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Toddler risk and protective characteristics: Common and unique genetic and environmental influences

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Abstract

The goal of this study was to disentangle the common and unique genetic and environmental influences on social-emotional competence, problem behavior, physiological dysregulation, and negative emotionality (NE) in toddlers. The sample consisted of 243 twin pairs (mean age = 31.94 months) rated by primary caregivers (>95% mothers) on the Children's Behavior Questionnaire and the Infant-Toddler Social and Emotional Assessment. A multivariate Cholesky Decomposition revealed three shared environmental factors, with one set of environmental influences common to competence, problem behavior, and physiological dysregulation, a second common to problem behavior and physiological dysregulation, and a third common to physiological dysregulation and NE. Also, there were two additive genetic factors, with one explaining variance in competence, NE, and a small amount of variance in problem behavior, and a second explaining variance in problem behavior and NE. Given the common shared environmental factors across outcomes, these results suggest that toddlerhood could be a particularly important time to intervene, as interventions could simultaneously improve competencies and reduce problem behaviors. This study also highlights the need for genetically informed research to examine the etiology of multiple outcomes and address overlap.

Keywords

behavior problems; competencies; toddler; twin; genetic

Toddlerhood and early childhood are times of rapid development across multiple domains, including emotional reactivity and regulation, physiological regulation (e.g., sleep consolidation), and a range of early social, emotional, and cognitive competencies. Emotional or physiological dysregulation, or more serious mood and behavioral problems may also become evident during these ages, potentially interfering with the acquisition of developmental competencies. Risk factors are often studied, but less is known about the etiology of positive outcomes such as early competence, which may promote later social or academic adaptation and protect against risk for psychopathology. Further, despite being

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related and commonly studied domains of children's socioemotional development, early competence, behavior problems, physiological dysregulation, and negative emotionality (NE) are usually studied separately, making it difficult to determine the extent to which they represent at least partially independent risk and protective factors, as opposed to different indicators of a broad continuum of adaptation (Carter, Briggs-Gowan, Jones, & Little, 2003).

One source of information on the common versus distinct etiology of related traits is examining genetic and environmental influences on their covariance. For example, if genetic influences on NE are distinct from those underlying eating and sleep dysregulation, it would indicate that rather than different manifestations of broad emotional and physiological dysregulation, these traits are at least partially biologically distinct and may require different strategies for intervention. Further, by disentangling genetic and environmental influences, twin studies can provide support for environmental explanations in a way that non-genetically informed research cannot. The goal of this study was to examine the common and unique genetic and environmental influences on competence, problem behavior, physiological dysregulation, and NE in toddler-aged twins. As one of the few twin studies to consider multiple negative outcomes and competence together, we aimed to obtain a more holistic understanding of toddler outcomes.

Competence

Competence refers to the presence of age-appropriate skills, indexing positive development across multiple domains, including compliance, self-regulation, prosocial behaviors, empathy, and mastery motivation (Carter et al., 2003). Competence in age-appropriate tasks increases the likelihood of later competence and likely minimizes the emergence and maintenance of maladaptive behavioral patterns, whereas lags in social-emotional competence likely increase risk for social-emotional and behavioral problems.

The importance of parenting and the home environment for toddler competence is well established (e.g., Brophy-Herb et al., 2011; Lang et al., 2014), but few studies have examined competence using a genetically informed framework, and most have focused on only one component of competence. Therefore, it is unknown how the etiology of competence is related to other toddler outcomes. For example, children's empathy and prosocial behavior are genetically influenced, with some studies also finding moderate shared environmental influences (e.g. Knafo & Plomin, 2006; Saudino, Ronald, & Plomin, 2005; Scourfield, John, Martin, & McGuffin, 2004). The few studies examining the etiology of toddler competence as a whole are mixed. Van Hulle, Lemery-Chalfant, and Goldsmith (2007) found strong genetic influences (61%–67%) and moderate shared environmental influences (22%–26%), whereas another study found only modest genetic influences (21%–15%), with most variability in overall competence due to shared environments (73%–78%; Saudino, Carter, Purper-Ouakil, & Gorwood, 2008). In addition, the extent to which genetic and environmental influences are unique to competence or shared with negative toddler outcomes is unknown.

Problem Behavior

Problem behaviors deviate from the normative developmental course and cause problems for children (Carter et al., 2003). These behaviors, consisting primarily of externalizing (e.g., aggression) and internalizing (e.g., anxiety, depression) problems, can be reliably identified early in childhood and show considerable stability across age (e.g., Briggs-Gowan, Carter, Bosson-Heenan, Guyer, & Horwitz, 2006; Saudino et al., 2008). Toddlers who exhibit internalizing and/or externalizing behaviors are at risk for anxiety, mood disorders, antisocial behavior, and substance use in adulthood (e.g., Caspi, Moffitt, Newman, & Silva, 1996).

