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Genetic and Environmental Contributions to Associations between Infant Fussy Temperament and Antisocial Behavior in Childhood and Adolescence

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Abstract

Previous research suggests that fussy temperament in infancy predicts risk for later antisocial behavior (ASB) in childhood and adolescence. It remains unclear, however, to what extent infant fussiness is related to later ASB through causal processes or if they both reflect the same family risk factors for ASB. The current study used two approaches, the comparison of siblings and bivariate biometric modeling, to reduce familial confounding and examine genetic and environmental influences on associations between fussiness in the first two years of life and ASB in childhood and late adolescence. Analyses were conducted on data from a prospective cohort (9,237 at 4-9 years and 7,034 at 14-17years) who are the offspring of a nationally representative sample of U.S. women. In the full sample, fussiness predicted both child and adolescent ASB to small but significant extents, controlling for a wide range of measured child and family-level covariates. When siblings who differed in their fussiness were compared, fussiness predicted ASB in childhood, but not ASB during adolescence. Furthermore, results from a bivariate Cholesky model suggested that even the association of fussiness with childhood ASB found when comparing siblings is attributable to familial factors. That is, although families with infants who are higher in fussiness also tend to have children and adolescents who engage in greater ASB, the hypothesis that infant fussiness has an environmentally mediated impact on the development of future ASB was not strongly supported.

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Youth antisocial behavior (ASB) in the form of rule breaking, delinquent, and aggressive behaviors has enormous financial and societal costs and is a predictor of many negative outcomes in adulthood, including criminal behavior, intimate partner violence, reduced occupational stability, poverty, depression, substance abuse, and physical health problems (Odgers et al. 2007; Pulkkinen Lyyra and Kokko, 2009; Reid et al. 2002). These associations suggest that preventing the development of ASB in childhood and adolescence has the potential to enhance public health. Nevertheless, effective prevention of ASB largely depends upon the identification of malleable causal factors that operate very early in life, which have proven difficult for epidemiologic research to clearly distinguish from non-causal risk markers.

Infant fussiness, or irritability, is one promising candidate for a child characteristic that causes future ASB. Several longitudinal studies have shown that fussy temperament in infancy predicts ASB in childhood (e.g., Bates 1989; Lahey et al. 2008) and adolescence (Olson et al. 2000; Teerikangas et al. 1998). In addition, infant fussiness appears to be influenced by environmental factors unique to the child (not experienced in common with one's siblings), as Goldsmith, Lemery, Buss, and Campos (1999) found that non-shared environmental influences (and measurement error) contributed to 42% of the variance in infant fussiness in their sample. These findings suggest that fussy temperament may be an effective target for psychosocial interventions aimed at preventing ASB. It remains unclear, however, if individual differences in infant fussiness play a causal role in influencing the development of ASB during childhood and adolescence or if fussiness and later ASB simply reflect common familial (i.e., genetic and environmental) background factors.

A number of genetically informed studies have examined links between temperament and later ASB. Findings from previous studies are difficult to interpret, however, because these studies are characterized by at least one of the following: (1) used measures of temperament that do not clearly differentiate fussiness/irritability from other, associated aspects of temperament, such as fearfulness (e.g., Gjone and Stevenson 1997); (2) did not consider ASB in adolescence separately from ASB in childhood (Singh and Waldman 2010); or (3) did not use methods that adequately control for factors that may correlate with offspring temperament and ASB and thus may confound their association (e.g., Schmitz et al. 1999). In addition, past genetically informed studies are based on relatively homogenous samples in terms of racial/ethnic diversity. The current study used a combination of sibling-comparison and multivariate biometric analyses to address these limitations and advance understanding of genetic and environmental contributions to associations between temperamental fussiness and ASB in childhood and late adolescence.

Distinguishing Fussiness from Fearfulness

There are several reasons to study fussiness separately from the more general construct of negatively emotionality. First, factor-analytic studies of infant temperament have found that negative emotionality can be divided into two subdimensions: fussiness, alternatively referred to as irritable distress, anger/frustration, or distress to limitations; and fearfulness, alternatively referred to as distress to novelty, fearful distress, or unadaptability (Rothbart and Bates 1998). Indeed, a previously conducted factor analysis of mother-reported infant

temperament data from the present sample has supported this distinction (Lahey et al. 2008). Second, fussiness and fearfulness are thought to have distinct biological substrates and situational triggers (Rothbart et al. 1994), and, third, biometric analyses have shown that genetic and environmental influences on these dimensions are almost entirely unique to each trait (Goldsmith et al. 1999).

The fourth and perhaps most important reason to consider fussiness apart from fearfulness when investigating risk for ASB is that these temperament dimensions, although positively correlated, have been found to be differentially associated with later ASB. Infant fussiness has been found to predict increased risk for conduct problems (CPs) in childhood (e.g., Bates et al. 1985, Morris et al. 2002, Gilliom and Shaw 2004), and, to a lesser extent, in late adolescence (e.g., Olson et al. 2000). In contrast, fearfulness has been found to predict *decreased* risk for ASB or to have a nonsignificant association. For example, Rothbart, Ahadi, and Hershey (1994) found an inverse association between infant fearfulness and aggression several years later in childhood, and others have reported an inverse association when predicting a broader index of ASB in early adolescence (Keiley et al. 2003). In addition, Lahey et al. (2008) found that infant fussiness, but not fearfulness, predicted ASB in childhood, while Teerikangas et al. (1998) found the same pattern of findings as Lahey when predicting ASB in adolescence. Nevertheless, previous genetically informed studies of temperament-ASB links have considered the superordinate construct of negative emotionality rather than fussiness specifically (e.g., Singh and Waldman 2010).

