which these associations vary according to children's genetic predisposition for level of language performance.

Data for this study was drawn from a large and diverse twin sample collected as part of the Early Childhood Longitudinal Study- Birth Cohort (ECLS-B), a nationally representative dataset following over 10,000 children and families from birth to school entry. This study employs an analytic strategy similar to that of Jaffee et al. (2005) in determining twins' genetic predisposition for language performance. Environmental variables of income, parental education, parental provision of cognitive stimulation, parental emotional supportiveness, and indices of environmental risk and advantage are continuous, and child sex is categorical. To the knowledge of the author, this is the first study to examine the role of gene–environment interaction as a contributor to language performance in environmental conditions of both risk and advantage. The specific research questions and hypotheses guiding this study are as follows:

Research Question 1: Are the environmental variables of income, parental education, parental provision of cognitive stimulation, parental emotional supportiveness, and child sex significant predictors of preschool children's language performance?

Hypothesis 1: Consistent with prior research, it is expected that income, parental education, parental provision of cognitive stimulation, parental emotional supportiveness, and female sex will be positively associated with preschool children's language performance.

Research Question 2: Are the associations between these singular environmental variables and children's language scores moderated by children's genetic predisposition for language performance?

Hypothesis 2: Associations between these singular environmental variables and children's language scores will be moderated by children's genetic predisposition for language performance. Given the inconclusive nature of research findings into gene–environment interaction in the area of cognition, it is predicted that the diathesis-stress model will be demonstrated for children with a genetic predisposition for low language performance, and the bioecological model will be demonstrated for children with a genetic predisposition for high language performance.

Research Question 2a (Diathesis-Stress Model): Are environmental variables more or less predictive of language scores for children with a higher genetic predisposition for low language performance?

Hypothesis 2a: According to the diathesis-stress model, riskier (versus more advantageous) environments will be more predictive of language scores for children with a higher genetic predisposition for low language performance.

Research Question 2b (Bioecological Model): Are environmental variables more or less predictive of language scores for children with a higher genetic predisposition for high language performance?

Hypothesis 2b: According to the bioecological model, more advantageous (versus riskier) environments will be more predictive of language scores for children with a higher genetic predisposition for high language performance.

Research Question 3: Are indices of environmental risk and environmental advantage significant predictors of children's receptive and expressive language performance?

Hypothesis 3: It is expected that the index of environmental advantage will be positively associated with children's receptive and expressive language performance, and the index of environmental risk will be negatively associated with children's receptive and expressive language performance.

Research Question 4: Are the associations between indices of environmental risk and advantage and children's language scores moderated by children's genetic predisposition for language performance?

Hypothesis 4: Associations between the indices of environmental risk and advantage and children's language scores will be moderated by children's genetic predisposition for language performance.

Research Question 4a (Diathesis-Stress Model): Are indices of environmental risk and advantage more or less predictive of language scores for children with a higher genetic predisposition for low language performance?

Hypothesis 4a: According to the diathesis-stress model, riskier (versus more advantageous) environments will be more predictive of language scores for children with a higher genetic predisposition for low language performance.

Research Question 4b (Bioecological Model): Are indices of environmental risk and advantage more or less predictive of language scores for children with a higher genetic predisposition for high language performance?

Hypothesis 4b: According to the bioecological model, more advantageous (versus riskier) environments will be more predictive of language scores for children with a higher genetic predisposition for high language performance.

Research Question 5: Are there significant three-way interactions between genetic predisposition for language, indices of environmental risk or advantage, and child sex?

Hypothesis 5: It is expected that findings will reveal significant three-way interactions among genetic predisposition for language, environmental risk and advantage indices, and child sex; that is, that interactions between genetic predisposition and risk or advantage variables will vary by child sex.

Research Question 5a (Diathesis-Stress Model): Are indices of environmental risk more or less predictive of language scores for children with a higher genetic predisposition for low language performance, and does this pattern differ for males and females?

Hypothesis 5a: According to the diathesis-stress model, riskier (versus more advantageous) environments will be more predictive of language scores for children with a higher genetic predisposition for low language performance, and males with this higher genetic predisposition will be more negatively affected by environmental risk as compared to females.

Research Question 5b (Bioecological Model): Are indices of environmental advantage more or less predictive of language scores for children with a higher genetic predisposition for high language performance, and does this pattern differ for males and females?

Hypothesis 5b: According to the bioecological model, more advantageous (versus riskier) environments will be more predictive of language scores for children with a higher genetic predisposition for high language performance, and females with this

higher genetic predisposition will be more receptive to environmental advantage as compared to males.

CHAPTER V

METHODOLOGY

Data Source and Analysis Sample

Data used for this study is from the Early Childhood Longitudinal Study- Birth Cohort (ECLS-B), a nationally representative sample of children born in 2001 and followed through kindergarten. The ECLS-B includes oversamples of twins, children of various racial/ethnic backgrounds, and children with low and very low birth weight. Among other variables, multiple aspects of children's cognitive and socio-emotional development were measured at 9 months, 2 years, preschool, and kindergarten. For the current study, all predictor and outcome variables are evaluated at the preschool data collection point only, with the exception of one control variable, status of low birth weight, which was measured at the nine-month data collection point. Environmental variables– including household income and parents' highest level of education– assessed both mothers and fathers if both were present in the home. Parental provision of cognitive stimulation and parental emotional supportiveness were assessed for mothers mostly. Children's preschool receptive and expressive language performance serve as the focal outcome variables. Receptive vocabulary and expressive language are always examined separately, as these two aspects of language are conceptually independent.

A twin sample can be analyzed in various ways. One method is that of selecting one twin from each family so as to avoid creating observations that are non-independent.

In order to maintain the benefit of a large twin sample, a different method is employed, that of including matched pairs of twins, i.e., twins from the same family, within a sample and adjusting for dependence of observations. Jaffee et al. (2005) employed this later strategy, using a sandwich or Huber/White variance estimator in STATA 7.0. To check the integrity of their findings, Jaffee et al. reran analyses examining a randomly selected twin from each twin pair. For the current study, all twins from the ECLS-B will be analyzed, using twin pairs from the same families; adjustments for dependence of observations will be made using the Huber/White variance estimator in STATA 9.0. All analyses will be conducted a second time, randomly selecting one twin within each twin pair in order to compare the initial findings with those using a sample with independent observations.

Unweighted data were used given that the primary intent of this study was to explore gene–environment interactions rather than to generate populations estimates. All numbers reported within this study are rounded to the nearest 50 in accordance with the stipulations of reporting data from the ECLS-B.

The final analysis sample includes all cases with complete data across all study variables. Approximately 500 cases were dropped from the original 1650 children in the twin subsample of the ECLS-B because of missing data. The majority of these cases did not have the video-taped parent-child interaction assessed with the Two Bags Task. The second most common reason for removing cases was that of missing receptive or expressive language scores, which may be attributable to attrition of this longitudinal study. Missing data on other demographic study variables was infrequent.

Demographic characteristics of the analysis sample and means, standard deviations, and ranges for all variables in the proposed study are shown in Table 1. The analysis sample consists of 1150 preschool-aged MZ and DZ twins. 200 twins (17%) are MZ, and 950 twins (83%) are DZ. To determine zygosity, parents and observers were asked during the two-year data collection time point about twins' hair color, hair texture, eye color, complexion, facial appearance, shape of ear lobes, and blood type. Parents were also asked about the similarity in timing when their twins' first teeth erupted and if the twins had ever been mistaken for each other by family members or strangers; if parents responded positively, they reported how frequently this occurred.

Measures

The following section describes measures proposed first for capturing language performance and second for capturing environmental variables hypothesized to influence children's language performance. Means and standard deviations are provided for the twin sample.

Language Measures: Preschool language assessment consisted of a group of vocabulary items from the Peabody Picture Vocabulary Test (PPVT) Third Edition (Dunn and Dunn, 1997) and the PreLAS 2000 Let's Tell Stories (Duncan and DeAvila, 1998). The PreLAS Simon Says, the PreLAS Art Show, and the first five items of the PPVT served as a screening device to determine whether children's language assessment should be conducted in English. The PreLAS Simon Says language screener consisted of ten receptive vocabulary items, in which children were asked in English to execute a series of simple movements. The PreLAS Art Show language screener consisted of nine

expressive vocabulary items, in which children were asked in English to identify objects shown in seven pictures and state the function of three of the objects. In order to qualify for assessment in English, children must have answered at least one non-practice item from the first five items of Simon Says, Art Show, or the PPVT. If children did not respond correctly to any of these items, and children's parents verified that the children understood Spanish, test administrators provided the preschool language assessment in Spanish.

For the formal language assessment, receptive vocabulary was directly assessed by the PPVT. Children's receptive vocabulary scores were based on 15 of 16 PPVT receptive vocabulary items. One item was removed because of differential item functioning. Children were asked to choose one of four pictures that represented the stimulus word. Scores were continuous, ranging from 0-15. Average reliability (percent agreement) for receptive vocabulary was .83 (Snow, Thalji, Derecho, Wheeless, Lennon, Kinsey, et al., 2007). For receptive vocabulary the mean was equal to 8.54, and the standard deviation was equal to 1.84.

