Genetic and Environmental Influences on Early Social Competence:

Moderation by Parental Social Support

by

Sierra Clifford

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Graduate Supervisory Committee:

Kathryn Lemery-Chalfant, Chair
Leah Doane
Michelle Shiota
Kevin Grimm

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ABSTRACT

This study examined whether social support available to parents moderated the heritability of parent-reported social approach at 12 months (N = 286 twin pairs, 52.00% female) and social competence at 30 months (N = 259 twin pairs, 53.30% female). Genetic and environmental covariance across age is also reported. Social support consistently moderated genetic influences on children's social approach and competence, such that heritability was highest when parents reported low social support. Shared environment was not moderated by social support and explained continuity across age. Findings provide further evidence that genetic and environmental influences on development vary across context. When parents are supported, environmental influences on children's social competence are larger, perhaps because support helps parents provide a broadly promotive environment.

DEDICATION

To all my family and friends for all their kindness, generosity, and inspiration, and in particular, to my sister and brothers, for their strength and perseverance. There is no one who inspires me more than you.

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CHAPTER 1

INTRODUCTION

Overview

Social competence is a central and early-emerging developmental competency that promotes adaptive development in multiple domains, with social competence during the preschool years predicting positive peer relationships, lower internalizing and externalizing behavior problems, and academic adjustment across childhood (Bornstein, Hahn, & Haynes, 2010; Hay et al., 2004; Ladd & Price, 1987). Toddlerhood and early childhood are also a time when children are first beginning to acquire foundational aspects of social competence (e.g., basic social skills and ability to sustain positive interactions with others), and social competence during this time may be more malleable than at later ages, when children's social trajectories are more firmly established (Hay et al., 2004; Eckerman & Peterman, 2001; Santos, Vaughn, Peceguina, Daniel, & Shin, 2014). This is also a period when the development of social competence may be particularly open to intervention, and individual and environmental factors that promote or interfere with early social competence (e.g., temperament or parenting) can have important long-term implications (Rubin, Bukowski, & Parker, 2006). The goal of this study was first to use the twin design to examine the role of genetic and environmental factors in development of children's social competence during infancy and toddlerhood, including the extent to which genetic and environmental influences on positive social approach at 12 months are shared with broader social competence at 30 months. Secondly, we test whether primary caregivers' perceived social support acts as a

moderator of latent genetic and environmental influences on children's social approach in infancy and social competence during toddlerhood.

Although many children are beginning to make the transition to out-of-home childcare or preschool, primary caregivers still play a key role in shaping most children's environment during toddlerhood and early childhood (Beauchamp & Anderson, 2010), and factors such as social support that influence parental wellbeing may also have a broad impact on children's development. Social support available to parents is related to children's social competence through parental stress and parenting (Ensor & Hughes, 2010; Melson, Ladd, & Hsu, 1993; Ostberg & Hagekull, 2013), and may also be important for other aspects of children's social experiences such as exposure to social situations outside the home (Cochran & Niego, 2002). However, the importance of parental social support for children's social competence may go beyond simple main effect associations. Social competence has been found to show substantial heritability in toddlerhood and early childhood (e.g., Van Hulle, Lemery-Chalfant, & Goldsmith, 2007), indicating that factors other than the early environment must be taken into account, but it is also likely that genetic and environmental influences vary across different environmental contexts and experiences (Rutter, Moffitt, & Caspi, 2006). Indeed, one adoption study of older children found that sensitive parenting may interact with genetic predisposition to predict social competence (Van Ryzin, Leve, Neiderhiser, Shaw, Natsuaki, & Reiss, 2015). However, no study to date has considered moderation of genetic and environmental influences on social competence during infancy and

toddlerhood, a time of rapid development in social skills and cognition combined with a social environment relatively more defined by caregivers than at later ages.

Social Development in Toddlerhood

The first three years of life are a time of rapid maturation in children's social, cognitive, and regulatory abilities, all of which support the development of social competence with peers (Eckerman & Peterman, 2001). By the end of the first year of life, infants appear to recognize others as active agents (Tomasello & Haberl, 2003), and display clear evidence of social interest and positive social behaviors directed towards peers (e.g., social smiling, proximity, affectionate gestures; Eckerman & Peterman, 2001; Rubin et al., 2006). Although the ability to establish joint attention is present by the end of the first year, many infants of this age cannot consistently maintain joint engagement even in play with mothers (Aureli & Presaghi, 2010), and they lack the capacity for coordinated interaction with unfamiliar peers (Brownell, Ramani, & Zerwas, 2006). This is a capacity that emerges by approximately 20-24 months of age, and social interaction increases rapidly in skill, frequency, and complexity across the third year of life (Brownell et al., 2006; Eckerman & Peterman, 2001). By 32 months, children are capable of sustained coordinated action (e.g., games or working toward a shared goal), integrated verbal and nonverbal interaction, sociodramatic play, and collaborative problem solving (Eckerman & Peterman, 2001). Although toddlers lack the linguistic and cognitive ability for complex representations of friendship, peer preferences are relatively stable, salient, and common among toddlers who are exposed to peers outside the home (Eckerman & Peterman, 2001). Friendships become more complex and reciprocal with age, and are

thought to both contribute to and rely on the development of basic social skills and prosocial behaviors such as coordinated social interaction, turn-taking, sharing, and empathy (Vandell, Nenide, & Van Winkle, 2006). Positive social experiences and adaptation during infancy and toddlerhood may provide the background for social adaptation during the transition to preschool and kindergarten (Hay et al., 2004), and evidence for cascading pathways from preschool-age social competence to later childhood internalizing and externalizing problems (Bornstein et al., 2010) highlights the importance of early social competence for a range of childhood outcomes.

Social Competence

Social competence has been defined in many ways, but can be thought of as the ability to effectively achieve personal and interpersonal goals within social settings, both short-term and over developmental time (Rose-Krasnor, 1997). This broad definition encompasses several more concrete resources and behaviors enabling positive, regulated, and flexible social interaction, including emotional regulation and understanding, social skills, and adaptive social goals (Rose-Krasnor, 1997). We focus on two areas of social competence: prosocial and age-appropriate social behavior (e.g., turn-taking), and social interest and engagement, both of which promote positive peer relationships and further opportunities for children to learn a range of interpersonal skills (Hay et al., 2004). Individual differences in socially competent behavior including prosocial behavior and social motivation are evident as early as toddlerhood (Rubin et al., 2006), and socially competent play in early childhood predicts continuing social competence, lower withdrawal, and lower aggression at age nine (Hay et al., 2004). In addition, social

competence defined as a combination of social motivation, skills, and peer acceptance is highly stable within the same childcare setting across early childhood (Santos et al., 2014), and social competence as a composite of prosocial, compliant, and positive social behaviors is highly stable from 24 to 36 months (Barnett, Gustafsson, Deng, Mills-Koonce, & Cox, 2012). Further, social behaviors such as social smiling in the first year of life predict social competence at 30 months of age (Parlade, Messinger, Delgado, Kaiser, Vaughan Van Hecke, & Mundy, 2009). Such findings suggest that despite considerable change in social abilities from 12 to 30 months, early indicators of social interest and approach may be precursors of toddlers' more complex social competencies.

Individual differences in social competence have many possible sources. The early social and family environment is thought to play a role in young children's acquisition of social competence by promoting or disrupting the development of early childhood competencies such as self-regulation and emotional understanding, as well as by children's internalization of schemas and expectations for the self and others (Rubin et al., 2006). Early parental warmth and responsiveness are related to aspects of social competence including prosocial behavior and social skills (e.g., Barnett et al., 2012; Lengua, Honorado, & Bush, 2007), although not all studies find a main effect of parenting on social competence (e.g., Van Ryzin et al., 2015). Parents, caregivers, and preschool teachers also directly teach and model social and emotional behavior, and interactions with peers and siblings provide an opportunity to practice social and conflict resolution skills (Hay et al., 2004; Rubin et al., 2006). At a more distal level, parenting stress and parental depression are both associated with children's social competence

deficits, likely via disruptions in parenting, negativity and conflict in the home, and fewer opportunities for positive social experiences (Goodman & Gotlib, 1999; Ostberg & Hagekull, 2013; Wang & Dix, 2015).

Twin studies of social competence in toddlerhood and early childhood point to the importance of genetic as well as environmental factors, for both broad social competence and narrower facets such as prosocial behavior and social interest (Beaver, Boutwell, Barnes, & Schwartz, 2014; Knafo & Plomin, 2006a; Van Hulle et al., 2007). Two studies using data from the Early Childhood Longitudinal Study—Birth Cohort find strong genetic influences on peer interaction at age 4 (74%) and a broad measure of social competence in kindergarten (77%), with little evidence for shared environmental factors (Beaver et al., 2014; Roisman & Fraley, 2012), although when supportive parenting at 24 months was included in bivariate models, genetic and shared environmental factors each explained approximately half (47%) of the modest correlation between parenting and broad social competence (Roisman & Fraley, 2012). In addition, the shared environment has been found to explain the majority of the correlation between prosocial behavior and positive parenting, whereas genetic factors primarily account for the correlation with parental negativity (Knafo & Plomin, 2006b). Another study finds that mother and father report of social interest in toddlerhood show high heritability, with consistency across reporter explained by genetic factors, as well as modest reporter-specific shared environmental influences (Van Hulle et al., 2007). Finally, there is some evidence for genetic influences on both continuity and change in young children's positive social behavior. For both prosocial empathy and sociability from 14 to 20 months, continuity is

primarily explained by genetic factors, with novel genetic influences on sociability at 20 months (Plomin et al., 1993). Continuity in prosocial behavior across two, three, and four years is explained by both genetic and shared environmental factors, although shared environmental influences become modest by age four and negligible in middle childhood whereas heritability remains substantial (Knafo & Plomin, 2006a).

