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Childhood Family Instability and Young Adult Health

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Abstract

American children live in a variety of family structures throughout their childhoods. Such instability in family arrangements is common and has important demonstrated implications for short-term child outcomes. However, it is not known whether family instability experienced in childhood has enduring health consequences across the life course. Using demographic, social, and biological data from the National Longitudinal Study of Adolescent to Adult Health, we investigate the family stress model, testing the relationship between parental family instability in childhood and four biological markers of health in young adulthood. This is the first study to examine whether the accumulation of family change in childhood leaves a lasting physiological residue. While family instability is associated with poorer short-term behavioral and socioeconomic outcomes as documented in previous research, we find no evidence of deleterious young adult health consequences. These findings are robust across different measures of physiological health risk and types of family transitions.

Keywords

biosocial; family; health; instability; stress

The landscape of American families is diverse and dynamic. Overlapping demographic and social trends of later age at marriage, greater acceptability of divorce and nonmarital childbearing, and more cohabitation mean a declining share of American children live with two biological married parents. According to the 2010 census, 29% of American children live with only one biological parent, and 3.5% reside with neither biological parent (Vespa, Lewis, and Kreider 2013). Roughly one quarter of all children live with a divorced or separated parent or step-parent (Kreider and Lofquist 2014).

Such cross-sectional estimates, however, belie the true complexity and dynamism of family structures. Research on family instability is intended to capture changes in family structure over time, focusing primarily on coresidence of biological and social parents. It is estimated that a quarter of children experience at least one family transition by age six (Cavanagh and Huston 2006). Furthermore, the likelihood of instability varies depending on the family

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SUPPLEMENTAL MATERIAL

Appendix A is available in the online version of the article.

structure at birth; 81% of children born into nonmarital cohabiting families will experience at least one family transition by age five, with more than 22% experiencing two or more (Graefe and Lichter 1999). Moreover, family instability has increased during the past decade among children born to single mothers (Brown, Stykes, and Manning 2016).

Family instability not only is an increasingly common feature of American children's lives but also is consequential. Much of the literature on family instability examines the implications of changes in family structure for sexual behavior, finding that women who experience family instability have earlier sexual debut (Fomby, Mollborn, and Sennott 2010), have earlier union formation (Fomby and Bosick 2013), and are more likely to have a premarital birth (Wu 1996). Children from unstable families have more behavioral problems (Osborne and McLanahan 2007). Finally, family instability has negative implications for cognitive (Cooper et al. 2011) and educational outcomes (Cavanagh and Fomby 2012). This evidence underscores the importance of examining cumulative and dynamic measures of family structure, such as family instability, rather than snapshot measures of the experience of divorce or parental absence by a given age.

Despite the advances made by research on family instability, the existing literature largely focuses on short-term behavioral and cognitive outcomes. Family instability is a relatively recent area of research in family studies, and the life course data required to investigate adult outcomes are often unavailable either because studies with adults do not have the detailed information on childhood family structure necessary to measure family instability or because studies with family instability measures focus on younger respondents. The few studies examining how family instability is related to health in childhood and adolescence rely on self- or parent-reported health measures and focus primarily on obesity (Bzostek and Beck 2011; Hernandez et al. 2014; Schmeer 2012). We improve on these studies by using objective measures of health risk based on biomarkers and investigating the association between childhood family instability and young adult health.

In this article, we integrate sociological research on family stress with biosocial research on the physiological consequences of childhood exposure to stress to investigate the association between childhood experience of family instability and young adult health risk. Differentiating among types of family transitions, we contribute to the emerging literature on heterogeneity in the effects of family instability. Given that family disruption is now a normative experience for children growing up in America, the longer-term health consequences of family instability have important implications for contemporary disease onset and aging processes.

BACKGROUND

Stress Models of Instability

Extending the well-established research on family structure, research on family instability shifted the focus from static measures of family status to a dynamic conceptualization of the family process across the life course. Family instability research draws on stress models to argue that changes in family structure are often preceded or followed by a variety of stressors that may prevent successful and healthy child development (Coleman, Ganong, and

Fine 2000). Indeed, measures of family instability were designed to capture the stresses that accompany family disruption (Wu and Martinson 1993).

Changes in family structure result in the renegotiation and reestablishment of boundaries, goals, patterns of interaction, roles, and values (McCubbin and Patterson 1983), and new roles in nontraditional family configurations may be particularly uncertain (Cherlin 1978). Such changes are stressful for parents and children and may lead to inconsistent parenting, emotional insecurity, and fluctuation in resources (Lee and McLanahan 2015; Osborne, Berger, and Magnuson 2012). Furthermore, each transition requires additional renegotiation and adjustment, leading to an accumulation of stressors (Fomby and Cherlin 2007).

There is evidence that family instability increases parental stress, which may lower parenting quality and reduce investment in and supervision of children (Cooper et al. 2011; Osborne et al. 2012). Furthermore, family transitions are often accompanied by fluctuations in resources and residential mobility, increasing uncertainty and stress (Adam and Chase-Lansdale 2002). Reconfigurations in family structure may result in a chaotic home environment, wherein children are insecure about the availability of parental support, influencing the child's emotional attachments (Coldwell, Pike, and Dunn 2006). Consistent with the stress model of instability, greater change is associated with poorer behavioral, emotional, and socioeconomic outcomes in childhood and early adulthood (Cavanagh, Crissey, and Raley 2008; Cavanagh and Fomby 2012; Cooper et al. 2011). In sum, theoretical and empirical research predicts family instability is a stressful childhood experience due to antecedent and resulting processes that create uncertainty and unpredictability in children's lives, and that is hypothesized to predict poorer outcomes compared to stable family environments.

Biological Embedding of Childhood Stress

Assuming that family instability is stressful for the child, due to either general uncertainty and unpredictability or resulting changes in parental behavior or resource availability, how would such childhood stress exposure influence health in adulthood? One robust mechanism through which social stress exposures are hypothesized to get under the skin is biological stress response. When an individual experiences a stressful event, the body mounts a biological response that activates the sympathetic nervous system (SNS) and network of the hypothalamus, pituitary gland, and adrenal glands (HPA axis). SNS releases adrenaline into the bloodstream, increasing heart rate, pulse, and blood pressure (McEwen and Lasley 2002). The HPA axis regulates the production of cortisol, suppressing immune function and increasing vascular tone (Thompson 2014).

Although such responses are a necessary and helpful reaction to immediate threats, chronic stressors elicit repeated activation of the stress response system and result in physiological costs (Hostinar and Gunnar 2013; Taylor, Repetti, and Seeman 1997). Chronic daily stress keeps the HPA axis and SNS activated, keeping cortisol and adrenaline hormone levels elevated, which impairs the immune system, increases inflammation, contributes to the buildup of fat tissue, damages blood vessels and arteries, and increases blood pressure (McEwen and Lasley 2002; Seeman et al. 1997). Thus, stress-related biological effects on immune, metabolic, and cardiovascular systems are evident in measures of inflammation, body mass index (BMI) or obesity, and hypertension, all of which indicate poor general

health with critical implications for future disease (Singer and Ryff 2001). Indeed, the health consequences of childhood chronic stress are not short-lived. Unsupportive childhood environments are associated with greater morbidity throughout adulthood (Wickrama, Lorenz, and Conger 1997), and low socioeconomic status (SES) in childhood is associated with worse adult health (Miller et al. 2009; Steptoe 2012).