Distinguishing Child and Adolescent ASB

In addition to considering fussiness apart from fearfulness when predicting ASB, it is also important to distinguish childhood and adolescent ASB as outcome variables. Childhood CPs include behaviors such as cheating, lying, disobedience, and bullying, which reflect violations of social norms but do not typically constitute illegal behaviors, whereas delinquency includes behaviors such as assault, stealing, and school truancy, which reflect status offenses and more serious forms of illegal behaviors. Furthermore, correlations between CPs in childhood and delinquency in late adolescence have been found to be small to moderate in size (e.g., Van Hulle et al. 2009), indicating that many children who exhibit high levels of CPs in childhood do not go on to engage in delinquent behaviors in adolescence, and many children who exhibit delinquency do not have a history of CPs in childhood.

Other evidence suggesting that childhood CPs should be studied separately from delinquency in adolescence comes from genetically informative analyses of individual differences in the two phenotypes. Although a meta-analysis found evidence for genetic influences on ASB in both childhood and adolescence (Rhee and Waldman, 2002), these genetic influences may not be shared in common. Van Hulle et al. (2009) explored the possibility that different genetic influences may contribute to childhood CPs and adolescent delinquency by fitting a series of multivariate biometric models to ASB in childhood and adolescence using data from the National Longitudinal Survey of Youth (NLSY79) and children of the NLSY79 (CNLSY). The best fitting model indicated that additive genetic influences on childhood CPs were distinct from additive genetic influences on delinquency

in late adolescence, even after ruling out the potential biasing effects of rater differences across age periods. In addition, Van Hulle et al. found that only a small proportion of the nonshared environmental influences on delinquency in late adolescence was shared with CPs in childhood. These findings strongly suggest that CPs in childhood and delinquency in adolescence have partially distinct etiologies and that there is value in studying them as distinct outcomes.

The Current Study

Because previous genetically informed analyses have considered fussiness as part of the superordinate dimension of negative emotionality and/or have not clearly distinguished ASB in childhood from ASB in adolescence as outcomes, the genetic and environmental contributions to associations between infant fussiness and ASB in childhood and adolescence currently are not well understood. In addition, non-genetically informed studies that have specifically examined fussiness have used analytical techniques that may not effectively rule out factors that could confound the relation between fussiness and ASB. In an effort to address these limitations, the current study uses two genetically informed analytic approaches, the comparison of siblings (Lahey and D'Onofrio 2010) and bivariate biometric models (Neale and Cardon 1992), to advance understanding of the associations between infant fussiness and ASB in childhood and late adolescence.

The first approach, the comparison of siblings within families, is an increasingly popular quasi-experimental design that can more closely approximate the causal association between temperament and ASB than is possible with conventional, between-family comparisons. The hypothesis that temperament has influence on ASB would be supported if fussier infants are more likely to develop ASB than their less fussy siblings. In contrast to between-family comparisons, sibling comparisons automatically rule out as alternative explanations for the association between fussiness and ASB all influences from any source that differs between nuclear families but are shared by siblings within nuclear families (Rodgers et al. 2000; Rutter 2007). Notable among the confounding influences controlled by sibling comparisons is passive gene-environment correlation. This is the case because parents' genes are randomly distributed among siblings, thereby eliminating any passive correlation between variations in siblings' genes and the caregiving environment (D'Onofrio and Lahey 2010).

Although sibling comparisons substantially reduce the potential for confounding, they do not rule out genetic and environmental influences that simultaneously 1) differ between siblings, 2) correlate with variations in infant temperament, and 3) correlate with ASB (Lahey and D'Onofrio 2010; Rutter 2007), and, as a result, cannot demonstrate causality. These uncontrolled influences would include variations in exposure to environmental risk factors that are correlated with sibling differences in temperament, perhaps resulting from evocative and active effects of temperament on the social environment, and variations in genetic influences on fussy temperament that constitutute genetic main effects on risk for ASB. Nevertheless, when measured selection factors that differ between siblings (e.g., maternal age at childbirth) are included as statistical covariates, it is possible to further minimize the potential for confounding. As such, sibling-comparisons can provide a more

rigorous test of the hypothesis that infant fussiness influences risk ASB in childhood and adolescence than can be achieved by comparing unrelated individuals.

The second approach, bivariate biometric modeling (Neale and Cardon 1992), allows for the partitioning of covariation between fussiness and ASB into genetic, shared environmental, and nonshared environmental sources. The estimate of covariation due to the nonshared environmental influences shares similarities with the estimate derived from the comparison of siblings, in that both indicate whether sibling differences in fussiness are associated with sibling differences in ASB. One difference between biometric modeling and sibling comparisons, however, is that biometric modeling separates *all* additive genetic influences from environmental influences (Plomin et al. 1980). As noted above, sibling comparisons cannot fully distinguish environmental influences from genetic influences that make siblings different in their ASB. Thus, an association found when comparing siblings might reflect genetic influences rather than or in addition to influences that stem solely from environmental exposures. Despite its advantages in disentangling genetic and environmental influences, valid interpretation of variance and covariance components from biometric models assumes equal similarity in environments across sibling types and no assortative mating, which if violated can cause estimates of heritability to be biased upwards and downwards, respectively (Barnes et al. 2014). Furthermore, the nonshared environmental source of covariance between temperament and ASB, like sibling-comparisons, does not automatically control confounding environmental influences that are associated with sibling differences in temperament and ASB (Turkheimer and Harden 2014).

