Early Childhood Family Stressors and Immune System Dysregulation in Adolescence

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Abstract

Exposure to stress is one way in which social disadvantages during childhood may alter biological and psychological systems with long-term consequences. Family social and economic conditions are critical for early childhood development and exposure to difficult family conditions may have lasting physiological effects. Yet there is little research linking early childhood conditions with physiological indicators of stress and system dysregulation in adolescence. In this study, we assess how family social and economic hardships that occurred in early childhood (birth to age 5) are associated with immune system dysregulation in adolescence, as indicated by reactivation of the Epstein-Barr virus (EBV). We utilize a novel biomarker of EBV reactivation obtained through saliva, a non-invasive method of collecting immune-system biomarkers, in 674 adolescents 11-17 years old. Multivariate regression results indicated that experiences of moving into a new caregiver household and economic hardship during early childhood were associated with elevated risk for EBV reactivation in adolescence. The findings held even when accounting for adolescents' current family conditions. The results suggest family instability and economic insecurity in early childhood may increase chronic stress exposure during childhood with lasting consequences for immune system dysregulation in adolescence.

Keywords: Epstein-Barr virus; virus reactivation; childhood stress; adolescent health

Introduction

Research on the social determinants of health suggests that social environments are important upstream determinants of health. Social environments experienced during childhood may be particularly critical for embedding social and economic disadvantages under the skin (Berens et al., 2017), with lasting consequences for individuals' health and population health disparities. Exposure to stress is one way in which social disadvantages during childhood may alter biological and psychological systems and produce health disparities. Biological embedding can occur through repeated stress exposures that result in dysregulated neuroendocrine and immune systems (Berens et al., 2017; Kuhlman et al., 2017). Both types of dysregulation in childhood have been associated with long-term health consequences (Barr, 2017; Berens et al., 2017). Further, research on adults suggests relatively common stressors may affect immune responses (Kiecolt-Glaser, 1999), and that the precursors to dysregulated immune systems likely begin during childhood (Elwenspoek et al., 2017). However, we know relatively little about social contexts and stress in children, particularly those that result in physiological changes that last through later stages of the life course.

In this study, we consider how family instability and insecurity in early childhood may result in biological embedding of family disadvantages through the dysregulation of children's immune systems. We test this idea by assessing how family instability during early childhood years is associated with Epstein-Barr virus (EBV) reactivation during adolescence. EBV is a ubiquitous virus in the U.S.; an estimated 80% of youth have been exposed to EBV by age 19 years (Dowd et al., 2013). After primary exposure, EBV remains latent in the body but can be reactivated as indicated by increases in replication of the viral DNA and antiviral antibodies (Ford and Stowe, 2017, 2013; Stowe et al., 2010). EBV reactivation has been posited as one

indication the individual's immune system has been dysregulated due, in part, to chronic stress exposure (Glaser et al., 1991). Building on family and biosocial research, we pose the following research questions: (1) Are disruptions in family structure during childhood associated with salivary shedding of EBV DNA in adolescence? (2) Are family economic hardships during childhood associated with EBV DNA shedding in adolescence? (3) Do adolescents' current family disadvantages account for the effects of childhood family instability or family economic hardship on EBV reactivation?

To address our research questions, we use data from a recent study of adolescents in and around Columbus, Ohio, known as the Adolescent Health and Development in Context (AHDC) study. The data used here include youth (ages 11-17 years) and caregiver survey data, as well as biomarkers of EBV obtained through saliva samples from a representative subsample of the youth (N=674) who participated in the Linking Biological and Social Pathways to Adolescent Health and Well-Being sub-study. The survey data provide retrospective information about family instability and economic insecurities faced by the youth during childhood as reported by the caregiver. For the purposes of this study, we focus on family-level changes that were reported as part of a life events list and whether these changes are associated with later salivary shedding of EBV DNA in adolescence.

Background

In conceptualizing children's social environments at various levels, the Bronfenbrenner bioecological model of development emphasizes family contexts as the most proximate influences on child health and development (Bronfenbrenner, 2001). Children interact directly with their family environments, particularly in early childhood, and children need supportive, nurturing and stable family environments to support their physical, mental, and emotional health.

Even as children grow and experience other environments, families continue to serve as important buffers between children and the larger communities and world in which they live.