However, genetic and environmental influences do not act in isolation, and whether and how genetically-influenced traits are expressed may depend on the environmental context. An enriched environment may protect against genetic risk, or a genetic predisposition may be triggered by exposure to an environmental risk (Rutter et al., 2006; Shanahan & Hofer, 2005). More generally, the heritability of a trait tends to be higher when the environment does not restrict the expression of that trait, allowing genetically-influenced predispositions to drive behavior (e.g., Lemery-Chalfant, Kao, Swann, & Goldsmith, 2013; Shanahan & Hoffer, 2005). For example, a recent meta-analysis shows that in US studies (but not studies from Western Europe or Australia, where access to social programs is higher), genetic influences on IQ become significantly stronger as socioeconomic status increases (Tucker-Drob & Bates, 2016). In contrast, heritability may be lower in environments that channel emotion and behavior toward a more narrow range, whether positive or negative. For example, negative emotionality is less heritable in an environment characterized by higher physical safety and structure (Lemery-Chalfant et al., 2013), potentially reflecting an environment less likely to enable or evoke genetically-influenced negativity. Thus, while social competence is heritable (e.g., Roisman & Fraley, 2012; Van Hulle et al., 2007), the extent to which children's

social outcomes are attributable to genetic or environmental factors may itself differ according to the environment (e.g., Van Ryzin et al., 2015). We use the twin design to examine moderation of latent genetic influences on social competence at 12 and 30 months of age, focusing on parental social support as a factor that may have broad influences on the development of children's social behavior.

Parental Social Support and Social Development

Social support is among the most important protective and promotive factors identified in the resilience literature, associated with physical and mental health, emotional wellbeing, and resilience to stress across the lifespan (Taylor, 2011). Social support is multifaceted, and may include instrumental (e.g., childcare or financial assistance), informational, or emotional support, from both informal (e.g., relatives, friends) and formal (e.g., doctor, social worker) sources (Taylor, 2011). It has been assessed in multiple ways, including perceived access to and quality of support, participants' report of actual support received, or characteristics such as size and integration of participants' support networks (Haber, Cohen, Lucas, & Baltes, 2007; Taylor, 2011). Perceived and received support are significantly but seldom highly correlated, and perceived support is often more strongly and consistently associated with mental health outcomes than received support (Haber et al., 2007), although one study finds size of parental support networks, but not satisfaction with support, to be associated with children's social competence (Melson et al., 1993).

In addition to positive outcomes at the individual level, social support available to parents has broad implications for the family climate and children's environmental

context in ways that are relevant to social development and the acquisition of social competence. For instance, parents' access to and satisfaction with social support are associated with lower parenting-related and general stress and reduced risk of depression, anxiety, and general psychological distress (Cutrona & Troutman, 1986; McConnell, Breitkreuz, & Savage, 2011; Ostberg & Hagekull, 2013), with one study finding lack of social support to be among the strongest predictors of high-escalating trajectories of depression in mothers across twenty-three years (Kingsbury, Hayatbakhsh, Mamum, Clavarino, Williams, & Naiman, 2015). Parental social support is also consistently associated with differences in parenting, including higher parental warmth, responsiveness, and efficacy, and lower punitive or intrusive parenting (Burchinal, Follmer, & Bryant, 1996; Ensor & Hughes, 2010; McConnell et al., 2011; Melson et al., 1993), and has been found to attenuate the link between parents' psychological distress and their negative parenting (Heberle, Krill, Briggs-Gowan, & Carter, 2015). In addition, parental access to social support has also been theorized to promote children's social competence by providing greater opportunity for children to observe and participate in social interactions outside the family (Cochran & Niego, 2002), although empirical evidence is limited.

Consequently, it is unsurprising that researchers have found parental social support to be associated with positive social and emotional outcomes for children, including multiple aspects of social competence (e.g., peer acceptance, assertiveness and cooperation; prosocial behavior; Ensor & Hughes, 2010; Melson et al., 1993; Oravecz, Koblinski, & Randolph, 2008; Ostberg & Hagekull, 2013). Findings are sometimes

mixed or nonsignificant (e.g., Melson et al., 1993), and much of this research has been conducted in school or preschool-age rather than infant children, but parental social support is related to children's social competence across multiple sources and measures of perceived and objective support (e.g., Ostberg & Hagekull, 2013; Serrano-Villar, Huang, & Calzada, in press).

However, the potential importance of parental social support may not be limited to relations with mean levels of children's social outcomes. Together, the range of promotive factors associated with social support (e.g., positive parenting and mental health, material and emotional resources, social integration in the community (Balaji, Claussen, Smith, Visser, Johnson Morales, & Perou, 2007; Cochran & Niego, 2002) may contribute to an environment that buffers the impact of genetic risk for low social competence (i.e., *social context as compensation*; Shanahan & Hofer, 2005). In contrast, not only is an environment characterized by low support less likely to compensate for genetic risk, but when children are high in social competence, it is more likely to be for genetic reasons. One adoption study in middle childhood suggests that genetic influences on social competence may play a stronger role in an adverse environment, with high birth mother sociability having a protective effect on children's broad social competence when adoptive parents were low in responsiveness, whereas children who experienced highly responsive adoptive parenting showed similar levels of social competence regardless of birth mother sociability (Van Ryzin et al., 2015). However, no study to date has examined gene-environment interplay in social competence at younger ages, or using a twin design.

The Current Study

The primary goal of the current study was to examine whether and how genetic and environmental influences on social competence in toddlerhood differ as a function of social support available to parents. Heritability indexes the proportion of total variance attributable to genetic influences within a particular population at a particular time, and may differ according to characteristics of the population (e.g., age or level of exposure to trait-relevant environmental factors). We used Purcell's (2002) method of examining moderation of heritability, which allows latent genetic and environmental variances to differ across levels of a measured environmental variable, such that, for example, social competence is more genetically-influenced among toddlers whose parents report lower social support. We also examined parental social support as a moderator of the heritability of social approach in infancy, as a potential precursor of toddler social competence, and considered the extent to which infant social approach is related to toddler social competence for genetic and environmental reasons. We expected that:

1) Social approach in infancy and social competence in toddlerhood would be moderately heritable and longitudinally related for both genetic and shared environmental reasons.

2) In both infancy and toddlerhood, higher perceived parental social support would be associated not only with higher mean levels of social competence, but with lower total variance and lower variance attributable to genetic factors.

CHAPTER 2

METHOD

Participants

Participants were drawn from the Arizona Twin Project, a longitudinal study of twin children born in the state of Arizona. Primary caregivers (95.70% mothers) were recruited from hospital birth records and took part in assessments when twins were approximately 12 months of age (*M* = 12.63, *SD* = 1.36 months) and 30 months of age (*M* = 31.88, *SD* = 2.83 months). At 12 months, participants were 572 twins (286 pairs; 52.00% female, 27.80% MZ, 37.70% same sex DZ, 34.40% opposite sex DZ) with completed zygosity assessments (14 twins with missing zygosity data were not included in the current study). Of these twins, 480 (240 pairs) also had data at 30 months, and 38 twins (19 pairs) were assessed at 30 months but not 12 months, for a full 30 month sample of 518 twins (259 pairs; 53.30% female, 25.50% MZ, 37.50% same sex DZ, 37.10% opposite sex DZ). At twelve months, twins were primarily Non-Hispanic Caucasian (59.60%) and Hispanic (25.80%), with the remaining twins falling into the categories of Asian or Asian American (6.60%), African American (4.60%), or other (3.30%). Annual household income ranged from under $20,000 to over $100,000 at both assessments (Median = $60,000-$80,000), and the majority of mothers reported either a completed college degree (36.20%; 30.00% of fathers), two or more years of graduate school (2.70%; 1.10% of fathers), or a completed graduate or professional degree (15.10%; 18.80% of fathers), with the remaining mothers reporting either some college (28.50%; 27.10% of fathers), a high school degree or equivalent (14.80%; 19.50% of

fathers), or less than a high school degree (2.70%; 3.60% of fathers). At 30 months, 40.02% of mothers had a completed college degree (30.02% of fathers), 2.50% reported two or more years of graduate school (1.30% of fathers), 17.60% reported a completed graduate or professional degree (20.00% of fathers), 27.20% reported some college (25.80% of fathers), 10.90% reported a high school degree or equivalent (19.60% of fathers), and 1.70% reported less than a high school degree (3.10% of fathers).

Procedure.

Primary caregivers completed two hour-long phone or online interviews assessing twins' temperament, competencies, problem behaviors, and environment at 12 and 30 months. Twin zygosity and demographic information including income, education, and race/ethnicity were collected at 12 months.

Measures.

Social Competence at 12 and 30 months. At both 12 and 30 months, twins' social competence was measured using the Infant and Toddler Social and Emotional Assessment (ITSEA; Carter, Briggs-Gowan, Jones, & Little, 2003), a measure designed for children between the ages of 1-3 years which assesses early behavioral problems and competencies on a scale of 0-2, where 0 = Not True/Rarely and 2 = Very True/Often). At 12 months, social competence was measured using the 5-item Social Relatedness scale (Cronbach's alpha = .58), which taps social affiliation (e.g., "Is Twin A interested in other babies and children?"). At 30 months, both Social Relatedness (Cronbach's alpha = .61) and the 5-item Prosocial Peer Relations scale (e.g., "Does Twin A take turns when playing with others?"; Cronbach's alpha = .65) were used. Scale-level reliability was low, possibly

due to the low number of items, but consistent with past research (Carter et al., 2003). The two scales were moderately correlated (r = .40, p < .001) at 30 months, and a mean composite was formed (Cronbach's alpha = .71). Unfortunately, Prosocial Peer Relations was not available at 12 months due to the age-inappropriateness of some items, and so a more reliable composite could not be formed.