Although individually neither sibling comparisons nor bivariate biometric analyses can control all confounding genetic and environmental influences, each design rules out and encompasses partially distinct alternative explanations, together providing a powerful test of the influence of fussy temperament on later ASB. Convergence in findings across the two approaches would provide strong evidence that fussy temperament has influence on ASB. On the other hand, divergence in findings may be informative as to the possible mechanisms underlying links between temperament and ASB. In either case, the use of both approaches to explore a common hypothesis provides a valuable opportunity to directly compare the performance of two popular quasi-experimental approaches for evaluating quasi-causal hypotheses.

Method

Participants

The National Longitudinal Survey of Youth (NLSY79) initially included a nationally representative sample of 6,111 individuals and an over-sample of 3,652 Hispanics and African Americans 14–22 years of age. Because all eligible individuals in each household were selected, multiple females from the same homes were included. Participants were assessed annually from 1979 to 1994 and biennially since then. NLSY79 response rates have consistently been at or above 90%. Sampling weights allow for estimates from analyses to be generalized to the original nationally-representative NLSY79 sample. Other studies provide additional details about the NLSY79 sample (Baker and Mott 1989).

The Children of the NLSY79 (CNLSY) includes the biological offspring of the NLSY79 women. Biennial assessments of CNLSY participants began in 1986 and are ongoing. Most mothers participated in each assessment: 95% in the initial assessment and 90% in subsequent waves (Chase-Lansdale et al. 1991). Analyses of childhood conduct problems (CPs) were based on data from 9,237 youths with mother-reported CPs from at least one assessment between ages 4-9 years (51.5% also having fussiness data), and analyses of adolescent delinquency were based on 7,034 youths who provided self-reported delinquency from at least one assessment between ages 14-17 years (54.7% also having fussiness data). A total of 6,804 youths provided data for both the child and adolescent analyses, meaning that 97% percent of the adolescent sample were included in the child analyses and 74% of the child sample were included in the adolescent analyses.

Characteristics of the NLSY79 mothers for the childhood CPs subsample and the adolescent delinquency subsample included in the present analyses are presented in Table 1. The samples were ethnically diverse. In the childhood CP sample, 18.6% of the mothers were African-American, 26.1% were Hispanic, and 55.3% were non-African-American, non-Hispanic. In the delinquency sample, 19.7% were African-American, 30.4% were Hispanic, and 49.8% were non-African-American, non-Hispanic.

The subsamples included in the present analyses were compared to those families who were excluded owing to missing data on the childhood CP or adolescent delinquency outcomes. First, the 9,237 offspring included in models predicting CP data were compared to the 2,186 not included in these models. The included offspring were born to older mothers than the offspring not included in the analyses (Ms = 25.46 and 23.64, respectively). In addition, the mothers of children included in the childhood CP models had lower intellectual abilities (Ms = 37.46 vs. 40.01), more years of education (Ms = 14.03 vs. 13.05), and were more likely to be Hispanic (18.59% vs. 12.04%). No statistically significant differences were found in terms of income at age 30, maternal delinquency, or in likelihood of being African-American.

Next, the 7,034 offspring included in models predicting adolescent delinquency were compared to the 4,389 who were not included. The offspring included in the delinquency models were born to younger mothers than the offspring not included in the analyses (Ms = 24.88 and 25.27, respectively). In addition, the mothers of children included in the delinquency models had lower intellectual abilities (Ms = 36.84 vs. 39.78), more years of education (Ms = 14.18 vs 13.27), were more likely to be African-American (30.40% vs. 19.22%), and were more likely to be Hispanic (19.74% vs. 13.12%). In contrast, no statistically significant differences were found in terms of income at age 30 or maternal delinquency.

In summary, relative to excluded participants, some evidence was found that the analyzed samples experienced greater exposure to some risks for ASB (e.g., lower maternal intellectual abilities, earlier maternal age at childbirth (CP subsample only)), lesser exposure to other risks (e.g., later maternal age at childbirth (delinquency subsample only), and no differences in exposure to other important risks (e.g., maternal delinquency). All of the risk factors included in the above comparisons were included as covariates in the population and

(when applicable) in the sibling comparison models to help account for potential sample biases. In addition, NLSY79 sample weights were included in the statistical models to further enhance the representativeness of the estimates.

Measures

Fussy temperament—Mothers rated their infants between 0-23 months of age (depending on the age of their infant when the biennial assessment occurred) on a subset of 17 items from the Infant Behavior Questionnaire (IBQ; Rothbart 1981). The items were intended to measure five temperament dimensions: fussiness (often fussy or irritable; trouble soothing infant when crying or upset; often cries or fusses compared to most babies; cries or becomes upset in response to noise), positive affect (smiles or laughs when you play with him or her; smiles or laughs when plays alone; smiles or laughs in the bath), fearfulness (cries or turns away from strangers; cries or turns away from unfamiliar dog or cat; cries when left alone in a room; cries or turns away from a doctor, dentist, or nurse), activity level (squirms and kicks during feeding; waves arms during feeding; moves around in the crib during sleep), and predictability (sleepy about the same time each evening; hungry about the same time each evening; wakes up in the same mood each morning). Predictability and activity level items were collected only from mothers rating their children between ages 0-11 months. Previously reported factor analysis of temperament items collected on infants between ages 0-11 months supported the five-factor structure just described over alternative specifications with fewer factors (Lahey et al. 2008). Confirmatory factor analysis was conducted with the current data including all offspring assessed between 0-23 months in order to confirm that the factor structure was not influenced by the addition of data from the older subset of children. Items were residualized on age of the infant (in months) at the time of assessment. The fit of the five-factor model was compared to a series of models conforming to four-factor, three-factor, two-factor, and one-factor structures (see Lahey et al. 2008 for further details regarding the factor structure of the alternative models).

