Family Structure Instability, Genetic Sensitivity, and Child Well-Being[1]

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The association between family structure instability and children's life chances is well documented, with children reared in stable, two-parent families experiencing more favorable outcomes than children in other family arrangements. This study examines father household entrances and exits, distinguishing between the entrance of a biological father and a social father and testing for interactions between family structure instability and children's age, gender, and genetic characteristics. Using data from the Fragile Families and Child Wellbeing Study and focusing on changes in family structure by age (years 0–9), the authors show that father exits are associated with increases in children's antisocial behavior, a strong predictor of health and well-being in adulthood. The pattern for father entrances is more complicated, with entrances for the biological father being associated with lower antisocial behavior among boys and social father entrances being associated with higher antisocial behavior. Child's age does not moderate the association; however, genetic information in the models sharpens the findings substantially.

INTRODUCTION

Children's exposure to family structure instability—defined as having a parent or parent figure move into or out of the household—has increased

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dramatically during the past few decades because of high rates of divorce and rising rates of cohabitation and nonmarital childbearing (Cherlin 2005). Over half of U.S. children born to married or cohabiting parents in the late 1990s are expected to experience the exit of a biological parent (usually a father) from the household before age 18 (Bumpass and Lu 2000). Similarly, more than two-thirds of children born to unmarried, noncoresident parents are expected to experience the entrance of a biological or social father into the household (Bzostek, McLanahan, and Carlson 2012). High levels of family structure instability are of interest to sociologists who care about the institution of the family. They also are of interest to those who care about inequality and mobility. Children from disadvantaged backgrounds are much more likely than other children to experience family structure instability, suggesting that recent trends may be lowering the future mobility of children with low socioeconomic status (SES) born in the past few decades (McLanahan 2004).

A large literature examines what happens to children when a biological father exits the household. This literature, which focuses primarily on divorce, finds that father exits are associated with a host of negative outcomes throughout the life course, including lower cognitive tests scores and more conduct problems in early and middle childhood; lower rates of high school completion and higher rates of delinquency and unintended pregnancy in adolescence; and more mental health problems, higher marital instability, and lower earnings in adulthood (McLanahan, Tach, and Schneider 2013). Although some of the association between divorce and poor child outcomes is due to factors that predate family change, a recent review of the literature suggests that divorce itself plays a causal role in shaping child outcomes, especially antisocial behaviors such as aggression and rule breaking (McLanahan et al. 2013).

A second literature examines what happens when a social father moves into the household, through either marriage or the formation of a cohabiting union. Theoretically, the impact of a father's entrance into the household is ambiguous. On the one hand, the entrance of a second adult should increase the amount of parental time and economic resources available to the child; on the other hand, an entrance may disrupt household routines and create tension in parent-parent and parent-child relationships (Hetherington et al. 1992). In general, the empirical literature finds that children in social father families do about as well as children in single-

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parent families, suggesting that the gains in economic resources are offset by other factors.

In addition to documenting a link between family structure change and a wide range of outcomes in childhood and adulthood, the literature points to a good deal of heterogeneity in children's responses to family structure change. There is evidence, for example, that the negative outcomes associated with family structure instability are more pronounced for young children as compared with older children (Sigle-Rushton and McLanahan 2004) and for boys as compared with girls (Cooper et al. 2011). In this article, we test for differences by age and gender, and we also examine a new source of potential heterogeneity in children's response to family instability: genetic sensitivity. Studies based on animals as well as humans find that genes connected to the dopaminergic and serotonergic systems play an important role in shaping individuals' responses to their environments, with some genotypes showing much more negative responses than others to difficult environments (Bennett et al. 2002; Karg et al. 2011; Klauke et al. 2012). There is also evidence of "differential genetic sensitivity," in which genotypes showing more negative responses to difficult environments also show more positive responses to positive environments (Ellis and Boyce 2008; Belsky and Pluess 2009; Ellis et al. 2011).

We use data from the Fragile Families and Child Wellbeing Study to examine whether changes in family structure are associated with increases in children's antisocial behaviors (aggression and rule breaking) and whether these associations are moderated by the type of change (exit or entrance), father's biological status, child's gender, and age at exposure. We also examine whether children with certain gene variants respond more strongly to changes in the family environment than other children. Antisocial behaviors in childhood, such as aggression and rule breaking, are associated with delinquency, dropping out of high school, and childbearing in adolescence and with low earnings, marital instability, and criminal activity in adulthood. Indeed, Nobel Prize winner James Heckman argues that the improvements in adult health and labor market outcomes among low-income children who participated in high-quality preschool programs are due in large part to reductions in childhood aggression and rule-breaking behavior (Heckman, Pinto, and Savelyev 2013).

BACKGROUND

Family Structure Instability and Children's Antisocial Behavior

A large body of research finds that children who grow up in stable, two-parent families fare better across a wide range of outcomes than children who grow up in unstable families (for reviews of this literature, see Seltzer [1994], Amato [2001], Sigle-Rushton and McLanahan [2004], and McLana-

han et al. [2013]). The link between family structure instability and offspring well-being is especially pronounced for outcomes involving social adjustment or conduct problems, such as rule breaking and aggression in childhood; delinquency, truancy, and early pregnancy in adolescence; and mental health problems and family instability in adulthood (Sigle-Rushton and McLanahan 2004; Waldfogel, Craigie, and Brooks-Gunn 2010). Whereas the early literature on family instability focused primarily on divorce and remarriage, more recent studies have focused on entrances into and exits from cohabiting unions as well as multiple changes in mothers' partnerships. These studies find that each partnership transition is associated with an increase in a child's problem behaviors, even after controlling for factors that affect selection into instability (Wu and Martinson 1993; Wu and Thomson 2001; Cavanagh and Huston 2006; Osborne and McLanahan 2007; Cavanagh, Crissey, and Raley 2008; Goodnight et al. 2013).

Although the exact pathways for these associations are still being debated, most researchers agree that the loss of economic resources, disruptions in family routines, and the loss of parental social capital are important mechanisms. With respect to economic resources, children who live with two parents have access to more resources in terms of parental time and money. Simple arithmetic tells us that, on average, the loss of a parent leads to a decline in household income. Economic theory also posits that two-parent households are more productive than one-parent households because of specialization (Becker 1974). These ideas are supported by a large literature showing that divorce is associated with a substantial loss of income for mothers and children (Holden and Smock 1991).

In contrast to the resource model, the household disruption model argues that change per se is hard for adults and children because it creates uncertainty and requires adjustment to new situations (Hetherington et al. 1992). In the case of divorce and remarriage, changes in family composition are expected to lead to disruptions in family routines, which may lead to less maternal involvement, less interaction among family members, and lower-quality interactions (Hetherington, Cox, and Cox 1985). Empirical studies provide considerable support for the argument that divorce and remarriage are associated with disruptions in family routines. Most recently, Beck et al. (2010) find evidence that coresidential relationship transitions—including both entrances and exits—are associated with significantly higher rates of maternal parenting stress and harsh discipline and lower-quality mother-child relationships.