Existing research in this area suffers from four limitations that this article addresses. First, many measures of childhood environment are retrospectively reported. Second, most measures of childhood environments ignore family structure completely. Third, many studies employ cross-sectional measures of childhood environments that conceal important variation in circumstances across childhood, overlooking dynamic contexts. Finally, most study samples are not nationally representative, limiting the generalizability of the findings. This article considers the association between a dynamic aspect of family context—family instability—and adult physiological health risk in a longitudinal, nationally representative sample.

Heterogeneity in Stress Response

Taken together, the research on family instability and childhood stress exposure predicts that family structure transitions in childhood will be associated with worse adult health.

However, this prediction assumes that all family change is stressful. Although much of the research on family instability proposes that the stress produced by the number of changes is most relevant (Fomby and Cherlin 2007; Wu and Martinson 1993), more recently there has been a call to differentiate family structure trajectories, paying greater attention to the types of transitions. Recent research demonstrates that the influence of family instability depends on both the type of transition and the outcome examined. For example, parental exits are worse for behavioral outcomes, while entrances are worse for child achievement (Magnuson and Berger 2009; Osborne et al. 2012). The departure of a biological parent from a two-parent family is associated with worse child cognition and externalizing behavior, whereas the entrance of a step- or social father into a single-mother family either is not associated with these child outcomes or is associated with improved outcomes (Lee and McLanahan 2015). Some types of change may actually improve child well-being; biological father entrances promote prosocial behaviors (Mitchell et al. 2015).

Furthermore, research on coping and resilience suggests exposure to stressful environments may not always be harmful (Antonovsky 1996; Schetter and Dolbier 2011). Concentrating on mental health, Thoits (1995) suggests that not all “undesirable” events are stressful. Some changes, including divorce, may actually alleviate ongoing strains or conflicts (Kelly and Emery 2003; Thoits 2010). Research on childhood economic deprivation finds conditional effects of adverse experiences depending on parent and child behaviors and characteristics (Elder and Caspi 1988). Individual beliefs about the meanings derived from challenging experiences may shape outcomes (Antonovsky and Sourani 1988) and even protect physical health (Taylor et al. 2000).

These findings are corroborated in studies that identify heterogeneous effects of subjective versus objective measures of adversity on health. In a meta-analysis of social integration, subjective and objective measures are correlated but independently predict mortality (Holt-

Lunstad, Smith, and Layton 2010). Subjective perception of social isolation is a stronger prediction of gene expression profiles consistent with increased inflammation (Irwin and Cole 2011). This research demonstrates that if stressors are not appraised as stressful they will not elicit the same physiological response, suggesting that objectively measured family instability may not predict elevated health risk if individuals have positive subjective experiences of the changes.

There also is evidence that not all stress response results in long-term health insults. Research on physiology and aging suggests that exposure to moderate stressors may have potential health benefits (Del Giudice, Ellis, and Shirtcliff 2011; Gems and Partridge 2008). Animal models demonstrate that some biological consequences of exposure to stressful childhood rearing environments may dissipate over time if the animal is returned to a supportive environment (Provencal et al. 2016). This research suggests that the health risk associated with family instability may depend on whether the stress experienced is moderate.

Family Instability and Health

While family stress theory and chronic stress response predict that family instability will be associated with worse health in adulthood, research on resilience and subjective appraisals of stress suggest that findings may be more heterogeneous. Existing empirical evidence is similarly mixed. Four recent studies examine the health consequences of family instability. In the Fragile Families and Child Wellbeing Study, family instability is associated with shorter telomere length (Mitchell et al. 2014), higher rates of asthma, worse mother-rated child health (Bzostek and Beck 2011), and greater BMI gains (Schmeer 2012). Hernandez and colleagues (2014) use data from the National Longitudinal Survey of Youth to examine the relationship between family instability from birth to age 18 and self-reported BMI at age 20. They find that girls who experience family instability in childhood are more likely to be obese, but boys are less likely to be obese.

Although these studies generally support a health detriment associated with family instability, there are three major limitations that must be addressed. First, the adverse health consequences of childhood stress may take decades to manifest (Miller et al. 2011), necessitating longitudinal study designs that follow children into adulthood. Second, children with elevated burdens of early-life stress exposures are at increased risk for many health problems (Felitti et al. 1998) due to stress-activated biological mechanisms that affect multiple systems. Therefore, studies of the health consequences of family instability must evaluate adult health outcomes across multiple health domains. Third, it is possible that child health confounds the relationship between instability and health in adulthood. Studies of the long-term health consequences of family instability must account for the possibility of health selection. The present study addresses these limitations by examining the association between childhood family instability and markers of physiological health risk across multiple biological systems in adulthood, controlling for childhood health status.

We use data from the National Longitudinal Study of Adolescent to Adult Health (Add Health) to evaluate the hypothesis that childhood exposure to family instability creates stress that will be evident in adult physiology and emerging chronic health problems. Specifically,

we predict that greater exposure to family instability (more frequent changes) leads to repeated activation of the stress response system, which will elicit physiological dysregulation in response and will be associated with worse health in adulthood. We also hypothesize that this relationship will vary depending on the type of family transition.

DATA AND METHODS

Data

Add Health is an ongoing national longitudinal study of the social, behavioral, and biological linkages in health and developmental trajectories from early adolescence into adulthood (Harris 2010). The data are representative of U.S. adolescents in grades 7 through 12 in 1994 to 1995. The initial sample included 20,745 adolescents aged 12 to 20; since the start of the study, participants have been interviewed at home in four data collection waves. At Wave I, a subsample of respondents' parents also completed in-home interviews ($n = 17,670$). At the most recent round of data collection (Wave IV) in 2008 to 2009, respondents were aged 24 to 32 ($n = 15,701$, 80.3% response rate) and were asked to participate in biological specimen collection (more than 95% provided specimens, almost 15,000). Our analyses included respondents who participated in Waves I and IV. Table 1 presents descriptive statistics for the analytic sample. We restricted our analyses to cases with complete information on all predictors ($n = 10,355$); differences in sample sizes across models are due to missing data on the biomarker outcomes.

Measures of Family Structure Change

We measured childhood parental family instability as the count of family structure changes experienced from birth to adolescence. To construct this longitudinal measure, we first constructed an annual measure of family structure using several sources of data: parents' self-reported union history and current union status (Wave I), youth's reports of the duration of life spent with current household members (Waves I and II), and whether and for how long a youth lived with a nonresident biological parent (Waves I, II, and III). We verified and supplemented these reports with retrospective reports of parental death, incarceration, adoption, and fosterage (Waves I, II, and III). Combining these responses, we constructed a measure of family structure focusing on biological and step-/social parent coresidence for each year of the child's life from birth to age at last adolescent interview (Wave I or II). Due to age variation in the sample, family structure may have been observed for some individuals until age 18 and for others only until age 13. Our results are robust to restricting the measure to age 13 (the age at which all respondents have complete family structure histories) and to restricting the analysis to only individuals observed to age 18. The measure of instability captures both cohabiting and marital relationships between parents but does not differentiate between them. We used a count measure of family instability ranging from zero to nine transitions with a mean of .77 (see Table 1).