Consistent with Lahey et al.'s analysis of CNLSY temperament data from 0-11 months, the five-factor model including temperament data from 0-23 months provided adequate fit to the data (Satorra-Bentler scaled χ^2=800.25, df=109, p<.001; CFI=.91; RMSEA=.03) and fit better than all models with fewer factors. Items from the fussiness scale were averaged together according to the corresponding factor and z-score standardized for use in the analyses predicting ASB outcomes. One item, cries or becomes upset in response to noise, was excluded from the fussiness scale because it had a low factor loading (standardized loading=.26) and, as noted by Lahey et al. (2008), is more consistent with the concept of sensory modulation than with irritability (Goldsmith et al. 2006). Factor loadings for the three fussiness items from a CFA conducted on just those items were .43, .69, and .81, for trouble soothing infant when crying or upset, often cries or fusses compared to most babies, and *often fussy or irritable*, respectively. Scale reliability was computed using CFA in order to provide less biased estimates relative to Cronbach's alpha, which may be biased downward if assumptions are not met (e.g., equal sensitivity of items, noncorrelated error terms; Brown 2006; Furr and Bacharach 2008). The reliability estimate (intraclass correlation equivalent) for fussiness was .69.

The NLSY79 included all individuals in the original 1979 households and the CNLSY assesses every offspring of the women in the NLSY79, resulting in a combined dataset with three levels of nesting: the NLSY79 household level, the individual women in the NLSY79 (the mothers in the current study), and the offspring of the women in the NLSY79 (the CNLSY sample). This clustering enables variation in temperament to be broken down at each level. Intra-class correlations (ICCs) divide the variation in a temperament scale at each level by the total variation in the temperament scale, providing estimates of the extent to which variability in temperament is shared with one's cousins, one's siblings, or is unique to individuals within a nuclear family. ICCs were calculated using a multilevel model to estimate random intercepts at the sibling and cousin levels. Previous behavioral genetic analysis of IBQ temperament scales have reported ICCs ranging from .28 to .61 between dyzygotic twins (Goldsmith et al. 2006). In the current study, it was found that 12% of the variance in the fussiness scale was shared among cousins in extended families. In addition, an additional 17% was shared by siblings within nuclear families. Summing the estimates, siblings shared a total of 29% of the variance in fussiness, falling within the range reported by Goldsmith et al. The remaining 71% of the variance in fussiness was found to be unique to individuals within a nuclear family (i.e., was not also shared with one's siblings or cousins). The considerable degree of variation found between siblings within families allowed for the use of sibling comparisons for testing effects of fussy temperament on ASB.

The clustering of CNLSY participants within families also allowed for biometric modeling of the covariance between fussiness and ASB. Each unique sibling pair within a family was included in these analyses, providing varying degrees of genetic relatedness. Different categories of sibling pairs (e.g., full vs. half siblings) were explicitly distinguished as part of NLSY79 assessments beginning in 2006. In addition, the genetic relatedness of siblings who have not participated since before 2006 has also been established on the basis of responses to questions that implicitly identify kinship links, such as yearly living status in relation to biological mother and father, and validated using data on highly heritable characteristics such as height (Rodgers et al. 1999).

Mother-reported childhood conduct problems—Mothers reported on their children's adjustment between ages 4-9 via the Behavior Problem Index (BPI; Peterson and Zill 1986) at each assessment. Mothers reported on whether each of the items were not true (0), somewhat or sometimes true (1), or very true or often true (2) for the individual child under consideration. The BPI includes 13 items selected from the externalizing scale of the Child Behavior Checklist (Achenbach 1978). Three factors have been identified from among these items (D'Onofrio et al. 2008): conduct problems (CPs), attention-hyperactivity-impulsivity problems, and oppositional problems. The items corresponding to the CP factor were as follows: cheats or lies; breaks things on purpose or deliberately destroys his/her own or another's things; disobedient at home; disobedient at school; has trouble getting along with teachers; does not feel sorry after misbehaving; bullies other children. CP scores across 4-9 years were calculated by first summing the CP items at each assessed year, and then calculating the average of the summed CP scales across ages 4-9. The resulting measure of childhood CPs was z-score transformed and modeled as a continuously distributed outcome. Previous analyses of CNLSY data have shown stability in levels of CPs across childhood

and have shown that the measure of childhood CPs is valid in the sense of predicting adolescent delinquency and criminal convictions (Lahey et al. 2006). Cronbach's alpha for the CP scale across ages 4-9 years ranged from .67 to .76, with a median value of .72.

Youth-reported delinquency—Offspring between ages 14 and 17 were administered 7 items from the self-reported delinquency (SRD) questionnaire in each assessment. The questionnaire included the following items scored 0 = no; 1 = yes: hurt someone bad enough to need bandages or a doctor; lied to parent about something important; taken something from a store without paying for it; intentionally damaged or destroyed property that didn't belong to you; had to bring your parent(s) to school because of something you did wrong; skipped a day of school without permission; and ran away from home. The 7 SRD delinquency items correlate highly with more serious forms of delinquency from the SRD, and the measure has been shown to predict later criminal convictions in the CNLSY sample with no evidence of sex differences in criterion validity (Lahey et al. 2008). Average levels of self-reported delinquency from ages 14-17 were highly negatively skewed with a large percentage (26%) of participants having a score of zero. Thus, they were rounded to the nearest whole number for use as count outcomes in the models. Cronbach's alpha for ages 14-17 ranged from .57 to .67, with a median value of .65.