Expressive language was directly assessed by the Let's Tell Stories subtest of the PreLAS 2000. Children were read two short stories, Story 1 and Story 2. After each story, children were asked to retell the story making references to given picture prompts. Children's responses were tape recorded, and coders at RTI scored children's responses using holistic scoring instructions provided for the items. Scores were continuous, ranging from 0-5, (0= no response or "I don't know," 1=short isolated phrases, 2=disconnected thoughts, at least one complete sentence, many grammatical errors, 3=

recognizable story line, limited detail, grammatical errors, 4= a recognizable version of a story in coherent, fluent sentences, and 5= articulate, detailed sentences, vivid vocabulary, and complex constructions). Average reliability (percent agreement) for expressive language was .83 (Snow, Thalji, Derecho, Wheeless, Lennon, Kinsey, et al., 2007). For expressive language the mean was equal to 2.38 and the standard deviation was equal to .97.

Household Income: During the parent interview, parents were asked to report their annual household income using categories ranging from 1 ($5,000 or less) to 13 ($200,001 or more). This item includes both mothers' and fathers' (or guardians') income if both are present in the home. If only one parent or guardian is present in the home, this item reflects the income of the single parent or guardian. Household income is represented as continuous variable in the analysis. For the preschool twin sample the mean was equal to 8.69 and the standard deviation was equal to 3.45 (8 = $35,001-$40,000; 9 = $40,001-$50,000).

Parental Education: Parents' level of education reflects the highest level of education of either parent or guardian living in the household. If the household only had one parent, the parent education variable was equal to the highest level of education held by either parent or guardian living with the child. Parents' highest level of education at the preschool data collection time point was based upon two items on the parent instrument. The first item asks for the highest grade completed, and the second asks about the completion of a high school diploma or equivalent. The variable for parental education is continuous, ranging from 1-9, which represents a spectrum from 8th grade or

below to a doctorate or professional degree. The mean of the composite variable for both mothers' and fathers' highest level of education is equal to 5.35, and the standard deviation is equal to 2.00.

Proximal Environmental Factors: Parental provision of children's cognitive stimulation and parental emotional supportiveness were assessed by the Two Bags Task in preschool. Mothers were most often assessed to capture these constructs; if mothers were not available, fathers or other guardian figures were assessed. The Two Bags Task, a modification of the Three Bags Task used in the Early Head Start Research and Evaluation Project (Love et al., 2002) and in the National Institute of Child Health and Human Development (NICHD) Study of Early Child Care and Youth Development (1999), provides a standardized, semi-structured play context, during which parent-child interaction can be observed. The Two Bags Task was administered separately for each child in a twin pair.

During the preschool ECLS-B Two Bags Task, the parent and child are videotaped while playing for 10 minutes with items from two different bags. The first contained the children's picture book Corduroy (Freeman, 1968) and the second bag contained Play-Doh and cookie cutters. During the interaction, the parent is instructed to begin with the first bag before playing with the second bag. The parent is told to interact with her child as usual; the parent was told that she has 10 minutes to interact with her child. Interviewers read instructions to the parents verbatim. The interaction is captured on DVD and coded using the system developed in conjunction with the Early Head Start Evaluation study. This system provides ratings of the quality and quantity of parent and

child behaviors. Scores were ascertained from five parental behaviors (i.e., parental emotional supportiveness, parental provision of cognitive stimulation, parental intrusiveness, parental negative regard, and parental detachment) and three child characteristics (i.e., child engagement of parent, child quality of play, and child negativity toward parent). Two parental behaviors, those of parental provision of cognitive stimulation and parental emotional supportiveness are of interest to the current study.

Parental Provision of Cognitive Stimulation: The ECLS-B conceptualizes parental provision of cognitive stimulation as parents' teaching that seeks to increase their child's language, perceptual, and cognitive development. Parents that stimulate their children's development are characterized as those who are cognizant of their child's level of development and seek to raise their child up to the next developmental level. One item from the Two Bags Task, parents' provision of cognitive stimulation, is used in this study. This construct is measured on a 7-point Likert-type rating scale, where parents' provision of cognitive stimulation is rated from very low (1) to very high (7). Average reliability (percent agreement) for parental provision of cognitive stimulation was .97 (Snow, Thalji, Derecho, Wheeless, Lennon, Kinsey, et al., 2007). The mean score for this sample is 4.35, and the standard deviation is .95.

Parental Emotional Supportiveness: The ECLS-B characterizes parental emotional supportiveness as involving parents' provision of a secure base from which their child feels safe to explore, in addition to parents' display of emotional support and enthusiasm of their child and their independent play or work. One item from the Two Bags Task is used to measure parents' emotional supportiveness toward their children in

the 10-minute videotaped interaction used in this study. This construct seeks to capture the extent to which parents are emotionally available to their children and the extent to which parents exhibit physical and affective presence during the interaction task. This construct is measured on a 7-point Likert-type rating scale, where parental emotional supportiveness is rated from very low (1) to very high (7). Average reliability (percent agreement) for parental provision emotional supportiveness was .97 (Snow, Thalji, Derecho, Wheeless, Lennon, Kinsey, et al., 2007). The mean score for this sample is 4.58, and the standard deviation is .92.

Indices of Environmental Risk and Advantage: Environmental variables including household income, parental education, and parental provision of cognitive stimulation were used to create an index of multiple risks and advantages experienced by twins. Parental emotional supportiveness was considered as a candidate for the risk and advantage index variables. Upon creating composite variables with household income, parental education, parental provision of cognitive stimulation, and parental emotional supportiveness, the risk and environmental advantage variables did not significantly interact with genetic predisposition variables, as was found for the composite environmental variable that excluded parental emotional supportiveness. Regression analyses indicated that parental emotional supportiveness did not significantly predict either children's receptive or expressive language scores, and thus it appears that this construct's main effect, in addition to its interaction with genetic predisposition, was less important, or at least not statistically significant as a contributor to children's receptive and expressive language.

Household income, parental education, and parental provision of cognitive stimulation were dichotomized as close to the bottom quartile of the sample distribution as possible (as allowed by the division of scores) to indicate risk. Household income was dichotomized at the 25.9% of the sample distribution, indicating income at $30,000 and lower. Parental education was dichotomized at 24.9% of the sample distribution, indicating education of a high school diploma/equivalent and lower. Parental provision of cognitive stimulation was dichotomized at 16.7% of the distribution, indicating parents who received a score of 4 or less on the cognitive stimulation item in the Two Bags Task. These dichotomous variables were combined to create a variable that captures index of risk, which ranges from 0 (no risk) to 3 (risk in all three domains). The mean of the risk index for this sample is .68, and the standard deviation is .92.

Household income, parental education, and parental provision of cognitive stimulation were dichotomized as close to the upper quartile of the sample distribution as possible (as allowed by the division of scores) to indicate advantage. Household income was dichotomized at 77.6% of the sample distribution, indicating income levels of $75,001 and higher. Parental education was dichotomized at 93% of the sample distribution, indicating education levels of a Master's degree and higher. Parental provision of cognitive stimulation was dichotomized at 90.8% of the sample distribution, indicating parents who received a score of 5 or higher on the cognitive stimulation item in the Two Bags Task. These dichotomous variables were combined to create a variable that captures index of advantage, which ranges from 0 (no advantage) to 3 (advantage in

all three domains). The mean of the advantage index for this sample is 1.02, and the standard deviation is .99.

Genetic Predisposition for Language Performance: To analyze gene–environment interaction with precision, researchers must articulate the genotype that interacts with specific environmental variables (Jaffee et al., 2005). When such genetic information is not available, data from MZ and DZ twins can be used to capture gene–environment interaction (Jaffee et al., 2005; Andrieu & Goldstein, 1998; Kendler & Kessler, 1995; Ottman, 1994). Jaffee et al. (2005) stated that twins' genetic risk for a disorder can be estimated as a function of both their zygosity (i.e., MZ or DZ) and their co-twins' diagnostic status. If an MZ twin's co-twin has been diagnosed with a disorder, the individual's genetic risk is considered high; if an MZ twin's co-twin has not been diagnosed for a disorder, genetic risk is considered low. Studies into conduct disorder and language disability have indicated twins' genetic risk by providing each child with a score based on their zygosity and co-twin's diagnostic status (e.g., genetic risk if MZ co-twin has disorder= 3; if DZ co-twin has disorder= 2; if DZ co-twin does not have disorder= 1; if MZ co-twin does not have disorder= 0). Thus, each twin can be placed on a continuum of genetic predisposition for a trait based on the co-twin's performance on the outcome of interest and the focal twin's zygosity.

The proposed method for measuring genetic predisposition for high and low language performance is modeled after the measure used by Jaffee et al. (2005). This study will use co-twins' language scores and zygosity to create a proxy for genetic predisposition for language performance. Each child will be categorized according to

their own language score, i.e., their membership within or outside the lowest 25% of language scores and the highest 25% of language scores, and separate analyses will be conducted for receptive and expressive language scores. If the focal twin's co-twin scored in the in the lowest or highest 25th percent and the pair is MZ, the focal twin receives a value of 4 for genetic predisposition in the regression equation. If the focal twin's co-twin scored in the lowest or highest 25th percent and the pair is DZ, the focal twin receives a value of 3. If the focal twin's co-twin did not score in the lowest or highest 25th percent and the pair is DZ, the focal twin receives a value of 2. If the focal twin's co-twin did not score in the lowest or highest 25th percent and the pair is MZ, the focal twin receives a value of 1. Table 2 shows the percentages of twins located in each genetic predisposition for language (high/low, receptive/expressive) category.