Social Support. Social support at both 12 and 30 months was measured using the Family Support Scale (FSS; Dunst, Jenkins, & Trivette, 1984) and the Medical Outcomes Study (MOS) Social Support scale (Sherbourne & Stewart, 1991). The FSS is an 18-item scale (Cronbach's alpha = .75 at 12 months; .68 at 30 months) assessing primary caregivers' perceived support from formal (e.g., doctor, childcare provider) and informal (e.g., parents, friends) sources on a scale from 1 (Not at all) to 3 (Extremely helpful). MOS Social Support consists of 5 items assessing the availability of tangible and emotional support from various sources (e.g., "Someone who shows you love and affection") on a scale of 1 (Never) to 5 (Always), along with a single item assessing the number of friends and relatives living nearby (ranging from 0 to 12+). For MOS Social Support, the 6 items were standardized using Z-score transformations to put them on a common metric prior to forming a mean composite (Cronbach's alpha = .87 at 12 months; .83 at 30 months). In addition, I computed mean composites of the FSS across 12 and 30 months (r = .66, p < .001), and the MOS across 12 and 30 months (r = .62, p < .001) in order to examine more stable measures of social support as moderators.

Zygosity. Zygosity was measured using the Zygosity Questionnaire for Young Twins (Goldsmith, 1991), a 32-item parent-report questionnaire that uses physical

similarities and differences between twins (e.g., hair color and texture) and other information (e.g., chorionicity) to estimate zygosity. The Zygosity Questionnaire for Young Twins has been found to show over 93% agreement with zygosity assessed by genotyping (Price, Freeman, Craig, Petrill, Ebersole, & Plomin, 2000).

Overview of Quantitative Genetic Analyses.

Quantitative genetic analyses parse the total phenotypic variance in one or more traits into latent additive genetic influences (A), shared environmental influences (C), and nonshared environmental influences (E), based on differences in the phenotypic resemblance of individuals with different degrees of genetic relatedness, such as monozygotic (MZ) and dizygotic (DZ) twin pairs. Because MZ twins share 100% of their genetic influences, whereas DZ twins share an average of 50% of their segregating genes, a stronger phenotypic covariance between MZ twins than between DZ twins suggests the presence of genetic influences on a trait, and the proportion of variance within a population attributable to genetic influences can estimated from the magnitude of that difference. However, although A is labeled additive genetic influences, this component encompasses all factors that increase the resemblance of MZ twins relative to DZ twins, including unaccounted for dominance and gene-by-gene interaction, as well as the effects of gene-environment interaction with unmeasured family-level moderators, and as such is better thought of as a broad-sense heritability. By the same token, the C component encompasses all factors that contribute equally to MZ and DZ twin covariance, and E encompasses all non-genetic factors that increase differences between twins, including measurement error.

The classic biometric ACE model is a multi-group structural equation model that estimates A, C, and E components from observed cross-twin covariances in MZ and DZ groups, and models the genetic correlation between twins differently for MZ and DZ groups. Specifically, the A component is fixed to correlate 1.0 between MZ twins and 0.50 between DZ twins, the C component is fixed to correlate 1.0 between twins for both MZ and DZ groups, and the E component is uncorrelated between twins by definition. After the full ACE model is fit, the significance of A and C paths is tested by dropping them from the model and comparing the fit of the reduced nested model to the full model, although E is always retained in the model because it includes measurement error. The significance of reduced models is tested using the -2 log likelihood chi-square test of fit with degrees of freedom equal to the number of dropped parameters.

The bivariate cholesky decomposition is an extension of the univariate ACE model that parses phenotypic covariance between two phenotypes into A, C, and E components using differences in the cross-twin, cross-trait covariance between MZ and DZ groups. Similar to a univariate ACE model, the bivariate Cholesky estimates a single set of A, C, and E influences on the first phenotype, which may be shared with or independent of the second, as well as a set of A, C, and E influences unique to the second phenotype (Neale & Cardon, 1992). Because the A, C, and E influences on the first phenotype are not parsed into shared and unique variance, the order of phenotypes matters for interpretation, but not for model fit or the significance of shared paths. In the current study, the bivariate Cholesky was used to estimate the extent to which A, C, and E influences on 12 month social approach were shared with 30 month social competence.

Finally, the classic ACE model estimates heritability as a population-level statistic that averages across heterogeneity in genetic and environmental influences within that population, but the heritability of a trait may differ within a population according to differences in the genetic or environmental context. Purcell's (2002) model introduces a measured environmental moderator to the univariate ACE twin model which is allowed to affect the strength of the paths from the latent A, C, and E components, allowing for an estimation of how latent G (or E) components differ across values of the moderator. In the current study, family-level moderators (social and family support) were used. Purcell's (2002) model is similar to a phenotypic regression, but instead of predicting mean levels of a trait, the effect of the moderator *M* on A, C, and E variance is estimated. Specifically, the additive genetic variance A is a modeled as $A = (a + \beta x M)^2$, where a is the genetic path estimate, M is the level of the observed moderator, and βx is a coefficient representing the effect of the moderator on A. Similarly, $C = (c + \beta y M)^2$ and $E = (e + \beta z M)^2$. If moderation is not present for A (or C, or E), βx is 0, and genetic variance is calculated as $A = a^2$, as in the univariate ACE model. Like the univariate ACE model, the significance of moderation on each path can be tested using the -2 log likelihood chi-square test of fit by fixing the β coefficient to 0.

The moderator may also be included in the means model (i.e., allowed to have a main effect on the phenotype). In this case, the moderator acts as a covariate in a typical linear regression, and the ACE model becomes a decomposition of the residual variance after the effects of the moderator on the phenotype are regressed out. Including the moderator in the means model accounts for the presence of gene-environment correlation

(i.e., all genetic effects that are shared between the moderator and the phenotype are controlled for), and prevents the detection of ACE moderation in the presence of gene-environment correlation when the genetic influences are shared between moderator and phenotype (Purcell, 2002).

In fitting moderated models, the first step was to compare the fit of a model where the moderator was included in the means model to a reduced model in which the effect of the moderator on the mean is fixed to 0. The best-fitting model from this step was then used as the full model against reduced models were compared. The second step was to test the significance of βx, βy, and βz in turn, and then to test the significance of any unmoderated a or c paths. The significance of the a or c paths was not tested if βx or βy, respectively, could not be dropped from the model, because significant moderation implies that A or C influences may be significant at one value of the moderator but not another.

CHAPTER 3

RESULTS

Preliminary analyses.

Descriptive Statistics and Twin Intraclass Correlations. Means and standard deviations, zero-order correlations, and twin ICCs are reported in Table 1. No variables exceeded recommended cutoffs for skewness or kurtosis (+/- 2.00 and 7.00, respectively; Muthén & Kaplan, 1985). Social approach at 12 months and social competence at 30 months were moderately related to each other and modestly but significantly related to family and social support within age. Across age, earlier social support was related to later social competence, but early social approach was unrelated to later social support. MZ twin correlations were high at both 12 (*ICC* = .87) and 30 (*ICC* = .92) months. DZ correlations were lower than MZ correlations at both ages, but still higher than half the value of MZ correlations (*ICC* from .62-.72), suggesting the presence of both additive genetic and shared environmental variance.

Saturated Models. Prior to quantitative genetic analyses, we fit saturated models for 12 month social approach and 30 month social competence to examine whether means, variances, and cross-twin covariances could be equated across sex, and whether means and variances could be equated across zygosity group. If the assumptions of the twin design are met, means and variances should not differ between MZ and DZ twin groups (cross-twin covariance is allowed to differ across zygosity, as a higher covariance between MZ twins relative to DZ twins is expected when genetic effects are present). Beginning with a five group model freely estimating means, variances, and covariances

for male MZ, female MZ, male DZ, female DZ, and opposite sex DZ twins, we then fit a series of models constraining parameters to be equal across group.

For social approach at 12 months, it was possible to equate means and variances across sex and zygosity group, and cross-twin covariances across sex ($\Delta\chi^2(21) = 14.11$, $p = .86$). For social competence at 30 months, it was possible to equate means and variances across zygosity group, and variances and covariances across sex ($\Delta\chi2(19) = 29.73$, $p = .06$). However, means could not be equated across sex ($\Delta\chi2(21) = 42.94$, $p = < .001$), with females showing higher social competence. Because ACE models analyze variances and covariances rather than means, a significant sex difference in social competence will not affect parameter estimates. Nevertheless, the effects of age, sex, and ethnicity (non-Hispanic white =1; all other racial and ethnic categories = 0) were regressed out of social approach and social competence prior to model fitting, as is standard practice when it is infeasible to incorporate additional covariates into quantitative genetic models (McGue & Bouchard, 1984).

Univariate and Bivariate Heritability of Social Approach and Competence.

The full ACE model provided the best fit for both social approach at 12 months and social competence at 30 months (Table 2). At 12 months, 37% of the variance in social approach was explained by additive genetic factors, and 48% by shared environmental factors, with the remainder explained by nonshared environmental influences (15%). Similarly, social competence at 30 months was primarily explained by moderate additive genetic (45%) and shared environmental (44%) factors, as well as modest nonshared environmental variance (11%).