Family and child-specific covariates—Sibling comparison analyses included several maternal and child-specific covariates that had the potential to confound the effects of child temperament on CPs and delinquency due to their possible correlations with temperament and the outcome variables. In 1980 the mothers completed the Armed Services Vocational Aptitude Battery of intellectual assessments, which provided estimates of mothers' intellectual abilities. Mothers also indicated their highest grade completed by 2010. Also, when the mothers were 15–22 years old, their participation in 12 delinquent behaviors was assessed using items from the Self-Reported Delinquency (SRD) interview (Elliott and Huizinga 1983). Other family-level covariates were mothers' race-ethnicity, coded as Hispanic, African American, or non-African American/non-Hispanic, and family income at mother age 30 (log-transformed). Several child-specific variables were also included as covariates in the models, including offspring sex, birth order, and maternal age at birth. All covariates were collected from NLSY79 mothers. Correlations between fussy temperament, covariates, and the CP and delinquency outcome variables are presented in Table 2.

Statistical Analyses

Population and sibling-comparison analyses—All population and sibling-comparison models were analyzed using multilevel generalized linear models in Mplus 6.12 (Muthén and Muthén 1998-2010) to accommodate the nested structure of the data. Childhood CPs and adolescent delinquency were tested in separate models. Sex was included as a covariate in the analyses, but was not tested as a moderator given the limitations in statistical power in testing interactions in sibling-comparison and biometric models of sibling data.

Population-level models estimated the predictive association between infant fussiness and later ASB (either childhood CPs or adolescent delinquency) after controlling for measured

covariates. Sibling-comparison models estimated the adjusted association between fussiness and ASB after controlling for all measured and unmeasured confounded environmental and genetic factors that make siblings similar (Lahey and D'Onofrio 2010; Rodgers et al. 2000), as well as several measured covariates that differ between siblings. Sibling-comparisons were calculated by taking the difference between the individual fussiness score of each sibling from the average score for all siblings in the same family. This statistical approach provides correct within-family estimates (Neuhaus and McCulloch 2006) and yields the same parameter estimates as less flexible fixed-effect models (Greene 2003).

Mother-reported childhood CPs were treated as a normally-distributed outcome with estimation of standard errors that are robust to violations of normality (Satorra and Bentler 2001), whereas adolescent-reported delinquency was analyzed as count outcomes. Full information maximum-likelihood estimation was used in all analyses to accommodate missing data on the temperament variables and the covariates (but not the outcome variables), and sampling weights were applied so that estimates could be generalized to the original nationally-representative NLSY79 sample.

Bivariate biometric models—In order to address the limitation that sibling comparisons only partially account for genetic influences, we also used bivariate Cholesky decomposition models (Neale and Cardon 1992) to estimate the genetic and environmental influences that childhood CPs (or adolescent delinquency) share in common with fussiness, and those influences that are unique to childhood CPs (or adolescent delinquency). These models provided separate estimates of covariance between fussiness and ASB attributable to additive genetic influences (A), shared environmental influences (C), and nonshared environmental influences and measurement error (E), as well as residual ACE influences unique to the outcome variable. The full bivariate biometric model is shown in Figure 1.

The bivariate biometric modeling was conducted in Mplus version 6.12 software (Muthén and Muthén 1998-2010). The within-pair genetic correlation for fussiness (rA_F) and outcome variable (rA_O) was fixed at 0.5 in DZ pairs/full siblings and 0.25 in maternal half siblings, as DZ twins and full siblings share an average of 50% of their segregating additive genetic influences, whereas maternal half siblings share an average of 25%. Within-pair correlations for shared environmental influences (rC_F and rA_O) were fixed at 1.0 in all sibling pairs. Nonshared environmental influences (rE_F and rE_O) were uncorrelated in all sibling pairs. Residual variation for the observed fussiness and outcome variables were fixed at 0, and intercepts for each of these variables were equated across siblings 1 and 2 within a pair. We fixed the variance of all latent ACE factors at 1.0 and obtained path estimates from the ACE factors to each observed variable (e.g., a_F, c_F, and e_F). The proportions of variance due to A, C, and E are obtained by squaring the estimated values of each path estimate. Total variance in fussiness is equal to 1.0 and is the sum of a_F^2, c_F^2, and e_F^2. The proportion of variance in the outcome shared with fussiness is the sum of a a_{cov}^2, c_{cov}^2, and e_{cov}^2. Residual variance in the outcome is sum of a_O^2, c_O^2, and e_O^2. The total sum of the residual variance in the outcome and shared variance between fussiness and the outcome is equal to 1.0.

A significant e_{cov} estimate (i.e., the amount of shared variance due to nonshared environment) would be consistent with the hypothesis that fussiness has a causal influence on the antisocial behavior outcome through some form of environmental process (Turkheimer and Harden, 2014). Nonetheless, a significant e_{cov} estimate would also be consistent with the alternative hypothesis that environmental influences that are not shared by siblings (e.g., variations in stress due to fluctuations in family income over time) influence both infant fussiness and antisocial behavior, creating the correlation between them. In contrast, significant a_{cov} and/or c_{cov} estimates, without a significant e_{cov} estimate, would indicate that the shared variance between fussiness and the outcome is due to common genetic and/or shared environmental influences (e.g., caregiving behaviors that are consistent over time) that make related individuals similar. Separate bivariate models estimated the influences on shared variance between (A) fussiness and childhood CPs and (B) fussiness and adolescent delinquency.

Results

Population-level and Sibling-comparison Analyses

Table 3 presents the results of the population and sibling-comparison models exploring the association between infant fussiness and ASB in childhood and adolescence. We first examined associations between fussiness and childhood CPs. In population-level analyses that controlled for measured child and family-level confounds, a 1 SD unit increase in fussiness predicted a 0.113 SD unit increase in childhood CPs ($b = 0.113$, $p < 0.001$). In addition, among the covariates included in the model, maternal age at childbirth ($b = -0.021$) female sex ($b = -0.283$), maternal delinquency ($b = 0.072$), African-American ethnicity ($b = 0.071$), and family income ($b = -0.116$) had statistically significant associations (ps <.05) with childhood CPs.