Research on the changing family contexts of children in the U.S. has indicated family transitions and instability (i.e., changes in living arrangements, parental residency status and household economic resources) are present in the lives of many children, due to high divorce, cohabitation, imprisonment rates, and unstable job markets (Brown et al., 2016; Desmond and Perkins, 2016; Perkins, 2017; Ruggles, 2015). Changes in children's family contexts, particularly changes in parent or caregiver status, may have both social and economic consequences. Socially, children may experience less time and emotional support and harsher parenting during and after family transitions (Beck et al., 2010), with the potential to increase their stress levels. Children may also experience stress indirectly through changes in interactions with their caregivers due to increases in caregiver stress that may occur with family instability/transitions (Beck et al., 2010; Dush, 2013). Economically, family transitions are often accompanied by declines in income (Avellar and Smock, 2005; Tach and Eads, 2015) and can increase the risk of material hardship (Osborne et al., 2012), which, in turn, may increase caregiver stress and depression (Williams and Cheadle, 2016), and reduce resources needed to provide adequate shelter, care and food for children.

Across several studies, family instability has been linked to worse health and developmental outcomes among children (Bzostek, 2008; Lee and McLanahan, 2015; Schmeer, 2011) and adolescents (Brown, 2006; Cavanagh, 2008). However, a recent review suggests that the effects on children may depend on the type of transition, outcome considered, and stage of childhood (Hadfield et al., 2018). A recent study also highlights the importance of non-parental related changes in household composition (Perkins, 2017). Moving in or out of a grandparent

household may be particularly important, as an estimated 30% of U.S. children ever co-reside with a grandparent and this is more likely to occur during early childhood (Amorim et al., 2017). Further, grandparent households with children appear to be associated with increased material hardship such as food insecurity (Ziliak and Gundersen, 2016).

Whether unstable or insecure family environments create sufficient stress among children to alter their biological systems, (i.e. biologically embedding), has yet to be determined. Most research in this area has focused on "toxic" stress in children, or those exposures that are severe in nature, such as abuse and neglect (Berens et al., 2017; Shonkoff and Garner, 2012). However, these sources of stress occur in a relatively small portion of the population and are unlikely to explain the large disparities in health found in the U.S.

Research is beginning to show that more common stressors related to low family SES, for example, are associated with inflammation and hair cortisol (biomarkers of stress) in children (Dowd et al., 2010; Schmeer and Yoon, 2016; Vliegenthart et al., 2016). Research also suggests that children's physiological responses to difficult family contexts begin as early as infancy and may be affected by more typical family stressors. For example, a study of 1135 young children (mostly low-income) followed from 7-48 months found that poor housing quality and lower levels of positive caregiving behavior were associated with higher levels of basal salivary cortisol (a physiological response to stress via the hypothalamic-pituitary-adrenal [HPA] axis pathway) over the time period studied. Further, two or more adult exits from the home was associated with higher basal salivary cortisol levels at all four time points from 7-48 months of age. Perceived high economic insufficiency (top 25% of sample) was associated with resting salivary cortisol at 7 months, and a lower decline in resting cortisol between the 7 month and subsequent waves compared to those below the 25th percentile in economic insufficiency (Blair

et al., 2011). Another study of 201 low income 2-year old children found increased basal salivary cortisol levels among those experiencing more family instability, using a composite measure of instability (caregiver changes, residential changes, caregiver partner changes, job loss and family member deaths), retrospectively reported for the prior 3 years (Sturge-Apple et al., 2017). These studies, suggest that both social and economic aspects of family instability may be important for the development of children's stress response and related physiological systems.

These stressful conditions, and resulting biological changes, may be long lasting, as research is beginning to point to long-term effects of stress during childhood for later biological responses (Elwenspoek et al., 2017). Recently, a study found significantly more DNA methylation among adolescents who had been orphaned and then adopted during childhood compared with those who were non-orphans, net of their health during adolescence (Esposito et al., 2016). Thus, early experiences related to inadequate or insecure caregiving suggests that stressful family conditions can alter children's physiology with long-term implications. Early childhood family conditions may be critical determinants of biological embedding due to the rapidly developing neuroendocrine and immune systems during this period of the life course (McEwen and McEwen, 2017).

Social adversity and virus reactivation.