Measure of Adolescent Health

Measures of adolescent health collected at the Wave I interview were limited to self-report. To investigate whether there is any evidence of health associations more proximate to the timing of family instability, and the possibility of differential attrition across waves, we used

a measure of BMI in adolescence constructed from self-reported height and weight. We also used this measure as a control in models of adult health given its relevance for physiological health risk. Average BMI in adolescence in the sample was 22.4 (see Table 1).

Measures of Adult Health Risk

We expected that the biological toll associated with cumulative stress experienced in childhood resulting from family instability would manifest across multiple biological systems in adulthood. Therefore, we analyzed the adult physical health risk of Add Health respondents using four measures derived from bio-marker assessments taken at the Wave IV interview. We analyzed three individual measures—high-sensitivity C-reactive protein (CRP), BMI, and hypertension—and one composite measure—metabolic syndrome. We examined these specific measures because they are indicative of the physiological response to stress across multiple biological systems (i.e., inflammatory, metabolic, and cardiovascular). In addition, the use of multiple measures of adult health would help test the robustness of our findings across multiple domains of health risk. Data collection procedures and biomarker validation are available elsewhere (Hussey et al. 2015; Nguyen et al. 2011; Whitsel et al. 2012).

CRP is produced by the liver in response to inflammation and measures immune function; elevated CRP indicates systemic inflammation. CRP was assayed from dried blood spots collected during the Wave IV interview in Add Health. CRP levels greater than 3 milligrams per liter are considered high risk for cardiovascular disease. We used a continuous measure of CRP, log-transformed to normalize the distribution. The mean level of CRP in the analytic sample was 4.66 (see Table 1). All models for CRP included controls for use of anti-inflammatory drugs, recent illness, and infection. Of the analytic sample, 30% reported recent or current use of anti-inflammatory drugs; results excluding this control are similar to those presented.

Using measured height and weight, we computed BMI, defined as weight in kilograms divided by height in meters squared. The mean BMI for the analytic sample respondents was 29.02, which is on the high end of the overweight categorization. We constructed a measure of hypertension from the average of the second and third systolic and diastolic blood pressure measurements; mean systolic blood pressure 140 or greater or mean diastolic blood pressure 90 or greater are considered hypertensive. We also considered those who self-reported a hypertension diagnosis or used antihypertensive medication as hypertensive. Of the sample, 28% were hypertensive. BMI and hypertension estimates in Add Health have been shown to have high reliability and validity (Hussey et al. 2015; Nguyen et al. 2011).

We constructed an indicator of metabolic syndrome using measures of blood pressure, blood glucose, high-density lipoprotein cholesterol, triglycerides, and waist circumference. When possible, for each biomarker we defined the high-risk threshold according to the guidelines established by the National Cholesterol Education Program Expert Panel. When exact measures were not available, we used an approximation (Bohr, Laurson, and McQueen 2016). High-risk blood pressure was defined as stage 1 or 2 hypertension from measured blood pressure or self-report of doctor-diagnosed hypertension. Glycated hemoglobin levels at 5.7% or greater were defined as high risk. The bottom decile of the sample distribution of

high-density lipoprotein was defined as high risk for men, while the bottom two deciles were defined as high risk for women. A measured value of triglycerides in the top decile of the sample distribution was defined as high risk. Finally, a measured waist circumference of 88 centimeters or greater for women and 102 centimeters or greater for men was defined as high risk. Metabolic syndrome was an indicator, defined as having high-risk levels on three or more of the component risk factors; 14% of the analytic sample was classified as having metabolic syndrome.

Stressors of Instability

There are a number of cascading factors associated with family instability that may lie on the pathway between family instability and poor health. In this article, we are interested primarily in the physiological stress pathway resulting from childhood family instability. This pathway may involve a variety of mechanisms, including parent/child relationship quality, residential instability, and restricted socioeconomic attainment as well as cumulative stress more generally resultant from the uncertainty and unpredictability of the childhood environment (Amato and Patterson 2017). In our analyses, we first included a minimal number of covariates to estimate the unmediated association of family instability with health risk, subsequently adding controls for measurable environmental stressors.

We controlled for potential stressors associated with family instability using measures of SES, parent-child relationship closeness, and residential stability. We used measures of parental and adult educational attainment to control for SES. At Wave I, adolescents reported on feelings of closeness with the biological mother and biological father ranging from 1 (*not at all*) to 5 (*very*). Important to note, this measure refers to the time of survey and not the time of family disruption or particular subjective assessments of the family structure change. We measured residential stability using an indicator for whether the child had lived in the current household since birth.

Health Selection

The relationship between family instability and adult health also may result from health selection, whereby poor child health induces both family instability and poor adult health. To address the possibility of health selection, we controlled for parent reports of birthweight and whether the child was ever breastfed (McDade et al. 2014; Richardson, Hussey, and Strutz 2011).

Behavioral and Socioeconomic Measures

To validate our measure of family instability, we also examined the behavioral and socioeconomic outcomes associated with family instability in existing research. We analyzed four behavioral outcomes—externalizing behavior, internalizing behavior, age at first sex, and number of sexual partners—and two socioeconomic outcomes—years of education and adult income. Externalizing behavior was measured as a count of the number of problem behaviors that the adolescent reported at Wave I. Internalizing behavior was measured similarly as a count of the number of anxiety and depressive symptoms the adolescent reported at Wave I. We constructed a measure of age at first sex by combining adolescent report of sexual debut at Wave I with adult retrospective report of age at first sex at Wave IV

for respondents who had not yet had sex at Wave I. At Wave IV, respondents reported the total number of sexual partners they had had to interview date. Finally, we used the Wave IV adult report of the highest level of education completed and household income. To ensure that the predictor of family instability preceded the outcomes measured at Wave I, in these models we restricted the measure of instability to the number of family structure changes that occurred before Wave I.

METHODS

We analyzed CRP and BMI using linear regressions and analyzed hypertension and metabolic syndrome using logistic regression. In Model 1, we included an indicator for two-biological-parent family structure at birth and BMI in adolescence as well as demographic controls for male gender, age at Wave IV, and a five-category race-ethnicity variable derived from self-report, with categories of white non-Hispanic, black non-Hispanic, Asian, other, and Hispanic. We found no evidence of differential associations depending on gender or race-ethnicity or on age at family change (results not shown). In Model 2, we introduced controls for instability stressors in addition to the Model 1 controls. In Model 3, we added controls for health selection. All analyses used appropriate sampling weights to adjust for unequal probability of selection into the sample and attrition over time, and standard errors were corrected for the school-based clustered design of Add Health (Chen and Chantala 2014).