To examine genetic and environmental relations between 12 month social approach and 30 month social competence, we fit a bivariate cholesky decomposition. Fit statistics and parameter estimates for the full and most reduced models are reported in Table 2. As in the univariate models, 12 month social approach and 30 month social competence were both moderately heritable (.36-.44), but none of this genetic variance was shared across age. Instead, the covariance was fully accounted for by shared environmental influences, with approximately half of the shared environmental influences on social competence at 30 months shared with earlier social approach.

Moderation of Genetic and Environmental Influences.

We tested two measures of social support available to caregivers (the FSS and the MOS) as concurrent moderators of genetic and environmental influences on children's social approach at 12 months and social competence at 30 months. We began with a model estimating moderation of the a, c, and e paths and including the moderator in the means model, and first tested whether the moderator could be dropped from the means model (see Table 3 for fit statistics for 12 month models, and Table 4 for 30 months). Using the best-fitting model from this step as the full model, we then tested whether we could drop the effect of the moderator on the a, c, and e paths in turn to arrive at the final best fitting model. We did not test the significance of A or C variance components because univariate analyses found the full ACE model to provide the best fit at both ages.

Moderation of Social Approach at 12 Months. Unstandardized a, c, and e path estimates and β coefficients indicating the effect of the moderator on each path are reported in Table 3 (12 months) and Table 4 (30 months) for the full and final best-fitting

models, and fit statistics are reported for all models tested. Figure 1 shows moderation of standardized and unstandardized variance according to the full model. For the model examining the FSS as moderator, it was possible to drop the effect of the moderator from the means model and the c and e paths, but not the a path, such that genetic variance was lowest when caregivers reported high family support (see Table 4 for standardized and unstandardized genetic and environmental variance components at mean levels of family support and one standard deviation above and below the mean). In the model examining moderation by the MOS, the moderator could not be dropped from the means model without significant loss of fit. The MOS significantly moderated the e path, but not the a or c paths, such that nonshared environmental variance was lowest when caregivers reported high social support (see Table 4 for standardized and unstandardized variance components at the mean and one standard deviation above and below).

Moderation of Social Competence at 30 Months. Fit statistics and parameter estimates are reported in Table 3 (12 months) and Table 4 (30 months), variance components are reported in Table 6 at the mean and one standard deviation above and below the mean, and Figure 1 shows moderation of standardized and unstandardized variance according to the full model. For both the FSS and the MOS, it was necessary to retain the effect of the moderator in the means model. Similar to findings at 12 months, the FSS moderated the a path, such that genetic variance in social competence was lowest at high levels of support. In addition, the FSS weakly but significantly moderated the e path, such that nonshared environmental variance was highest when caregivers reported high levels of support, although the effect of the moderator could be dropped from the c

path without loss of fit. For the model examining the MOS as moderator, only the a path was significantly moderated, again with genetic variance lowest at high levels of social support.

Moderation of Social Competence at 30 Months by Longitudinal Social Support. Fit statistics and parameter estimates for longitudinal moderation models are reported in Table 5, and variance components are reported in Table 6 at the mean and one standard deviation above and below the mean. For the FSS, the variance-only model did not converge, but the model including the moderator in the means model could be successfully estimated, and was used as the full model against which reduced models were compared. For the MOS, the model retaining the moderator in the means model fit significantly better, and thus was retained as the full model. For both the FSS and the MOS, moderation of the c and e paths could be dropped, but it was necessary to retain moderation of the a path, such that genetic variance was highest at low levels of social support.

Examination of Moderation Under Full and Reduced Models. Although Purcell's (2002) model performs well in correctly detecting moderation, and full models accurately recapture the correct parameters, reduced models have limited power to differentiate between moderation of genetic, shared environmental, and nonshared environmental paths. Purcell (2002) recommends using the plot of variance components under the full model as a guide for final model selection. We provide plots of unstandardized and standardized variance components under the full model in Figure 1 for concurrent 12 and 30 month models, and Figure 2 for longitudinal models. Examination of full models in

comparison to final models indicated that the pattern of variance components across level of social support typically changed little, suggesting that our final models accurately captured the pattern of moderation. One exception is the model for moderation by MOS support at 12 months, where the full model suggests moderation of A, C, and E components and the reduced model, dropping A and C moderation, results in a notably different standardized solution. Lack of A and C moderation by MOS social support at 12 months should be interpreted cautiously. In addition, full models examining moderation by the longitudinal FSS and MOS composites across 12 and 30 months both suggested the presence of moderation of the c path, such that C variance was highest at high levels of social support, although only moderation of the a path was significant.

CHAPTER 4

DISCUSSION

Summary and Interpretation of Findings

The goal of this study was to examine latent genetic and environmental influences on social approach and competence in infancy and toddlerhood, and whether these influences are moderated by primary caregivers' perceived social support. Consistent with past twin research in this age range (e.g., Van Hulle et al., 2007), social approach at 12 months and social competence at 30 months were influenced by both genetic and shared environmental factors. However, heritability depended on primary caregivers' perceived social support, such that both total variance and variance attributable to genetic factors were highest when social support was low. This pattern was largely but not entirely consistent across age and measurement of social support, although moderation of genetic influences was not significant for MOS social support at 12 months. In contrast, shared environmental influences were consistently substantial and unmoderated by social support, and these influences fully explained continuity between 12 month social approach and 30 month social competence. Past research shows mean-level associations between parents' access to social support and children's social competence, via multiple possible pathways (e.g., Ensor & Hughes, 2010; Ostberg & Hagekull, 2013; Serrano-Villar et al., in press). We are the first to find that parental social support also moderates the broad genetic and environmental etiology of infant and toddler social competence, perhaps because parental social support is associated with a range of contextual factors important for the development of social competence (Cochran & Niego, 2002).

The pattern of moderation of heritability found in this study has multiple possible interpretations. First, because social support is related to positive parenting and lower parental stress and depression (Burchinal et al., 1996; Kingsbury et al., 2015; McConnell et al., 2011), the expression of genetic risk for poor social competence may be constrained in environments characterized by high parental social support. Social support is an important source of material, emotional, and informational resources, and parents with access to these resources may be more able to provide an environment conducive to the development of social competence for all children, regardless of genetic predisposition (Cochran & Niego, 2002; Taylor, 2011). In addition, some heritable traits associated with low social competence, such as negative reactivity (e.g., Sallquist et al., 2009), may make children both more difficult and stressful to parent, and more vulnerable to lack of sensitive parenting (Dix & Yan, 2014). Parents with high access to social support may be more focused on and effective in providing an environment tailored to the needs of these children, better enabling them to achieve the same high levels of social competence as other children. Indeed, social support is associated with higher parental sensitivity in mothers of irritable infants (Crockenberg & McCluskey, 1986), and lower rates of depression in mothers of infants with difficult temperament (Cutrona & Troutman, 1986). In addition, to the extent that parental social support does increase children's likelihood of being exposed to a range of people outside the family and social situations outside the home (Cochran & Niego, 2002), such early positive experiences may be most important for children who are not genetically predisposed to social approach.

At the same time, there are likely important genetic as well as environmental influences on a number of basic competencies and traits important for the development of social competence, including ability to perceive and respond to emotions and social cues, face recognition, sociable temperament, and executive functions such as attentional regulation (Iarocci, Yager, & Elfers, 2007). When the environment is not conducive to the development of social competence, genetic influences on such traits may be the primary reason why some children nevertheless show higher levels of social competence.

A second possible interpretation is gene-environment interaction (GxE) in a molecular genetic sense. GxE between genetic variants and family-level factors acts to decrease the similarity of individuals (such as DZ twins) who share the same environment but different genes, and consequently registers as additive genetic influence in the classic ACE twin model. Thus, our finding of higher genetic influences at lower levels of social support would be consistent with a diathesis-stress pattern in which genetic risk only manifests in a more adverse environment (Shanahan & Hofer, 2005). Molecular genetic approaches to GxE have limitations such as a high false-positive rate in published candidate gene research and a multitude of practical challenges, including small effect sizes of individual genetic variants, genetically heterogeneous phenotypes, and the complex genetic and environmental etiology of psychological traits (Clifford & Lemery-Chalfant, 2015; Rutter et al., 2006). However, there is strong evidence that GxE does occur and has the potential to explain individual differences in adaptation to stress (Rutter et al., 2006). Adequately-powered research that attempts to address these challenges by, for example, examining more proximal biological endophenotypes of

complex psychological traits, or examining theoretically-relevant functional gene systems rather than individual genes in isolation, may still provide valuable insights (Clifford & Lemery-Chalfant, 2015). For social competence and approach, systems of interest may include the neurotransmission of oxytocin and arginine vasopressin, implicated in social bonding and behavior, as well as dopaminergic approach and reward response circuits which may play a role in extraversion (Clifford & Lemery-Chalfant, 2015; Depue & Morrone-Strupinski, 2005). With attention to replication and greater knowledge of genetic functionality, these systems may be promising targets for methodologically-sound research on gene-environment interplay in the development of young children's social competence.