Biosocial research has long supported the idea that one way individuals respond physiologically to stress is through a reactivation of latent viruses (Glaser et al., 1991). This can be assessed among individuals with prior exposure to viruses, such as herpes simplex virus (HSV), cytomegalovirus (CMV), or Epstein-Barr virus (EBV). These viruses are often acquired during childhood/adolescence and remain latent with few symptoms in the body unless a physical or psychosocial threat activates the immune system. Studies have established

associations between viral reactivation in adulthood with childhood neighborhood (Ford and Browning, 2015) and family adversity (Elwenspoek et al., 2017; Janicki-Deverts et al., 2014), well as concurrent marital stress (Kiecolt-Glaser, 1999; Kiecolt-Glaser et al., 2011, 2010).

Regarding reactivation during adolescence, studies of youth in the U.S. have found that severe childhood conditions, such as abuse and institutionalization, were linked with reactivation of HSV during adolescence (Shirtcliff et al., 2009). Further, in a national study, lower SES and early experiences of abuse during childhood were associated with higher EBV reactivation levels (antibody titers) in young adulthood. Among those exposed to physical abuse, those who were abused in early childhood (ages 3-5) had higher EBV levels than those first abused during adolescence (Slopen et al., 2013a, 2013b). As a whole, this body of research suggests the possible importance of either early life experiences or the cumulative effects of stressors beginning in early childhood for later viral reactivation.

We know less about how other, more common, sources of family stress may affect children's developing immune systems. More regularly occurring events, such as changes in family structure during childhood may produce physiological changes that result in adolescent immune system dysregulation if children face repeated stress due to these conditions over time or during a sensitive developmental period.

In this study, we assess the role of family transitions during early childhood (birth to age 5) as a sensitive developmental period and a time when children are highly dependent on stable family environments and positive caregiver interactions (Shonkoff and Garner, 2012). We consider various types of family change reported for birth to age 5, including changing parental partners and children who move into or out of new parent/caregiver households, and their associations with EBV reactivation during adolescence (ages 11-17 years). We also assess early

childhood economic hardship as a potential additional source of family instability during this period, and its implications for EBV DNA shedding during adolescence. Finally, given that early family instability could set children on a track for increased family structure or economic disadvantages during adolescence, we assess whether family conditions during adolescence are pathways through which early childhood family transitions and/or economic hardship affect the risk of EBV reactivation in adolescence.

Methods

Data and Sample

The AHDC emphasizes the interplay of social, psychological, and biological processes in shaping youth developmental outcomes such as health risk and pro-social behavior, mental and physical health, and educational outcomes. During 2014-2016 the study collected data on multiple contexts of youth development from a representative sample of households with adolescents ages 11-17 residing in an urbanized area of Franklin County, OH (containing a majority of the city of Columbus and several suburban municipalities) using a prospective cohort design. Informed consent was obtained from all individual participants included in the study. The data were collected over a week, beginning with entrance surveys with the focal youth and his/her caregiver, followed by a 7-day period when smartphones were used to obtain ecological momentary assessment (EMA) data. The data collection was concluded with an end of the week in-home exit survey, which included the collection of saliva, later analyzed for presence of EBV VCA IgG antibodies and EBV DNA in a representative sub-sample of the youth (N=674). A full description of the EBV sample and analytical procedures for extracting EBV DNA can be found in a previously published study (Ford and Stowe, 2017). Given our interest in stress as indicated by a reactivation of the previously acquired EBV, we limit our sample to adolescents with prior

exposure to EBV (youth who had a salivary EBV VCA IgG antibody level greater than 0.02 were considered EBV positive as noted in Ford & Stowe, 2017), which was approximately 80% of the EBV sample (N=540). We also conducted supplementary analyses using the full EBV sample to assess whether the results found could be attributed to higher likelihood of contracting EBV (first exposure). The findings from these analyses are discussed in the results section and tables are available upon request.

Measures

Our dependent variable - salivary shedding of EBV DNA is a dichotomous measure created to compare youth who had 10 or more copies (the lower bound detectable level) of EBV DNA in their saliva (yes=1) to those youth who were below the detectable level. Salivary shedding of the virus indicates replication of the DNA of the virus and is suggestive of EBV reactivation. As Table 1 indicates, approximately 65% of the salivary EBV positive youth had evidence of EBV reactivation.