RESULTS

Childhood Experience of Family Instability

The majority of Add Health children were raised in stable family structures from birth to adolescence (see Table 2). However, the experience of family instability varies greatly by family structure at birth. Roughly two thirds of children born into two-parent families continue to live in that stable family structure across childhood, compared to less than one fifth of children born into single-parent families who experience no family structure transitions. Parental death and incarceration are similarly rare, regardless of family structure at birth. Note that this category includes only instances wherein the change in family structure resulted from a parental death or incarceration with no subsequent transitions; parents who were nonresident at the time of death or incarceration are not captured.

The majority of children from two-parent families who experience one family structure transition do so through parental divorce (9%). Two transitions are actually more common than one (15% vs. 10%, respectively), with the largest sequence category occurring when two biological parents divorce and the coresident biological parent remarries (10%). Of children born into two-parent families, 2% experience another common sequence, from parental divorce to remarriage to the dissolution of the second union. All remaining sequences are combined into categories by number of transitions (other two transitions, other three transitions, and four or more transitions).

The most common family structure sequence for children born into single-parent families is one transition occurring with the entrance of a step-parent (31%). Fourteen percent of

children experience the entry of a biological parent only. A total of 9% of children experience two transitions resulting from a step-parent entry and subsequent exit, and only 1% of children experience an additional step-parent entry for a total of three transitions. All remaining sequences are combined into categories by number of transitions (other two transitions, other three transitions, and four or more transitions).

Descriptive Results

To examine the relationship between childhood family instability and adult health, we tested differences in mean levels and proportions of self-reported BMI in adolescence and physiological health risk in adulthood across family instability categories using adjusted Wald tests and chi-squared tests (see Table 3). There are almost no significant differences in physical health across the number of family structure transitions. The only exception is the level of CRP, which is significantly higher for adults who experienced one transition compared to those who experienced no transitions. In descriptive bivariate associations, there is no evidence of differential health risk in adolescence or adulthood by childhood experience of family instability.

Multivariate Results

Table 4 presents estimates of the association between family instability and each of the four adult physiological health-risk measures. Model 1 controls only for family structure at birth, BMI in adolescence, and respondent demographic characteristics. All outcomes were coded so that increasing values are indicative of poorer health (i.e., higher BMI suggests worse health); therefore, a negative coefficient/odds ratio less than 1 implies better health. We find that family instability is negatively associated with hypertension, with each additional transition associated with 7% lower odds of having hypertension.

In Model 2 (see Table 4), we include controls for the types of stressors that may lie on the pathway from family instability to adult health. The negative association between family instability and adult physiological health is statistically significant for hypertension and metabolic syndrome. Given that the negative association between family instability and health risk is statistically significant when we control for this pathway, the social stressors that are associated with family instability suppress the negative association between family instability and health risk. With each additional change in family structure, individuals have approximately 9% lower odds of being hypertensive and 8% lower odds of having metabolic syndrome compared to those with the same educational attainment, demographic characteristics, and adolescent SES. The magnitude of this association is approximately equivalent to the reduction in the odds of hypertension associated with a two-year increase in education.

It is possible that parents selectively navigate family structure transitions depending on the health of their children. We control for the possibility of health selection in Model 3 (see Table 4), including measures of infant health. There is no substantive change when controlling for birthweight and breastfeeding.

Heterogeneity by Type of Family Transition and Relationship Quality

Next, we examine whether the association depends on the type of family transition for children born into two-biological-parent families (see Table 5). Children who experience the death of a coresident biological parent have marginally higher risk of hypertension in adulthood compared to peers who remain in stable, two-parent families throughout childhood; note the number of respondents in this category is small ($n = 75$). There is no significantly associated change in health risk for children who experience only biological parent divorce. The risk of both hypertension and metabolic syndrome is lower for individuals who experienced four or more transitions. A child born to a two-parent family who experienced four or more family structure transitions by adolescence is expected to have roughly 50% lower odds of having hypertension as an adult and 46% lower odds of having metabolic syndrome compared to his or her counterpart who lived in a stable, two-parent family. Note that the coefficients for hypertension are consistently in the negative direction for all transition categories except death/incarceration, although statistically significant only for four or more transitions.

We examine the possibility that the association between family instability and adult health risk depends on subjective assessments of the experience of transition. As presented in the Methods section, we did not have a direct measure of reported sentiment regarding family change at the time of event. Instead, we used reported relationship quality with biological parents in adolescence as a proxy, assuming current relationship captures, in part, previous feelings. There is no main effect of parental closeness, and while the interaction term between parental closeness and family instability is consistently negative across all models (greater closeness with higher instability reduces health risk), it is not statistically significant (see Table 6). We find no evidence of heterogeneity in the association between family instability and adult health risk by level of reported closeness to biological parents.

Robustness Checks

Thus far we have demonstrated that family instability is associated with lower hypertension among all respondents as well as lower hypertension and metabolic syndrome among those born into two-parent families. We consider two potential explanations in testing the robustness of our findings: the possibility that our measure of instability is not capturing the hypothesized stress and selective attrition in the sample over time.

Validating instability.—One possible explanation is that the measure of family instability is not valid. To test this, we examined the relationship between family instability and the behavioral and socioeconomic outcomes documented in the existing research (see Table 7). The short-term behavioral outcomes in adolescence are all significantly associated with family instability in the expected direction. Greater exposure to family instability is associated with more externalizing and internalizing behavior in adolescence and earlier age at sexual debut. The longer-term outcomes are similarly consistent with existing research. Individuals who experience more family structure transitions in childhood have more sexual partners in adulthood. Children from unstable families also achieve lower educational attainment and have lower earnings in adulthood. All results are robust to the inclusion of additional controls for the stressors of instability and health selection (not shown). These

results replicate the findings of previous research and validate our measure of family instability.

Selective attrition.—Contrary to the existing literature on family instability, which documents deleterious consequences associated with greater numbers of family structure changes, we find family instability is not associated with worse adult health risk. Another possible explanation for our findings is selective attrition across waves. For selective attrition to explain our findings, attrition would need to be greater among respondents with high instability and poor health, and/or low instability and good health. In Appendix A (which can be found with the online version of the article), we investigate this possibility using measures of health and instability at Wave I to predict missingness at Wave IV. Using obesity as a measure of health at Wave I, we find no evidence of selective attrition. Respondents with no experience of instability and normal weight at Wave I are significantly *less* likely to be missing at Wave IV than respondents with no experience of instability who are obese. To the extent that health at Wave I is predictive of health at Wave IV, we find no evidence of selective attrition that would explain our findings.

DISCUSSION

This is the first study to test for adult physiological evidence of the family stress model underlying the family instability hypothesis. Consistent with the family stress model, family instability is associated with negative behavioral and socioeconomic outcomes across the transition from adolescence to adulthood. However, contrary to the predictions derived from the family stress model, we find no evidence that family instability is associated with young adult physical health detriments. Individuals who experienced multiple family structure changes during childhood experience no deleterious metabolic, cardiovascular, or inflammatory consequences in young adulthood compared to their peers in stable, two-parent families throughout childhood. In fact, we find that greater childhood exposure to family structure changes is significantly associated with lower levels of hypertension, suggesting a cardiovascular protection associated with family instability.