In sibling-comparison analyses that controlled for measured child-level and unmeasured family-level covariates, the magnitude of the association between fussiness and childhood CPs was attenuated but still statistically significant, with a 1 SD unit increase in fussiness predicting a 0.052 SD unit increase in childhood CPs ($b = 0.052$, $p < 0.05$). Among the child-level covariates included in the model, maternal age at childbirth ($b = -0.026$), female sex ($b = -0.282$), and birth order ($b = .029$) had statistically significant associations with childhood CPs.

We next examined associations between infant fussiness and adolescent delinquency. In population-level analyses, a 1 SD unit increase in fussiness predicted a 12% increase in adolescent delinquency ($b = .112$, $p = .008$). In addition, maternal age at childbirth ($b = -0.037$) female sex ($b = -0.24$), birth order ($b = 0.056$), maternal delinquency ($b = 0.053$), and Hispanic ethnicity ($b = 0.097$) had statistically significant associations (ps <.05) with adolescent delinquency. In contrast, in the sibling-comparison analysis, the association between fussiness and adolescent delinquency was not statistically significant ($b = -0.01.$, $p = .922$). Among the child-level covariates, maternal age at childbirth ($b = -0.041$), female sex ($b = -0.240$), and birth order ($b = .071$) had statistically significant associations with adolescent delinquency.

Biometric Models

Bivariate Cholesky models were used to estimate genetic, shared environmental, and nonshared environmental influences on shared variance between infant fussiness and ASB in childhood and adolescence. Table 4 presents estimates from these models.

In the model of fussiness and childhood CPs, total variance of fussiness was estimated as a_F^2=0.225, c_F^2=0.212, and e_F^2=0.564. The variance in childhood CPs shared with fussiness was due to a_{cov}^2=0.073, c_{cov}^2=0.023, and e_{cov}^2=0.000, summing to a 9.6% overlap, which was primarily due to genetic variation as well as shared environmental influences, but not nonshared environmental influences. Residual variation in childhood CPs was estimated as a_O^2=0.590, c_O^2=0.000, and e_O^2=0.314. In other words, 69% of the variance in childhood CPs was due to familial influences ($a_{cov}^2 + c_{cov}^2 + a_O^2 + c_O^2$)/1.0, most of which was due to genetic variation, and approximately 10% of this variance was shared with fussiness.

In the model of fussiness and adolescent delinquency, total variance of fussiness was estimated as a_F^2=0.276, c_F^2=0.197, and e_F^2=0.527. Note that estimated influences on variance of fussiness differed slightly between the two models due to slight differences in the sample of pairs included in each model, although the pattern of influences was similar. The variance in adolescent delinquency shared with fussiness was due to a_{cov}^2=0.032, c_{cov}^2=0.002, and e_{cov}^2=0.006, summing to a 4.0% overlap, which was primarily due to genetic variation; however, none of the estimated shared variance paths were statistically significant. Residual variation in adolescent delinquency was estimated as a_O^2=0.336, c_O^2=0.000, and e_O^2=0.624. In other words, 37% of the variance in adolescent delinquency was due to familial influences ($a_{cov}^2 + c_{cov}^2 + a_O^2 + c_O^2$)/1, most of which was due to genetic variation, and 4% of this variance was shared with fussiness.

In summary, nonshared environmental influences were not found to account for a significant portion of the shared variance between fussiness and either childhood CPs or adolescent delinquency.

Discussion

The current study used two genetically-informative designs to understand the predictive association between fussiness in infancy and ASB in childhood and late adolescence. Results from a sibling-comparison analysis of childhood CPs suggested that part of the association between fussiness and childhood CPs could not be explained by confounding family influences and might indicate that fussiness may play a causal environmental role in the transactional origins of childhood CPs. In contrast, bivariate biometric modeling failed to show evidence that nonshared environmental influences contributed to the covariation between infant fussiness and childhood CPs.

Taken together, these findings suggest that the association between infant fussiness and childhood CPs can be explained predominantly by environmental and genetic influences shared by family members. One possible explanation for differences in findings between sibling-comparison and the biometric model is that childhood CPs are a direct manifestation of the same temperamental extreme as fussiness, consistent with the spectrum model of

temperament-psychopathology association (Clark et al. 1994) and passive gene-environment correlation. Another possibility is that ASB is associated with fussy temperament as a result of intervening environmental exposures that are evoked (e.g., coercive parent-child interaction; Patterson, Reid, and Dishion 1992) or selected (e.g., deviant peer groups; Snyder et al. 2010) by children with fussy temperaments, consistent with evocative and active gene-environment correlations.

Analyses of adolescent delinquency were also consistent with family level genetic and environmental confounding of the effect of fussiness on ASB. Although results from a population-level analysis showed a predictive association between fussiness and adolescent delinquency, results from a sibling-comparison analysis that controlled both measured and unmeasured confounds showed no association of infant fussiness with adolescent delinquency. In addition, bivariate biometric modeling did not show evidence for nonshared environmental influences on the covariation between infant fussiness and adolescent delinquency. These findings suggest that the association between infant fussiness and adolescent delinquency may be explained by confounding familial influences.