Our independent variables of interest were assessed based on a series of questions asked of the caregiver about the family conditions of the adolescent during childhood, with particular reference to early childhood (birth to age 5). Our measures of family instability included whether the child experienced the following: parents' divorce, parent's partner moving into the household; parent's partner moving out of the household; the child moved into a grandparent household; the child went to live with new caregiver; the child moved into a different parent's household; and, the child moved out of a grandparent's household. We constructed a total instability score summing the instability measures and capping the top score at four, given the small number of children with more than four of these changes during the first 5 years of life. In conducting robustness checks, we modeled total family instability linearly, categorically and as a

dummy variable for "high" instability. All results were similar so we show only the linear variable results here. We also created five variables that represented particular types of change: parental divorce, partner moved in, partner moved out, any new family setting (child moved into a grandparent, new caregiver, or a new parent's home), and child moved out of a grandparent home. In all models, we also assessed whether the child had ever lived with only one adult in the household during this period, to separate out single-parent from family instability effects.

To answer our second research question, of whether these family instability effects were due to economic hardship during the same period, we measured caregiver reports of childhood economic adversity between birth and age five. This included whether the child experienced the following family hardships: homelessness, eviction/foreclosure, bankruptcy, trouble paying bills, receipt of government assistance, or parental job loss. We created a dummy variable indicating exposure to any of these family economic hardships during the early childhood period.

Finally, we assessed the adolescents' current family situation in terms of family structure and income, as possible additional effects of concurrent family conditions on EBV reactivation. The family measures included were: caregiver marital status (married, cohabiting, never married, divorced, separated, or widowed), number of children in the household, and total household income during the past year (defined as a categorical variable of below $30,000, $30-60,000, and above $60,000).

Our covariates included adolescent demographic characteristics: age, sex, race and ethnicity (white, black, Hispanic, other). We also included the adolescents' caregiver characteristics age, sex and education level (high school degree or less, some college, and college degree or more).

Table 1 shows the sample characteristics. Almost a quarter of the adolescents lived in a single-adult household at some point during early childhood. Most had no family instability events during this time (72%). Thirteen percent experienced one family structure change and almost 10% experienced two family changes. The remaining 5% experienced 3 or more changes in family structure as characterized in this study. Of the types of family change, the child moving into a new household (grandparent, new caregiver, or different parent) was the most common (12%). Ten percent experienced parental partners moving in and 8.5% partners moving out during early childhood. The least common family structure changes were parental divorce (6%) and moving out of a grandparent household (5%) between birth and age five. In addition to family structure instability, almost 44% of the youth experienced family economic hardship during these early years.

Insert Table 1 here.

Statistical Analysis

Of the 540 EBV positive youth, 60 had one or more independent variables missing. Although this is not a large number of missing cases, we preferred to impute the data to maintain as large of a sample as possible. We utilized multiple imputation with chained equations using the MI suite in Stata. The use of chained equations allowed us to make specific distributional assumptions for each covariate to ensure that plausible values were imputed for each variable. We created 25 imputed datasets for use in our analysis. The dependent variable was used in the imputation process but cases with missing EBV data were dropped from the analysis (von Hippel 2007).

Using the imputed data, we conducted logistic regression analysis, given the binary nature of our dependent variable, reactivated with evidence of salivary shedding of EBV DNA or

not. Models were also assessed using the linear values of the amount of viral shedding with the "non-activated" considered censored values (using Tobit regression). The results, which were consistent with the dummy variable models presented here, are not included but are available upon request. We tested gender and race interactions with family instability and found no significant interaction effects. Our results are presented as odds ratios (exponentiated coefficients) for ease of interpretation. We report significant levels up to $p<0.1$.

Results

Table 2 provides the results from the multiple regression models. Model 1 shows the associations between total family instability and living in a single-adult household during early childhood with the odds of EBV reactivation in adolescence, controlling for current adolescent and caregiver characteristics. Neither single adult nor total amount of family instability are significant. Further testing of the categorical or dummy variable for high instability showed similar null findings.

Insert Table 2 here.

In model 2 we separate out the family instability measure into various types of changes, in an effort to determine whether certain types of family structure change in early childhood are associated with later EBV reactivation. The results indicate that moving into a new family household (grandparent, new caregiver or new parent) during early childhood is associated with two and a half times the odds of EBV reactivation during adolescence. The other types of changes do not appear to be associated with later EBV reactivation. In order to rule out collinearity problems, models were tested with each type of change variable individually entered

and the findings were the same; of the types of change assessed, moving into a new family household was the only family instability event associated with later EBV reactivation.