Finally, sociological theory tells us that households composed of two biological parents who trust one another and are committed to one another and to the child generate more parental social capital than households composed of one biological parent or a biological parent and stepparent (Coleman 1988). Parents who live together are in a better position to coparent their

child (e.g., communicate, monitor behavior) than parents who live apart; parent-child relationships are also expected to be of higher quality when parents live with the child. Again, these ideas are consistent with empirical studies showing that divorce reduces parental monitoring and the amount of time and money fathers invest in their child while remarriage has mixed effects (McLanahan and Sandefur 1994).

All the models described above suggest that the exit of a biological father from the household should increase children's antisocial behavior. Of course, parents' decision to separate is not a random event, and the exit of a father may be a marker for a family that is not functioning well. Economic hardship or parental conflict or low father parenting quality may lead to a divorce and may also affect child well-being. In cases such as this the exit of the father may actually improve the home environment and increase child well-being (Amato, Loomis, and Booth 1995; Jaffe et al. 2003).

Whereas theory is consistent with respect to the exit of a father from the household, it is ambiguous with respect to an entrance, with the resource model predicting an improvement in child well-being, the disruption model predicting a decline in well-being, and the parental social capital model predicting mixed effects. An important limitation of the literature on family structure transitions is that studies of father exits almost always involve the exit of a biological father, whereas studies of father entrances almost always involve the entrance of a nonbiological or "social" father. Recent increases in nonmarital childbearing have made it possible to compare these two types of entrances. Whereas no study to date has distinguished between biological father entrances and social father entrances, a recent paper by Osborne, Berger, and Magnuson (2012) finds that father entrances during the first year following a nonmarital birth are positively associated with child well-being, whereas father entrances later in childhood show a negative association. Although these authors do not distinguish between entrances by biological fathers and entrances by social fathers, we would expect biological father entrances to be more common in the first year following a birth and social father entrances to be more common in later years. In the analyses that follow, we distinguish between biological fathers' entrances and social fathers' entrances.

Interactions by Timing of Event, Gender, and Genes

Life course theory argues that the impact of life events depends on the developmental stage and social context within which they occur. According to developmental theory, transitions that occur in early childhood should be more consequential than transitions that occur later in childhood or adolescence. Young children are less able to psychologically process family events and have fewer sources of nonfamily support (Hetherington,

Camara, and Featherman 1983). Early transitions also increase the risk that a child will experience additional transitions, resulting in the accumulation of disadvantage (Cavanagh and Huston 2008). Finally, negative experiences in early childhood may alter children's behavior in ways that create a negative feedback loop, reducing parents' subsequent investments (Heckman 2006). The empirical literature is largely consistent with the argument that early family transitions are more consequential than later transitions, although transitions during adolescence are also associated with poor outcomes.

Gender may also moderate the association between family structure instability and child well-being. Although boys and girls should have similar levels of exposure to family instability, there is some evidence that boys are more negatively affected than girls (Hetherington et al. 1985; Demo and Acock 1988; Biller 1993; Entwisle, Alexander, and Olson 1997; Cavanagh et al. 2008). One reason for expecting boys to respond more negatively is that the loss of a male role model may be more important for boys' identity (Allison and Furstenberg 1989). Also, postdivorce mother-son relationships are significantly worse than comparable mother-daughter relationships (Hetherington et al. 1985). There is also evidence that boys are more sensitive than girls to a variety of changes that often accompany family changes, such as parental conflict, loss of economic resources, and residential mobility (Davies and Lindsay 2001; Kling, Ludwig, and Katz 2005).

Finally, there are good reasons to expect the association between family instability and child outcomes to vary by child's genetic makeup. The literature on genetic influences on antisocial behavior is well established (Moffitt 2005). For many years this research relied on twin and adoption samples and focused on the main effects of genes, suggesting that between 45% and 55% of the variance in antisocial behaviors was due to additive genetic factors. More recently, as a result of the availability of molecular biology markers (i.e., measured genes), researchers have begun to examine how genetic characteristics may alter people's responses to their social environments. Most of this research has centered on the role of several neurotransmitter systems, the most prominent of which are dopamine and serotonin. Dopamine is a neurotransmitter—a chemical that transmits signals in between the nerve cells (neurons) of the brain—that helps regulate thought, movement, attention, motivation, and learning (Ungless, Magill, and Bolam 2004; Brischoux et al. 2009). Individuals with chronically high levels of dopamine typically remain in a heightened sense of alert, which may result in feelings of irritability, paranoia, and antisocial behavior (Zald et al. 2008). Serotonin is a neurotransmitter that helps to regulate the cognitive functions of memory, mood, and learning and is most often associated with internalizing behaviors, such as depression, anxiety,

and being withdrawn (Uher and McGuffin 2010). The serotonergic system is hypothesized to work on antisocial behaviors by inhibiting social actions and thereby lowering this type of more aggressive and rule-breaking behavior (Fox et al. 2005).

More importantly for this article, studies of human molecular genetics and social environment interplay have increased dramatically during the past decade. Most of these studies rely on the classic diathesis-stress model, which treats genetic variations and environments as being either "risky" or "protective" and argues that people with risky genes respond more negatively than their peers to difficult environments (Belsky and Pluess 2009). More recently, researchers have proposed a "genetic plasticity" or "biological susceptibility" model, which posits that some genotypes are highly susceptible to environmental influences whereas others are not (Boyce and Ellis 2005; Ellis and Boyce 2008; Belsky et al. 2009; Belsky and Pluess 2009; Mitchell et al. 2013). According to this model, those with more sensitive genes have more negative outcomes than others when the environment is "unfavorable" and more positive outcomes than others when the environment is "favorable" (Mitchell et al. 2013). This phenomenon is often referred to as the "orchid-dandelion hypothesis," with orchids referring to those with more sensitive genes and dandelions referring to those with less sensitive genes.

In the current study we focus on three markers of the dopamine system—the Taq1a polymorphism of the dopamine receptor 2 gene (DRD2, 11q23, rs1800497),[2] the Val154Met polymorphism of the Catechol-O-methyltransferase gene (COMT, 22q11.21, rs4680), and the 48bp VNTR in the third exon of the dopamine receptor 4 gene (DRD4, 11p15.5)—and two markers of the serotonin transporter gene (5-HTT, SLC6A, 17q11.2): 5-HTTLPR and STin2. The dopamine markers, COMT and DRD2, and DRD4 have been strongly tied to antisocial behavior (Benjamin, Ebstein, and Belmaker 2002; Miczek et al. 2002; Schmidt et al. 2002; De Almeida et al. 2005; Nikolova et al 2011). More importantly, all three markers have shown a responsiveness to environmental context influencing children's behavior (Guo, Roettger, and Shih 2007; Bakermans-Kranenburg and van IJzendoorn 2011). For example, a recent metanalysis found that the DRD2 and DRD4 polymorphisms moderate the association between parental health behaviors and marital status and attention throughout childhood (Bakermans-Kranenburg and van IJzendoorn 2011). Similarly, the COMT marker has been shown to moderate the influence of child maltreatment on various psychosocial outcomes (e.g., affect, startle reflex, etc.; Klauke et al. 2012).

[2] The Taq1a polymorphism is actually located in the nearby ANKK1 gene but still influences DRD2 expression (Lucht and Rosskopf 2008).