Twin studies reveal both genetic and environmental influences on early problem behavior, with internalizing and externalizing in infancy and early childhood influenced moderately by genes (46%–57% for Externalizing, 50%–59% for Internalizing) and modestly by the shared environment (27%–34% for Externalizing, 13%–29% for Internalizing; e.g., Bartels et al., 2004; Saudino et al., 2008). Research also has begun to examine genetic and environmental covariance between problem behavior and NE, and found genetic factors underlying the phenotypic prediction from toddler NE to behavior problems at 4 years (Schmitz et al., 1999), as well as the concurrent relation between NE and externalizing in twins 4–17 years old (Singh & Waldman, 2010). Conversely, a third study found that covariance between toddler NE and early to late childhood internalizing and externalizing symptoms was explained by shared environmental influences (Rhee et al., 2007). However, less is known about genetic and environmental influences underlying relations with competence or physiological dysregulation.

Physiological Dysregulation

Physiological dysregulation in toddlerhood includes sleeping and eating problems, problems regulating emotional states, and unusual sensory sensitivities (Carter et al., 2003). We focus on eating and sleep dysregulation to minimize conceptual overlap with NE. Physiological dysregulation is common but studied more rarely than NE or sensory sensitivity. Sleep problems (e.g., trouble falling or staying asleep, needing to be held to go to sleep) and eating problems (e.g., picky eating, spitting out food, refusing to eat certain foods) are reported to occur in 20–25% of young children (Owens, Spirito, & McGuinn, 2000; Wright, Parkinson, Shipton, & Drewett, 2007) and may impact toddler physical and social-emotional development greatly. Sleep problems are a risk factor for later development of psychopathology (Gregory & Sadeh, 2016), whereas eating problems identified in childhood can increase the later risk of developing an eating disorder (Kotler, Cohen, Davies, Pine, & Walsh, 2001). Early sleep and eating problems often co-occur, and share predictors including preterm birth, family adversity, and psychological stress (Schmid, Schreier, Meyer, & Wolke, 2011; Tauman et al., 2011). In addition, physiological dysregulation is related to problem behavior, attention regulation, and temperament (Haycraft, Farrow, Meyer, Powell, & Blissett, 2011; Stein, Mendelsohn, Obermeyer, Amromin, & Benca, 2001), necessitating joint consideration to understand sources of covariance.

Parent-reported sleep problems are moderately to highly heritable in toddlerhood (54–61%) with some evidence for shared environmental influences (30%; Saudino et al., 2008; Van

den Oord, Verhulst, & Boomsma, 1996). In contrast, very few twin studies have examined eating dysregulation in toddlers. In one, mother report of toddler's reactivity to food was influenced by shared family environment (Rowe & Plomin, 1977). However, Saudino et al. (2008) reported substantial genetic influence (64% for males, 71% for females) and little shared environmental influence (2% for males, 5% for females) on maternal report of young children's eating problems. The dearth of twin studies examining sleep and eating dysregulation highlights the need for further research.

Negative Emotionality

The temperament component Negative Emotionality (NE) is defined as a tendency to react with rapid, intense, or prolonged negative emotion, and is thought to index normative, not necessarily maladaptive, individual differences. Although NE is substantially correlated with both internalizing and externalizing psychopathology in adults (Krueger & Markon, 2006), some NE is typical in early childhood as toddlers learn how to regulate emotions (Carter et al., 2003). In addition, although NE and problem behavior are linked, they are distinct constructs, and measurement confounding does not account for relations between temperament and problem behavior in toddler- and preschool-age children (Lemery, Essex, & Smider, 2002).

NE is consistently heritable across twin studies (Saudino, 2005). For example, two studies considering broad NE factors report substantial heritability (42%–65%), with no significant shared environmental influences (Goldsmith, Buss, & Lemery, 1997; Saudino et al., 2008). However, for anger proneness, there was evidence for genetic (34%) and shared environmental (38%) effects, supporting the influence of the shared environment on some aspects of NE in toddlerhood (Goldsmith et al., 1997).

The Current Study

The primary aim of this study was to investigate common and distinct genetic and environmental sources of variance in competence, problem behavior, physiological dysregulation, and NE. We began by examining the univariate heritability of each outcome to understand how our sample compares to other twin panels. Next, we considered genetic and environmental influences underlying covariance among all four outcomes. We predicted that all outcomes would be influenced by genetic factors, that physiological dysregulation and problem behavior would also be influenced by the shared environment (e.g. Saudino et al., 2008; Van Hulle et al., 2007), and that NE and problem behavior would share genetic influence (Schmitz et al., 1999; Singh & Waldman, 2010).