It is possible that such family changes may occur due to marital and family conflict, and thus these transitions may actually relieve stress in affected children, with potential benefits to their adult cardiovascular health in particular. Moreover, children who experience family transitions may develop nonparental relationships and noncognitive resources that support their well-being or buffer against stress exposure. It is also possible that we do not observe any adult health detriment associated with family instability because any initial health insult at the time of transition is small and dissipates quickly. Indeed, we find no differences in self-reported BMI in adolescence by experience of family instability. Further research is needed to examine diverse objective health markers earlier in the life course to identify whether there is any immediate or short-term evidence of physical health effects.

We find that parental death is associated with risk of hypertension. This is consistent with a large literature on parental death and orphanhood, which characterizes this event as a childhood trauma rather than a chronic strain (Thoits 1995). Furthermore, it is possible that parental death experienced in childhood is indicative of shared familial premature aging,

morbidity, or mortality and is therefore predictive of worse adult health. With the exception of parental death, other types of family structure transitions do not appear to be differentially related to adult health risk. Future research must consider transitions out of single-parent households at birth as well, which we were unable to examine in this article due to a limited number of cases. While these findings may be surprising in light of a large literature on the negative cognitive and behavioral consequences of family instability, it is consistent with mixed results from the few studies that have examined health outcomes.

One potential explanation for the unexpected findings draws from the adaptive calibration model of stress response. Early-life experiences prime the individual's biological response system for the kinds of environments it can expect throughout the life course (Gunnar and Quevedo 2007; Thompson 2014). This general evolutionary model suggests that exposure to moderate stressors may allow the stress response system to better develop appropriate responses compared to those with no early-life exposure to stressful events (Del Giudice et al. 2011; Ellis and Del Giudice 2014). Moderate stressors include more commonplace events than severe stressors, such as conflict with parents or friends, academic failure, or difficult living situations (Edge et al. 2009). According to this hypothesis, moderate early-life stress may be associated with better behavioral and physiological responses in later life compared to severe or low stress because the individual is more adept at coping with stressors (Shapero et al. 2015).

There is some empirical evidence in support of this model. Repeated exposure to moderately stressful events in animal studies has demonstrated a down-regulation in subsequent stress response (Lyons and Parker 2007; Macri and Würbel 2007). Studies in humans have documented similar associations. Edge and colleagues (2009) use the Risky Family Questionnaire, including questions about family conflict and closeness, to retrospectively assess levels of moderate stress in early life. They find that moderate stress exposure is associated with lower levels of implicit anxiety in adulthood. Shapero and colleagues (2015) find that a history of moderate stressors in childhood, such as bad grades, arguments with parents, teasing, and moving, attenuates the depressive response to subsequent stressors in adolescence. If family instability represents a moderate stressor, children exposed to changes in family structure may have a better-calibrated stress response compared to children with no moderate early-life stress exposures, resulting in better physiological biomarkers of health risk. Future research should test this hypothesis, examining whether childhood exposure to family instability moderates response to later-life stressors.

It is possible that instability is biologically embedded through a mechanism other than stress response. Another related explanation for our findings is that increased exposure to family structure changes exposes individuals to more diverse microbial environments, resulting in a more robust immune system and lower inflammation (Belkaid and Hand 2014). This possibility is supported by our finding that the measured stressors presumed to accompany family instability suppress the relationship between instability and hypertension. In other words, once we control for measured stressors that may account for the stress response pathway, the microbial diversity pathway is strengthened. Such a pathway predicts that individuals who experience family instability would have a lower incidence of infection and a more resilient microbiome due to increased diversity in their social and physical

environments. Researchers with infection and microbiome data may be able to test this pathway directly.

The pathways between childhood family instability and adult health are complex. The results presented here control for a variety of early-life stressors predicted to accompany family instability and predict poor health outcomes. We find that these stressors actually suppress the negative association between family instability and young adult health risk. We also control for adolescent BMI to isolate the adult health association of family instability. If the children who experience family instability have lower BMI in adolescence, our results may understate the relationship between greater family instability and lower young adult health risk. We control for the possibility of health selection by including two retrospectively reported measures of early-childhood health, which are limited. We also demonstrate that family instability is associated with adverse adolescent and adult behavioral and socioeconomic outcomes (see Table 7), which predict worse adult health. While we do not include these controls in the final models presented, our conclusions are robust to including these additional measures (not shown). Thus, our findings overall suggest that despite the intervening factors that are associated with worse health outcomes, there are unmeasured factors that contribute to a net positive cardiometabolic benefit associated with family instability.

There is a need for future research to better conceptualize and measure exposure to moderate, chronic, mundane stressors rather than the acute severe stressors, such as abuse and maltreatment, that are commonly examined. Potential exposures include residential change, school change, and family complexity as well as other types of household instability to which children are exposed resulting from the entrance or exit of casual romantic partners, grandparents, siblings, and kin. Individual appraisal of such events as stressful would also be useful, as would measurement of mediating and moderating resources such as social support and noncognitive resources. Moreover, while this article investigates the stress pathway, more direct tests of this pathway using stress biomarkers such as cortisol response and recovery are necessary to better understand the nature of the family instability stressor and its physiological consequences.

Our findings suggest a modification of the family stress model with respect to physiological indicators of chronic stress exposure, which proposes family structure changes are by nature stressful and therefore deleterious for children. We find evidence consistent with the family stress model in our analysis of behavioral and socioeconomic outcomes. Individuals with greater family instability have more behavioral problems, riskier sexual behavior, and lower SES. In contrast, our analysis of adult health indicates no lasting physiological costs associated with childhood family instability. Since ours is the first study to analyze the medium-term consequences of family instability for objective measures of health in young adulthood to mid-adulthood, it is possible that any short-term biological dysregulation resulting from family instability stress exposure during childhood dissipates quickly.

It is also possible that family instability actually alleviates antecedent stressors occurring in families that are about to experience change (Thoits 2010). If family instability relieves stress compared to staying in unhealthy or tumultuous relationships, then this may improve

health in the long run. Indeed, once known stress factors associated with family instability are controlled, the protective association with health—hypertension in particular—increases. This reinforces the conclusion that something beneficial may be associated with family structure change in the longer term. Future research using prospective measures of parent-child and parental relationship quality before and after family change could investigate this possibility.

The results have important population-level implications for the socioeconomic gradient in health. While lower SES predicts childhood family instability and is subsequently predictive of lower adult SES, net of this disadvantage, family instability is not associated with worse health in adulthood. Further research is needed to better understand the nature of the stressors of family instability, potential mechanisms underlying the observed relationship, and whether such reduced health risks manifest in better health outcomes and healthier aging.

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Table 1.

Descriptive Statistics and Correlations ($n = 10,355$), National Longitudinal Study of Adolescent to Adult Health (Waves I and IV).