Finally, there is evidence that both polymorphisms of the 5-HTT gene interact with social context (including parenting, SES, child maltreatment, life stress, etc.) to influence a broad range of behaviors, including depressive behavior, emotional regulation, attachment, and negative emotionality (Auerbach et al. 1999; Barry, Kochanska, and Philibert 2008; Caspi et al. 2010; Karg et al. 2011; Mitchell et al. 2011; Pluess et al. 2011; Simons et al. 2011). In sum, there are good reasons to believe that each of the markers described above may moderate the association between family structure instability and children's antisocial behavior.

Hypotheses

On the basis of our reading of the literature, we propose the following hypotheses.

HYPOTHESIS 1.—*Father exits from the household are associated with increases in children's antisocial behavior.*

HYPOTHESIS 1A.—*Exits occurring in early childhood are more strongly associated with antisocial behavior than exits occurring in middle childhood.*

HYPOTHESIS 1B.—*The association between father exits and antisocial behavior is more pronounced among boys than among girls.*

HYPOTHESIS 2.—*Father entrances into the household are associated with increases in children's antisocial behavior.*

HYPOTHESIS 2A.—*Social father entrances are more strongly associated with increases in antisocial behavior than biological father entrances.*

HYPOTHESIS 2B.—*Entrances occurring in early childhood are more strongly associated with antisocial behavior than entrances occurring in middle childhood.*

HYPOTHESIS 2C.—*The association between father entrances and antisocial behavior is more pronounced among boys than among girls.*

HYPOTHESIS 3.—*The association between family structure instability and antisocial behavior is more pronounced among children with more "sensitive" genetic variants than among children without these variants.*

SAMPLE

Our data come from the Fragile Families and Child Wellbeing Study, which is based on a stratified, multistage, probability sample of children born in large U.S. cities between April 1998 and September 2000, with an oversample of children born to unmarried parents (Reichman et al. 2001). Because of the oversample, the families in this sample are disproportionately poor (or near poor) and may be at particular risk of family structure instability. This feature of the data affords us greater power to detect interactions with genes than an equally sized sample of all births. Baseline interviews with mothers and fathers were conducted within 48 hours of the

child's birth, and subsequent interviews were conducted when the focal child was 1, 3, 5, and 9 years old. Externalizing behavior was reported in years 3, 5, and 9. Saliva DNA samples were collected at the age 9 follow-up, using the Oragene•DNA sample collection kit (DNA Genotek Inc., Ontario). We use data from all five waves and restrict the analysis to children who live with their mothers most of the time all nine years (n = 4,697), for whom we have full genetic information (n = 2,772) and at least one measure of antisocial behavior (n = 2,673), and for whom coresidency at birth with the father is known (n = 2,493).

MEASURES

Antisocial Behavior

We utilized two subscales (aggression and nonaggressive rule breaking; see table 1 for descriptive statistics) from the Child Behavioral Checklist to assess children's antisocial behavior (Achenbach 1992; Achenbach and Rescorla 2001). For children this age, the combined subscale is referred to as "externalizing behavior." These measures were collected when the child was 3, 5, and 9 years old. Each item consists of a three-point Likert scale on which mothers report whether their child's behavior is true often or very (2), sometimes or somewhat (1), or never (0). The aggression subscale includes items such as disobedience at home or school, getting in many fights, attacking people, screaming, bullying, talking too much, changing mood suddenly, demanding a lot of attention, and being unusually loud. The rule-breaking scale contains items such as vandalizing, swearing, stealing, setting fire, lying, cheating, and not feeling guilty after misbehaving. Items are summed to form the "externalizing index" (year 3: 22 items, $\alpha = 0.87$, mean = 13.5; year 5: 30 items, $\alpha = 0.86$, mean = 12.8; year 9: 42 items, $\alpha = 0.89$, mean = 6.3). Some items, while covering the same general concept, changed somewhat across waves to better measure developmental changes in externalizing behaviors (particularly rule breaking). Substantive results of analyses were consistent between the raw, log transformed (to account for the positive skew), and standardized scores. We present results based on the standardized scores.

Family Structure Change

At each wave, information on family structure and family structure transitions was obtained from mothers and used to determine the timing of father entrances and exits during the first nine years of the child's life. On the basis of questions about where the child would live after leaving the hospital, we classified children as living in two biological parent families (cohabiting or married; n = 1,470) or single biological parent families (n =

TABLE 1
DESCRIPTIVE STATISTICS OF DEPENDENT AND INDEPENDENT VARIABLES

Variable	*N*	Mean	SD	Min	Max
Dependent variables:					
Year 3 externalizing	1,920	13.47	7.73	0	42
Year 5 externalizing	1,959	12.87	7.49	0	44
Year 9 externalizing	2,323	6.29	6.91	0	70
Controls at baseline:					
M's age at birth	2,493	25.02	5.94	14	47
Educational attainment	2,493	12.01	2.19	8	18
Race:					
Black	2,493	.49		0	1
White	2,493	.21		0	1
Hispanic	2,493	.27		0	1
Other	2,493	.03		0	1
Baseline ln(household income)	2,493	9.89	1.10	4.4	11.8
Child is female	2,493	.48		0	1
Low birth weight (<2.5 kg)	2,493	.09		0	1
Child is M's firstborn	2,493	.38		0	1
F resided with M at birth	2,493	.61		0	1
M discussed abortion	2,493	.37		0	1
M or F ever depressed	2,493	.49		0	1
M or F ever alcohol problem	2,493	.48		0	1
M or F ever incarcerated	2,493	.45		0	1
M's report of couple violence	2,493	.04		0	1
M lived with both parents at 15	2,493	.43		0	1
M's report of the relationship	2,493	11.26	4.4	4	16
M's report of overall health	2,493	2.89	.94	1	4
Time-varying controls 1, 3, 5 (average):					
Material hardship		1.21	1.60	0	10
M's report of the relationship	2,407	8.8	8.0	5	20
M report of violence	2,365	.07		0	1
M report of F parenting	2,398	3.21	1.3	1	4
Sensitivity measures:					
M's rating of C's age 1 temperament	2,395	15.55	4.58	6	30
F's rating of C's age 1 temperament	1,741	16.28	4.46	6	30
M's impulsivity score	2,363	11.81	3.73	0	18
F's impulsivity score	1,763	12.01	4.01	0	18

NOTE.—M=mother, F=father, C=child.

1,023). On the basis of their initial classification, children were then classified according to whether they experienced the exit of a biological father from the household, the entrance of a biological or social father into the household, or no transition. For mothers who missed a wave and responded to a later wave, we utilized the relationship histories to determine if and when a residential change occurred. We focused exclusively on first entrances and first exits since including higher-order changes is likely to confound events that occurred during the same time period.