Method

Participants

Participants were drawn from the Arizona Twin Project, a population-based panel of twins born in Arizona (Lemery-Chalfant, Clifford, McDonald, O'Brien, & Valiente, 2013). The twins and their mothers first participated when the twins were 12 months of age. The initial sample consisted of two hundred and ninety-one twin pairs who participated at 12 months

and had both interview and demographic data. Twenty-seven percent of participants were Hispanic, and 55% were European American (4% African American, 3% Asian American, 1% Native American, and 8% mixed ethnicity). Income ranged from under \$20K to over \$100K (median of \$60–80K). Parental education ranged from less than a high school diploma to a professional degree (median education was a college degree). At the 30-month follow-up (mean age = 31.94 months, $SD = .25$), 520 (89%) twins participated. Twins who were missing outcome data ($n = 6$), covariate data ($n = 2$), or had a severe medical condition or cognitive impairment ($n = 26$) were excluded from all analyses. Therefore, a total of 243 twin pairs were included in analyses. More specifically, 63 (26%) were monozygotic, 91 (37.6%) were same-sex dizygotic, and 89 (36.4%) were opposite-sex dizygotic.

Procedure

When the twins were 30 months old, primary caregivers completed two structured telephone interviews, performed by the same trained undergraduate or graduate research assistant and typically completed 1–2 weeks apart. The first assessed demographics, zygosity, and twins' social and emotional behavior, including temperament, competence, behavior problems, and physiological dysregulation. The second assessed developmental milestones, the home environment, and parenting. Primary caregivers answered each question for both twins at the same time.

Measures

Zygosity.—Zygosity was established using the Zygosity Questionnaire for Young Twins (Goldsmith, 1991). This 32-item questionnaire asks caregivers about the pregnancy (e.g., use of In Vitro Fertilization) and physical characteristics of the twins, and yields over 95% agreement with zygosity determined via genotyping (Forget-Dubois et al., 2003). Zygosity was further verified with pregnancy and birth medical records, including lab reports of the placenta, as well as comparing photos of the twins.

Competence, Problem Behavior, and Physiological Dysregulation.—The Infant Toddler Social and Emotional Assessment (ITSEA; Carter et al., 2006) is a parent-report questionnaire that assesses social-emotional and behavioral problems and competencies in 1- to 3-year olds. In our study, we administered all scales with the exception of Problem Behavior and Negative Emotionality. Competence (e.g., “Does Twin A/B put toys away after playing?”) was a mean composite of the Compliance, Attention, Imitation/Play, Mastery Motivation, Empathy, and Prosocial Peer Relations scales. Cronbach's alpha was .90 in our study. Physiological Dysregulation (e.g., “Twin A/Twin B has trouble falling asleep or staying asleep,”) was the mean composite of the Sleep Dysregulation and Eating Dysregulation scales. In our study, Cronbach's alpha was .78. We administered the Problem Behavior subscale of the brief version of the ITSEA (BITSEA; Briggs-Gowan, Carter, Irwin, Wachtel, & Cicchetti, 2002), which includes 29 items (e.g., “Does Twin A/Twin B hit, shove, kick, or bite other children?”) tapping externalizing, internalizing, atypical, and maladaptive behaviors. In our study, Cronbach's alpha was .79. The BITSEA has good psychometric properties, including excellent test-retest reliability, interrater agreement among mothers and fathers, and discriminant validity (Briggs-Gowan, Carter, Irwin, Wachtel, & Cicchetti, 2004), but does not support separate internalizing and externalizing

factors. However, previous research has demonstrated that internalizing and externalizing symptoms co-occur in children (Oland & Shaw, 2005), thus providing a rationale for examining internalizing and externalizing symptoms as a unitary construct in our study. For both the ITSEA and BITSEA, items were asked separately for each twin and rated on a 3-point scale: 0 = *not true/rarely*, 1 = *somewhat true/sometimes*, and 2 = *very true/often*, with a *non-applicable* option.

Negative Emotionality.—The Children’s Behavior Questionnaire—Short Form (CBQ-SF; Putnam & Rothbart, 2006) is a reliable and valid parent-report measure of temperament in young children. NE was a mean composite of the CBQ Anger/Frustration (e.g., “Gets quite frustrated when prevented from doing something s/he wants to do”, $\alpha = .76$ in our study) and Soothability (reversed) scales (e.g., “Is easy to soothe when s/he is upset”, $\alpha = .73$ in our study). These scales were significantly correlated, $r(513) = -.39$, $p < .001$, controlling for twin interdependence using the Type=Complex option in Mplus 7.0 (Muthén & Muthén, 2015). Primary caregivers rated twins on a 7-point scale from 1 = *extremely untrue of your child* to 7 = *extremely true of your child*.

Covariates.—Age, sex (0 = male, 1 = female), and ethnicity (0 = non-Hispanic European American, 1 = all other racial and ethnic groups) were included as covariates in descriptive analyses and regressed out prior to model fitting.