Model 3 adds economic hardship to the model to consider its association with EBV reactivation; and whether the early family instability is significantly associated with adolescent EBV reactivation net of economic hardships experienced during early childhood. The results indicate that adolescents who experienced economic hardship during early childhood have almost 1.5 higher odds of EBV reactivation than those who did not have such experiences. This effect appears to be additive to the association of moving into a new family household, which remains significant and similar in magnitude as in Model 2.

In model 4 we added current adolescent family characteristics that may also contribute to adolescents' EBV reactivation. Of the family structure and household income variables, only number of children in the household is significantly associated with reactivation of EBV among adolescents. The odds of reactivation increases by 26% for each additional child. The odds ratios for both moving into a new family household and economic hardship remained similar in magnitude and significance when including the adolescent family characteristics in the model.

Of the covariates included, older age is associated with a decrease and African American race with an increase in odds of reactivation during adolescence.

To further check the robustness of these results, we assessed the same independent variables in predicting the odds that the adolescent was ever exposed to EBV before the survey (i.e., salivary EBV VCA IgG positive status). The results indicated that family instability was not associated with the odds of being previously exposed to EBV. However, early childhood economic hardship did increase the odds of salivary EBV VCA IgG positive status by 90%. Of

the covariates only African American race was significantly associated with the odds of EBV positive status by adolescence.[a]

Discussion

The goal of this study was to assess the associations of early childhood (birth to age 5) family conditions with immune-system dysregulation in adolescence. We focused on Epstein-Barr virus (EBV) reactivation, as this virus is often acquired during childhood/adolescence and may be reactivated by a physical or psychosocial threat. Further, past studies have suggested that EBV reactivation in later stages of the life course are associated with childhood environments (Elwenspoek et al., 2017; Ford and Browning, 2015; Janicki-Deverts et al., 2014).

Our results support these studies, finding that reports of early childhood family instability and economic hardship are associated with increased odds of DNA shedding of EBV among salivary EBV VCA IgG positive adolescents. In particular, we found that family changes associated with the child moving into a new household, while also changing family members, was particularly salient as a predictor of later EBV reactivation. Family economic hardship experiences during this sensitive period had additive risks for EBV reactivation in adolescence.

When adding adolescent (current) family structure and economic conditions to the model, we saw little evidence that current family conditions affected adolescent EBV reactivation. Instead, our results point to the potential for early life family stressors to be associated with later immune system dysregulation in adolescence, net of family conditions during adolescence. This suggests the possibility of biological embedding of family disadvantage via the immune system reactivation during early childhood.

[a] Results not shown here but available upon request.

We recognize several limitations to this study. First, using retrospective data on family conditions that occurred possibly 14 years before the survey reports (for the average adolescent where the family condition occurred at birth) is not ideal. It may be that more stressful family changes during this early childhood period are more likely to be remembered by caregivers and have enduring effects. For example, it may be that a complete change of household and family members and economic hardship are more difficult, memorable, and time-defined events than a parental divorce that may be protracted and less defined by the time period elicited. Thus, we cannot rule out that other types of family instability might have contributed to these associations and went unreported or inaccurately recalled.

Second, we have not assessed the mechanisms through which early childhood family conditions might affect adolescent viral reactivation. Although we considered the role of adolescent family structure and household income with little changes in the early childhood effects, other types of changes in adolescents' lives between early childhood and adolescence may be what is driving our evidence of long-term effects of early childhood environments on adolescent EBV shedding.

Finally, we have not directly assessed stress during childhood. Instead, we have evaluated potentially stressful family circumstances associations with later immune system dysregulation. Thus, we have not documented long-term stress responses, but rather how early environments may shape later physiological dysregulation.

Nonetheless, the findings here are consistent with past research indicating the importance of early childhood for conditioning the stress response and immune system dysregulation in later stages of the life course (Cohen et al., 2004; McEwen and McEwen, 2017). Further, we provide this evidence in a diverse and population-based sample of youth. More research is needed to

further delineate the implications of family stressors occurring in early childhood and to understand the long-term consequences of EBV reactivation (and other indicators of immune-system dysregulation) during childhood and adolescence.