The left panel of table 2 shows the distribution of the timing of the biological father's first exit among children who began life living with two biological parents, either married or cohabiting. Only about half of the chil-

TABLE 2
DISTRIBUTIONS (Percentage) OF BIOLOGICAL AND SOCIAL RESIDENTIAL TRANSITIONS BY AGE AND TYPE OF TRANSITION

	Biological Father Exit Analysis (n = 1,470)		Father Entry Analysis (n = 1,023)	
			Biological Father Entry	Social Father Entry
Always two-parent . . .	48	Always single mother . . .	34	
Exit, ages 0–1	19	Ages 0–1	19	11
Exit, ages 1–3	13	Ages 1–3	9	9
Exit, ages 3–5	10	Ages 3–5	3	10
Exit, ages 5–9	10	Ages 5–9	3	2

dren in this group experienced a father exit by age 9. Generally speaking, father exits were most common in the first year of life (19%). The right panel of table 2 shows the distribution of father entrances for those who began life living with a single mother. Around one-third of the children in this group experienced a biological father entrance, another one-third experienced a social father entrance, and the remaining one-third never lived with a biological or social father. Table 2 shows that biological father entrances typically occur in the first three years of life, with entrances in the first year accounting for over half of all entrances. Social father entrances are more evenly distributed across all waves, although they too are most common during the first year after birth. Note that it would be incorrect to describe children who never experience a father entrance as living in a "stable" family since many of their mothers are in noncoresident (dating) partnerships that change over time (McLanahan and Beck 2010). Note also that the reduction in entrances in later childhood is partially a result of our restriction to first entrances.

Genes

Owing to the novelty of the biological susceptibility model, there is little guidance as to how to determine the sensitivity or reactivity of a genetic variant or polymorphism. To date most studies have utilized the fact that some genes have been classified as "risky" and reclassified them as "sensitive" (Belsky et al. 2009; Belsky and Pluess 2009; Mitchell et al. 2013).

Serotonin[3].—Although several genes regulate the serotonergic system, we use the one most often studied in the literature, the serotonin transporter

[3] Genotypes for 5-HTTLPR, Stin2, and DRD4 were obtained by PCR and gel electrophoresis, while the other dopamine genes were marked with an Illumina SNP chip.

gene (5-HTT). This gene codes for the protein that recycles serotonin from the synapses, which, in theory, allows for greater responsiveness to the environment. We utilize two well-examined polymorphisms of the serotonin transporter gene (see table 3 for distributions): (1) a functional polymorphism (5-HTTLPR) in the 5′ regulatory region and (2) a 17 base pair variable number tandem repeat (VNTR) in the second intron region (cSTin2 VNTR). For the 5-HTTLPR polymorphism, the most common alleles are the short (S) 14-repeat and long (L) 16-repeat, resulting in the genotypes LL, SS, or LS.[4] The S allele has been shown to be associated with less efficient transcription rates and is typically considered more sensitive than the L allele (Heils et al. 1996; Caspi et al. 2010). For the STin2 polymorphism, the two most common alleles are the 10 and 12 repeat, with the 12 repeat allele being associated with more environmental reactivity—at least for depression (Hranilovic et al. 2004; Mitchell et al. 2011).

Dopamine.—Unlike serotonin, for which we use two measures of the same gene (at different loci on the gene), for dopamine we use one measure each for three different genes along the dopaminergic system (see table 3 for distributions). Like the 5-HTT measures, the DRD4 VNTR is a length polymorphism and was obtained by PCR followed by gel or capillary electrophoresis. We code 6–10 repeats as "long" or 7R alleles (which make up 80% of long alleles) and call the short allele 4R because is constitutes 85% of the short (2R-5R) alleles. To date, this polymorphism has shown the highest level of replication for the 7R allele being the sensitive allele (Bakermans-Kranenburg and van IJzendoorn 2006, 2011). The other two dopamine markers are measured as single nucleotide polymorphisms. Like DRD4, Dopamine Receptor D2 (DRD2, 11q23) codes for proteins controlling the dopamine receptors in the synapse (Noble et al. 1991), and for the Taq1a polymorphism, people have either a C (for cytosine) or a T (for thymine), thus resulting in the genotypes CC, TT, or CT, where the TT genotype is typically assumed to be the sensitive genotype (Bakermans-Kranenburg and van IJzendoorn 2011). Catechol-O-methyltransferase (COMT, 22q11.21) codes for a major enzyme involved in the inactivation of dopamine in the synaptic cleft, and the Met allele of the Val158 Met polymorphism (rs4680) is known to decrease COMT activity by coding the amino acid methionine instead of valine and is typically coded as the sensitive allele (Lachman et al. 1996; Klauke et al. 2012).

[4] Recall that in all cases people have two copies of the gene (one from the father and one from the mother) so that three options are available: two homozygote genotypes (two copies of the same allele) and one heterozygote genotype (one of each allele).

TABLE 3
DISTRIBUTION OF GENOTYPES

	(1)	(2)	(3)
5-HTTLPR	LL	LS	SS[a]
	(42)	(42)	(16)
STin2	10/10	10/12	12/12[a]
	(10)	(40)	(50)
DRD2	CC	CT	TT[a]
	(45)	(42)	(13)
COMT	Val/Val	Val/Met	Met/Met[a]
	(38)	(48)	(14)
DRD4	4R/4R	4R/7R	7R/7R[a]
	(55)	(37)	(8)

[a] Homozygote sensitive genotype. Numbers in parentheses are %.

Controls

As is true for all studies based on observational data, we do not randomly assign families to different family structure transitions. Instead, parents choose whether or not to enter or exit a coresidential relationship. Thus any association we observe between family structure change and child well-being may be due to a third factor that is causing both the change and the poor outcome in the child.[5] For example, an abusive relationship between the parents may cause them to end their partnership and may also cause children to be aggressive or anxious. In this case, failing to take account of differences in violence will lead to an overestimate of the negative effect of family structure change. Fortunately, the Fragile Families data include a rich set of variables that allow us to control for many family and individual characteristics that are likely to affect parents' decisions to end or begin a coresidential union, including grandparents' characteristics (whether parents were raised in a two-parent household), parents' characteristics (race, age, education, employment status, income, health, mental health history, incarceration history, drug and alcohol history), parents' relationship quality (supportiveness, violence, whether they discussed having an abortion), and child's health (low birth weight, birth order). Each of these variables is measured at the baseline interview or retrospectively at the one-year interview. While our approach does not eliminate the possibility that an unmeasured, or at least an unaccounted for, characteristic is responsible for the association between family structure transitions and children's antisocial behavior, the rich set of control variables give us more confidence that

[5] It also is possible that having children with serious behavior problems may cause parents to end their relationship, although previous research using these data finds no evidence of such an effect (Cooper et al. 2011).

our estimates are due to the change in family structure rather than some other variable.

Further, we provide a separate sensitivity analysis that uses time-varying covariates to test alternative explanations for the association between father exits/father entrances and children's antisocial behavior. Here we focus on three covariates: (1) economic hardship (measured as whether parents had problems making ends meet in each of four domains: food, utilities, housing, and medical care), (2) couple relationship quality (physical and coercive violence, supportiveness), and (3) father's parenting quality (reported by mother).