Results

Preliminary Analyses

Table 1 contains descriptive statistics and correlations for study variables. Means and standard deviations of toddler outcomes were comparable to normative data on these measures (Carter, et al., 2003; Putnam & Rothbart, 2006), and no variable exceeded acceptable ranges of ± 2.00 for skewness or ± 7.00 for kurtosis (Muthén & Kaplan, 1985). Using the Type=Complex option in Mplus 7.0 to account for twin dependence, NE, physiological dysregulation, and problem behavior were positively correlated with one another, and negatively correlated with competence.

The twin design allows the estimation of genetic and environmental influences on a phenotype (Neale & Maes, 2004). Monozygotic (MZ) twins share 100% of their genes, whereas Dizygotic (DZ) twins, on average, share 50% of their segregating genes. Thus, MZ intraclass correlations (ICCs) higher than DZ ICCs suggest genetic influences and doubling the difference between MZ and DZ ICCs provides an estimate of heritability. Twin intraclass correlations (ICCs) are provided in Table 1. MZ twins were more similar than DZ twins for all variables, but DZ ICCs were higher than half the MZ ICCs, suggesting the role of both additive genetic and shared environmental influences. However, biometrical model fitting provides more precise estimates and allows the significance of genetic and environmental variance to be tested.

Biometrical structural equation model fitting was conducted using OpenMx (Neale et al., 2016). To test the assumptions of the twin design, we first fit saturated models testing zygosity differences in means and variances and found no significant differences.

We then fit univariate models decomposing the variance into latent additive genetic (A, the additive effect of multiple genes), shared environmental (C, environmental experiences that increase cotwin similarity), and non-shared environmental (E, environmental experiences that increase cotwin dissimilarity, plus measurement error) factors. To find the most parsimonious solution, models were tested by systematically dropping parameters, using the -2 log likelihood chi-square test of fit to compare reduced to full models. A significant difference in fit suggests that the dropped path is needed to reproduce the data. Because E contains measurement error, this path is never dropped. Table 2 contains fit statistics and standardized variance components for univariate ACE models, which yielded genetic estimates ranging from .35-.59 across outcomes, shared environmental estimates ranging from .18-.55, and nonshared environmental estimates ranging from .09-.37.

Cross-twin cross-trait ICCs are presented in Table 3. Comparing the MZ and DZ cross-twin correlations suggests that both A and C influences underlie the relation between Problem Behavior and Dysregulation, whereas the relation between Problem Behavior and NE is likely more influenced by A, and the relation between Dysregulation and NE is likely more influenced by C. However, examining cross-twin correlations between Competence and the other outcomes reveals that the DZ correlations are higher than MZ correlations for Problem Behavior ($r_{MZ} = .18$, $r_{DZ} = .25$) and Dysregulation ($r_{MZ} = .12$, $r_{DZ} = .18$). This is likely due to a combination of primarily shared environmental influences underlying covariance between these phenotypes and measurement error, especially as the MZ group contains only 63 twin pairs. Nevertheless, although it is not a violation of the assumptions of the twin design, this could be indicative of sibling effects.

Hypothesis Testing: Analytical Rationale

Next, a multivariate Cholesky Decomposition was conducted to estimate ACE influences on covariances across the outcomes (Figure 1; Neale & Maes, 2004). In a Cholesky decomposition with four outcomes, the first set of latent factors consists of genetic, shared environmental, and nonshared environmental influences on the first variable, which are also allowed to influence each subsequent variable. The second set of latent factors represents influences on the second variable that are independent of the first but allowed to influence the third and fourth variables. The third set are independent of the first and second, but also influence the fourth, and the fourth set consists of influences unique to the fourth variable. Variable order does not affect model fit or the significance of A, C, or E covariance between any two traits, but is relevant for the interpretation of each set of factors. We chose the following order: competence, problem behavior, physiological dysregulation, and NE. As the sole indicator of positive adjustment, competence was chosen as the first variable because we expected it to be broadly relevant for all other traits, whereas physiological dysregulation and NE were chosen as third and fourth, respectively, because we expected that as potential indicators of more general dysregulation, they might share genetic or environmental influences distinct from other traits.

After fitting the full model, we first dropped all paths estimated at or very near zero. Then, we systematically tested individual paths, only testing A or C variance on earlier traits after establishing that A or C covariance with subsequent traits could be dropped without loss of

fit. Finally, we tested combinations of paths to arrive at the most parsimonious final model, using confidence intervals and the AIC to inform final model selection when the $-2 \log$ likelihood test was insufficient to distinguish between models.

Multivariate Cholesky Decomposition Model Fitting

Table 4 and Table 5 include model fit and estimates for the full and most parsimonious final multivariate Cholesky Decompositions, respectively. After dropping non-significant paths, the final model (Figure 2) revealed that two genetic factors and three shared environmental factors were needed to account for covariances among the outcomes. The first genetic factor explained the majority of variance in competence (53%), and a small amount of variance for problem behavior (3%) and NE (9%). The second, which was independent of competence, explained 34% of the variance in problem behavior and 39% of the variance in NE.