Covariates: The following demographic variables are included in the analysis as covariates: child age (in months), birth weight status (not low birth weight/low birth weight), child race (non white/white). Child age is used as a covariate because differences in child age, and thus language developmental progress, can influence children's language scores. Child birth weight status is used as a covariate because substantial developmental literature indicates that low birth weight status is a major risk factor for children's development, and low birth weight status and can have considerable negative implications for neurological development. Literature has demonstrated that children of low birth weight have impeded growth of receptive vocabulary and poorer language skills in general as compared to children of normal birth weight (Stolt, Haataja, Lapinleimu, Lehtonen, 2009). Last, child race is used as a covariate to acknowledge that

there are cultural biases in standardized testing that often favor white culture, which is relevant to the language assessments administered in the ECLS-B; further, the cultural sensitivity of the Two Bags Task, which measures parental provision of cognitive stimulation and parental emotional supportiveness in the ECLS-B has not yet been determined.

CHAPTER VI

RESULTS

Preliminary Analyses

The assertion that language is genetically influenced warrants a comparison by zygosity of the correlation of language scores among twins. MZ twins share 100% of their genetic material, and DZ twins share, on average, 50% of their genetic material; thus if MZ twins demonstrate correlations of language scores that are higher than those of DZ twins, it can be concluded that genetics is playing some role in the manifestation of this skill. Table 3 shows the correlations between the study sample's MZ and DZ twins' receptive and expressive language performance. That MZ twins demonstrated language scores that are more highly correlated for both receptive and expressive language supports the assertion that genetics plays a role in language performance for twins in this study sample. Fischer's r-to-z transformation was used to determine if correlations of scores between MZ and DZ twins are significantly different. The difference between MZ and DZ twins for expressive language was significant ($z=2.71$), and the difference between MZ and DZ twins for receptive language was approaching significance ($z=1.83$).

Independent samples t-tests were conducted to test if the means of environmental variables, sex, controls, and language scores were significantly different between MZ and DZ twins. Table 4 displays the results of the independent t-tests. There were no significant differences between means of MZ and DZ twins across any of the variables

with the exception of household income. On average, DZ twins lived in families with higher annual income.

Table 5 shows correlations between all study variables. All environmental variables, which include household income, parental education, parental provision of cognitive stimulation, and parental emotional supportiveness, were all significantly correlated with receptive and expressive language at $p<.01$ in the expected direction. Parental emotional supportiveness demonstrated the lowest correlation to receptive ($r=.15$) and expressive language ($r=.11$) as compared to all other environmental variables. Parental education demonstrated the highest correlation with receptive vocabulary ($r=.37$), and household income demonstrated the highest correlation with expressive language ($r=.24$). Correlations between each environmental variable and receptive vocabulary were higher as compared to correlations between each environmental variable and expressive language. Risk and advantage were also significantly correlated with both receptive and expressive language in the expected directions. Child sex was significantly and positively correlated with both receptive ($r=.10$) and expressive ($r=.14$) language (child sex coded as 1=male and 2=female). The covariates of child age at the time of assessment, race, and birth weight status were all significantly correlated to receptive and expressive language in the expected direction, suggesting that their use as covariates is warranted. All genetic predisposition variables were significantly but not highly correlated with receptive and expressive language. The highest correlation among environmental variables was between household income and parental education ($r=.66$).

Gene–environment correlations can be examined by evaluating the associations between genetic predisposition variables and environmental variables (Jaffee, et al., 2005). Correlations demonstrated a small to moderate gene–environment correlation, similar to that found by Jaffee et al. (2005). Correlations between genetic predisposition variables and environmental variables were highest for household income and parental education, and these correlations were higher for genetic predisposition for receptive vocabulary as compared to expressive language. The strongest correlation among these variables was that of genetic predisposition for low receptive vocabulary and household income ($r=-.25$), which is followed by the correlation between genetic predisposition for high receptive vocabulary and parental education ($r=.24$), the correlation between genetic predisposition for high receptive vocabulary and household income ($r=.24$), and the correlation between genetic predisposition for low receptive vocabulary and parental education ($r=-.23$). These correlations were significant at $p<.01$. The correlations among genetic predisposition variables for expressive language and environmental variables were lower. The strongest correlation among expressive language variables was that between genetic predisposition for high expressive language and parental education ($r=.16$), which was followed by the correlation between genetic predisposition for high expressive language and household income ($r=.16$), the correlation between genetic predisposition for low expressive language and household income ($r=-.14$), and the correlation between genetic predisposition for low expressive language and parental education ($r=-.11$). These correlations were significant at $p<.01$.

Correlations between genetic predisposition variables and parental provision of cognitive stimulation and parental emotional supportiveness were lower than those involving income and parental education. Genetic predisposition for high receptive vocabulary was significantly correlated with parental provision of cognitive stimulation (r=.15) and parental emotional supportiveness (r=.11), and genetic predisposition for low receptive vocabulary was significantly correlated to parental provision of cognitive stimulation (r=-.14) and parental emotional supportiveness (r=-.82). These were significant at $p<.01$. Genetic predisposition for high expressive language was significantly correlated to parental provision of cognitive stimulation (r=.07) but not significantly correlated to parental emotional supportiveness. Genetic predisposition for low expressive language was significantly correlated to parental provision of cognitive stimulation (r=-.06) but not significantly correlated to parental emotional supportiveness.

Hierarchical Multiple Regression Analyses

A series of hierarchical multiple regression analyses were conducted to investigate gene–environment interaction as a contributor to children's language performance. The first set of analyses investigated the effects of singular environmental variables and gene–environment interaction, in which each of the four genetic predisposition variables (genetic predisposition for high receptive vocabulary, genetic predisposition for low receptive vocabulary, genetic predisposition for high expressive language, and genetic predisposition for low expressive language) were multiplied by each singular environmental variable. These four analyses are presented in Tables 6-9. The second set of analyses investigated the effects of indices of environmental risk and

advantage and gene–environment interaction, in which each of the four genetic predisposition variables were multiplied by environmental risk and environmental advantage. A third set of analyses expanded on these models to investigate three-way interactions among genetic predisposition, risk or advantage variables, and child sex. The second and third set of analyses were conducted in eight models, in which the first four analyses examined two- and three-way interactions involving environmental risk; the second four analyses examined two- and three-way interactions involving environmental advantage. These eight analyses are presented in Tables 10-17.

The twin sample of this study contains twin pairs belonging to the same family, and thus observations of children's language performance are non-independent. In order to correct for this non-independence, all regression analyses were conducted using a sandwich or Huber/White variance estimator in STATA 9.0 (Rogers, 1993; Williams, 2000; StataCorp, 2001). This strategy adjusts estimated standard errors, correcting for dependence of observations due to analyzing twin pairs within the same families. To further verify the findings from the Huber/White analysis, all analyses were rerun with a subsample created by randomly selecting one twin within each twin pair. There were no notable differences in findings across the Huber/White analyses and the analyses randomly selecting one twin within each twin pair. Results reported are those using the Huber/White analysis for the full twin sample (N=1150).

Gene–Environment Interaction Involving Singular Environmental Variables

To investigate the associations between singular environmental variables and children's language performance and the potential moderating role of genetic

predisposition, the first four analyses were conducted in four steps. Covariates were entered in Step 1, singular environmental variables were entered in Step 2, genetic predisposition variables were entered in Step 3, and interaction terms between genetic predisposition and singular environmental variables were entered in Step 4. All continuous main effect variables were centered at the mean with the purpose of reducing multicollinearity. Interaction terms were a product of centered genetic predisposition variables and centered or categorical singular environmental variables.

All four regression models were significant ($p<.001$). In every model, the addition of singular environmental variables to the covariates created a significant change in R^2. All singular environmental variables were shown to significantly predict both receptive vocabulary and expressive language performance, with the exception of parental emotional supportiveness; this variable was not a significant predictor of either receptive vocabulary or expressive language. For receptive language, the R^2 value indicates that the main effect of singular environmental variables and controls accounted for 28% of the variance in children's receptive vocabulary performance. For expressive language, the R^2 value indicates that the main effect of singular environmental variables and controls accounted for 14% of the variance in children's expressive language performance.

The main effects of these variables were analyzed to examine their relation to each language outcome, however, because probabilistic epigenesis posits that genetic and environmental forces inextricably interact, main effects are not emphasized in this study. Rather, interaction effects between genetic predisposition, environmental variables, and

child sex are the main focus of this study and will thus be explored in the subsequent analyses.

Within the genetic predisposition x singular environmental variable models, one interaction term, genetic predisposition for low expressive language x parental provision of cognitive stimulation, was shown to significantly predict children's expressive language performance (B=.11; $p<0.05$). Table 9 displays the results of this significant interaction. Hierarchical regression indicates that the controls accounted for an initial 7% of the variance in children's expressive language performance, the addition of singular environmental variables to controls enabled the model to account for 7% more of the variance, the addition of genetic predisposition to the model then accounted for another 6% of the variance, and finally the addition of gene–environment interaction terms at the last step was shown to account for an additional 1% of the variance in children's expressive language performance. The ΔR^2 for Step 4 indicates that the addition of this gene–environment interaction variable did not significantly increase this model's ability to account for variance in children expressive language scores over and above the statistical main effect of the genetic predisposition variable in concert with controls and environmental effects. No additional interaction terms between genetic predisposition and singular environmental variables were shown to be significant.