ANALYTICAL STRATEGY

Because we are interested in capturing the dynamic aspect of family structure change on children's behavior, we use latent growth curve modeling (Singer and Willett 2003; Bollen and Curran 2006). This analytic strategy assumes that children differ in their initial level of externalizing behavior and that variance in subsequent trajectories depends on father's residential status, genetic characteristics, and controls. A unique intercept (α), a linear, time-dependent slope (β), and some measurement error (ε) characterize each child's trajectory of externalizing behaviors. Thus, the level 1 equation,

$$y_{it} = \alpha_i + \beta_i t + \varepsilon_{it}, \tag{1}$$

represents within-individual i change over age t. As mentioned earlier, on average, children were interviewed around ages 3, 5, and 9. However, because there is variance in the exact timing of the interview and because of the rapid decline in antisocial behavior during these age periods, we allow for individually varying times of observation to avoid biasing the results (Horney, Osgood, and Marshall 1995). To incorporate the time-varying changes in the father's entry into or exit from a residential relationship with the mother on the child's externalizing behavior, we modify equation (1) as follows:

$$y_{it} = \alpha_i + \beta_i t + \gamma_{tt'} w_{it'} + \varepsilon_{it}, \tag{2}$$

where $\gamma_{tt'} w_{it'}$ represents the effect of each previous interwave time t' entry or exit on externalizing behavior at time t for each ith individual. In other words, externalizing behavior at age 3 can be influenced by changes in father's residential status between waves 1 and 2 (ages 0 and 1) and waves 2 and 3 (ages 1 and 3). Externalizing behaviors at age 5 are influenced by changes in father's residential status between ages 3 and 5, and externalizing behaviors at age 9 are influenced by changes in father's res-

idential status between ages 5 and 9. Each $\gamma_{tt'}$ represents a perturbation from the latent externalizing trajectory associated with a change in father's residential status at a specific point in time (Bollen and Curran 2006).[6]

The second level of the growth model allows the random intercepts (α_i) and slopes (β_i) to be a function of variables that differ across individuals i but do not change across age t. This level represents between-individual change over time. The level 2 equations are as follows:

$$\alpha_i = \alpha_0 + \alpha_1 G_i + \alpha_j \mathbf{X}_{ij} + u_i, \tag{3}$$

$$\beta_i = \beta_0 + \beta_1 G_i + \beta_j \mathbf{X}_{ij} + v_i. \tag{4}$$

In our model, genes affect both the random intercept and the random slope. In addition, a vector $\mathbf{X}$ of j control variables also influences both the intercept and slope. The intercept and slope for each externalizing behavior are directly regressed on these characteristics to assess for potential group differences in the means of the growth factors.

Finally, to estimate the interaction between genes and family structure changes, we substitute equations (3) and (4) into equation (1) and add an interaction term ($\lambda_{tt'}(\text{GENES} \times w_{it'})$):

$$\begin{aligned} y_{it} = {} & \alpha_0 + \alpha_1 \text{GENES}_i + \alpha_j \mathbf{X}_{ij} + \beta_0 t + \beta_1 \text{GENES}_i t + \beta_j \mathbf{X}_{ij} t \\ & + \gamma_{tt'} w_{it'} + \lambda_{tt'}(\text{GENES} \times w_{it'}) + u_i + v_i t + \varepsilon_{it}, \end{aligned} \tag{5}$$

where $\lambda_{tt'}$ represents the interactive effect of genes for family instability in time t' on externalizing behaviors in time t. This interactive effect is a more parsimonious version of a model that treats genes as a time-varying covariate and interacts them with family instability at each wave (Li, Duncan, and Acock 2000).

We use a robust maximum likelihood estimator that accounts for clustering of observations (by hospital) and uses all available data, even if not all waves are present (Muthén and Muthén 2007). This technique has been shown to produce less biased results than listwise deletion and performs similarly to multiple imputation methods (Schafer and Graham 2002). Because we have specific hypotheses about the direction of the biological and social father residential changes and the interactions with genes, we use one-tailed tests to assess statistical significance.

We begin by estimating a model that examines the association between father exits and children's antisocial behavior and whether the association varies by the age of the child and the child's gender. Next, we estimate a model that examines the association between father entrances and chil-

[6] A more complicated model allowing for a time-varying influence of both the slope and the intercept was tested, but the time-varying effects on the slopes appeared not to provide any additional insight, and therefore, the more efficient model is presented.

dren's antisocial behavior. Here we distinguish between the entrance of a biological father and the entrance of a social father. We also examine whether the associations differ by child's age and genetic sensitivity. We end with robustness checks for population stratification, gene-environment correlation, and alternative causal explanations.

RESULTS

We begin by testing our hypotheses about the association between father exits from the household and children's antisocial behaviors. We hypothesized that father exits would be positively associated with child's antisocial behavior, that exits during early childhood would show a stronger association than exits during middle childhood, and that the association would be stronger for boys than for girls. The results are shown in columns 1 and 2 of table 4. Looking first at column 1, we see that, for boys, a father exit is associated with an increase in antisocial behavior in every time period. The year-specific coefficients are not significantly different from one another. The last row, which presents the coefficient for all years combined,

TABLE 4
Time-Varying Regression of Externalizing Behavior on Recent Biological Father Residential Transition (Compared to Always Two-Parent)

	Biological Father Exit vs. Always Two-Parent		Biological Father Entry vs. Always Single		Social Father Entry vs. Always Single	
Intercept	Boys (1)	Girls (2)	Boys (3)	Girls (4)	Boys (5)	Girls (6)
Ages 0–1	.70*	.62***	−.57*	−.53	.21	.26
	(.34)	(.17)	(.33)	(.39)	(.68)	(.71)
Ages 1–3	.51*	.59*	−.60*	−.20	.27	.02
	(.31)	(.34)	(.31)	(.46)	(.55)	(.76)
Ages 3–5	.36	.28*	−.44	−.65	.50	.30
	(.25)	(.15)	(.38)	(.42)	(.32)	(.49)
Ages 5–9	.79**	.12	.20	−.31	.10	.31
	(.32)	(.31)	(.46)	(.44)	(.52)	(.44)
All ages together	.60***	.46**	−.51*	−.24	.29	.20
	(.17)	(.17)	(.24)	(.30)	(.33)	(.32)

Note.—Numbers in parentheses are SEs. All analyses control for race, mother's age and education, household income, child's gender, birth weight, birth order, report of if an abortion was discussed, both parents' report of how the relationship was going before the birth, parent's lifetime depression, parent's lifetime alcohol problem, if either parent had ever been incarcerated, father's residential status at birth, if there was any domestic violence during the pregnancy, mother's self-report of health, and if the mother lived with her parents at age 15.

* $P < .05$, one-tailed.

** $P < .01$.

*** $P < .001$.

indicates that a father exit is associated with a 0.60 increase in boys' antisocial behavior, which is slightly larger than the difference associated with being black rather than white but smaller than the association for being male rather than female. As shown in column 2, the pattern for girls is similar to that for boys, except that the coefficients for exits in early childhood are larger than the coefficients for exits after age 3. The coefficient for all years combined indicates that a father exit is associated with a 0.46 increase in antisocial behavior.

Next we test our hypotheses about the association between father entrances and child's behavior. We hypothesized that father entrances would be associated with increases in children's antisocial behavior, that entrances occurring in early childhood would show a stronger association than entrances occurring in later childhood, that the entrance of a social father would be more negative than the entrance of a biological father, and that the association between father entrances and antisocial behavior would be stronger among boys than among girls. The results are reported in columns 3–6. Looking first at column 3, we see that the entrance of a biological father into the household is associated with a decrease in boys' antisocial behaviors. The reference group is living with a single mother. The size of the coefficients is larger for early entrances as compared with later entrances, but the differences between the age-specific coefficients are not statistically significant. The average association across all years indicates that a biological father entrance is associated with a −0.51 decrease in boys' antisocial behavior. The pattern for girls is similar to the pattern for boys with respect to the size and direction of the coefficients, but none of the coefficients for girls are statistically significant.