Although the univariate model showed moderate additive genetic influences on physiological dysregulation (45%), physiological dysregulation in the multivariate model was explained by shared (44%) and nonshared (56%) environmental influences, with weak and nonsignificant A. This discrepancy across models is likely due to the pattern of cross-twin cross-trait correlations noted earlier, which require primarily C influences on covariance between dysregulation and the other phenotypes. In the multivariate model, the first shared environmental factor accounted for moderate variance in competence (38%), and minimal but significant variance in problem behavior (5%) and physiological dysregulation (4%), whereas the second explained moderate variance in problem behavior (46%) and dysregulation (18%). The final shared environmental factor accounted for variance in physiological dysregulation (22%) and NE (17%). Nonshared environmental variance was modest (2%–22%) for all scales except physiological dysregulation, and primarily scale-specific, although nonshared environmental influences on competence explained a small but significant amount of variance in problem behavior (2%). Table 6 includes the proportion of the phenotypic correlation accounted for by genetic and environmental factors for both the full and final model, which provides standardized contributions of each variable.

Discussion

We examined the multivariate genetic and environmental etiology of common toddler positive and negative outcomes. Covariance across these constructs was largely accounted for by the shared environment, with one set of environmental influences common to competence, problem behavior, and physiological dysregulation, a second common to problem behavior and physiological dysregulation, and a third common to physiological dysregulation and NE. We also found two additive genetic factors, with one explaining variance in competence and NE, along with a small amount of variance in problem behavior, and a second explaining variance in problem behavior and NE. Findings support the importance of the shared environment across multiple domains and suggest that specific environmental risk and protective factors may be uniquely related to particular combinations of toddler outcomes, such as emotional and physiological dysregulation.

Genetic and Environmental Influences on Trait Variances

Consistent with past research (Bartels et al., 2004; Goldsmith et al., 1997; Van Hulle et al., 2007), we found high heritability and significant shared environmental influences for toddlers' competence, modest genetic influences and moderate shared environmental influences for problem behavior, and moderate heritability and modest shared environmental influences for NE. Our estimates of shared environmental influences on problem behavior are higher than other studies, perhaps because of our heterogeneous sample (25% Hispanic). Twin studies typically include twins of Northern European descent (Polderman et al., 2015), but studies including less-studied groups have reported larger shared environmental influences (e.g., Zheng, Rijdsdijk, Pingault, McMahon, & Unger, 2016).

Physiological dysregulation in the univariate model was moderately heritable, with modest but significant shared environmental influences, which is consistent with past twin studies of sleep problems in toddlerhood (Saudino et al., 2008, Van den Oord et al., 1996), although the behavior genetic literature for eating problems is sparse and contradictory (e.g. Rowe & Plomin, 1977). Modest genetic influences on physiological dysregulation were present in the full multivariate model, but could be dropped from the final model. The discrepancy in results across models may be due to the DZ cross-twin cross-trait correlation between physiological dysregulation and competence that was higher than MZ correlations.

Genetic and Environmental Influences on Trait Covariances

We found that competence was related to problem behavior and physiological dysregulation for shared environmental reasons, with additional nonshared environmental influences explaining a small amount of covariance between competence and problem behavior but not physiological dysregulation. Multiple environmental factors could explain relations between competence, problem behavior, and physiological dysregulation, including parental warmth and discipline, or emotional expression. In one study of children ages 4–8 years, mothers' expression of both positive and negative emotion was associated with children's social competence and externalizing problems (Eisenberg et al., 2001). Parental positive expressivity could foster social competence, whereas frequent parental expression of hostile negative emotions could lead to overarousal in emotionally evocative situations (Buck, 1984), potentially impacting children's regulation of emotions, attention, and behavior.

Alternately, some environmental factors may be indirectly related to one domain through their influence on another. For example, sleep dysregulation could impact attentional focusing, which is included in our competence composite and may also be important for other facets of competence. Indeed, children with fragmented sleep demonstrated poorer performance on attentional focusing tasks (Sadeh, Gruber, & Raviv, 2002). If sleep and eating problems increase risk for behavior problems and lower competence, then environmental interventions targeting early sleep and eating may have cascading effects across multiple domains.

Finally, genetic influences on competence explained a relatively small proportion of the variance in NE, but fully accounted for the correlation between the two domains. Some aspects of competence such as attentional focusing may be important for children's ability to

regulate negative emotions (Rothbart & Bates, 2006). Conversely, NE may interfere with the development and expression of competencies such as self-regulation and mastery motivation (Valiente, Swanson, & Eisenberg, 2012). The ways in which genes translate to behavior are complex, but NE bears further investigation as a heritable, early-emerging trait that may disrupt the acquisition of competence.