Number	Variable	MI%	SD	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1	Adolescent BMI	22.43	4.43	1.00														
2	Adult BMI	29.02	7.41	.69	1.00													
3	Adult C-reactive protein	4.66	8.01	.19	.32	1.00												
4	Adult hypertension	28%	-	.23	.27	.07	1.00											
5	Adult metabolic syndrome	14%	-	.29	.40	.13	.46	1.00										
6	Family instability	.77	1.19	.03	.02	.02	-.03	-.01	1.00									
7	Male	50%	-	.08	.03	-.15	.19	.07	-.02	1.00								
8	Adult age	28.82	1.78	.18	.05	.01	.06	.08	.03	.06	1.00							
9	Parental education	13.59	2.47	-.10	-.11	-.06	-.04	-.06	-.06	.02	-.07	1.00						
10	Years of education	14.57	2.23	-.09	-.11	-.04	-.09	-.10	-.16	-.12	.00	.37	1.00					
11	Closeness to biological mom	4.51	.80	.00	.01	.00	.02	.02	-.07	.09	-.10	-.01	.03	1.00				
12	Closeness to biological dad	3.96	1.23	-.02	-.01	-.02	.02	.01	-.31	.14	-.13	.03	.09	.26	1.00			
13	Never moved from birth	22%	-	.02	.02	.02	.03	.00	-.20	.01	-.01	.00	.05	.01	.09	1.00		
14	Birthweight (ounces)	118.60	2.89	.06	.05	-.04	.00	-.02	-.05	.09	-.02	.04	.03	-.02	.05	.03	1.00	
15	Ever breastfed	46%	-	-.09	-.09	-.05	-.05	-.06	-.07	.01	-.11	.22	.17	.00	.06	-.04	.10	1.00

Note: BMI = body mass index.

Table 2.

Family Instability by Family Structure at Birth, National Longitudinal Study of Adolescent to Adult Health (Wave I).

Origin Structure	<i>n</i>	%	Number of Transitions
Two biological parents			
Stable	5,733	68	0
Parental death/incarceration	75	1	1
Divorce	735	9	1
Adoption	21	0	1
Divorce→remarriage	809	10	2
Other two transitions	403	5	2
Divorce→remarriage→divorce	181	2	3
Other three	185	2	3
Four or more	313	4	4+
<i>Number of unique sequences</i>	<i>210</i>		
<i>Longest sequence</i>	<i>9</i>		
<i>n</i>	<i>8,455</i>		
Single biological parent			
Stable	255	17	0
Parental death/incarceration	20	1	1
Biological parent entry	204	14	1
Step-parent entry	458	31	1
Biological parents switch custody	19	1	1
Adoption	90	6	1
Step-parent entry→divorce	132	9	2
Other two transitions	94	6	2
Step-parent entry→divorce→step-parent entry	12	1	3
Other three	136	9	3
Four or more	70	5	4+
<i>Number of unique sequences</i>	<i>132</i>		
<i>Longest sequence</i>	<i>8</i>		
<i>n</i>	<i>1,490</i>		

Table 3.

Physiological Health Risk by Family Instability, National Longitudinal Study of Adolescent to Adult Health (Waves I and IV).

Physiological Health Risk	Number of Family Transitions			
	0	1	2	3+
Adolescent BMI	22.43	22.68	22.60	22.51
Adult CRP	4.45	5.15 ^a	4.96	4.82
Adult BMI	28.70	29.35	29.02	28.69
Adult hypertension	.27	.31	.27	.22
Adult metabolic syndrome	.14	.16	.13	.12
Percentage	56	19	16	9
<i>n</i>	10,355			

Note: BMI = body mass index; CRP = C-reactive protein.

^aIndicates that the mean differs significantly ($p < .01$) from zero family transitions using adjusted Wald tests for CRP and BMI and chi-squared tests for hypertension and metabolic syndrome.

Table 4. Childhood Experience of Family Instability and Adult Health, National Longitudinal Study of Adolescent to Adult Health (Waves I and IV).

Childhood Experience	In(CRP)			BMI			Hypertension			Metabolic Syndrome		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Family instability	.02 (.02)	.01 (.02)	.01 (.02)	-.03 (.06)	-.06 (.06)	-.05 (.06)	.93 [*] [.88, .98]	.91 ^{**} [.86, .97]	.91 ^{**} [.86, .97]	.96 [.90, 1.02]	.92 [*] [.86, .99]	.92 [*] [.86, .99]
Two biological parents at birth	-.07 (.04)	-.05 (.05)	-.03 (.05)	-.28 (.17)	-.22 (.18)	-.22 (.19)	.82 [*] [.68, .99]	.86 [.71, 1.04]	.88 [.73, 1.06]	.85 [.67, 1.07]	.91 [.72, 1.15]	.92 [.73, 1.16]
Adolescent BMI	.08 ^{***} (.00)	.08 ^{***} (.00)	.08 ^{***} (.00)	1.18 ^{***} (.02)	1.17 ^{***} (.02)	1.16 ^{***} (.02)	1.11 ^{***} [1.09, 1.12]	1.10 ^{***} [1.09, 1.12]	1.10 ^{***} [1.09, 1.12]	1.16 ^{***} [1.14, 1.18]	1.15 ^{***} [1.14, 1.17]	1.15 ^{***} [1.14, 1.17]
Male	-.55 ^{***} (.03)	-.57 ^{***} (.03)	-.57 ^{***} (.03)	-.39 ^{***} (.15)	-.46 ^{***} (.14)	-.49 ^{***} (.14)	2.26 ^{***} [1.94, 2.62]	2.24 ^{***} [1.92, 2.61]	2.28 ^{***} [1.96, 2.65]	1.46 ^{***} [1.25, 1.71]	1.43 ^{***} [1.20, 1.69]	1.44 ^{***} [1.21, 1.70]
Age	-.02 [*] (.01)	-.02 (.01)	-.02 [*] (.01)	-.40 ^{***} (.04)	-.39 ^{***} (.04)	-.39 ^{***} (.04)	1.04 [†] [1.00, 1.08]	1.04 [†] [.99, 1.08]	1.03 [.99, 1.08]	1.03 [.98, 1.09]	1.03 [.98, 1.09]	1.03 [.98, 1.09]
<i>Race-ethnicity (white, non-Hispanic)</i>												
Black, non-Hispanic	-.02 (.05)	-.04 (.05)	-.08 (.06)	.61 ^{**} (.20)	.56 ^{**} (.20)	.56 ^{**} (.20)	1.13 [.94, 1.37]	1.11 [.92, 1.34]	1.07 [.89, 1.29]	1.24 [†] [.98, 1.58]	1.23 [†] [.97, 1.55]	1.20 [.95, 1.53]
Asian	-.42 ^{***} (.10)	-.37 ^{***} (.10)	-.36 ^{***} (.10)	-.122 ^{***} (.33)	-.107 ^{***} (.33)	-.94 ^{***} (.34)	1.06 [.77, 1.46]	1.10 [.81, 1.50]	1.08 [.79, 1.48]	1.50 [†] [.97, 2.33]	1.64 [*] [1.06, 2.54]	1.64 [*] [1.05, 2.55]
Other	-.06 (.16)	-.10 (.15)	-.09 (.15)	.57 (.78)	.48 (.78)	.51 (.77)	1.14 [.72, 1.81]	1.10 [.71, 1.72]	1.10 [.71, 1.70]	.71 [.33, 1.54]	.66 [.30, 1.50]	.66 [.29, 1.51]
Hispanic	.08 (.05)	.02 (.05)	.03 (.05)	.39 [†] (.23)	.14 (.24)	.19 (.24)	.90 [.72, 1.13]	.86 [.68, 1.08]	.86 [.68, 1.08]	1.11 [.87, 1.40]	1.00 [.79, 1.26]	1.00 [.79, 1.27]
Parental education		-.02 [*] (.01)	-.01 (.01)		-.11 [*] (.04)	-.10 [*] (.04)		.98 [.96, 1.01]	.99 [.96, 1.02]		.97 [†] [.94, 1.00]	.97 [†] [.94, 1.00]
Years of education		-.04 ^{***} (.01)	-.03 ^{***} (.01)		-.09 [*] (.04)	-.09 [*] (.04)		.96 [*] [.93, 1.00]	.96 [*] [.93, 1.00]		.92 ^{***} [.88, .96]	.92 ^{***} [.88, .96]
Closeness to biological mom		.06 [*] (.02)	.05 [*] (.02)		.01 (.12)	.02 (.12)		.99 [.91, 1.07]	.98 [.91, 1.07]		1.02 [.92, 1.14]	1.02 [.92, 1.13]
Closeness to biological dad		.01 (.02)	.01 (.02)		.05 (.06)	.05 (.06)		.97 [.92, 1.01]	.97 [.92, 1.01]		.97 [.90, 1.05]	.97 [.90, 1.06]
Never moved from birth		.04 (.04)	.03 (.04)		.02 (.16)	.01 (.16)		1.06 [.91, 1.24]	1.06 [.91, 1.24]		.94 [.77, 1.16]	.94 [.77, 1.16]
Birthweight (ounces)			-.00 [†] (.00)			.01 [*] (.00)			1.00 [*] [.99, 1.00]		1.00 [.99, 1.00]	1.00 [.99, 1.00]