Finally, columns 5 and 6 indicate that a social father entrance is not associated with a significant increase in children's antisocial behavior, compared to living with a stable single mother for either boys or girls. Nevertheless, the social father coefficients are in the expected (positive) direction; and when boys and girls are combined, the coefficient for a social father entrance between 3 and 5 is statistically significant (results not shown).

Interactions by Genotypes

The last set of analyses test our hypotheses about gene × environment interactions.[7] We hypothesized that the association between family structure instability and antisocial behavior is more pronounced among chil-

[7] Although not part of our main hypotheses to be tested, none of the genes had a significant main effect on externalizing behaviors, conditional on the controls and family transitions. This is not surprising since the genetic differential sensitivity theory implies a crossover (for better or for worse) model with no main effect of genes (Mitchell et al. 2013).

dren with more "sensitive" genetic variants than among children without these variants. Table 5 presents the results for the five genetic markers we examined. For this analysis we did not distinguish across age groups but used the combined measure. We did run separate models for boys and girls. Looking first at boys (col. 1), we see that four of the five genetic markers

TABLE 5
Regression Estimates for Externalizing Behavior Trajectories on Gene-Environment Interactions

	Biological Father Exit vs. Always Two-Parent		Biological Father Entry vs. Always Single Mother		Social Father Entry vs. Always Single Mother	
Genetic Polymorphism and Genotype	Boys (B) (1)	Girls (B) (2)	Boys (B) (3)	Girls (B) (4)	Boys (B) (5)	Girls (B) (6)
5-HTTLPR:						
LL	. . .	. . .	. . .	. . .	. . .	. . .
LS	.8 (.5)*	.1 (.5)	−.6 (.4)	−.1 (.5)	.5 (.6)	.1 (.6)
SS	1.3 (.6)*	.6 (.6)	−1.4 (.7)*	−.6 (.6)	1.8 (.7)**	.3 (.6)
χ^2 (2 df)	9.3*	2.1	6.0*	3.1	6.1*	.9
Stin2:						
10/10	. . .	. . .	. . .	. . .	. . .	. . .
10/12	.6 (.7)	.5 (.6)	−.8 (.7)	−.1 (.6)	.1 (.7)	.4 (.8)
12/12	.9 (.6)	.6 (.6)	−.7 (.6)	−.3 (.7)	.9 (.8)	−.1 (.7)
χ^2 (2 df)	3.4	3.1	3.8	1.3	1.89	.4
DRD2:						
CC	. . .	. . .	. . .	. . .	. . .	. . .
CT	.8 (.5)*	.6 (.4)*	−.4 (.5)	−.5 (.5)	1.1 (.6)*	.3 (.6)
TT	1.4 (.7)*	1.5 (.6)*	−.8 (.6)	−.4 (.8)	1.2 (.7)*	.8 (.8)
χ^2 (2 df)	8.0*	6.3*	3.6	2.6	6.1*	.8
COMT:						
Val/Val	. . .	. . .	. . .	. . .	. . .	. . .
Val/Met	.7 (.4)*	.0 (.3)	−.9 (.5)*	.0 (.5)	.5 (.4)	−.2 (.6)
Met/Met	1.2 (.6)*	−.1 (.7)	−.9 (.6)	.2 (.6)	.8 (.5)*	−.1 (.6)
χ^2 (2 df)	7.7*	1.3	5.4	1.9	5.0	.5
DRD4:						
4R/4R	. . .	. . .	. . .	. . .	. . .	. . .
4R/7R	1.4 (.6)*	1.1 (.5)*	−.9 (.5)*	−.1 (.5)	.8 (.6)	.0 (.7)
7R/7R	1.8 (.9)*	1.0 (1.0)	−.8 (.9)	−.8 (1.1)	1.2 (1.2)	.5 (1.1)
χ^2 (2 df)	9.7**	4.9	5.6	3.2	3.6	.1

Note.—Numbers in parentheses are SEs. All analyses control for race, mother's age and education, household income, child's gender, birth weight, birth order, report of if an abortion was discussed, both parents' report of how the relationship was going before the birth, parent's lifetime depression, parent's lifetime alcohol problem, if either parent had ever been incarcerated, father's residential status at birth, if there was any domestic violence during the pregnancy, mother's self-report of health, and if the mother lived with her parents at age 15.

* $P < .05$, one-tailed.

** $P < .01$.

*** $P < .001$.

show significant interactions with biological father exits: 5-HTTLPR ($\chi^2 = 9.3$, 2 *df*), DRD2 ($\chi^2 = 8.0$, 2 *df*), COMT ($\chi^2 = 7.7$, 2 *df*), and DRD4 ($\chi^2 = 9.7$, 2 *df*). Furthermore, all of the markers, including the smaller and insignificant interactions with Stin2, work in the expected direction such that the most sensitive genotypes have larger, positive associations compared with the least sensitive genotype. For girls, the pattern of the coefficients is the same as it is for boys, but only two of the interactions are statistically significant, and only one of the coefficients (DRD2) is of similar size to the coefficient for boys. These results indicate that boys with more sensitive genes respond more negatively to a father exit from the household than boys with less sensitive genes.

Columns 3 and 4 present the coefficients for the interaction between children's genetic characteristics and biological father entrances, while columns 5 and 6 show the interaction coefficients for genotype and social father entrances. As shown in column 3, all of the interaction coefficients are in the expected direction, and three of the five are statistically significant. In each case, boys with the more sensitive genetic variant respond more favorably to the entrance of their biological father into the household than boys without this gene variant. None of the interactions is significant for girls, although the coefficient for DRD4 is identical in size to the coefficient for boys The results for social father entrances show a similar pattern insofar as boys with the more sensitive gene variants show a stronger response to a change in family structure than boys with the less sensitive variants. Three of the interaction coefficients are statistically significant (the SS variant of 5-HTTLPR, both the CT and TT variants of DRD2, and the Met/Met variant of the DRD4 marker). Again, the coefficients for girls are smaller, and none are statistically significant.

The interaction results presented in table 5 are based on a model that does not differentiate by child's age. We chose this model because age differences in children's response to family structure change were not statistically significant. Since one might hypothesize that the G × E interactions might differ by age of child, even if the main effect of family change does not, we estimated another model that allowed the interactions of genes and father transitions to vary by age of the child (0–1, 1–3, 3–5, and 5–9). In results not shown, we found that the G × E interaction coefficients in early childhood (e.g., ages 1–3) were 50%–70% larger than the interaction coefficients in later childhood (i.e., 5–9). However, because of the partitioning of the age groups, the standard errors were large and the differences were not statistically significant. Nevertheless, given the strong theoretical and growing empirical evidence that early childhood experiences are especially important for shaping children's health and future well-being, these questions should be revisited in the future with a larger sample of children.