Although most variance in problem behavior was explained by environmental influences, problem behavior and NE had no environmental covariance. Instead, a second genetic factor independent of competence accounted for substantial variance in both traits. As a component of temperament, NE encompasses normative and partially biologically-influenced variation (Rothbart & Bates, 2006), with high emotional expressivity particularly common in toddlerhood (Carter et al., 2003). However, high NE may increase risk for more severe behavioral difficulties (Garstein, Putnam, & Rothbart, 2012), especially under stressful conditions. Our finding that problem behavior shares genetic covariance with NE suggests the possibility of NE as a heritable emotional core of problem behaviors, with the disordered extreme differentiated from normative variation by environmental factors shared across multiple domains. A study by Tackett and colleagues (2013) corroborates our findings. More specifically, the authors found that in children and adolescents, NE was more strongly associated with the general factor of psychopathology than with either specific internalizing or externalizing factors, and quantitative genetic analyses revealed additive genetic influences on the general factor, but not on the specific factors. Together, these results suggest that examining internalizing and externalizing symptoms together is appropriate for younger samples. Indeed, although we found no environmental covariance between NE and problem behavior, one study of a high-risk sample found that an environment characterized by multiple stressors (e.g., neighborhood dangerousness, overcrowding) exacerbated the relation between toddlers' NE and internalizing problems (Northerner, Trentacosta, & McLear, 2016), supporting the idea that relations between NE and more severe behavior problems can be environmentally-dependent under stress.

In contrast, NE was related to physiological dysregulation solely for environmental reasons. Physiological dysregulation and NE are commonly treated as two indices of infant dysregulation and combined into a single component, as seen in the ITSEA (Carter et al., 2003) and the construct of Difficult Temperament (Thomas & Chess, 1977). Our finding that NE is genetically distinct from eating and sleep dysregulation suggests that it might be more appropriate to conceptualize and study them as related but separate traits linked through environmental mechanisms such as parenting around eating and sleep (Mindell, Sadeh, Kohyama, & How, 2010; Savage, Fisher, & Birch, 2007). Challenging interactions during mealtimes and bedtimes may increase the use of negative parenting strategies, which may in turn elicit both dysregulated behavior and negative emotion. Additionally, environmental influences on sleep or eating (e.g., quality of the sleep environment) may indirectly influence NE, as poor sleep or nutrition may increase negative reactivity and interfere with regulation (Gruber & Cassoff, 2014; Wachs, 2000).

Our finding that the shared environmental covariance played a role in each outcome suggests it may be possible to affect functioning across multiple domains by targeting key aspects of the environment. For example, Van Zeijl et al. (2006) found that an intervention promoting

positive parenting in mothers of 1–3-year old children decreased children's problem behaviors via changes in maternal attitudes toward sensitive discipline. However, our finding of three shared environmental factors, each explaining covariance across different sets of traits, suggests that some environmental risk and protective factors are specific to narrower domains of functioning, and even factors such as parenting recognized as important for multiple domains may be most relevant to particular outcomes in specific contexts (e.g., parenting around sleep; Mindell et al., 2010). Similarly, the separate genetic factors influencing competence and problem behavior support multiple genetic and environmental pathways to adaptation and maladaptation in childhood, with high competence potentially compensating for the risk associated with early problem behavior.

Study Limitations

One limitation was the reliance on maternal report, which could have inflated estimates of the shared environment. However, other studies using maternal report assessing very similar constructs report no shared environmental influences, suggesting that rater bias is not accounting for the shared environmental influences (e.g. Gagne, Saudino, & Asherson, 2011; Gregory, Eley, & Plomin, 2004; Van Hulle et al., 2007). In addition, we found multiple C factors, as opposed to a single factor, and thus it is unlikely that an overarching characteristic of the primary caregiver accounts for all shared environmental influences. Primary caregivers reported on both twins in the same interview, therefore shared method effects may be occurring, as multivariate analyses showed shared environmental covariance among competence, problem behavior, and physiological dysregulation, all measured using the same instrument. Although we acknowledge this possibility, our multivariate analysis produced three shared environmental factors needed to account for covariances among the four outcomes, suggesting that we are capturing true covariance across outcomes in addition to shared method variance. An important way to advance this line of research is to use multi-method approaches.

Another question is whether results generalize to singleton children. Goldsmith and Campos (1990) found no differences between infant twins and singletons on temperament, and our means and standard deviations for the ITSEA and CBQ were similar to those reported in normative samples (Carter et al., 2003; Putnam & Rothbart, 2006), suggesting that our twin sample is representative of the overall population. Another assumption is that trait-relevant environments are not more similar for MZ than DZ twins, which would threaten internal validity. Research suggests that this assumption holds for temperament in infancy and childhood (Borkenau, Riemann, Angleitner, & Spinath, 2002; Goldsmith, Lemery, Buss, & Campos, 1999). Finally, gene-environment correlations and interactions were not examined in the current study.