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Tables

Table 1: Sample Descriptive Statistics. Seropositive EBV status adolescents. Adolescent Health and Development in Context Study. N=540

Variable	Mean	Std. Dev.	Min	Max
Birth to age 5 family conditions				
Child lived with only 1 adult	23.9%		0	1
Total family instability				
Total family instability events	0.5	0.9	0	4
Child experienced no instability	72.0%		0	1
Child experienced 1 event	13.2%		0	1
Child experienced 2 events	9.6%		0	1
Child experienced 3 events	2.8%		0	1
Child experienced 4 or more events	2.4%		0	1
Type of family instability				
Child's parents divorced	5.9%		0	1
Parent's partner moved in	10.4%		0	1
Parent's partner moved out	8.5%		0	1
Child moved into new family/caregiver household	12.0%		0	1
Child moved out of grandparent household	4.6%		0	1
Family Economic Hardship				
Child experienced economic hardship	43.7%		0	1
Variables assessed in adolescence				

EBV reactivation	65%		0	1
Adolescent age	14.4	1.8	11	17
Adolescent male	51.9%		0	1
Adolescent white	49.6%		0	1
Adolescent black	40.9%		0	1
Adolescent Latino	6.1%		0	1
Adolescent other race	3.3%		0	1
Caregiver age	45.9	8.2	26	81
Caregiver male	14%		0	1
Caregiver education				
High school or less	19.4%		0	1
Some college	34.4%		0	1
Bachelor's degree or more	46.1%		0	1
Caregiver marital status			0	1
Married	57.4%		0	1
Cohabiting	8.5%		0	1
Single (never married, divorced, separated)	32.6%		0	1
Widowed	1.5%		0	1
Number of children in household	2.1	1.2	1	8
Household income under $30K	32.8%		0	1
Household income $30-$60K	23.9%		0	1
Household income over $60K	43.3%		0	1

Table2: Regression results of EBV reactivation during adolescence on early childhood family conditions. Adolescent Health and Development in Context Study. N=540

Variables	Model 1	Model 2	Model 3	Model 4
Birth to age 5 family conditions				
Child lived with only 1 adult	1.15	1.20	1.14	1.32
	(0.31)	(0.33)	(0.31)	(0.38)
Total family instability	1.21			
	(0.15)			
Child's parents divorced		1.03	0.94	0.95
		(0.46)	(0.43)	(0.44)
Child moved into new family/caregiver household		2.50**	2.41**	2.38**
		(1.05)	(1.02)	(1.01)
Parent's partner moved in		1.06	1.06	1.10
		(0.42)	(0.42)	(0.45)
Parent's partner moved out		1.44	1.34	1.18
		(0.58)	(0.54)	(0.50)
Child moved out of grandparent household		0.47	0.45	0.38
		(0.30)	(0.29)	(0.24)
Family experienced economic hardship			1.46*	1.43*
			(0.30)	(0.30)
Variables assessed in adolescence				
Caregiver cohabiting[1]				1.40
				(0.57)
Caregiver single[1]				0.79
				(0.20)
Caregiver widowed[1]				1.39
				(1.51)
Number of children in household				1.26**
				(0.12)
Household income under $30,000[2]				1.18
				(0.38)
Household income $30,000-$60,000[2]				0.71
				(0.21)
Adolescent age	0.86***	0.87**	0.87**	0.89**
	(0.048)	(0.049)	(0.049)	(0.052)
Adolescent male	0.91	0.91	0.90	0.96
	(0.17)	(0.17)	(0.17)	(0.18)
Adolescent black[3]	1.60**	1.60**	1.57**	1.67**
	(0.35)	(0.36)	(0.35)	(0.42)
Adolescent Latino[3]	1.13	1.15	1.14	1.06

	(0.44)	(0.46)	(0.46)	(0.44)
Adolescent other race[3]	1.64	1.72	1.65	1.95
	(0.88)	(0.91)	(0.89)	(1.10)
Caregiver age	1.02	1.02	1.02	1.03**
	(0.013)	(0.014)	(0.014)	(0.015)
Caregiver male	0.96	0.97	0.97	0.96
	(0.27)	(0.28)	(0.28)	(0.28)
Caregiver some college[4]	0.93	0.95	0.95	0.98
	(0.25)	(0.26)	(0.26)	(0.28)
Caregiver bachelor's degree or more[4]	0.97	1.04	1.11	1.22
	(0.26)	(0.29)	(0.32)	(0.39)
Log likelihood	-340	-337	-335	-328

Robust SE in parentheses. *** p<0.01, ** p<0.05, * p<0.1
[1]Ref: Caregiver married; [2]Ref: Household annual income over $60,000; [3]Ref.: adolescent white; [4]Ref: caregiver high school degree or less.