Findings from the bivariate Cholesky models were not wholly consistent with previous research. Whereas the current study found evidence that genetic and/or shared environmental influences, but not nonshared environmental influences, contributed to the covariation between infant fussiness and later ASB, Singh and Waldman's (2010) study on the etiology of the association between the broader construct of negative emotionality and ASB found evidence for genetic (88%) and nonshared environmental influences (12%), but not shared environmental influences. It is possible that differences in findings for the bivariate models may be explained by differences in aspects of temperament under study (negative emotionality in previous studies versus fussiness in the present study), differences in the age range of temperament and ASB measures being considered, and differences in representativeness of the samples. It should also be noted that biometric studies of sibling data are limited in their ability to precisely separate shared environmental and genetic influences, which could also contribute to inconsistencies in findings. For example, it is possible that full siblings were more likely to live together or lived together longer than half siblings. However, to the extent that the environment would more similar for full siblings than for half siblings, the estimate for additive genetic influences would be biased upward and the estimate for shared environmental influences biased downward (Barnes et al. 2014).

The current study had several strengths that serve to advance understanding of the association between infant fussiness and ASB in childhood and adolescence. The use of a large, diverse sample from the nationally-representative NLSY79 and CNLSY enhances the generalizability of the current findings. In addition, the use of multiple prospective, genetically informative, and quasi-experimental methods enhanced our ability relative to previous studies to rigorously evaluate the hypothesis that fussiness is associated with ASB when confounding influences are controlled. Indeed, the current study is the first to use a sibling-comparison approach to help rule out the confounding influence of family background characteristics on links between temperament and ASB. Furthermore, the use of bivariate biometric modeling provided an opportunity to compare results from quasi-experimental methods that are frequently used to evaluate causal hypotheses (D'Onofrio et

al. 2013), but rarely in the same study. The results from the present analyses, in which an effect was found in support of an environmentally mediated effect of temperament in the sibling model but not in the biometric model, suggests that the inability of sibling comparisons to fully control genetic influences puts the approach in some cases at a disadvantage relative to biometric models for testing quasi-causal hypotheses.

The present study has several limitations. First, a large number of CNLSY participants were not included in the analyses owing to missing data on the outcome variables. Second, child temperament was assessed through maternal-report, which has a number of advantages and disadvantages relative to observational measures of temperament. A major advantage of maternal-report is that mothers observe their children over a much broader range of situations and longer periods of time than is possible with outside observers, allowing mothers to consider characteristics of their infants' emotional and behavioral patterns that may be difficult to elicit reliably in a laboratory setting or during a brief naturalistic observation (Bates 1989). An important disadvantage of maternal report, however, is that mothers may be more susceptible to a number of potentially biasing influences, such as social-desirability and contrast effects when rating multiple offspring, which in some cases are less likely to influence ratings made by outside observers. Third, child temperament and CPs were both reported by mothers, leading to the possibility that associations may be biased due to shared rater variance, and may explain why effects attributable to the shared environment were larger when predicting childhood CPs than when predicting adolescent delinquency. Fourth, the measurement of temperament in the present study was also limited by its brevity. Because the NLSY79 includes a very large, representative sample and has many aims beyond measuring child characteristics and family processes, only a select number items from the IBQ (Rothbart 1981) could be included. It is possible that the potential for unique environmental contributions to the association between temperament and ASB would have been greater if a broader set of temperament items had been used to measure fussiness.

In summary, the present study failed to find consistent evidence that the association between temperamental fussiness/irritability in the first two years of life and ASB in later childhood and adolescence is environmentally mediated, suggesting instead that family background characteristics account for the association. Nevertheless, further research is needed before it will be possible to reach stronger conclusions about the nature of the association between fussiness and ASB, as we have yet to clearly understand the processes underlying these genetically-influenced links.

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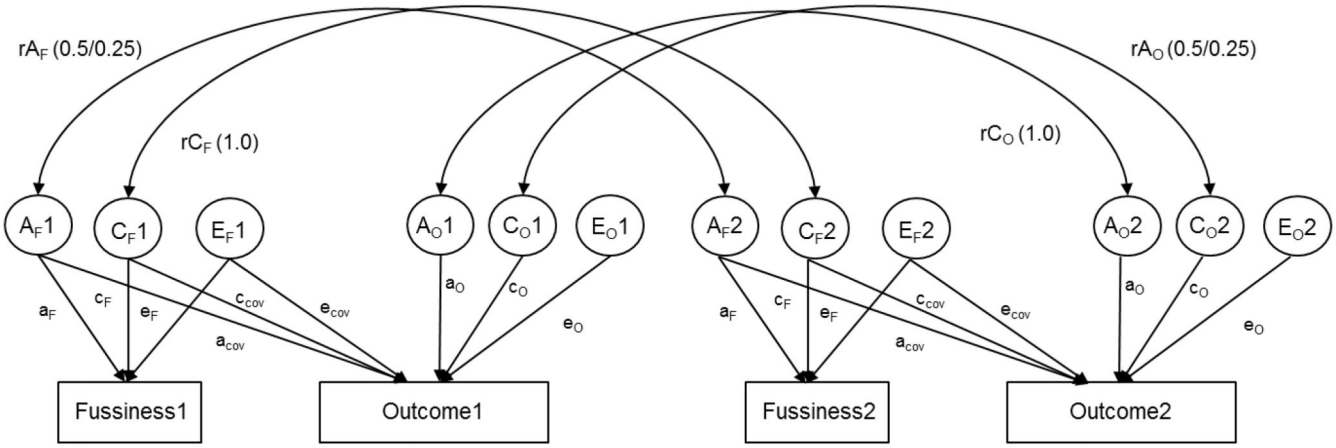

Fig. 1.