Data were plotted to examine how genetic predisposition for low expressive language moderates the association between parental provision of cognitive stimulation and children's expressive language scores. The scatter plot in Figure 1 shows that parental provision of cognitive stimulation appears to be most predictive of expressive

language performance for children with the highest genetic predisposition for low expressive language, as exhibited by this group's steepest slope. Parental provision of cognitive stimulation appears to be slightly less predictive of expressive language performance for children with the second highest genetic predisposition for low expressive language, as exhibited by this group's second steepest slope. Parental provision of cognitive stimulation appears to be even less predictive of expressive language performance for children with the third highest genetic predisposition for low expressive language, as exhibited by this group's third steepest slope. Last, parental provision of cognitive stimulation appears to be least predictive of expressive language performance for children with the least genetic predisposition for low expressive language, as exhibited by this group's smoothest slope.

A clear pattern is evident in this scatterplot: Parental provision of cognitive stimulation was most predictive of expressive language performance for children with the highest genetic predisposition for low expressive language, and incrementally less so for lower levels of genetic predisposition for low expressive language.

That the initial analyses could detect one gene–environment interaction term as a significant predictor when a singular environmental variable was involved is notable given scholars warnings that detecting gene–environment interaction is difficult and that genes interacting with indices of environmental factors are more likely to be detected by statistical analysis (Moffitt et al., 2006). Thus additional analyses were conducted to further investigate if indices of environmental risk and advantage interacted with genetic predisposition for language variables.

Gene–Environment Interaction Involving Environmental Risk and Advantage Indices

To investigate the associations between indices of environmental risk and advantage and children's language performance and the potential moderating role of genetic predisposition, eight analyses were conducted in four steps. Covariates were entered in Step 1, risk or advantage variables were entered in Step 2, genetic predisposition variables were entered in Step 3, and two-way interaction terms between genetic predisposition and risk or advantage variables were entered in Step 4. Risk and advantage variables were centered at the mean with the purpose of reducing multicollinearity. Interaction terms were a product of centered genetic predisposition variables and centered or categorical environmental variables.

All eight regression models were significant ($p<.001$). Within the genetic predisposition x indices of environmental risk and advantage models, one interaction term, genetic predisposition for low expressive language x environmental advantage, was shown to significantly predict children's expressive language performance (B=.12; $p<0.05$). Table 17 displays the results of this significant interaction. Hierarchical regression indicates that the control variables accounted for an initial 8% of the variance in children's expressive language scores, the addition of environmental advantage to the control variables enabled the model to account for 6% more of the variance, genetic predisposition then accounted for another 6% of the variance, and finally the addition of the two-way gene–environment interaction term at the last step was shown to account for 1% of the variance in children's expressive language performance. The ΔR^2 for Step 4 indicates that the addition of this gene–environmental variable significantly increased this

model's ability to account for variance in children's expressive language performance ($F=6.21$, $\Delta R^2=0.01$, $p<.05$). No additional interaction terms between genetic predisposition and environmental risk or advantage were shown to be significant.

Data were plotted to examine how genetic predisposition for low expressive language moderates the association between advantage and children's language scores. Figure 2 shows this significant interaction. The scatterplot shows that environmental advantage appears to be most predictive of expressive language performance for children with the highest genetic predisposition for low expressive language, as exhibited by this group's steepest slope. Environmental advantage appears to be slightly less predictive of expressive language performance for children with the second highest genetic predisposition for low expressive language, as exhibited by this group's second steepest slope. Environmental advantage appears to be even less predictive of expressive language performance for children with the third highest genetic predisposition for low expressive language, as exhibited by this group's third steepest slope. Last, environmental advantage appears to be least predictive of expressive language performance for children with the least genetic predisposition for low expressive language, as exhibited by this group's smoothest slope.

The pattern evident in this scatterplot mirrors the one observed in Figure 1: Environmental advantage was most predictive of expressive language performance for children with the highest genetic predisposition for low expressive language, and incrementally less predictive as genetic predisposition for low expressive language becomes incrementally lower.

Examining Three-way Gene x Environment x Sex Interaction

The third and final investigation was that of the interaction between genetic predisposition, indices of environmental risk and advantage, and child sex; that is, if interactions between genetic predisposition and indices of environmental risk and advantage vary as a function of child sex. These three-way interactions were examined because previous literature indicates that there are consistent differences in female and male language performance, in that females more often demonstrate higher language proficiency. There are various reasons for sex differences, and one prominent reason is that genes for language interact with sex hormones in disparate ways, yielding differences in language performance for males and females (Kovas et al., 2005).

In order to investigate interactions between genetic predisposition, indices of environmental risk or advantage, and child sex, these three-way interaction variables were added as Step 5 to the hierarchical regression models using indices of environmental risk and advantage. Within these three-way interaction models, one interaction, genetic predisposition for high receptive vocabulary x advantage x sex, was shown to significantly predict children's receptive language performance ($B = .28$; $p < .10$). Table 14 displays this significant interaction. Hierarchical regression indicates that controls accounted for an initial 19% of the variance in children's receptive vocabulary performance, the addition of singular environmental variables accounted for another 7% of the variance, and the addition of the following three blocks– which consists of genetic predisposition for high receptive vocabulary, the two-way interaction terms, and the three-way interaction term – accounted for an additional 7% of the variance in children's

receptive vocabulary performance. In Step 5 additional two-way interaction terms and the three-way interaction term are added to the model, and the ΔR^2 indicates that this addition significantly increased this model's ability to account for variance in children's receptive vocabulary performance ($F=2.64$, $\Delta R^2=0.004$, $p<.05$). No additional three-way interaction terms were shown to be significant.

Data were plotted to examine how genetic predisposition for high receptive vocabulary moderates the association between advantage and children's language scores and how this interaction varies by sex. Figure 3 shows this significant three-way interaction. To note similarities between the scatterplots for males and females, environmental advantage is most predictive of receptive vocabulary performance for males and females with the highest genetic predisposition for high receptive vocabulary, and environmental advantage is incrementally less predictive of receptive vocabulary performance as genetic predisposition for high receptive language becomes incrementally lower.

The most salient difference across males and females in the interaction among environmental advantage, genetic predisposition, and receptive language is that environmental advantage is most predictive for females with the highest genetic predisposition for high receptive vocabulary as compared to all other genetic predisposition groups of females and males. That environmental advantage is less predictive at lower levels of genetic predisposition for males and females indicates that this three-way interaction is largely generated by the steep slope for females with highest

genetic predisposition for high receptive language, in that this groups' slope differs substantially in comparison to the slopes of all other male and female groups.

CHAPTER VII

DISCUSSION

This study sought to respond to two prominent criticisms in the field of human development; first, there is currently a lack of concordance between researchers' complex theoretical frameworks and employed analytic strategies, and second, there is a dearth of research into gene–environment interaction in areas of well-being and school achievement (O'Brien, 2005; Moffit, Caspi & Rutter, 2006). The purpose of this study was to investigate the potential moderating role of genetic predisposition for language performance on various environmental variables previously linked to early language development. The specific environmental factors examined in this project included the more distal variables of household income and parental education and the more proximal variables of parental provision of cognitive stimulation and parental emotional supportiveness. Child sex was investigated as previous literature supports its relation to language performance. Risk and advantage were also evaluated as previous literature indicates that indices of environmental factors can elicit genetic potentials (Moffitt et al., 2007).

This study demonstrated that genetic predisposition for language is expressed in different ways depending on environmental context. The results of this study overall show tentative support for the notion of gene–environment interaction as articulated in the theory of probabilistic epigenesis (Gottlieb, 2007). In relation to theories of language

and language acquisition, these results may serve as preliminary evidence that language does not operate according to genetic determinism; rather, results support the notion of probabilistic theories of language (Werker & Tees, 2005). This study demonstrates that genetic predisposition may interact with both proximal environmental variables and indices of environmental risk and support (Moffitt et al., 2006). Findings of this study also indicate that sex may indeed influence children's language performance (Kovas et al., 2005), and that interactions between genetic predisposition and environmental factors can vary according to sex.

More specifically, this study found that gene–environment interaction terms can be detected as significant predictors of language performance and that such terms, when added to the main effects of their respective hierarchical multiple regression models, can improve their models' ability to account for variance in language performance. Significant gene–environment interactions surfaced at three different levels of analysis. One significant interaction term was found for genetic predisposition with singular environmental influence; however, the effect was small and did not significantly add to the model's ability to account for variance in children's language performance. One significant interaction was found for genetic predisposition with an index of environmental advantage; the ΔR^2 found for this second two-way interaction revealed that adding this interaction term to its respective model significantly increased this model's ability to account for variance in children's expressive language performance. The last significant interaction was found for genetic predisposition with an index of environmental advantage and child sex; the ΔR^2 for this three-way interaction revealed

that adding this interaction term to its respective model significantly increased this model's ability to account for variance in children's receptive vocabulary performance.