One potential proximal mechanism linking parental social support to differences in the etiology of children's social competence is exposure to social situations outside the family. Cochran and Niego (2002) argue that parents' integration into social networks and communities carries over into children's social integration, and this may be especially true for infants and toddlers, who are dependent on social management by parents and caregivers in a way that older children are not. It must be noted that enrollment in childcare is a major avenue through which many young children first interact with peers outside the home. In general, toddlers enrolled in high-quality group-based childcare show earlier development of more complex peer interactions, better social skills, and more positive peer relations, as well as relatively stable early friendships (Campbell, Lamb, & Hwang, 2000; Eckerman & Peterman, 2001; Huston et al., 2015; Ladd et al., 2012; NICHD, 2001). Further, positive peer experiences in childcare settings predict

declining reticence in children selected for high negative reactivity during infancy (Almas et al., 2011). However, the relations between childcare and children's social outcomes are complex, not always positive, and often mixed, and these relations depend on multiple independent and interacting factors, including average and cumulative time spent in care, type of child care (e.g., home-based versus center), size of the peer group, and children's own temperament and relationships with teachers and peers (Campbell et al., 2000; Huston et al., 2015). In contrast, while parental social support is both distal and indirect, it may also be a more straightforward protective factor.

In addition, a majority of twins in this sample (155 pairs; 67.7%) did not attend either preschool or out-of-home childcare at 30 months, below the average reported in the large-scale NICHD Study of Early Care and Youth Development (approximately 52% enrolled in group-based care by age 24 months; NICHD, 2008), and a majority of those who attended either preschool or childcare in this sample attended for 15 hours per week or under (53 pairs; 23.6%). Low rates of child care use in this sample may be because for parents of twins, one parent taking time off from work to care for the children can be more cost-effective than formal childcare arrangements. At the same time, one commonly reported motivation for parents who use childcare is to provide social experiences for their young children (Ladd, Profilet, & Hart, 1992), and parents who do not use childcare for pragmatic reasons may still be motivated to provide their children with peer socialization opportunities by other means such as informal playgroup or playdate arrangements.

Indeed, although childcare is the first source of peer socialization for many infants and toddlers, it is not the only source. Parents and caregivers play an important role as managers of young children's social experiences, in part by directly arranging community activities where children may meet peers (e.g., visits to the library or park), as well as informal playdates and playgroups (Ladd & Pettit, 2002). The limited empirical research on parental facilitation of young children's peer experiences suggests that it is common for parents to initiate or facilitate playdates for their preschool-aged children, although there is considerable variability between families in the frequency of such peer contacts and the proportion of visits initiated by the parent, as opposed to the child or a peer (Bhavnagri & Parke, 1991; Ladd & Golter, 1988; Ladd & Hart, 1992).

In addition, some evidence suggests that such parent-mediated peer socialization is related to children's social outcomes (Ladd & Pettit, 2002). Specifically, higher frequency of parent-initiated social contacts is associated with size and stability of children's preschool social networks, higher prosocial behavior, and lower non-social play (Ladd & Golter, 1988; Ladd & Hart, 1992), parent-report of preschoolers' experience with playgroups and playdates is related to greater social competence during a laboratory peer interaction (Lieberman, 1977), and preschoolers' greater exposure to peers in informal community settings is associated with lower anxiety in kindergarten (Ladd & Price, 1987), although these findings must be interpreted in light of possible gene-environment correlation between parents' and children's social predispositions. There is little evidence to suggest that child care use or family structure is associated with systematic differences in the frequency of parent-initiated playdates and playgroups

(Ladd & Hart, 1992), although few studies have examined this question. In this sample in particular, use of childcare as an indicator of young children's social experiences outside the home may not take into account the many other ways that parents and caregivers act as social gatekeepers and curators for their children.

Our finding of substantial and consistent shared environmental influences on children's social competence across age and level of perceived parental social support is also interesting. These shared environmental influences may be due to reporter bias or measurement effects, especially as we relied on primary caregiver report and used the same social approach items at 12 and 30 months. However, they may also reflect genuine environmental factors that influence children's social competence independent of both parental perceptions of social support and genetic influences. It is a common pattern in twin studies that shared environmental influences are most evident early in life, when children are most dependent on parents and the immediate family environment, and attenuate with age (e.g., Knafo & Plomin, 2006a), perhaps due to children's increasing ability to shape and select their own environments. However, early social competence is still important for peer relationships during the transition into preschool or kindergarten and later elementary school (Hay et al., 2004; Ladd & Price, 1987), and small individual differences may have long-lasting and cumulative effects, with early social competence and peer experiences potentially setting children on positive or risky social trajectories across later childhood (Bornstein et al., 2010, Hay et al., 2004). Consequently, understanding shared environmental influences on infants' and toddlers' social approach and competence is important, even if these influences are limited to early development.

The latent shared environmental influences estimated in the classic univariate twin design are uninformative about which aspects of the shared environment may influence social competence. However, past multivariate twin studies report that positive and negative parenting practices in infancy and early childhood are largely accounted for by the shared environment rather than children's genetic factors (Cheung, Harden, & Tucker-Drob, 2016), and that observed supportive parenting at 24 months (cognitive stimulation, warmth, sensitivity, low detachment; measured separately for each twin) shares environmental variance with children's social competence in kindergarten (Roisman & Fraley, 2012). This suggests that relations between social competence and positive parenting reported in phenotypic research (e.g., Burchinal et al., 1996) are at least partially environmentally rather than genetically mediated. Other possible environmental influences on toddlers' social competence include socioeconomic status and its more proximal correlates such as preschool classroom quality or neighborhood safety, as well as opportunities for social interaction outside the home (Beauchamp & Anderson, 2010; NICHD Early Child Care Research Network, 2002; Oravecz et al., 2008).

Finally, our findings highlight the importance of parents' well-being and environmental experiences for children's development. It is common for research and intervention aimed at improving children's outcomes to focus on parenting behavior and skills training, and measuring, understanding, and working to alter such proximal mechanisms is necessary and effective. However, good parenting requires not only knowledge, but the emotional and instrumental resources to effectively implement that knowledge, and social support is one avenue for providing those resources (Balaji et al.,

2007; Cochran & Niego, 2002). Increasing parents' access to social support may affect the environment they create for children in a range of ways, including lower stress and mental illness, more sensitive and less harsh and controlling parenting, and more time and energy for parenting, especially when parents report high stress or children are highly negative or difficult to parent. All of these factors are likely to be especially import when children are in infancy and toddlerhood, when their own emotional experience, regulation, and ability to acquire foundational social skills is highly dependent on parents (Beauchamp & Anderson, 2010). Further, our findings suggest that providing adequate social support to parents may be especially important when children are not genetically predisposed to social competence.

Limitations and Future Directions.

This study has a few limitations and we suggest future directions to address them. First, it would be valuable to directly measure possible mediators between parental social support and children's competence, such as interactions with peers and exposure to social situations, both as mean-level predictors and as moderators of the heritability of social outcomes. Our study was limited by a reliance on parent-report for children's social approach and competence, and the use of a relatively brief measure of social competence that taps some aspects of social competence in infancy and toddlerhood (e.g., prosocial peer behavior, social interest and approach), but not others (e.g., ability to read and respond to social cues). Multiple dimensions of social competence are likely important, and future genetically-informed research should examine a broader range of indicators of social competence using multiple informants (e.g., teachers, peers, observers).

In addition, genetic influences on social competence also likely play a role in parents' social support, and modeling both moderation and rGE in a larger twin sample may be informative. Purcell's (2002) method of estimating moderation of heritability does not allow estimation of gene-environment correlation (rGE) when using a family-level moderator. Including the moderator in the means model removes genetic confounding, ensuring findings are not due to passive rGE, but will fail to find moderation of genetic factors that are correlated with the moderator (Purcell, 2002). Price and Jaffee's (2008) method allows estimation of both passive rGE and moderated heritability, but our power was insufficient to support this method. Finally, despite their strengths, all twin studies are subject to assumptions, including the assumption that findings in twin samples can be generalized to singletons and the assumption that trait relevant environments experienced by MZ twins are not more similar than those experienced by DZ twins (the Equal Environments Assumption). These assumptions may not be supported for social development. Some studies find differences in social behavior and experience between MZ and DZ twins in childhood (Thorpe & Gardner, 2006). It is unknown whether differences between MZ and DZ twins also exist during the toddler and preschool years, but it is possible that greater similarity of social experiences among MZ twins results in an overestimation of genetic variance in social competence. Future studies should continue to test these assumptions.

Conclusion

This study adds to a growing body of literature (e.g., Lemery-Chalfant, 2013; Tucker-Drob & Bates, 2016; Van Ryzin et al., 2015) showing that the relative importance

and manifestation of additive genetic, shared environmental, and nonshared environmental influences on a trait may vary across individuals within a sample, for reasons including environmental differences. One implication of this research as a whole is that estimates of heritability from community samples may not be applicable to high-risk samples, and even within community samples high heritability should not be used as evidence for the unimportance of the environment. Changing the environments that children are exposed to, either at the level of proximal factors such as parenting or broader social or family factors such as socioeconomic status or parental access to social support, has the potential to influence the development of even highly heritable traits. At the same time, our findings should not be used to dismiss the importance of heritable factors for young children's social competence, which were substantial at mean levels of parental social support in our study and have been documented in past twin research across infancy and later childhood (Beaver et al., 2014; Knafo & Plomin, 2006a; Roisman & Fraley, 2012; Van Hulle et al., 2007). Understanding the biological underpinnings of social competence and behavior may be important for promoting positive social development in children who are genetically or temperamentally vulnerable to poor social outcomes. It will be important for research to focus on the ways in which genetic and environmental factors act in concert to influence children's social competence, and to understand at a more proximal level how the expression and implications of heritable traits may differ across environmental contexts.