Sensitivity Analyses

As noted earlier, a major concern of studies using observational data is that the predictor of interest is a marker of some other variable that is causing both the predictor and the outcome of interest. To address this concern, all of our models include a rich set of control variables measured at birth. We also conducted additional analyses that used time-varying covariates to measure family's economic hardship, parents' relationship quality (supportiveness and violence), and father's parenting quality in the year prior to a father exit from the household. These three constructs were chosen because they are frequently proposed as alternative explanations for the association between father exits and children's behavior problems. Economic hardship, parental conflict, and low or negative fathering are strong predictors of union dissolution as well as poor child outcomes.

Row 1 of table 6 shows the coefficients for father exits for three different groups: (1) all boys, (2) boys with the 5-HTTLPR LL genotype, and (3) boys with the 5-HTTLPR SS genotype. Column 1 in each of the three sections corresponds to the main effect of a father exit on externalizing shown in table 5. These estimates are slightly different from the ones reported in table 5 because here we estimate separate models for boys with the LL and SS genotypes. According to these estimates, the association between a father exit and children's antisocial behavior is much smaller for boys with the LL marker (0.1) than for boys with the SS marker (1.4). The inclusion of economic hardship in the year prior to the exit reduces the coefficient by between 14% and 33%. The inclusion of couple violence (col. 3) and couple supportiveness (col. 4) in the year prior to the exit actually increases the coefficient for father exit by about 30%, suggesting that differences in parents' relationship quality are suppressing the effect of a father exit. And the inclusion of father's parenting quality (col. 5) only slightly reduces (13%–17%) the size of the father exit coefficient. In the final column we control for the full set of time-varying covariates. Taken together, economic hardship, parents' relationship quality, and father's parenting quality appear to counterbalance each other such that the final coefficient is similar to the original coefficient. Most importantly, the association between father exits and children's antisocial behavior persists even after taking these alternative explanations into account. Of course this finding does not mean that some other unmeasured variable is not accounting for the association between father exits and antisocial behavior. It does, however, mean that something besides prior economic hardship, parental conflict, and fathers' parenting must be operating.

Like table 6, table 7 reports the association between biological father entry and externalizing behavior for all boys, 5-HTTLPR LL boys, and 5-HTTLPR SS genotypes, controlling for prior economic hardship, relationship quality, and fathers' parenting quality. By examining the first

TABLE 6

BIOLOGICAL FATHER EXIT WITH TIME-VARYING EFFECTS OF SES, RELATIONSHIP QUALITY, AND FATHER PARENTING

	ALL BOYS						BOYS WITH 5-HTTLPR LL GENOTYPE						BOYS WITH 5-HTTLPR SS GENOTYPE					
	(1)	(2)	(3)	(4)	(5)	(6)	(1)	(2)	(3)	(4)	(5)	(6)	(1)	(2)	(3)	(4)	(5)	(6)
Biological father exit	.6**	.4*	.8***	.8***	.5**	.4*	.1	.0	.2	.1	.0	.0	1.4*	1.2	1.8*	2.0*	1.2*	1.5*
	(.2)	(.2)	(.2)	(.2)	(.2)	(.2)	(.4)	(.4)	(.4)	(.4)	(.4)	(.5)	(.8)	(.8)	(.8)	(.9)	(.8)	(.9)
Household income		X				X		X				X		X				X
Couple violence			X			X			X			X			X			X
Couple supportiveness				X		X				X		X				X		X
Supportive parenting					X	X					X	X					X	X

NOTE.—Numbers in parentheses are SEs. All analyses control for race, mother's age and education, household income, child's gender, birth weight, birth order, report of if an abortion was discussed, both parents' report of how the relationship was going before the birth, parent's lifetime depression, parent's lifetime alcohol problem, if either parent had ever been incarcerated, father's residential status at birth, if there was any domestic violence during the pregnancy, mother's self-report of health, and if the mother lived with her parents at age 15.

* $P < .05$, one-tailed.

** $P < .01$.

*** $P < .001$.

TABLE 7
BIOLOGICAL FATHER ENTRY WITH TIME-VARYING EFFECTS OF SES, RELATIONSHIP QUALITY, AND FATHER PARENTING

	ALL BOYS						BOYS WITH 5-HTTLPR LL GENOTYPE						BOYS WITH 5-HTTLPR SS GENOTYPE					
	(1)	(2)	(3)	(4)	(5)	(6)	(1)	(2)	(3)	(4)	(5)	(6)	(1)	(2)	(3)	(4)	(5)	(6)
Biological father entry	−.5*	−.4*	−.5*	−.5*	−.4*	−.4	.0	.0	.0	.0	.0	.0	−1.4*	−1.2*	−1.5*	−1.5*	−1.4*	−1.2*
	(.2)	(.2)	(.2)	(.2)	(.2)	(.2)	(.4)	(.4)	(.4)	(.4)	(.5)	(.6)	(.7)	(.7)	(.7)	(.7)	(.7)	(.7)
Household income		X				X		X				X		X				X
Couple violence			X			X			X			X			X			X
Couple supportiveness				X		X				X		X				X		X
Supportive Parenting					X	X					X	X					X	X

NOTE.—Numbers in parentheses are SEs. All analyses control for race, mother's age and education, household income, child's gender, birth weight, birth order, report of if an abortion was discussed, both parents' report of how the relationship was going before the birth, parent's lifetime depression, parent's lifetime alcohol problem, if either parent had ever been incarcerated, father's residential status at birth, if there was any domestic violence during the pregnancy, mother's self-report of health, and if the mother lived with her parents at age 15.

* $P < .05$, one-tailed.

** $P < .01$.

*** $P < .001$.

column of each of the three groups, we see that for boys with the LL genotype, a father entry has no association with antisocial behavior (0.0), while for boys with the SS genotype, the coefficient is large and negative (−1.4). As was true for father exits, controlling for economic hardship in the previous time period reduces the coefficient for father effect of the entry by about 14%–20%. Controlling for couple relationship quality and father parenting quality, however, does not change the coefficient. This finding suggests that some of the positive association between father entrances and lower antisocial behavior is due to the fact that single mothers with more economic resources (fewer hardships) are more likely to have a biological father move into the household. However, the basic finding still holds (especially for the sensitive genotypes). Unfortunately, we do not have complete information on all social fathers prior to their moving in with the mother; rather we have information only on men who were in a romantic relationship with the mother at the time of the previous interview. Thus we cannot adjust for parents' relationship quality or father's parenting quality for social fathers who enter the household. However, we can control for mothers' economic hardship in the previous year, and doing so does not change the coefficient for social father entry (not shown).

In addition to moderating environmental influences, genes may also play an important role in shaping people's environments. For this reason, some analysts may argue that gene-environment interactions are actually due to gene-environment correlation (rGE; Plomin et al. 2008). This argument is similar to concerns about reverse causality and omitted variable bias in the social science literature, only here the omitted variable is genes. There is some evidence, for example, that temperamentally difficult children evoke less paternal involvement and negatively influence parental relationship quality (Lewin-Bizan 2006), which may result in union dissolution. In this case, children's genetically related behavior may be causing the family disruption rather than vice versa. We test this hypothesis by regressing parents' reports of child's temperament (EAS temperament scale) at age 1 on child's dopaminergic and serotonergic genes, conditional on controls. We find strong evidence that mother's and father's reports of more difficult temperament are positively associated with the dopamine genes and that father's report of a difficult temperament is positively associated with the serotonin genes. However, when used to predict subsequent father entrances or exits, there is no significant (or substantive) effect of either temperament or the number of dopamine or serotonin genes on family structure change.[8] This finding suggests that although some of our genetic markers

[8] Although the temporal ordering is murkier, the child's EAS temperament at age 1 does not predict the biological father's entry or exit between birth and age 1.

may be related to temperament (which is not surprising), they do not seem to be a cause of family structure change, at least not in these data.