Future Directions

One future direction is to determine which environmental factors contribute to covariance across toddler outcomes, and whether their influence is strongest for specific sets of outcomes (e.g., consistency and routine for physiological dysregulation and NE). Parenting interventions targeted toward parents with toddlers and preschool-aged children reduce problem behaviors and increase areas of competence (e.g. Gardner, Shaw, Dishion, Burton,

& Supplee, 2007), but it is unknown whether they simultaneously affect physiological dysregulation, or which environmental factors might be uniquely important for the link between physiological dysregulation and NE. Finally, objective measurement of multiple aspects of children's environment (e.g., physical safety, enrichment, and parenting) is needed.

A second future direction is to examine longitudinally mediation and direction of effect. For example, some environmental influences may predict other outcomes via eating and sleep dysregulation, or children high in NE may evoke particular parenting strategies surrounding eating and sleep that increase risk for physiological dysregulation. Longitudinal modeling across early childhood would also allow an examination of continuity and change in genetic and environmental influences. The shared environment often plays a larger role in childhood, with heritability increasing with age (Knopik, Neiderhiser, DeFries, & Plomin, 2016), suggesting shared environmental influences on our outcomes may attenuate with age, but fewer studies have examined genetic and environmental influences on covariance across outcomes over time. It may be that the environmental factors underlying concurrent covariance continue to explain relations among these outcomes across age.

Conclusion

This study examined common and unique genetic and environmental influences among competence, problem behavior, physiological dysregulation, and NE in toddlerhood. Key findings include the importance of the shared environment for these toddler outcomes, and that multiple environmental factors underlie relations among different sets of traits, suggesting the utility of designing interventions targeted to specific combinations of outcomes such as NE and physiological dysregulation. In addition, our results show that competencies are genetically distinct in etiology, and the larger literature shows they are protective for children at-risk for problem behaviors and physiological dysregulation, supporting a holistic approach to toddler development that includes positive as well as negative outcomes.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

Example multivariate Cholesky Decomposition. A = additive genetic influences; C = common environmental influences; E = nonshared environmental influences. A1, C1, and E1 represent all ACE influences on Competence, which may be shared with all other phenotypes. A2, C2, and E2 represent influences on Problem Behaviors that are independent of Competence but may be shared with Physiological Dysregulation and Negative Emotionality (NE). A3, C3, and E3 represent influences on Dysregulation that may be shared with NE, and A4, C4, and E4 represent influences on NE.

Figure 2. Best-fitting multivariate Cholesky Decomposition with significant standardized parameter estimates. A = additive genetic influences; C = common environmental influences; E = nonshared environmental influences.

Table 1.

Zero-Order Correlations, Descriptive Statistics, and Twin Intra-class Correlations

	1	2	3	4	MZ	DZ
1. Competence	-				.93	.60
2. Problem Behaviors	-.36**	-			.87	.73
3. Physiological Dysregulation	-.18**	.44**	-		.59	.43
4. Negative Emotionality	-.33**	.49**	.30**	-	.71	.47
Mean	1.53	0.35	0.44	3.81		
Standard Deviation	0.25	0.20	0.32	0.70		
Actual Minimum	0.41	0.00	0.00	1.83		
Actual Maximum	2.00	1.21	1.86	6.92		
Possible Minimum	0.00	0.00	0.00	1.00		
Possible Maximum	2.00	2.00	2.00	7.00		
Skewness	-0.87	1.01	0.94	0.18		
Kurtosis	1.21	1.68	0.88	-0.08		

Note:

**
p < 0.01. MZ = monozygoticDZ_{SS} = same-sex dizygotic; DZ_{OS} = opposite sex dizygotic.

Table 2.

Univariate ACE models and fit statistics

Scale	Model	-2LL	df	AIC	A	C	E
Competence	ACE	1158.36	482	194.36	.42 (.29-.58)	.49 (.33-.69)	.09 (.05-.14)
Problem Behaviors	ACE	1163.04	482	199.04	.35 (.22-.52)	.55 (.38-.74)	.10 (.05-.16)
Physiological Dysregulation	ACE	1324.14	482	360.14	.45 (.12-1.00)	.18 (.01-.59)	.37 (.20-.59)
Negative Emotionality	ACE	1299.70	480	339.70	.59 (.30-.97)	.18 (.02-.50)	.23 (.11-.39)

Note. -2LL = -2 log likelihood, *df* = degrees of freedom, AIC = Akaike's Information Criterion. A = additive genetic influence, C = shared environmental influence, and E = nonshared environmental influence. Standardized estimates are given, with confidence intervals in parentheses.