Path diagram for bivariate Cholesky decomposition of the covariation between fussiness and antisocial behavior outcome. Subscripts $_F$ and $_O$ = fussiness and outcome variable, respectively. Subscripts $_1$ and $_2$ indicate sibling 1 and sibling 2. A_F, C_F, and E_F = latent additive genetic, shared environmental, and nonshared environmental factors for fussiness; A_O, C_O, and E_O = latent additive genetic, shared environmental, and nonshared environmental factors for outcome variable. rA_F and $rA_{O\,=}$ correlation of additive genetic factors within sibling pair for fussiness and outcome, fixed at 0.5 in full siblings and 0.25 in half siblings. rC_F and $rC_{O\,=}$ correlation of shared environmental factors within sibling pair for fussiness and outcome, fixed at 1.0 in all sibling pairs. a_F, c_F, and e_F = path estimates from A, C, and E latent factors for fussiness to observed fussiness variable. a_O, c_O, and e_O = path estimates from A, C, and E latent factors for outcome variable to observed outcome variable. a_{cov}, c_{cov}, and e_{cov} = path estimates from A, C, and E latent factors for fussiness to observed outcome variable.

Table 1

Demographic Characteristics of Mothers from the Two Subsets of the National Longitudinal Survey of Youth Sample used in the Separate Analyses of Childhood Conduct Problem and Adolescent Delinquency Outcomes in their Offspring

Maternal Variables	Conduct Problems Subsample			Delinquency Subsample		
	N(mothers)	M	SD	N(mothers)	M	SD
Age at first birth	3830	23.18	5.38	2928	22.78	4.91
Intellectual ability	3674	37.46	27.04	2824	36.84	27.24
Years of education	3830	14.03	2.61	2930	14.45	3.94
Income at 30 years old	3372	$33897	$75898	2703	$32171	$67981
Delinquency (1980)	3659	.001	1.48	2801	−.02	1.43

Note. Delinquency is the number of the mother's delinquent activities during the previous year regressed on mother's age when she completed the Self-Reported Delinquency Interview. Intellectual ability is percentile scores from the AFQT.

Table 2

Bivariate Correlations of Offspring Fussy Temperament during Infancy with Outcome Variables and Continuous Covariates

	N for correlation	Correlation with infant fussiness
Outcome variables		
Childhood CPs	4754	.19***
Adolescent Delinquency	3847	.07***
Covariates		
Birth Order	5329	.08***
Mother's Age at Birth	5329	−.09***
Mother's Delinquency[1]	3059	.04*
Mother's Highest Grade[1]	3199	−.10***
Mother's Estimated IQ[1]	3080	−.23***
Family Income (at Mother Age 30)[1]	2778	−.17***

[1] Sibling-average scores were used for these correlations, as these covariates were invariant across individual siblings.

* p<.05.

** p<.01.

*** p<.001.

Table 3

Unstandardized Regression Coefficients from Population and Sibling-Comparison Analyses Predicting Child Conduct Problems and Adolescent Delinquency

	Outcome: Child Conduct Problems				Outcome: Adolescent Delinquency			
	Population estimates		Sibling-comparison estimates		Population estimates		Sibling-comparison estimates	
Predictor Variables	**b**	**SE**	**b**	**SE**	**b** [1]	**SE**	**b** [1]	**SE**
Main Effects								
Fussiness	.113***	.019	.052*	.024	.112**	.042	−.010	.101
Sex (male=0, female=1)	−.283***	.020	−.282***	.020	−.240***	.031	−.240***	.030
Covariates								
Birth Order	.011	.014	.029*	.013	.056***	.016	.071***	.015
Maternal Age at Childbirth	−.021***	.003	−.026***	.003	−.037***	.004	−.041***	.004
Hispanic (EA=0, hisp= 1)	−.003	.038			.097*	.044		
Afr. Amer. (EA=0, AA = 1)	.071*	.036			.012	.038		
Mother's IQ	.000	.001			−.001	.001		
Mother's Highest Grade	−.007	.006			−.006	.008		
Mother's Income at Age 30	−.116***	.017			−.006	.007		
Mother's Delinquency	.072***	.010			.053***	.011		

Note. Sample size was 9,237 for prediction of conduct problems and 7,034 for delinquency;

[1] Coefficients expressed as logits.

* p<.05.

** p<.01.

*** p<.001.

Table 4

Bivariate Cholesky Models Representing Association of Fussiness with (a) Childhood Conduct Problems and (b) Adolescent Delinquency

	Fussiness			Childhood Conduct Problems					
	Total Variance			Shared Variance			Residual Variance		
		Est.	Est.^{2}		Est.	Est.^{2}		Est.	Est.^{2}
(A)	a_F	0.474**	0.225	a_{cov}	0.271*	0.073	a_{CCP}	0.768***	0.590
	c_F	0.460***	0.212	c_{cov}	0.151**	0.023	c_{CCP}	−0.001*	0.000
	e_F	0.751***	0.564	e_{cov}	−0.006	0.000	e_{CCP}	0.560***	0.314
	Sum		1.000			0.096			0.904
	Fussiness			Adolescent Delinquency					
	Total Variance			Shared Variance			Residual Variance		
		Est.	Est.^{2}		Est.	Est.^{2}		Est.	Est.^{2}
(B)	a_F	0.525**	0.276	a_{cov}	0.178	0.032	a_{AD}	0.580***	0.336
	c_F	0.444***	0.197	c_{cov}	0.040	0.002	c_{AD}	0.000***	0.000
	e_F	0.726***	0.527	e_{cov}	−0.079	0.006	e_{AD}	0.790***	0.624
	Sum		1.000			0.040			0.960

Est., obtained path estimate; Est.^{2}, derived square of path estimate; a, additive genetic influence; c, shared environmental influence; e, nonshared environmental influence; F, fussiness; cov, covariation; CCP, childhood conduct problems; AD, adolescent delinquency.

Note. Sample sizes for models were (A) 6,689 full-sibling pairs and 3,329 half-sibling pairs and (B) 6,233 full-sibling pairs and 3,122 half-sibling pairs.

*p<.05.

**p<.01.

***p<.001.