The first significant interaction was found between genetic predisposition for low expressive language and parental provision of cognitive stimulation. That a significant interaction between genetic predisposition and a proximal environmental factor was found is supported by literature that explains that statistically significant gene–environment interaction is more likely for proximal, as opposed to distal, environmental factors because proximal factors have a more direct influence on individuals' neurobiological systems (Moffitt, Caspi, Rutter, 2006). For example, parental facilitation or lack of facilitation of children's language development can directly and specifically enable children to acquire language and form neural connections in language learning areas of the brain. Further, distal environmental factors may play an important role in language learning and neural connections, yet the effects of these variables are often mediated by more proximal factors (Moffitt, et al., 2006). This notion can in part help to explain why other gene–distal environmental interactions were not found in this study (e.g., for household income and parental education). Further, the notion that gene–environment interactions are harder to detect for singular, as opposed to multiple, environmental factors can also help to explain why other regression analyses addressing genetic predisposition interaction with singular environmental variables failed to detect significant interactions.

The scatterplot for this significant two-way interaction indicates that parental provision of cognitive stimulation appears to be most predictive of expressive language

performance for children with the highest genetic predisposition for low expressive language, and that parental provision of cognitive stimulation appears to be incrementally less predictive of expressive language performance as level of genetic predisposition for low expressive language becomes incrementally lower.

A second significant two-way interaction was found between genetic predisposition for low expressive language and environmental advantage. Similar to the previous pattern, the scatterplot for this interaction indicated that environmental advantage appears to be most predictive of expressive language performance for children with the highest genetic predisposition for low expressive language, and that environmental advantage appears to be incrementally less predictive of expressive language performance as level of genetic predisposition for low expressive language becomes incrementally lower.

The third and final interaction was found between genetic predisposition, environmental advantage, and child sex. In the scatterplot, a pattern of sensitivity was generally demonstrated across males and females, in that environmental advantage appears to be most predictive of receptive vocabulary performance for males and females with the highest genetic predisposition for low expressive language, and that environmental advantage appears to be incrementally less predictive of receptive language performance as level of genetic predisposition for low expressive language becomes incrementally lower. Distinctively, environmental advantage was most predictive for females in the highest genetic predisposition group as compared to all other male and female genetic predisposition groups. This form of gene–environment

interaction may be explained by the notions of sex differences in language performance articulated by Kovas et al. (2005).

The results of this study do not appear to provide unequivocal support for one particular model of gene–environment interaction. The most salient support for a particular gene–environment interaction model is found in the three-way interaction between genetic predisposition for high receptive vocabulary, advantage, and sex, which demonstrates a rather clear pattern of bioecological gene–environment interaction. In this case, environmental advantage appeared to yield the greatest positive influence for children with higher levels of genetic predisposition for high language performance and yield lesser influence for children with lower levels of genetic predisposition for high language performance. In other words, in environments with features that promote language success, children with higher levels of genetic predisposition for high receptive vocabulary performance may be more sensitive or receptive to such features as compared to children with lower levels of this genetic predisposition.

Explanations for the first two significant interactions are slightly more complex. The first two interactions appear to demonstrate a diathesis-stress pattern, in that parental provision of cognitive stimulation and environmental advantage were more predictive of expressive language for children with higher levels of genetic predisposition for low expressive language. In these cases, positive environmental conditions appear to yield the greatest positive influence for children with the highest genetic predisposition for low expressive language performance. In other words, in environments with features that promote language success, children with higher levels of genetic predisposition for low

expressive language may be more sensitive or receptive to such features in comparison to children with lower levels of this genetic predisposition.

The patterns found for these two-way interactions can also be interpreted through a third gene–environment interaction lens, which is that of differential susceptibility. The theory of differential susceptibility asserts that "something else" is going on, which is that individuals with certain genetic predispositions may be more plastic or sensitive not only in environments of risk, but also in environments of advantage (Belsky & Pluess, 2009, p. 885). That is, certain genes may not necessarily function solely to make individuals more vulnerable to environmental risk, but enable individuals to be more malleable or sensitive in the context of both environmental risk and advantage. The theory of differential susceptibility can be useful for interpreting the findings of these first two interactions: This theory offers the explanation that parental provision of cognitive stimulation and environmental advantage are more predictive for children with higher levels of genetic predisposition for low language performance because such children may be more sensitive to both positive and negative environmental conditions. This theory also explains that positive environmental conditions are less predictive for children with lower genetic predisposition for low expressive language because such children may be less sensitive or receptive to both positive and negative environmental conditions.

It is important to note that no significant interactions involving this genetic predisposition variable were detected in the context of environmental risk, thus this study cannot determine how predictive environments of risk would be for children with higher levels of genetic predisposition for low expressive language. Further, an evaluation of the

advantage scatterplot simply provides a view how associations vary along a continuum of advantage, wherein a value of zero simply represents a lack of advantage, providing no useful information pertaining to level of risk. If this study expanded its analysis to capture the full range of environmental factors from risk to advantage, data might reveal that environments of risk are also most predictive for children with the highest genetic predisposition for low expressive language. This lack of information in this study indicates that additional research is needed to further explore how predictive environments of risk are for children with higher levels of genetic predisposition for low receptive and expressive language.

The second interaction between genetic predisposition for low expressive language and advantage was a mismatch configuration of gene–environment interaction that was not considered when formulating the hypotheses for this study. That is, the consideration of genetic risk in the context of environmental advantage is not as common in the literature as the investigation of genetic risk in the context of environmental risk and genetic advantage in the context of environmental advantage. Indeed, hypotheses predicted that the diathesis-stress model would be demonstrated for children with a genetic predisposition for low language performance, and the bioecological model would be demonstrated for children with a genetic predisposition for high language performance. The mismatch interaction found in this study underscores the importance of moving beyond dual-risk and dual-advantage models when formulating gene–environment interaction hypotheses. A dual-risk gene–environment interaction model examines genetic predisposition for poor outcomes in the context of environmental risk.

A dual-advantage gene–environment interaction model examines genetic predisposition for positive outcomes in the context of environmental advantage. Formulating hypotheses from these two models alone can limit researchers' understanding of the full range of gene–environment interaction processes, some of which can be found in other configurations, such as genetic predisposition for low performance in the context of environmental advantage (as demonstrated in the current study) or genetic predisposition for high performance in the context of environmental risk (which would test for resilience).

That individuals with a genetic predisposition for low expressive language exhibited more sensitivity to environmental advantage, is a noteworthy finding. This suggests that individuals with genetic risk should not be evaluated solely in environmental risk, but also in environmental advantage. That individuals with genetic risk can exhibit greater plasticity, or more sensitivity to environmental advantage, underscores the importance of interventions for individuals with genetic vulnerabilities, in that such individuals may exhibit considerable progress given appropriate environmental support.

It is important to acknowledge that this study employed a rather conservative test of gene–environment interaction, measuring the contribution of gene–environment interaction effects over and above any statistical main effect of genes and environment. It may very well be that capturing main effects of genetic predisposition variables (that is, that which is necessitated as a separate step of hierarchical multiple regression models), is a statistical possibility but a theoretical and empirical impossibility, because such

separation and measurement is in contradiction to the notion of inextricable gene–environment interaction (Gottlieb, 2000). Because the statistical main effect of genetic predisposition was considered prior to gene–environment interaction variables, it may be that actual, not statistical, gene–environment interaction makes a larger contribution to language performance than that which is presented in this study. Nonetheless, the emphasis of this study is that hierarchical multiple regression modeling detected gene–environment interaction variables as significant predictors of children's language performance, and that scatterplots revealed that in the context of particular environmental experience, language performance varies according to genetic predisposition.

These findings should be interpreted with an acknowledgment of this study's limitations and strengths. The most prominent limitation of this study is the lack of access to children's specific genetic material related to language. This information was not available in this dataset, thus it was not possible to measure interactions between specific genes for language and the selected environmental factors. Access to specific genetic material related to language in large datasets is currently sparse, and thus use of the large twin sample and alternative proxy for genetic predisposition for language performance in this study was warranted. The employed dataset also did not provide information about parents' language performance, which could have provided an additional indicator of children's genetic endowment for language performance. Similar to all gene–environment interaction studies using twins raised in the same family, a more accurate understanding of gene–environment interaction could have been achieved if twins were separated at birth and raised in disparate adoptive families. This was not the case for

twins in this dataset, and thus this indicates a potential direction for future investigations into gene–environment interaction. Within the twin sample, there were also a small number of twins with a genetic predisposition for high expressive language (1.3% of the total sample). This was attributed to less incremental scoring of expressive language, which prevented this variable from actually capturing the highest 25th percent of the expressive language distribution. This variable was divided at the highest 14th percent of the expressive language distribution, as the next highest division point would be the highest 46th percent of the distribution, which is farther from 25 percent and less representative of actual high expressive language performance. This problem was not encountered for low expressive language, nor for high or low receptive vocabulary.

Beyond limitations of the dataset, there are certain limitations in this study's methods. In order to deviate from a focus on disadvantage and poor outcomes (e.g., language disability), this study sought to examine genetic predisposition for low and high language performance by examining membership in the upper and lower quartiles of language performance. This choice is not without its problems, in that much less can be said about the children with language scores in the middle 50 percent of the sample. Additionally, creating a dividing line at the upper and lower quartiles may have created a false sense of difference for those twin pairs that fell close to the dividing lines (e.g., wherein one twin fell closely below the 25th percentile and the co-twin fell closely above the 25th percentile). This strategy can be defended, however, in that other studies of gene–environment interaction, e.g., those in the field of psychopathology, have often relied on

a diagnostic criteria based on number of symptoms exhibited by each child, thus creating a similar line of division (Jaffee et al., 2005).