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Table 1

Descriptive statistics, zero-order correlations, and twin intraclass correlations

	1	2	3	4	5	6	7	8	9	10	MZ	SS DZ	OS DZ
1. Social Approach (12)	1.00	.34**	.12**	.20**	-.02	.05	.03	.12*	.04	-.01	.87	.62	.72
2. Social Competence (30)		1.00	.24**	.16**	.23**	.15**	.23**	.23**	.13**	-.01	.92	.64	.69
3. Family Support (12)			1.00	.48**	.66**	.38**	.92**	.46**	.04	-.06			
4. MOS Social Support (12)				1.00	.36**	.62**	.90**	.46**	.03	-.12**			
5. Family Support (30)					1.00	.45**	.90**	.45**	.02	-.10*			
6. MOS Social Support (30)						1.00	.90**	.47**	.02	-.03			
7. Family Support (12 & 30)							1.00	.51**	.04	-.07			
8. MOS Social Support (12 & 30)								1.00	.04	-.05			
9. Sex									1.00	.01			
10. Ethnicity										1.00			
N	572	504	538	568	500	510	430	456	572	572			
M	1.65	1.55	.73	.004	.69	-.01	.70	.01	.52	.40			
SD	.33	.31	.33	.77	.29	.74	.29	.67	.50	.49			
Min	.40	.20	.11	-2.88	.17	-2.53	.19	-2.71	0	0			
Max	2.00	2.00	1.92	.84	1.58	.88	1.60	.86	1	1			
Skewness	-.84	-.99	.83	-1.24	.64	-1.17	.71	-1.27	-.08	.42			
Kurtosis	.21	1.46	.89	1.33	.18	.818	.19	1.71	-2.00	-1.83			

Note. 12 = assessed at 12 months; 30 = assessed at 30 months; 12 & 30 = mean composite of the scale assessed at 12 and 30 months. Sex is coded male = 0, female = 1. Ethnicity is coded 0 = non-Hispanic white, 1 = all other ethnicities. Twin intraclass correlations are reported on the right. MZ = monozygotic; SS DZ = same sex dizygotic, OS DZ = opposite sex dizygotic twin pairs.
*$p < .05$; **$p < .01$.

Table 2

Univariate ACE and bivariate Cholesky model fit and parameter estimates

Univariate ACE Models										
Scale	Model	-2LL	df	Δ -2LL	Δ df	*p*	AIC	A	C	E
12 Month Social Approach	**ACE**	**110.59**	**564**	–	–	–	**-1017.41**	**.04/.37 (.23-.54)**	**.05/.48 (.31-.69)**	**.02/.15 (.11-.20)**
	AE	134.82	565	24.23	1	< .001	-995.18			
	CE	127.61	565	17.02	1	< .001	-1002.39			
	E	338.5	566	227.91	2	< .001	-793.5			
30 Month Social Competence	**ACE**	**15.58**	**497**	–	–	–	**-978.42**	**.04/.45 (.31-.63)**	**.04/.44 (.26-.65)**	**.01/.11 (.07-.15)**
	AE	34.15	498	18.57	1	< .001	-961.85			
	CE	41.58	498	26.00	1	< .001	-954.42			
	E	230.40	499	214.81	2	< .001	-767.6			

Bivariate Cholesky Decomposition: 12 Month Social Approach and 30 Month Social Competence													
Model	-2LL	df	Δ -2LL	Δ df	*p*	AIC	Age	A1	C1	E1	A2	C2	E2
Full	95.42	1058	–	–	–	-2020.58	12 Months	.04/.36 (.22-.54)	.05/.49 (.32-.69)	.02/.15 (.11-.20)			
							30 Months	.00/.00 (-.01-.01)	.02/.18 (.05-.39)	.00/.00 (.00-.01)	.04/.45 (.31-.61)	.02/.26 (.09-.52)	.01/.11 (.07-.15)
Final	**96.47**	**1060**	**1.05**	**2**	**.59**	**-2023.53**	12 Months	**.04/.36 (.22-.54)**	**.05/.49 (.32-.69)**	**.01/.15 (.11-.20)**			
							30 Months		**.02/.19 (.08-.35)**		**.04/.44 (.31-.61)**	**.02/.26 (.10-.48)**	**.01/.11 (.07-.15)**

Note. -2LL = -2 log likelihood; df = degrees of freedom; Δ = change; *p* = probability; AIC = Akaike's Information Criterion; A, C, and E represent additive genetic, common environment, and nonshared environment factors, respectively. Unstandardized squared parameter estim ates are reported first, followed by standardized squared parameter estimates. Standardized confidence intervals are reported in parentheses. The most parsimonious final model is indicated in bold.

Table 3

12 month Social Approach moderated by the Family Support Scale

Model	-2LL	df	Δ -2LL	Δ df	*p*	AIC	a path	c path	e path	βx	βy	βz
Mean & Variance	82.64	502				-921.36						
Variance	85.38	503	2.73	1	.10	-920.62	.28 (.18-.38)	.23 (.12-.33)	.14 (.11-.18)	-.12 (-.24- -.01)	-.00 (-.10-.10)	-.02 (-.06-.01)
No A moderation	89.31	504	3.93	1	.05	-918.69						
No C moderation	85.38	504	.00	1	.94	-922.62						
No E moderation	86.7	504	1.33	1	.25	-921.30						
No C or E moderation	**86.7**	**505**	**1.33**	**2**	**.51**	**-923.30**	**.29 (.22-.37)**	**.22 (.18-.27)**	**.13 (.11-.15)**	**-.15 (-.25- -.05)**		
No moderation	96.79	506	11.41	3	.01	-915.21						
12 month Social Approach moderated by the MOS Social Support Scale												
Mean & Variance	71.28	530				-988.72	.21 (.17-.25)	.19 (.13-.25)	.12 (.10-.14)	-.04 (-.09-.01)	.08 (-.03-.19)	-.04 (-.06- -.02)
Variance	80.48	531	9.21	1	< .001	-981.52						
No A moderation	72.86	530	1.58	1	.21	-988.14						
No C moderation	73.92	531	2.64	1	.10	-988.08						
No E moderation	83.52	531	12.25	1	< .001	-978.48						
No A or C moderation	**73.94**	**532**	**2.66**	**2**	**.26**	**-990.06**	**.19 (.16-.23)**	**.22 (.17-.26)**	**.12 (.10-.14)**			**-.05 (-.07- -.03)**
No moderation	95.44	533	24.16	3	< .001	-970.56						

Note. -2LL = -2 log likelihood; df = degrees of freedom; Δ = change; *p* = probability; AIC = Akaike's Information Criterion; a, c, and e are unstandardized path estimates, and βx, βy, and βz represent the effect of the moderator on the A, C, and E variance, respectively. Unstandardized variance is calculated with the formula $A = (a + \beta xM)^2$, where M is the level of the moderator. Confidence intervals for unstandardized parameter estimates are reported in parentheses. The first step of model fitting was to test the effect of the moderator on the mean; t he best-fitting model was used as the full model against which reduced models were compared. The most final parsimonious final model is indicated in bo ld. Unstandardized and standardized variance is reported in Table 4 for values of the moderator at the mean and one standard deviation above and below.

Table 4

Fit statistics and parameter estimates for parental social support as a moderator of toddler social approach and toddler competence

Model	-2LL	df	Δ -2LL	Δ df	*p*	AIC	a path	c path	e path	βx	βy	βz
30 month Social Competence moderated by the Family Support Scale												
Mean & Variance	1.50	476				-950.50	.32 (.23-.41)	.19 (.07-.31)	.05 (.02-.08)	-.16 (-.28- -.03)	.00 (-.14-.14)	.06 (.01-.11)
Variance	9.45	477	7.95	1	< .001	-944.55						
No A moderation	7.74	477	6.24	1	.01	-946.26						
No C moderation	**1.50**	**477**	**.00**	**1**	**.97**	**-952.50**	**.32 (.25-.39)**	**.19 (.15-.24)**	**.05 (.02-.08)**	**-.16 (-.26- -.05)**		**.06 (.01-.11)**
No E moderation	6.53	477	5.03	1	.02	-947.47						
No moderation	13.09	479	11.59	3	.01	-944.91						
30 month Social Competence moderated by the MOS Social Support Scale												
Mean & Variance	-11.44	486				-983.44	.20 (.17-.24)	.18 (.13-.23)	.09 (.08-.11)	-.08 (-.14- -.03)	.03 (-.07-.13)	.02 (.00-.04)
Variance	1.56	487	13.00	1	< .001	-972.44						
No A moderation	-3.08	487	8.36	1	< .001	-977.08						
No C moderation	-10.99	487	.45	1	.50	-984.99						
No E moderation	-9.05	487	2.4	1	.12	-983.05						
No C or E moderation	**-8.85**	**488**	**2.59**	**2**	**.27**	**-984.85**	**.20 (.17-.24)**	**.19 (.15-.23)**	**.10 (.08-.11)**	**-.06 (-.10- -.02)**		
No moderation	1.43	489	12.87	3	< .001	-976.57						

Note. -2LL = -2 log likelihood; df = degrees of freedom; Δ = change; *p* = probability; AIC = Akaike's Information Criterion; a, c, and e are unstandardized path estimates, and βx, βy, and βz represent the effect of the moderator on the A, C, and E variance, respectively. Unstandardized variance is calculated with the formula $A = (a + \beta xM)^2$, where M is the level of the moderator. Confidence intervals for unstandardized parameter estimates are reported in parentheses. The first step of model fitting was to test the effect of the moderator on the mean; the best-fitting model was used as the full model against which reduced models were compared. The most final parsimonious final model is indicated in bold. Unstandardized and standardized variance is reported in Table 4 for values of the moderator at the mean and one standard deviation above and below.