Table 3.

Cross-Twin Cross-Trait Correlations

MZ								
	Within-twin Cross-trait Correlations				Cross-twin Cross-trait correlations			
	1.	2.	3.	4.	1.	2.	3.	4.
1. Competence	1.00	.22	.16	.28	.91	.18	.12	.30
2. Problem Behavior		1.00	.49	.48		.88	.42	.44
3. Dysregulation			1.00	.33			.49	.26
4. Negative Emotionality				1.00				.69

DZ								
	Within-twin Cross-trait Correlations				Cross-twin Cross-trait correlations			
	1.	2.	3.	4.	1.	2.	3.	4.
1. Competence	1.00	.35	.14	.35	.66	.25	.18	.22
2. Problem Behavior		1.00	.33	.45		.70	.33	.23
3. Dysregulation			1.00	.25			.39	.22
4. Negative Emotionality				1.00				.45

Note. MZ = monozygotic; DZ = dizygotic.

Table 4.

Model for Full and Final Models (Four-Variable Models)

Model	-2LL	df	AIC	-2LL	df	p
Full	4632.91	1908	816.91			
Final	4647.32	1921	805.32	14.40	13	.35

Note. -2LL = -2 log likelihood; *df*= degrees of freedom; = change; AIC = Akaike's Information Criterion.

Table 5.

Genetic and Environmental Contributions to Variance and Covariance across Outcomes (Four-Variable Models)

Full model										
	A1	C1	E1	A2	C2	E2	A3	C3	E3	E4
Competence	.49 (.35-.65)	.42 (.25-.63)	.09 (.06-.12)							
Problem Behavior	.02 (.00*-.09)	.08 (.01-.22)	.02 (.00*-.15)	.36 (.25-.48)	.44 (.28-.62)	.09 (.06-.12)				
Dysregulation	.00 (-.05,-.05)	.06 (.00*-.23)	.00 (-.03-.04)	.00 (-.06-.06)	.18 (.05-.38)	.02 (.00-.09)	.06 (-.25-.99)	.15 (.01-.49)	.53 (.38-.71)	
Negative Emotionality	.14 (.03-.31)	.02 (-.01-.12)	.00 (-.02-.02)	.27 (.11-.50)	.00 (-.02-.05)	.01 (.00-.06)	.06 (-.63-1.55)	.10 (.00 [†] -.32)	.00 (-.01-.02)	.21 (.14-.29)

Final model										
	A1	C1	E1	A2	C2	E2	A3	E3	A4	C4
Competence	.53 (.39-.69)	.38 (.22-.57)	.09 (.06-.13)							
Problem Behavior	.03 (.00*-.09)	.05 (.00*-.17)	.02 (.00*-.05)	.34 (.23-.46)	.46 (.33-.62)	.10 (.07-.13)				
Dysregulation	-	.04 (.00*-.13)	-	-	.18 (.09-.30)	-	.22 (.12-.34)	.56 (.47-.67)		
Negative Emotionality	.18 (.09-.29)	-	-	.39 (.25-.57)	-	-	.17 (.08-.30)	-	-	.26 (.19-.34)

Note. ACE components of variance for each phenotype are standardized according to total variance of that phenotype. ACE components of covariance are standardized according to the total variance of the second phenotype.

^{*}The lower bound of the confidence interval is not 0, but a very small positive number (.001-.004) rounded down. Attempting to drop this path from the model led to significant loss of fit

[†]The lower bound of the confidence interval is not 0, but a very small positive number (.003) rounded down. The path can be dropped without significant loss of fit, but only if A43 is retained in the model

Table 6.

Proportion of the phenotypic correlation accounted for by A, C, and E factors

Full Model												
	Competence			Problem Behavior			Dysregulation			Negative Emotionality		
	A	C	E	A	C	E	A	C	E	A	C	E
Competence	.49	.42	.09									
Problem Behavior	.33	.54	.12	.38	.51	.11						
Dysregulation	.00	.97	.03	.00	.89	.11	.06	.39	.55			
Negative Emotionality	.75	.25	.00	.79	.13	.08	.22	.62	.16	.66	.12	.22

Final Model												
	Competence			Problem Behavior			Dysregulation			Negative Emotionality		
	A	C	E	A	C	E	A	C	E	A	C	E
Competence	.53	.38	.09									
Problem Behavior	.40	.46	.14	.37	.51	.12						
Dysregulation	.00	1.00	.00	.00	1.00	.00	.00	.43	.57			
Negative Emotionality	1.00	.00	.00	1.00	.00	.00	.00	1.00	.00	.57	.17	.26

Note. Diagonal elements represent the proportion of variance in each phenotype accounted for by A, C, and E factors. Off-diagonal elements represent the proportion of the correlation between two phenotypes accounted for by A, C, and E factors.