That parental emotional supportiveness was not found as a significant predictor of children's language performance was unexpected, given that previous research shows support for this association (Tamis-LeMonda, Bornstein, & Baumwell, 2001). This non-significance may in part be an artifact of method, in that this construct was measured by a single item. Parental provision of cognitive stimulation, however, was also measured by a single item, and this construct was found to significantly predict children's language performance. In comparison to parental provision of cognitive stimulation, parental emotional supportiveness as a construct may require consideration of additional features (e.g., the presence or absence of parental harshness), thus warranting an index of parental emotional supportiveness features. An index variable for parental emotional supportiveness was not used in this study, but if it is was, this index may have had a greater likelihood of significantly predicting language performance and/or interacting with genetic predisposition to predict children's language performance.

A considerable weakness of this study is the inability to detect more statistically significant gene–environment interactions. To place the findings of this study in a broader context, this study examined thirty-six potential gene–environment interactions to investigate the potential moderating role of genetic predisposition; of these thirty-six gene–environment interaction terms, only three gene–environment interaction terms were detected as significant predictors. Thus the overall pattern across these models provides some, but not unequivocal, support for the theory of inextricable gene–environment

interaction as a contributor to children's language performance. It could be argued that gene–environment interaction may be taking place in all conditions evaluated and that gene–environment interaction was too hard to detect. Indeed, scholars have warned about the difficulty of detecting gene–environment interaction (Moffit et al., 2006), and other researchers examined and plotted interaction effects that occurred at a trend level of $p<.15$ (McGrath et al., 2007). Nonetheless, the ratio of significant to non-significant interactions indicates that this study could not detect significant gene–environment interaction as expected across each hypothesis. The significant and nonsignificant findings in this study indicate that continued research into gene–environment interaction as a contributor to children's language performance and development is warranted.

Last, it is important to note that the findings of this study are not to be generalized to the population as a whole. Even though the ECLS-B dataset was created for the generalization of findings, the twin subsample was trimmed down to 1150 to achieve equal n's for each predictor in the regression equations. Additionally, twins often demonstrate delays in language performance and are thus not appropriate indicators of the overall preschool language learning population.

Despite these weaknesses, this study has considerable strengths. This study achieved concordance between its complex conceptualization of human development and the employed analytic strategy, addressing O'Brien's (2005) suggestion for contemporary research. Second, this study extended research of gene–environment interaction into the field of language, addressing Moffitt et al.'s (2006) suggestion to examine gene–environment interaction beyond the field of psychopathology and into areas of well-being

and school achievement. Further, the conceptual model, that of using genetic predisposition as a moderator of environmental influence, and the strategy of measuring genetic predisposition by use of zygosity and co-twins' performance was recommended by the same group of scholars in the field of gene–environment interaction, further indicating consistency of conceptualization and employed analytic strategy (Moffitt, Caspi, and Rutter, 2006; Jaffee, Caspi, Moffitt, Dodge, Rutter, Taylor, and Tully, 2005). Thus there is also reason to have confidence in this study's genetic proxy and measurement of genetic predisposition for language performance, despite the lack of access to specific genetic information.

This gene–environment interaction study deviated from a focus on environmental risk, and, to the knowledge of the author, is the first study in the field of language research to examine genes interacting with both advantage and risk. The use of index variables of risk and advantage is also considered a strength, as one could argue that variables never quite exist in isolation in "real life." As noted by O'Brien (2005), disadvantage and advantage tends to co-occur (e.g., parents with lower education tend also to have lower income and fewer resources to provide cognitive stimulation for their children). Further, the focus of this study was not solely on poor outcomes or a diagnostic category; gene–environment interaction was used to predict the full range of language performance.

The use of the ECLS-B dataset was a major asset to this study, as it provided a large twin subsample and key distal and proximal environmental variables previously linked to children's language performance. The large sample size provided enough power

to detect gene–environment interaction, which is noteworthy given that previous scholars lamented the difficulty of detecting statistical interactions between any two factors in environmental science (McCall, 1991; McClelland & Judd, 1993), and, much more, scholars provided warning concerning the difficulty of detecting gene–environment interaction (Moffit, Caspi, and Rutter, 2006). The important finding that gene–environment interaction was detected for both proximal and index variables was enabled by the comprehensive measurement of children's learning environments in the ECLS-B.

Each evaluation across the various levels of complexity of gene–environment interaction detected a significant interaction, enabling an evaluation of gene–environment interaction in the context of singular environmental variables, indices of environmental risk and advantage, and risk and advantage indices with variation by sex. An important strength of this study is that of testing configurations of genetic predisposition in environments of risk and advantage, which yielded the notable finding that gene–environment interaction may not necessarily conform to either a diathesis-stress or bioecological model, but may in fact operate in more complex ways, such as that of differential susceptibility.

Recommendations for future research includes continuing researching gene–environment interaction as a contributor to children's language performance to build upon the results of this study; this will increase researchers' current yet limited understanding of this area of gene–environment interaction. Importantly, future research into gene–environment interaction in language should examine the interaction of specific genetic material with specified environmental factors as contributors to children's

language performance, in addition to the interaction of genetic material with sex hormones in environments of both risk and advantage. Future studies should examine genetic predisposition for language performance across the entire spectrum of language performance rather than simply the upper and lower quartiles, as examined in the current study. Gene–environment interaction should also be examined as a contributor to children's language development, which necessitates longitudinal study designs. And last, this study indicates that researchers should not be limited to a dual-risk/dual-advantage conceptualization of gene–environment interaction patterns, but test a configuration of gene–environment interaction, wherein genetic predisposition for positive and poor outcomes are both examined in the context of risk and advantage.

This study contributes to gene–environment and language literature as an initial investigation into the contribution of gene–environment interaction to language performance. Overall, this study demonstrates that gene–environment interaction can be detected as a significant predictor of children's language performance. Most notably, this study underscores the importance of researchers' continued and innovative investigations into gene–environment interaction as a contributor to children's language performance and development.

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Appendix A

Tables

Table 1. Descriptive Data of the Study Sample (N=1150)

	Mean	SD	Range
Zygosity[a]	1.85	.36	1-2
Household Income	8.69	3.45	1-13
Parental Education	5.35	2.00	1-9
Parental Provision of Cognitive Stimulation	4.35	0.95	1-7
Parental Emotional Supportiveness	4.58	0.92	2-7
Child Sex[b]	1.51	0.50	1-2
Child Assessment Age (Months)	52.67	0.58	44.50-63.70
Child Race[c]	0.66	0.47	0-1
Low Birthweight Status	0.58	0.49	0-1
GP for High Rec Voc	2.16	0.66	1-4
GP for Low Rec Voc	2.16	0.66	1-4
GP for High Exp Lng	2.00	0.55	1-4
GP for Low Exp Lng	2.13	0.66	1-4
Risk[d]	0.68	0.92	0-3
Advantage[e]	1.02	0.99	0-3
Receptive Vocabulary	8.54	1.84	0-15
Expressive Language	2.38	0.97	0-5

Notes. [a] 1=MZ (200); 2=DZ (950).
[b] 1 = male; 2 = female;
[c] 0 = Not White; 1 = White
[d] and [e] Indices of Risk and Advantage (derived from income, parental education, and parental provision of cognitive stimulation)

Table 2. Percentages of twins in each genetic predisposition for language category

Genetic Predisposition (GP)	Lowest	Low	High	Highest
GP for High Receptive Vocabulary	11.70	63.90	21.00	3.40
GP for Low Receptive Vocabulary	11.40	65.00	19.90	3.70
GP for High Expressive Language	13.80	73.50	11.40	1.30
GP for Low Expressive Language	11.40	67.10	17.80	3.60

Table 3. Correlations of Language Performance Between MZ and DZ Twins

	Monozygotic	Dizygotic
Receptive Vocabulary	.73**	.65**
Expressive Vocabulary	.58**	.41**

**p<.01

Table 4. Testing for Significant Mean Differences Between MZ and DZ Twins

Variable	Zygosity	Mean	SD	t
Household Income	MZ	8.15	3.62	
	DZ	8.79	3.41	-2.15*
Parental Education	MZ	5.14	2.13	
	DZ	5.39	1.97	-1.55
Parental Provision of Cognitive Stimulation	MZ	4.28	.94	
	DZ	4.36	.95	-.98
Parental Emotional Supportiveness	MZ	4.55	.92	
	DZ	4.59	.92	-.42
Child Age	MZ	52.82	4.24	
	DZ	52.65	3.99	.50
Receptive Language	MZ	8.43	1.89	
	DZ	8.56	1.83	-.84
Expressive Language	MZ	2.34	.91	
	DZ	2.39	.98	-.63