Another type of rGE may occur if parent's genes are correlated with both family instability and child's behavior. For example, a parent's genes may make him or her impulsive or difficult to get along with, which, in turn, may produce an unstable family environment as well as high levels of externalizing behavior in children. While this argument seems plausible, recall that our interactions showed that children with the same genetic makeup have very different and opposite responses to the biological father's entering or exiting the household. So while a common genetic factor might explain one of these responses, it is hard to see how such a factor would explain both. Nevertheless, we tested the plausibility of this argument by including mother's genetic makeup is our models to see if this altered our estimates of children's responses to family instability. Importantly, there was no noteworthy change in the G × E coefficient when we controlled for mother's genes. Note that even though mothers' genes directly contribute to children's genes, this is not a linear combination because (1) the father's genotype is not available and (2) only half of the mother's genotype is used for any child.

Because we do not have father's DNA, we were unable to conduct the same analysis with father's genotype. However, since dopamine and, to a lesser extent, serotonin are related to impulsivity, we can use parents' impulsivity scores as controls in the same way we did for genes. Here we find a moderate association (although not statistically significant) between mother's impulsivity and her dopamine genotype, and we might expect the association to be higher for men (Congdon, Lesch, and Canli 2008). However, when we include both parents' impulsivity scores as controls in the G × E models of child's externalizing behavior, we find no notable changes in the interaction coefficients. Again, this finding suggests that passive rGE does not account for the G × E effects reported in table 5.

Finally, as part of our sensitivity analyses, we allowed the G × E interactions to differ by race. Because of concerns about population stratification (differences in the distribution of genotypes by ancestry), it is common practice in genetic studies to stratify analyses by racial ancestry, in this case, whites and blacks. Doing so results in smaller sample sizes and larger standard errors, but the pattern of the coefficients is similar for both groups. Because self-identified race and genetic ancestry are not perfectly correlated, we cannot entirely rule out the possibility that ancestry differences account for some of the interactions we observe. Similarly, we should note that the genes we measure may not be the true causal mechanisms; rather they may simply be correlated with other genes that are the true causes of the interaction. Our choice of these particular genes is based

on biological theory and previous literature, but more research is needed to certify that these particular genes are the primary genetic factors in the interaction.

DISCUSSION

Our article tested several hypotheses about the link between family structure instability and children's antisocial behavior. Consistent with much past research, we found that family structure transitions were generally associated with increases in children's antisocial behavior, with one important exception: the entrance of a biological father into the household—a transition not studied in prior research—was associated with a decrease in antisocial behavior. This finding is likely due to the fact that the biological father has been part of the child's environment since birth, and thus he or she would have benefited from the increase in the family's economic resources and parental social capital while experiencing little or none of the stress associated with a disruption in family routines and relationships. Indeed, in our sample, the vast majority of biological fathers who entered the child's household were romantically involved with the mother at birth and planning to help raise the child.

We also hypothesized that the association between family structure instability and children's antisocial behavior would depend on the age and gender of the child. The evidence for age differences was mixed. In the models without the genetic information, we found some evidence that early father exits were worse than later exits, but the differences were not statistically significant. In the models with genes, which were estimated only for boys, the coefficients for early father exits were larger (by about 50%) than the coefficients for later exits; however, the differences were not statistically significant. The fact that the difference in the size of the coefficients was substantial suggests that, with a larger sample, they might have been statistically significant. Although we did not find strong evidence of age differences in children's response to family change, we did find evidence for gender differences, with boys showing stronger and more consistent responses to father exits and entrances than girls.

Finally, we found strong evidence that children's reaction to changes in family structure were moderated by their genetic makeup. Although gene by environment interactions have been examined in prior studies, ours is the first to show how genetic characteristics shape children's responses to family structure instability. We found that boys with genetic variants that make them more "sensitive" to their environments responded more negatively to the exit of a father from the household and more positively to the entrance of a biological father into the household. This finding, which was

robust across several genetic markers, across different races, and to multiple alternative explanations, is consistent with the "differential genetic sensitivity" model (Boyce and Ellis 2005; Belsky and Pluess 2009).

Implications.—The results presented here have a number of implications for how we think about research on family structure, genes, and children's life chances. First, our findings show that there is considerable heterogeneity in children's responses to family conditions and that biological variables can enrich our ability to understand this phenomenon. As shown here, estimates from regression analyses that omit genetic markers may significantly underestimate the consequences of family instability for some groups of children while overestimating it for others. Further research is needed to determine how widespread this problem may be and the extent to which genetic sensitivity is environment specific or person specific.

Second, our findings lend additional support to the argument that stress is an important mechanism in explaining the link between family structure changes and children's antisocial behaviors. They do so not only by showing significant associations between changes in family structure and children's behavior but also by showing that children whose genes make them more sensitive to stress respond more strongly to family change than children whose genes make them less sensitive. Indeed, children without this biological sensitivity show very little increase in antisocial behavior when exposed to family structure instability. The extent to which these particular genetic markers are the true interactive variants—or simply strongly correlated with other nearby genetic markers—is not tested here. However, these variants do have a large literature supporting their use. Moreover, insofar as the variants we use are simply markers of other genes, our results are biased downward. The evidence we present on differential genetic sensitivity is unusually powerful. To the best of our knowledge, no other research has been able to show significant positive and negative reactions being moderated by genetic endowment for two separate events (exit/entrance) with opposite implications (positive/negative) using the same sample. The greater reactivity of those with sensitive alleles, differentially responding both positively to positive family transitions and negatively to negative family transitions, is powerful evidence for the genetic differential susceptibility model.

Our finding of a crossover effect also has implications for how we think about social mobility more broadly. For example, the emerging evidence from research on G × E interactions teaches us that things are much worse than we thought for a substantial portion of the children exposed to difficult environments. At the same time, it tells us that the potential payoff to improving the environments of these children is much greater than we may have expected. Furthermore, the fact that none of the genetic markers we examined had a significant main effect on children's antisocial behavior

underscores the importance of the social environment in determining how genes affect children's future mobility.

Finally, and more broadly, our findings highlight how the new research on measured genes and gene × environment interactions, which is leading to a paradigm shift in the debate between "nurture versus nature," should be of great interest to sociologists whose primary concern is the social environment. Ultimately, this new research may provide empirical support for sociological ideas that have been rejected in the past because of subgroup heterogeneity. Given that the associations between certain social environments and outcomes of interest vary across genotype, and given that the sensitive markers are often the less common variant, failing to incorporate genetic information into our models can lead to substantial measurement error, biasing coefficients for social environments toward zero and resulting in type I errors. In sum, while we recognize that many sociologists are skeptical of the emerging interest in genomics, we would argue that this fear is largely misplaced and that, if anything, the new research is providing strong support for the role of the social environment in shaping how genes are expressed and when and where they matter.

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