Table 5

Fit statistics and parameter estimates for longitudinal parental social support as a moderator of toddler social approach and competence

Model	-2LL	df	Δ -2LL	Δ df	*p*	AIC	a path	c path	e path	βx	βy	βz
30 month Social Competence moderated by the Family Support Scale (Composite across 12 and 30 months)												
Mean & Variance	-1.40	408	–	–	–	-817.40	.38 (.29-.48)	.09 (-.09-.27)	.06 (.02-.10)	-.23 (-.34- -.11)	.09 (-.09-.28)	.05 (-.00 -.10)
No A moderation	7.47	409	8.87	1	< .001	-810.53						
No C moderation	-.02	409	1.39	1	.24	-818.02						
No E moderation	1.32	409	2.72	1	.10	-816.68						
No C or E moderation	**2.68**	**410**	**4.09**	**2**	**.13**	**-817.32**	**.33 (.26-.40)**	**.18 (.13-.22)**	**.10 (.08-.11)**	**-.16 (-.26- -.06)**	–	–
No moderation	13.58	411	14.98	3	< .001	-808.42						
30 month Social Competence moderated by the MOS Social Support Scale (Composite across 12 and 30 months)												
Mean & Variance	-8.28	434	–	–	–	-876.28	.22 (.18-.25)	.17 (.11-.23)	.09 (.08-.11)	-.08 (-.15- -.02)	.03 (-.08-.14)	.02 (-.00-.04)
Variance	1.37	435	9.64	1	< .001	-868.63						
No A moderation	-1.63	435	6.65	1	.01	-871.63						
No C moderation	-7.93	435	.34	1	.56	-877.93						
No E moderation	-6.09	435	2.19	1	.14	-876.09						
No C or E moderation	**-5.89**	**436**	**2.39**	**2**	**.30**	**-877.89**	**.21 (.18-.25)**	**.18 (.13-.22)**	**.10 (.08-.11)**	**-.06 (-.11- -.02)**	–	–
No moderation	2.63	437	10.90	3	.01	-871.37						

Note. -2LL = -2 log likelihood; df = degrees of freedom; Δ = change; *p* = probability; AIC = Akaike's Information Criterion; a, c, and e are unstandardized path estimates, and βx, βy, and βz represent the effect of the moderator on the A, C, and E variance, respectively. Unstandardized variance is calculated with the formula $A = (a + \beta xM)^2$, where M is the level of the moderator. Confidence intervals for unstandardized parameter estimates are reported in parentheses. The first step of model fitting was to test the effect of the moderator on the mean; the best-fitting model was used as the full model against which reduced models were compared. The most final parsimonious final model is indicated in bold. The Variance-only model was not estimated for the Family Support Scale 12 and 30 month composite, because the model failed to converge. The Mean & Variance model is used as the full comparison model. Unstandardized and standardized variance is reported in Table 4 for values of the moderator at the mean and one standard deviation above and below.

Table 6

Moderated A, C, and E components at the mean and one standard deviation above and below the mean of social support

12 month Social Approach									
	-1 Standard Deviation			Mean			+1 Standard Deviation		
Moderator	A	C	E	A	C	E	A	C	E
Family Support Scale	.05/.45	.05/.41	.02/.14	.03./33	.05/.50	.02/.17	.02/.21	.05/.59	.02/.20
MOS Social Support	.04/.33	.05/.44	.03/.23	.04/.36	.05/.49	.02/.15	.04/.40	.05/.53	.01/.07
30 month Social Competence (Concurrent)									
Family Support Scale	.07/.61	.04/.34	.01/.05	.04/.49	.04/.41	.01/.10	.03/.36	.04/.48	.01/.16
MOS Social Support	.06/.57	.04/.34	.01/.09	.04/.46	.04/.42	.01/.12	.02/.35	.04/.51	.01/.14
30 Month Social Competence (Longitudinal)									
Family Support Scale	.07/.62	.03/.29	.01/.09	.05/.53	.03/.36	.01/.11	.03/.41	.03/.45	.01/.14
MOS Social Support	.06/.60	.03/.31	.01/.10	.04/.51	.03/.38	.01/.12	.03/.40	.03/.46	.01/.14

Note. A, C, and E represent additive genetic, common environment, and nonshared environment, respectively, under the best-fitting moderated model. A, C, and E values are first reported as unstandardized moderated variance components, followed by standardized variance components. Unstandardized variance is calculated with the formula $A = (a + \beta xM)^2$, where a is the additive genetic path estimate, βx is the effect of the moderator on the additive genetic path, and M is the level of the moderator. Values for a, c, and e paths and βx, βy, and βz coefficients are reported in Table 3, and the means and standard deviations are reported in Table 1.

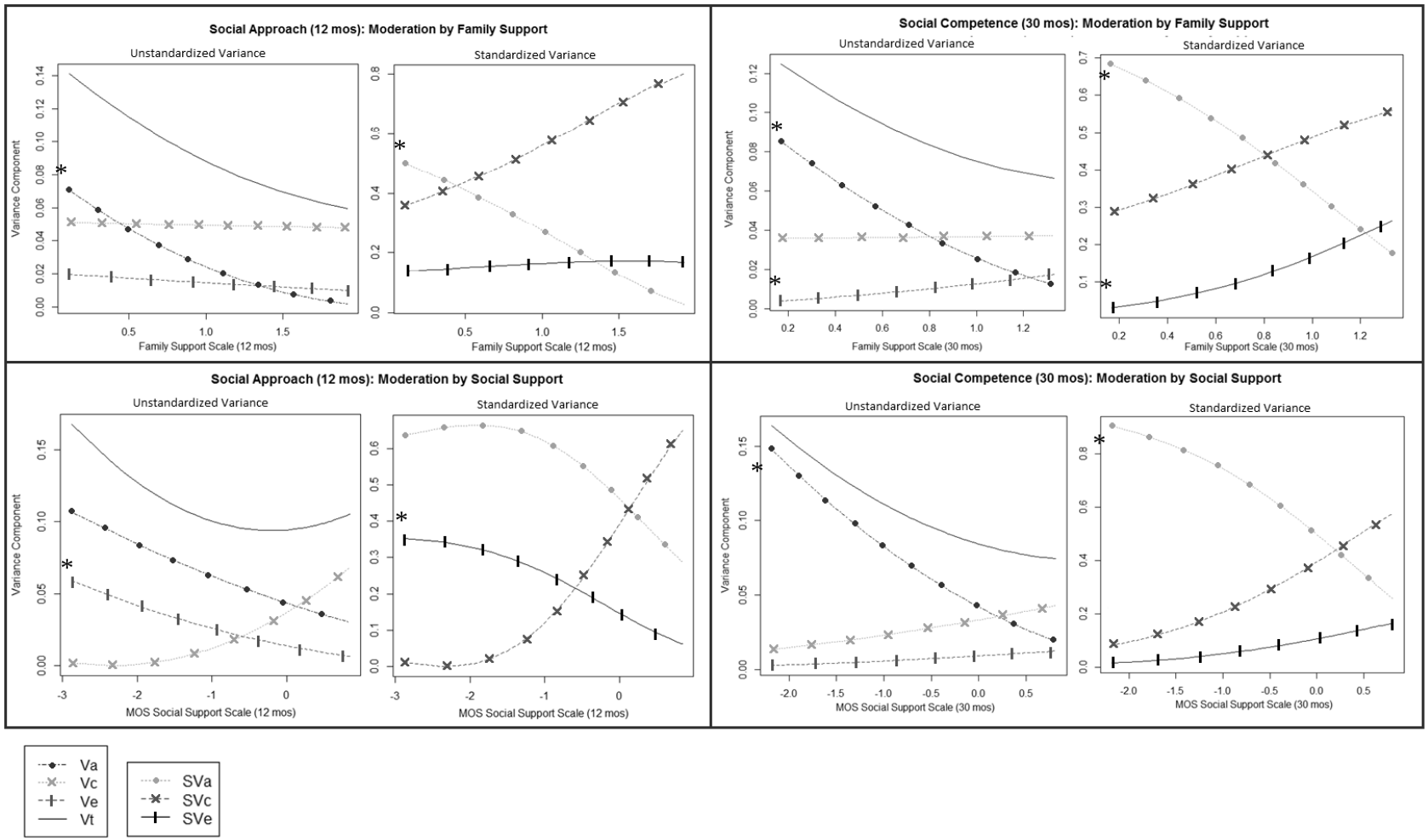

Figure 1. Moderation of unstandardized (left) and standardized (right) variance components according to the full model. Va, Vc, Ve, and Vt represent unstandardized additive genetic, shared environmental, nonshared environmental, and total variance, respectively, and SVa, SVc, and SVe represent standardized additive genetic, shared environmental, and nonshared environmental variance. Paths that are significant in the final model are marked with an asterisk.