*p<.05

Table 5. Correlations Among Study Variables

	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.
1. Receptive Vocabulary	.48**	.03	.40**	-.45**	.22**	-.23**	.37**	.37**	.22**	.15**	.10**	.24**	-.08**	.36**	-.31**	.36**
2. Expressive Language	--	.02	.23**	-.27**	.20**	-.31**	.248**	.22**	.17**	.11**	.14**	.20**	-.10**	.17**	-.22**	.24**
3. Zygosity		--	.31**	.27**	.56**	.27**	.07*	.05	.03	.01	-.03	-.02	-.01	.03	-.05	.02
4. GP for High Rec Voc			--	-.20**	.37**	-.09**	.24**	.24**	.15**	.11**	.04	.13**	-.03	.20**	-.20**	.24**
5. GP for Low Rec Voc				--	.00	.41**	-.25**	-.23**	-.14**	-.08**	-.04	-.14**	.03	-.24**	.23**	-.22**
6. GP for High Exp Lng					--	.00	.16**	.16**	.07*	.02	.01	.06	-.05	.08**	-.14**	.14**
7. GP for Low Exp Lng						--	-.14**	-.11**	-.06*	-.02	-.05	-.15**	.07*	-.06*	.13**	-.13**
8. Household Income							--	.66**	.34**	.33**	.03	.04	-.034	.43**	-.80**	.65**
9. Parental Education								--	.36**	.32**	-.01	-.01	.00	.33**	-.73**	.73**
10. Parental Provision of Cognitive Stimulation									--	.58**	.00	-.05	-.02	.23**	-.56**	.62**
11. Parental Emotional Supportiveness										--	.05	-.07*	-.04	.19**	-.43**	.41**
12. Sex											--	.07*	.07*	-.02	-.00	-.01
13. Age (Months)												--	-.03	-.01	.02	.01
14. Birth Weight Status													--	-.10**	.03	-.02
15. Race (Not White/White)														--	-.38**	.35**
16. Risk															--	-.58**
17. Advantage																--

* $p < .05$. ** $p < .01$.

Table 6. Hierarchical Regression Analyses Using Singular Environmental Variables to Predict Receptive Vocabulary (GP for High Rec Voc)

	B	SE	ΔR^2	FΔ
Step 1: *df*= 3				
Age	0.11**	0.02		
Birth Weight	-0.12	0.12		
Race	1.40**	0.13		
Step 2: *df*= 5			0.09	18.87**
Income	0.05*	0.02		
Parental Education	0.20**	0.04		
Parental Provision of Cognitive Stimulation	0.17*	0.07		
Parental Emotional Supportiveness	-0.08	0.07		
Sex	0.35**	0.10		
Step 3: *df*= 1			0.06	56.61**
GP for High Rec Voc	0.72*	0.10		
Step 4: *df*= 5			0.00	0.17
Income x GP for High Rec Voc	0.00	0.037		
Education x GP for High Rec Voc	-0.00	0.063		
Cognitive Stimulation x GP for High Rec Voc	0.06	0.10		
Emotional Supportiveness x GP for High Rec Voc	0.01	0.10		
Sex x GP for High Rec Voc	0.02	0.17		

† $p < .10$. * $p < .05$. ** $p < .01$.

Table 7. Hierarchical Regression Analyses Using Singular Environmental Variables to Predict Receptive Vocabulary (GP for Low Rec Voc)

	B	SE	ΔR^2	FΔ
Step 1: *df*= 3				
Age	0.11**	0.02		
Birth Weight	-0.12	0.12		
Race	1.40**	0.13		
Step 2: *df*= 5			0.09	18.87**
Income	0.05*	0.02		
Parental Education	0.20**	0.04		
Parental Provision of Cognitive Stimulation	0.17*	0.06		
Parental Emotional Supportiveness	-0.08	0.07		
Sex	0.35**	0.10		
Step 3: *df*= 1			0.08	86.72**
GP for Low Rec Voc	-0.85**	0.09		
Step 4: *df*= 5			0.00	0.5
Income x GP for Low Rec Voc	-0.02	0.03		
Education x GP for Low Rec Voc	0.01	0.05		
Cognitive Stimulation x GP for Low Rec Voc	-0.00	0.10		
Emotional Supportiveness x GP for Low Rec Voc	-0.00	0.10		
Sex x GP for Low Rec Voc	-0.17	0.16		

† $p < .10$. * $p < .05$. ** $p < .01$.

Table 8. Hierarchical Regression Analyses Using Singular Environmental Variables to Predict Expressive Language (GP for High Exp Lng)

	B	SE	ΔR^2	FΔ
Step 1: *df*= 3				
Age	0.05**	0.01		
Birth Weight	-0.16*	0.06		
Race	0.33**	0.07		
Step 2: *df*= 5			0.07	14.18**
Income	0.03*	0.01		
Parental Education	0.05*	0.02		
Parental Provision of Cognitive Stimulation	0.12**	0.04		
Parental Emotional Supportiveness	-0.04	0.06		
Sex	0.26**	0.06		
Step 3: *df*= 1			0.02	18.20**
GP for High Exp Lng	0.26**	0.06		
Step 4: *df*= 5			0.01	0.97
Income x GP for High Exp Lng	0.03	0.03		
Education x GP for High Exp Lng	-0.05	0.04		
Cognitive Stimulation x GP for High Exp Lng	0.10	0.08		
Emotional Supportiveness x GP for High Exp Lng	-0.06	0.07		
Sex x GP for High Exp Lng	0.12	0.11		

† $p < .10$. * $p < .05$. ** $p < .01$.

Table 9. Hierarchical Regression Analyses Using Singular Environmental Variables to Predict Expressive Language (GP for Low Exp Lng)

	B	SE	ΔR^2	FΔ
Step 1: *df*= 3				
Age	0.05**	0.01		
Birth Weight	-0.16**	0.06		
Race	0.33**	0.07		
Step 2: *df*= 5			0.07	14.18**
Income	0.03*	0.01		
Parental Education	0.05*	0.02		
Parental Provision of Cognitive Stimulation	0.12**	0.04		
Parental Emotional Supportiveness	-0.04	0.04		
Sex	0.26**	0.06		
Step 3: *df*= 1			0.06	52.28**
GP for Low Exp Lng	-0.37*	0.05		
Step 4: *df*= 5			0.01	1.63
Income x GP for Low Exp Lng	0.01	0.02		
Education x GP for Low Exp Lng	-0.01	0.04		
Cognitive Stimulation x GP for Low Exp Lng	0.11*	0.06		
Emotional Supportiveness x GP for Low Exp Lng	0.04	0.06		
Sex x GP for Low Exp Lng	-0.01	0.10		

† $p < .10$. * $p < .05$. ** $p < .01$.

Table 10. Hierarchical Regression Analyses Using Risk Variable to Predict Receptive Language (GP for High Rec Voc)

	B	SE	ΔR^2	FΔ
Step 1: *df*= 3				
Age	0.11**	0.02		
Birth Weight	-0.12	0.12		
Race	1.40**	0.13		
Step 2: *df*= 2			0.045	22.90**
Risk	0.42**	0.07		
Sex	0.34**	0.11		
Step 3: *df*= 1			0.08	69.62**
GP for High Rec Voc	0.80**	0.10		
Step 4: *df*= 1			0.00	0.16
Risk x GP for High Rec Voc	0.05	0.11		
Step 5: *df*= 3			0.00	1.46
Risk x Sex	-0.22*	0.11		
Sex x GP for High Rec Voc	-0.10	0.18		
Risk x Sex x GP for High Rec Voc	-0.14	0.20		

† $p < .10$. * $p < .05$. ** $p < .01$.

Table 11. Hierarchical Regression Analyses Using Risk Variable to Predict Receptive Language (GP for Low Rec Voc)

	B	SE	ΔR^2	FΔ
Step 1: *df*= 3				
Age	0.11**	0.02		
Birth Weight	-0.12	0.12		
Race	1.40**	0.13		
Step 2: *df*= 2			0.045	22.90**
Risk	-0.41	0.07		
Sex	0.34**	0.11		
Step 3: *df*= 1			0.10	96.75**
GP for Low Rec Voc	-0.91**	0.10		
Step 4: *df*= 1			0.00	2.07
Risk x GP for Low Rec Voc	0.12	0.10		
Step 5: *df*= 3			0.00	1.19
Risk x Sex	-0.08	0.11		
Sex x GP for Low Rec Voc	-0.12	0.17		
Risk x Sex x GP for Low Rec Voc	-0.15	0.17		

† $p < .10$. * $p < .05$. ** $p < .01$.

Table 12. Hierarchical Regression Analyses Using Risk Variable to Predict Expressive Language (GP for High Exp Lng)

	B	SE	ΔR^2	FΔ
Step 1: *df*= 3				
Age	0.05**	0.01		
Birth Weight	-0.16**	0.06		
Race	0.33**	0.07		
Step 2: *df*= 2			0.049	26.25
Risk	-0.20**	0.40		
Sex	0.26**	0.06		
Step 3: *df*= 1			0.02	20.86
GP for High Exp Lng	0.28**	0.06		
Step 4: *df*= 1			0.00	0.02
Risk x GP for High Exp Lng	-0.01	0.07		
Step 5: *df*= 3			0.00	0.41
Risk x Sex	0.01	0.06		
Sex x GP for High Exp Lng	0.13	0.11		
Risk x Sex x GP for High Exp Lng	0.04	0.14		

† $p < .10$. * $p < .05$. ** $p < .01$.