Childhood Experience	ln(CRP)			BMI			Hypertension			Metabolic Syndrome		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Ever breastfed			-.13 *** (.04)			-.30 * (.14)			.94 [.83, 1.06]			.96 [.79, 1.16]
Adjusted R^2 / F -adjusted test statistic	.14	.15	.16	.49	.49	.49	.70	.62	2.05	2.75	6.56	4.25
n		9,125			10,049			9,916			8,717	
Model specification		Linear			Linear			Logit			Logit	

Note: BMI = body mass index; CRP = C-reactive protein. Standard errors are in parentheses for linear models; 95% confidence intervals are in brackets for nonlinear models. All models include appropriate sample weights and correct for clustered sampling design. CRP models include controls for recent infections and use of anti-inflammatory drugs.

[†] $p < .10$,

* $p < .05$,

** $p < .01$,

*** $p < .001$.

Table 5.

Type of Family Transition from Two-parent Family and Adult Health, National Longitudinal Study of Adolescent to Adult Health (Waves I and IV).

Variables	ln(CRP)	BMI	Hypertension	Metabolic Syndrome
<i>Type of transition (stable)</i>				
Parental death/incarceration	.03 (.17)	-.57 (.58)	1.99 [†] [.91, 4.37]	1.23 [.50, 3.01]
Divorce	.01 (.07)	-.13 (.32)	.83 [.64, 1.09]	.87 [.60, 1.27]
Divorce/remarry	.02 (.08)	.26 (.28)	.97 [.73, 1.28]	.75 [†] [.53, 1.06]
Other two	-.01 (.09)	-.39 (.49)	.92 [.63, 1.33]	.73 [.45, 1.18]
Divorce/remarry/divorce	-.19 (.13)	-.41 (.47)	.72 [.45, 1.15]	.92 [.48, 1.75]
Other three	.13 (.15)	.21 (.64)	.88 [.56, 1.36]	1.37 [.74, 2.53]
Four or more	.17 [†] (.09)	-.40 (.40)	.49** [.30, .80]	.54* [.32, .92]
Adolescent BMI	.08*** (.00)	1.17*** (.03)	1.10*** [1.09, 1.12]	1.16*** [1.14, 1.19]
Male	-.60*** (.03)	-.44** (.15)	2.30*** [1.97, 2.70]	1.48*** [1.21, 1.81]
Age	-.02 [†] (.01)	-.41*** (.05)	1.03 [.98, 1.08]	1.02 [.96, 1.09]
<i>Race-ethnicity (white, non-Hispanic)</i>				
Black, non-Hispanic	-.05 (.06)	.93*** (.23)	1.16 [.93, 1.44]	1.18 [.90, 1.55]
Asian	-.31** (.12)	-.70 [†] (.40)	1.20 [.87, 1.64]	1.72 [†] [1.00, 2.96]
Other	-.09 (.19)	.17 (.96)	1.02 [.57, 1.81]	.62 [.22, 1.80]
Hispanic	.03 (.06)	.11 (.29)	.83 [.64, 1.08]	.96 [.73, 1.26]
Parental education	-.02* (.01)	-.11* (.05)	.98 [.95, 1.01]	.97 [.93, 1.01]
Years of education	-.03** (.01)	-.10* (.04)	.97 [.93, 1.01]	.93** [.89, .97]
Closeness to biological mom	.07* (.03)	.08 (.15)	.99 [.90, 1.08]	1.08 [.95, 1.22]
Closeness to biological dad	.02 (.02)	.07 (.08)	.98 [.92, 1.04]	.96 [.88, 1.04]
Never moved from birth	.05 (.04)	.02 (.17)	1.07 [.90, 1.27]	.94 [.76, 1.16]
Birthweight (ounces)	-.00** (.00)	.01* (.00)	1.00 [.99, 1.00]	1.00 [.99, 1.00]
Ever breastfed	-.10** (.04)	-.28 [†] (.15)	.92 [.80, 1.06]	.93 [.75, 1.16]

Variables	ln(CRP)	BMI	Hypertension	Metabolic Syndrome
Adjusted R^2 /F-adjusted test statistic	.17	.50	.89	2.15
n	7,448	8,206	8,103	7,115
Model specification	Linear	Linear	Logit	Logit

Note: BMI = body mass index; CRP = C-reactive protein. Standard errors are in parentheses for linear models; 95% confidence intervals are in brackets for nonlinear models. All models include appropriate sample weights and correct for clustered sampling design. CRP models include controls for recent infections and use of anti-inflammatory drugs.

[†] $p < .10$,

* $p < .05$,

** $p < .01$,

*** $p < .001$.

Table 6.

Family Instability by Feelings of Closeness to Parents, National Longitudinal Study of Adolescent to Adult Health (Waves I and IV).