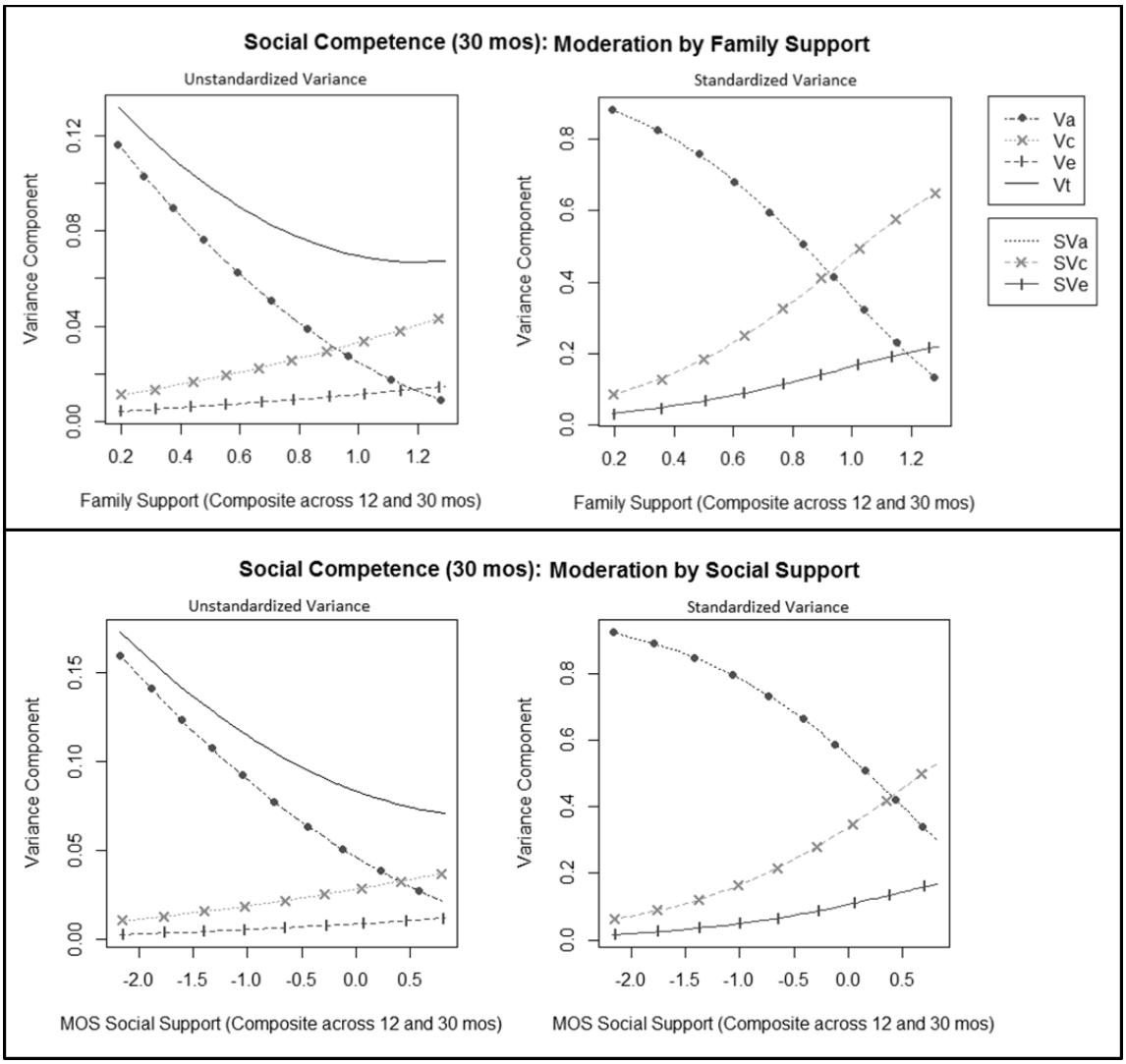

Figure 2. Moderation of unstandardized (left) and standardized (right) variance components according to the full model. Va, Vc, Ve, and Vt represent unstandardized additive genetic, shared environmental, nonshared environmental, and total variance, respectively, and SVa, SVc, and SVe represent standardized additive genetic, shared environmental, and nonshared environmental variance. Paths that are significant in the final model are marked with an asterisk.

APPENDIX A

MEASURES

Infant and Toddler Social and Emotional Assessment (ITSEA): Selected Scales (Carter, Briggs-Gowan, Jones, & Little, 2003)

Social Approach (12 and 30 months):

		Not true/ Rarely 0	Somewhat true/ sometimes 1	Very true/ often 2
1. Twin A likes to be cuddled, hugged, kissed by loved ones.	**Twin A**	O	O	O
	Twin B	O	O	O
2. Twin A is affectionate with loved ones.	**Twin A**	O	O	O
	Twin B	O	O	O
3. Twin A smiles back at you from across room.	**Twin A**	O	O	O
	Twin B	O	O	O
4. Twin A hugs people with a squeeze or a pat.	**Twin A**	O	O	O
	Twin B	O	O	O
5. Twin A is interested in other babies and children.	**Twin A**	O	O	O
	Twin B	O	O	O

Prosocial Peer Relations (30 months only):

		Not true/ Rarely 0	Somewhat true/ sometimes 1	Very true/ often 2
1. Does Twin A ask for things nicely when playing with children?	**Twin A**	O	O	O
	Twin B	O	O	O
2. Does Twin A play well with other children?	**Twin A**	O	O	O
	Twin B	O	O	O
3. Does Twin A take turns when playing with others?	**Twin A**	O	O	O
	Twin B	O	O	O
4. Does Twin A have at least one favorite child friend other than his/her cotwin (a child)?	**Twin A**	O	O	O
	Twin B	O	O	O
5. Does Twin A play 'house' with other children?	**Twin A**	O	O	O
	Twin B	O	O	O

Family Support Scale
(Dunst, Jenkins, & Trivette, 1984)

Next, I will list people and groups that oftentimes are helpful to members of a family raising young children. Please indicate how helpful each source has been to your family during the past 3 to 6 months. There are three response choices: not at all helpful, somewhat helpful, or extremely helpful.

	Not at all helpful	Somewhat helpful	Extremely helpful
1. Parents	**1**	**2**	**3**
2. Spouse or partner's parents	**1**	**2**	**3**
3. Your relatives/kin	**1**	**2**	**3**
4. Spouse or partner's relatives/kin	**1**	**2**	**3**
5. Spouse or partner	**1**	**2**	**3**
6. Your friends	**1**	**2**	**3**
7. Spouse or partner's friends	**1**	**2**	**3**
8. Your own children	**1**	**2**	**3**
9. Other parents	**1**	**2**	**3**
10. Co-workers	**1**	**2**	**3**
11. Parent groups	**1**	**2**	**3**
12. Social groups/clubs	**1**	**2**	**3**
13. Church members/minister	**1**	**2**	**3**
14. Your family or your children's physician	**1**	**2**	**3**
15. Early childhood intervention program	**1**	**2**	**3**
16. School-day-care provider	**1**	**2**	**3**
17. Professional helpers (social workers, therapists, teacher, etc.)	**1**	**2**	**3**
18. Professional agencies (public health, social services, mental health, etc).	**1**	**2**	**3**
19. Other _____	**1**	**2**	**3**

Medical Outcomes Study (MOS) Social Support Scale
(Sherbourne & Stewart, 1991)

Answer these questions about the support that you receive. We want to know how often each of the following kinds of support is available to you if you need it. The responses to each question will be on a scale from 1 to 5 where 1 means 'never' and 5 means 'always.' Please answer 'Not Applicable' if the question does not apply to you.

Never	Rarely	Sometimes	Frequently	Always	NA

1. Someone to take you to the doctor if you needed it

1 2 3 4 5 6

2. Someone who shows you love and affection

1 2 3 4 5 6

3. Someone to have a good time with

1 2 3 4 5 6

4. Someone to confide in or talk to about yourself or your problems

1 2 3 4 5 6

5. Someone to help you with daily chores if you were sick

1 2 3 4 5 6

6. About how many close friends and close relatives do you have that live nearby?

0 1 2 3 4 5 6 7 8 9 10+

APPENDIX B

12 MONTH IRB APPROVAL LETTER

ARIZONA STATE UNIVERSITY
Research Compliance Office
Office for Research & Sponsored Projects Administration
P.O. Box 873503
Tempe, AZ 85287-3503

Phone
(480) 965-6788
Facsimile
(480) 965-7772

To:	Kathryn Lemery PSYCHOLOGY
From:	Mark Roosa, Chair Soc Beh IRB
Date:	03/20/2008
Committee Action:	**Expedited Approval**
Approval Date:	03/20/2008
Review Type:	Expedited F5 F7
IRB Protocol #:	0803002735
Study Title:	Arizona Twin Project
Expiration Date:	03/18/2009

The above-referenced protocol was approved following expedited review by the Institutional Review Board.

It is the Principal Investigator's responsibility to obtain review and continued approval before the expiration date. You may not continue any research activity beyond the expiration date without approval by the Institutional Review Board.

Adverse Reactions: If any untoward incidents or severe reactions should develop as a result of this study, you are required to notify the Soc Beh IRB immediately. If necessary a member of the IRB will be assigned to look into the matter. If the problem is serious, approval may be withdrawn pending IRB review.

Amendments: If you wish to change any aspect of this study, such as the procedures, the consent forms, or the investigators, please communicate your requested changes to the Soc Beh IRB. The new procedure is not to be initiated until the IRB approval has been given.

Please retain a copy of this letter with your approved protocol.

APPENDIX C

30 MONTH IRB APPROVAL LETTER

ASU Knowledge Enterprise Development

Office of Research Integrity and Assurance

To:	Kathryn Lemery PSYCHOLOGY
From:	Mark Roosa, Chair SM Soc Beh IRB
Date:	06/11/2010
Committee Action:	**Exemption Granted**
IRB Action Date:	06/11/2010
IRB Protocol #:	1006005211
Study Title:	Arizona Twin Project

The above-referenced protocol is considered exempt after review by the Institutional Review Board pursuant to Federal regulations, 45 CFR Part 46.101(b)(2) .

This part of the federal regulations requires that the information be recorded by investigators in such a manner that subjects cannot be identified, directly or through identifiers linked to the subjects. It is necessary that the information obtained not be such that if disclosed outside the research, it could reasonably place the subjects at risk of criminal or civil liability, or be damaging to the subjects' financial standing, employability, or reputation.

You should retain a copy of this letter for your records.