Table 13. Hierarchical Regression Analyses Using Risk Variable to Predict Expressive Language (GP for Low Exp Lng)

	B	SE	ΔR^2	FΔ
Step 1: *df*= 3				
Age	0.05**	0.01		
Birth Weight	-0.16**	0.06		
Race	0.33**	0.07		
Step 2: *df*= 2			0.05	26.25**
Risk	-0.20**	0.04		
Sex	0.26**	0.06		
Step 3: *df*= 1			0.06	54.05**
GP for Low Exp Lng	-0.38**	0.05		
Step 4: *df*= 1			0.00	1.43
Risk x GP for Low Exp Lng	-0.06	0.05		
Step 5: *df*= 3			0.00	0.03
Risk x Sex	-0.01	0.05		
Sex x GP for Low Exp Lng	-0.01	0.10		
Risk x Sex x GP for Low Exp Lng	0.02	0.10		

† $p < .10$. * $p < .05$. ** $p < .01$.

Table 14. Hierarchical Regression Analyses Using Advantage Variable to Predict Receptive Language (GP for High Rec Voc)

	B	SE	ΔR^2	FΔ
Step 1: *df*= 3				
Age	0.11**	0.02		
Birth Weight	-0.12	0.12		
Race	1.40**	0.13		
Step 2: *df*= 2			0.07	38.17**
Advantage	0.49**	0.06		
Sex	0.35**	0.10		
Step 3: *df*= 1			0.07	63.39**
GP for High Rec Voc	0.75**	0.09		
Step 4: *df*= 1			0.00	0.04
Advantage x GP for High Rec Voc	-0.02	0.08		
Step 5: *df*= 3			0.00	2.64*
Advantage x Sex	0.15	0.10		
Sex x GP for High Rec Voc	-0.13	0.18		
Advantage x Sex x GP for High Rec Voc	0.28†	0.15		

† $p < .10$. * $p < .05$. ** $p < .01$.

Table 15. Hierarchical Regression Analyses Using Advantage Variable to Predict Receptive Language (GP for Low Rec Voc)

	B	SE	ΔR^2	FΔ
Step 1: *df*= 3				
Age	0.11**	0.02		
Birth Weight	-0.12	0.12		
Race	1.40**	0.13		
Step 2: *df*= 2			0.07	38.17**
Advantage	0.49**	0.06		
Sex	0.35**	0.10		
Step 3: *df*= 1			0.10	96.70**
GP for Low Rec Voc	-0.88**	0.09		
Step 4: *df*= 1			0.00	5.80*
Advantage x GP for Low Rec Voc	0.16	0.07		
Step 5: *df*= 3			0.00	0.88
Advantage x Sex	0.08	0.10		
Sex x GP for Low Rec Voc	-0.18	0.17		
Advantage x Sex x GP for Low Rec Voc	-0.02	0.07		

† $p < .10$. * $p < .05$. ** $p < .01$.

Table 16. Hierarchical Regression Analyses Using Advantage Variable to Predict Expressive Language (GP for High Exp Lng)

	B	SE	ΔR^2	FΔ
Step 1: *df*= 3				
Age	0.05**	0.01		
Birth Weight	-0.16*	0.06		
Race	0.33**	0.07		
Step 2: *df*= 2			0.06	32.49**
Advantage	0.20**	0.03		
Sex	0.27**	0.06		
Step 3: *df*= 1			0.02	20.60**
GP for High Exp Lng	0.27**	0.06		
Step 4: *df*= 1			0.00	0.91
Advantage x GP for High Exp Lng	0.05	0.05		
Step 5: *df*= 3			0.00	0.35
Advantage x Sex	0.01	0.06		
Sex x GP for High Exp Lng	0.11	0.11		
Advantage x Sex x GP for High Exp Lng	-0.05	0.10		

† $p < .10$. * $p < .05$. ** $p < .01$.

Table 17. Hierarchical Regression Analyses Using Advantage Variable to Predict Expressive Language (GP for Low Exp Lng)

	B	SE	ΔR^2	FΔ
Step 1: *df*= 3				
Age	0.05**	-0.01		
Birth Weight	-0.16*	-0.06		
Race	0.33**	-0.07		
Step 2: *df*= 2			0.06	32.49**
Advantage	0.20**	-0.03		
Sex	0.27**	-0.06		
Step 3: *df*= 1			0.06	54.55**
GP for Low Exp Lng	-0.38**	-0.05		
Step 4: *df*= 1			0.01	6.21*
Advantage x GP for Low Exp Lng	0.12*	-0.05		
Step 5: *df*= 3			0.00	0.10
Advantage x Sex	0.02	-0.06		
Sex x GP for Low Exp Lng	0.02	-0.10		
Advantage x Sex x GP for Low Exp Lng	0.04	-0.10		

† $p < .10$. * $p < .05$. ** $p < .01$.

Appendix B

Figures

Figure 1. Interaction of Genetic Predisposition for Low Expressive Language and Cognitive Stimulation

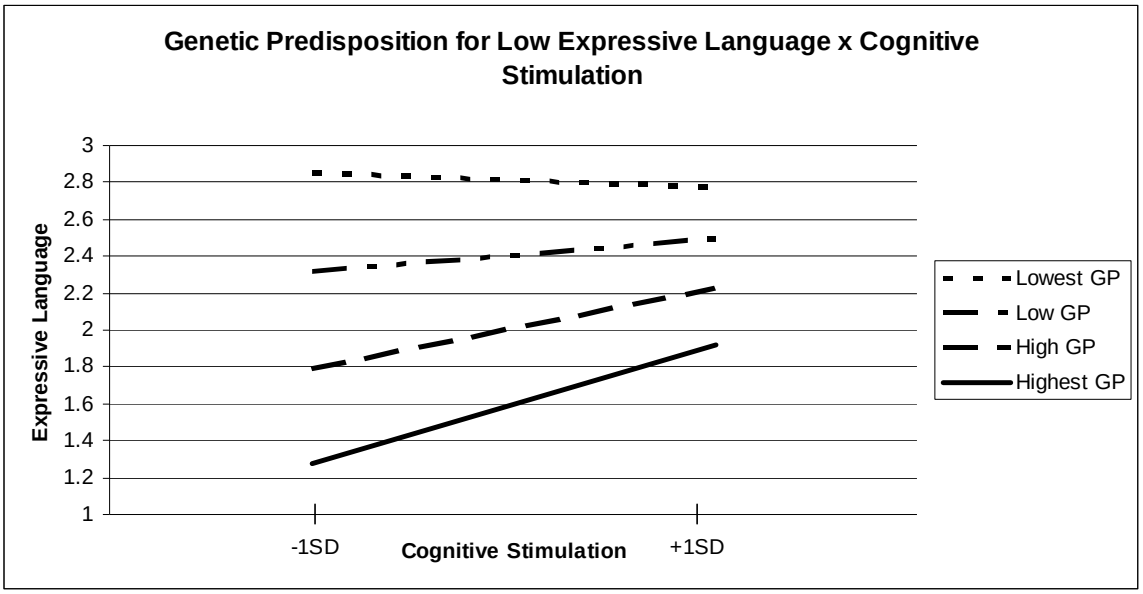

Figure 2. Interaction of Genetic Predisposition for Low Expressive Language and Advantage

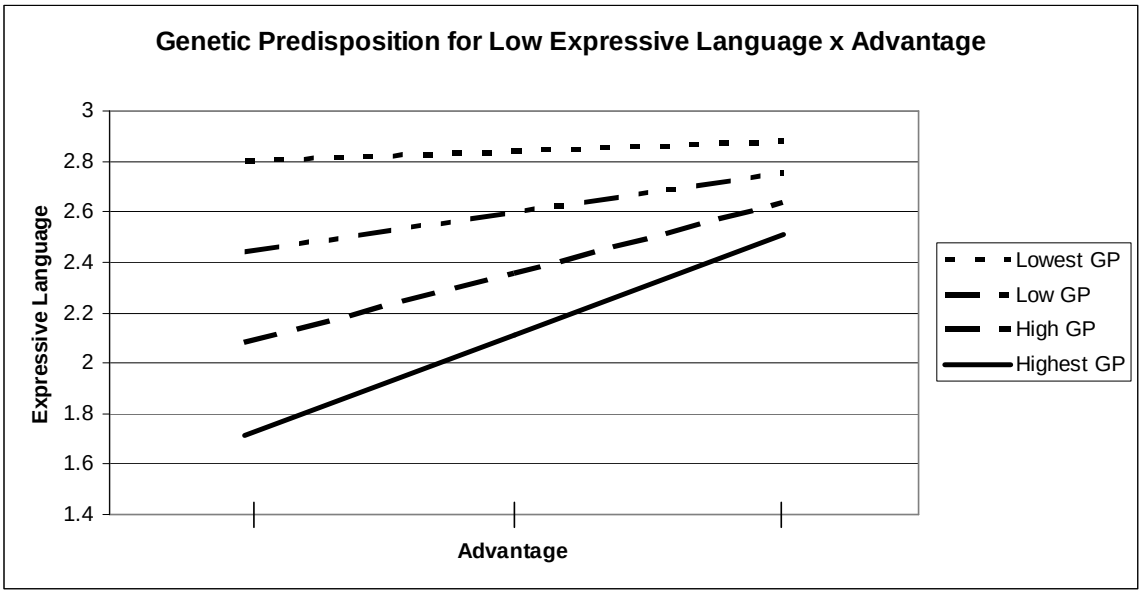

Figure 3. Interaction of Genetic Predisposition for High Receptive Vocabulary, Advantage, and Sex