Variables	ln(CRP)		BMI		Hypertension		Metabolic Syndrome	
	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
Family instability	.10 (.08)	.01 (.05)	.10 (.32)	-.09 (.18)	.99 [.74, 1.33]	1.00 [.87, 1.16]	.95 [.65, 1.40]	.99 [.82, 1.18]
Closeness to biological mom	.07** (.03)	.05* (.02)	.05 (.15)	.02 (.12)	1.00 [.91, 1.10]	.98 [.90, 1.06]	1.03 [.89, 1.19]	1.02 [.91, 1.13]
Closeness to biological dad	.01 (.02)	.01 (.02)	.04 (.06)	.04 (.08)	.98 [.91, 1.05]	1.00 [.94, 1.06]	.99 [.91, 1.08]	.99 [.90, 1.10]
Family instability by closeness to mom	-.02 (.02)		-.03 (.07)		.96 [.92, 1.02]		.97 [.90, 1.06]	
Family instability by closeness to dad		-.00 (.01)		.01 (.05)		.97 [.93, 1.01]		.98 [.93, 1.03]
Two biological parents at birth	-.03 (.05)	-.03 (.05)	-.21 (.19)	-.21 (.18)	.88 [.73, 1.06]	.86 [.71, 1.05]	.92 [.73, 1.16]	.91 [.72, 1.15]
Adolescent BMI	.08*** (.00)	.08*** (.00)	1.16*** (.02)	1.17*** (.02)	1.10*** [1.09, 1.12]	1.10*** [1.09, 1.12]	1.15*** [1.14, 1.17]	1.15*** [1.14, 1.17]
Male	-.57*** (.03)	-.57*** (.03)	-.49*** (.14)	-.49*** (.14)	2.28*** [1.96, 2.65]	2.28*** [1.96, 2.65]	1.44*** [1.21, 1.70]	1.44*** [1.21, 1.70]
Age	-.02 [†] (.01)	-.02* (.01)	-.39*** (.04)	-.39*** (.04)	1.03 [.99, 1.08]	1.03 [.99, 1.08]	1.03 [.98, 1.09]	1.03 [.98, 1.09]
<i>Race-ethnicity (white, non-Hispanic)</i>								
Black, non-Hispanic	-.08 (.06)	-.08 (.06)	.56** (.20)	.56** (.20)	1.07 [.89, 1.29]	1.08 [.89, 1.30]	1.20 [.95, 1.53]	1.21 [.95, 1.53]
Asian	-.36*** (.10)	-.36*** (.10)	-.94*** (.35)	-.94*** (.34)	1.08 [.79, 1.48]	1.09 [.80, 1.48]	1.64* [1.05, 2.56]	1.64* [1.05, 2.56]
Other	-.10 (.15)	-.09 (.15)	.51 (.77)	.51 (.77)	1.10 [.71, 1.69]	1.10 [.71, 1.70]	.66 [.29, 1.50]	.66 [.29, 1.50]
Hispanic	.03 (.05)	.03 (.05)	.20 (.24)	.19 (.24)	.86 [.68, 1.08]	.86 [.68, 1.08]	1.00 [.79, 1.27]	1.00 [.79, 1.27]
Parental education	-.01 (.01)	-.01 (.01)	-.10* (.04)	-.10* (.04)	.99 [.96, 1.02]	.99 [.96, 1.02]	.97 [†] [.94, 1.00]	.97 [†] [.94, 1.00]
Years of education	-.03*** (.01)	-.03*** (.01)	-.09* (.04)	-.09* (.04)	.96* [.93, 1.00]	.96* [.93, 1.00]	.92*** [.88, .96]	.92*** [.88, .96]
Never moved from birth	.03 (.04)	.03 (.04)	.01 (.16)	.01 (.16)	1.06 [.91, 1.24]	1.06 [.91, 1.24]	.94 [.77, 1.16]	.94 [.77, 1.16]

Variables	ln(CRP)		BMI		Hypertension		Metabolic Syndrome	
	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
Birthweight (ounces)	-.00 [†] (.00)	-.00 [†] (.00)	.01 [*] (.00)	.01 [*] (.00)	1.00 [*] [.99, 1.00]	1.00 [*] [.99, 1.00]	1.00 [.99, 1.00]	1.00 [.99, 1.00]
Ever breastfed	-1.13 ^{***} (.04)	-1.13 ^{***} (.04)	-.30 [*] (.14)	-.30 [*] (.14)	.94 [.83, 1.06]	.94 [.83, 1.06]	.96 [.79, 1.16]	.96 [.79, 1.16]
Adjusted R^2/F -adjusted test statistic	.16	.16	.49	.49	1.48	1.32	4.17	3.59
n	9,125		10,049		9,916		8,717	
Model specification	Linear		Linear		Logit		Logit	

Note: BMI = body mass index; CRP = C-reactive protein. Standard errors are in parentheses for linear models; 95% confidence intervals are in brackets for nonlinear models. All models include appropriate sample weights and correct for clustered sampling design. CRP models include controls for recent infections and use of anti-inflammatory drugs.

[†] $p < .10$.

^{*} $p < .05$.

^{**} $p < .01$.

^{***} $p < .001$.

Table 7.

Family Instability on Behavioral/Socioeconomic Outcomes, National Longitudinal Study of Adolescent to Adult Health (Waves I and IV).

Variables	Wave I		Age at First Sex	Wave IV		
	Externalizing Behavior	Internalizing Behavior		Number of Sexual Partners	Years of Education	Income
Family instability	.22 *** (.02)	.21 *** (.04)	1.03 *** [1.02, 1.04]	1.07 *** [1.04, 1.10]	-.29 *** (.03)	-.06 *** (.01)
Two biological parents at birth	-.22 *** (.04)	-.53 *** (.10)	.97 ** [.95, .99]	.85 *** [.78, .92]	.70 *** (.07)	.16 *** (.03)
Male	.23 *** (.03)	-1.00 *** (.09)	.99 [.98, 1.01]	1.55 *** [1.46, 1.64]	-.57 *** (.05)	.34 *** (.03)
Age	.21 *** (.01)	.29 *** (.03)	.99 *** [.98, .99]	1.01 [.99, 1.03]	.04 (.03)	.05 *** (.01)
<i>Race-ethnicity (white, non-Hispanic)</i>						
Black, non-Hispani	.11 (.07)	.38 ** (.13)	1.05 ** [1.01, 1.09]	1.16 ** [1.06, 1.28]	-.34 * (.17)	-.25 *** (.05)
Asian	-.02 (.10)	1.10 *** (.24)	.92 ** [.87, .97]	.68 *** [.54, .85]	.65 ** (.20)	.19 ** (.06)
Other	.15 (.13)	.38 (.44)	.96 [.89, 1.05]	1.21 [.89, 1.64]	-.22 (.29)	-.13 (.09)
Hispanic	.24 *** (.07)	.72 *** (.14)	.98 [.94, 1.02]	.90 [†] [.81, 1.00]	-.64 *** (.12)	-.04 (.04)
Specification	OLS	OLS	Survival	Poisson	OLS	OLS
R ²	.12	.05	.08	.05	.08	.06
n	14,694	14,754	13,842	13,976	14,750	13,037

Note: OLS = ordinary least squares. Standard errors are in parentheses for linear models; 95% confidence intervals are in brackets for nonlinear models. All models include appropriate sample weights and correct for clustered sampling design.

[†] $p < .10$,

* $p < .05$,

** $p < .01$,

*** $p < .001$.